IV. Provisions for Disposition of Existing Stocks

The Agency has authorized the registrants to sell or distribute product under the previously approved labeling for a period of 18 months the effective date of use deletions.

List of Subjects

Environmental protection, Pesticides and pests, Product registrations.

Dated: September 20, 1999.

Richard D. Schmitt,

Acting Director, Information Resources Services Division, Office of Pesticide Programs.

[FR Doc. 99–29078 Filed 11–9–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–896; FRL–6388–3]

Notice of Filing Pesticide Petitions to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

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 docket control number PF–896, must be received on or before December 10, 1999.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION'' section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–896 in the subject line on the first page of your response.

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

Product Manager	Office location/telephone number/e-mail address	Address	Petition number(s)
Cynthia Giles-Parker (PM 22).	Rm. 247, CM #2, 703–305–7740, e-mail: giles-parker.cynthia@epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA	PP 8F4998
Shaja Brothers	Rm. 237, CM #2, 703–308–3194, e-mail: brothers.shaja@epamail.epa.gov.	Do.	PP 9E3810, 9E3813, OE3912, 9E5075, and 9E6061

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS	Examples of potentially affected entities
Industry	111	Crop production
	112	Animal production
	311	Food manufacturing
	32532	Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the ''FOR FURTHER INFORMATION CONTACT'' section.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**--Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number PF–896. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–896 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal

Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by E-mail to: ''*opp-docket@epa.gov* ,'' or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–896. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified in the ''FOR FURTHER INFORMATION CONTACT'' section.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.
2. Describe any assumptions that you used.
3. Provide copies of any technical information and/or data you used that support your views.
4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.
5. Provide specific examples to illustrate your concerns.
6. Make sure to submit your comments by the deadline in this notice.
7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

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.

List of Subjects

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

Dated: November 1, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

The 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. GMJA Specialties

8F4998

EPA has received a pesticide petition (8F4998) from GMJA Specialties, 10001 13th Avenue, East Bradenton, FL proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of PT807-HCl N,N-Diethyl-N-2-(4-methybenzyloxy)ethylamine hydrochloride in or on the raw agricultural commodity (RAC) oranges at 0.01 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of PT807-HCl in plants and animals is understood. In plants (oranges), unchanged parent is the only residue identified in fruit. Valencia orange trees were treated with ^{14}C PT807-HCl at a nominal rate of 1,000 ppm (approximately 60x the maximum recommended application rate). Fruit from the previous season's crop present on the tree at the time of application was harvested 50 days after treatment (DAT) and mature fruit (not present on the tree at application) was harvested 370 DAT. Total radioactive residue (TRR) levels were 0.538 ppm in 50 DAT orange samples and were 0.051 ppm in 370 DAT orange samples. Most of the radioactivity was present on the peel (88.63% TRR or 0.475 ppm in the 50 DAT fruit, and 64.19% TRR or 0.033 ppm in the 370 DAT fruit). Unchanged parent PT807-HCl was detected in 50 DAT mature fruit (0.386 ppm), but not in the 370 DAT mature fruit (less than 0.001 ppm).

The metabolism of PT807-HCl in oranges has been determined. The only significant metabolite is unchanged parent. No detectable residues of PT807-HCl are anticipated in oranges treated at the recommended application rate.

^{14}C PT807-HCl was extensively metabolized and readily eliminated in the urine and feces following oral administration to a lactating goat. The efficient elimination process resulted in neglible to modest retention of radioactive residues in milk and tissues (less than 0.2% of the administered dose). No residues of unchanged parent were identified in tissues or milk. The rapid elimination of PT807-HCl and its metabolites coupled with the highly exaggerated dose (approximately 3,600x the dietary burden) clearly indicate that no detectable residues of PT807-HCl will accumulate in milk and tissues.

2. *Analytical method*. An analyticial method capable of extracting PT807-HCl from whole oranges, juice, and dried pulp using organic solvents has been validated. Extracted PT807-HCl residues are analyzed using high performance liquid chromatography (HPLC) with a ultraviolet (UV) detector. The limit of

quantitation (LOQ) of the method is 0.01 ppm.

3. *Magnitude of residues*. Seventeen field trials were conducted using various varieties of oranges in California (4 trials), Florida (12 trials), and Texas (1 trial). Two of the trials (1 in California and 1 in Florida) were declined studies with sampling intervals of 0, 7, 14, 30, and 60 days after application. For all other trials, oranges were harvested at the earliest possible time for normal commercial harvest after a single application with PT807-HCl at the maximum recommended application rate, 6 gram active ingredient per acre (g/ai/A). At some of the test sites (depending on the variety of oranges), the previous season's crop was present on the tree at application for these trials, oranges were collected 0 to 68 DAT. In all other trials, fruit were not present on the trees at applications and mature oranges were collected at normal harvest (197 to 359 DAT). Samples were analyzed for residues of PT807-HCl by HPLC with UV detection. Residues of PT807-HCl were nondetectable (less than 0.01ppm) in all treated and control samples.

A processing study was conducted using oranges treated at 5x the maximum application rate in California. The harvested oranges were from the previous season's crop and were on the tree at the time of application. Therefore, the application represents the maximum possible residues. No detectable residues were measured in whole oranges, juice, or oil. Residues of PT807-HCl were detected in dried pulp at 0.015 and 0.017 ppm (average 0.016 ppm). Correcting the measured residues for the exaggerated application rate, no detectable residues are likely in any processed product of oranges.

Residues of PT807-HCl were determined to be stable in whole orange, fruit, oil, juice, and dried pulp stored frozen up to 113 days.

B. Toxicological Profile

1. *Acute toxicity*. PT807-HCl exhibits low acute oral and dermal toxicity (Toxicity Category III, LD_{50} of 531 milligrams/kilograms (mg/kg) and greater than 2,525 mg/kg, respectively) and inhalation toxicity (Toxicity Category IV, LC_{50} of greater than 2.08 milligrams per liter (mg/L). PT807-HCl is minimally irritating to the eyes, only slightly irritating to the skin (Toxicity Categories III and IV, respectively), and is not a dermal sensitizer. An acute neurotoxicity study in rats showed no specific evidence of neurotoxicity; transient non-specific signs of toxicity were observed in this study.

2. *Genotoxicity*. The genotoxic potential of PT807-HCl has been assessed in an *Ames Salmonella* assay, a Chinese hampster ovary (CHO) hypoxanthine guanine phophoribosyl transferase (HGPRT) gene mutation assay, mouse micronucleus assay, an *in vitro* CHO assay for chromosomal aberrations, and an *in vivo* unscheduled DNA synthesis (UDS) assay. The *in vitro* chromosomal aberration assay was positive with and without metabolic activation; however, all of the remaining assays were negative, indicating very low genotoxic potential of PT807-HCl. The contribution of the positive *in vitro* chromosomal aberration assay is weakened by the negative finding in an *in vivo* study (mouse micronucleus) measuring a similar endpoint.

3. *Reproductive and developmental toxicity*. Based on currently available data, PT807-HCl does not present a unique hazard to infants or children and there is no evidence that children are likely to be more sensitive to the toxic effects of PT807-HCl. A 2-generation reproductive toxicity study with PT807-HCl in rats showed developmental delays in pups associated with decreased weight gain at 2,000 and 4,000 ppm, doses which were also toxic to the adult animals. PT807-HCl showed evidence of developmental effects in rats only at a severely maternally toxic dose level. No evidence of developmental toxicity was seen in rabbits.

4. *Subchronic toxicity*. Studies have been conducted with PT807-HCl in mice, rats, and dogs. In dietary studies in rats and dogs, the most notable findings include decreased food consumptions and a consequent decrease in body weight gain (resulting primarily from poor palatability of the test material). Dogs also showed a trend toward anemia, and males showed arrested or delayed sexual maturation at the high dose (equivalent to approximately 222 mg/kg/day). Marked weight loss and decreased weight gain was observed at this dose, and this dose level is considered to have exceeded, a maximum tolerance dose (MTD). Rats dosed by gavage showed signs of neurotoxic effects (tremors in coordination changes in activity) at doses greater than or equal to 300 mg/kg/day. These clinical signs disappeared 2-4 hours post-dosing. Rats receiving dietary administration of up to 5,000 ppm PT807-HCl for 13 weeks did not exhibit any neurotoxic effects. In mice, treatment-related decreased food consumption and body weight gain were seen in males at 7,000 ppm highest dose tested (HDT). No treatment-related toxicity was evident at dietary doses up to 3,500 ppm (479 and 635 mg/kg/day for males and females respectively).

5. *Chronic toxicity*. Ecolyst is not oncogenic when administered to rats at dietary concentration of up to 10,000 ppm for 24 months, and when administered to mice at doses up to 7,000 ppm (equivalent to 1,050 mg/kg/day/(male) 1,250 mg/kg/day(female) for 18 months. In the rat, survival was increased in the treated animals. Systemic toxicity was evident from decreased body weight gains and increased incidences of hepatocellular hypertrophy and foci cellular alteration of hepatocytes in both rats and mice receiving dietary levels of 5,000 and 10,000 ppm of PT807-HCl. In the mouse, decreased body weights were noted in males at 7,000 ppm (1,050 mg/kg/day) HDT. No other treatment-related effects were noted. There were no treatment-related effects of dietary administration of PT807-HCl to dogs at doses up to 5,000 ppm (equivalent to 152 male/136 female mg/kg/day) except for a transient decrease in body weight and food consumption in the first few weeks of the study, and food consumption in the first few weeks of the study, primarily at the 5,000 ppm level, due to poor palatability of the test diet.

6. *Plant and animal metabolism*. Valencia orange trees treated with approximately 470 mg ^{14}C PT807-HCl in 400 ml spray solution/tree. Samples were extracted and radioactivity was partitioned into organic, aqueous, and non-extractable fractions. Extractable, radioactivity was analyzed by HPLC to separate parent and metabolites. Unchanged parent PT807-HCl was detected in leaves (14.191 ppm), immature fruit (0.093), and mature fruit (0.386 ppm) from the previous season's crop that was harvested approximately 50 DAT, but not in mature fruit (less than 0.001 ppm) harvested 370 DAT. ^{14}C PT807-HCl is extensively metabolized and readily eliminated by animals as indicated in a lactating goat study. A lactating goat was dosed with ^{14}C PT807-HCl once a day for 5 consecutive days at a target rate of 10 ppm in the diet. Approximately 100% of the total dose was recovered. Most of the radioactivity (approximately 100% of the total dose was recovered. Most of the radioactivity (approximately 93.8% of the administered dose) was excreted in the urine and approximately 5.6% of the dose was excreted in the feces. Tissues and milk contained less than 0.2% of the administered dose. Unchanged parent compound was not detected in any of the tissue. The rapid elimination of PT807-HCl and its metabolites coupled with the highly

exaggerated dose (approximately 3,600x the dietary burden) clearly indicates that no detectable residues of PT807-HCl will accumulate in milk and tissues.

7. *Metabolite toxicology*. PT807-HCl was rapidly excreted from the rat following oral administration. Approximately 70-80% of the administered dose as excreted from the urine and 10-20% was excreted from the feces. Minimal radioactive residue remained in the tissue. A small quantity of the unchanged parent ^{14}C PT807-HCl (M-14) was detected in urine and feces of the treated rats. The metabolism of PT807-HCl occurs through a variety of pathways, including oxidation, reduction, hydroxylation, deamination, N-dealkylation, and conjugation.

8. *Endocrine disruption*. No evidence of endocrine disruption, including estrogenic or anti-estrogenic activity was present in the animal studies. The developmental toxicity studies showed no effects suggesting endocrine disruption (e.g., change in fetal sex ratios, or malformed or altered reproductive organ development). Maturational delays were seen in both sexes of pups in the reproductive toxicity study at high dose levels; these findings correlated with the decreased body weight gain at these doses. There were no effects on anogenital distance, estrous cyclicity of adult females or on reproduction and fertility. F_O females at 2,000 and 4,000 ppm showed histopathological evidence of decreased cyclicity at weaning of their litters; no such findings were apparent in the F_1 females which were necropsied 1-2 weeks after weaning. The findings in the F_O females attributed to the combined stress of weaning and weight loss. As described below, high dose dogs given a dose exceeding an MTD and showing marked weight loss, showed evidence of maturational arrest of the germinal epithelium and absence of sperm in the epidydimides. All four high dose female dogs were in anestrus (as compared to two of the four control females). These findings are considered related to the marked weight loss and weight gain decrease in this study at the high dose level. No similar findings were seen in a chronic dog study at dose levels up to 5,000 ppm.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. There are no anticipated dietary exposures to PT807-HCl outside of those requested in this tolerance petition. The chronic dietary exposure from the consumption of oranges and its processed products, treated with PT807-HCl is very low. The exposure is only 0.5% of the reference dose (RfD) (0.000063 mg/kg/day) for the most high exposed population, children 1 to 6 years old. The dietary exposure is only 0.17% of the reference dose (RfD) (0.000021 mg/kg/day) for the U.S. population.

ii. *Drinking water*. There are no registered uses of PT807-HCl at this time; thus, the only potential source of residues in drinking water is this requested use on oranges. Available data suggest that PT807-HCl will not be a ground water contaminant because it does not exhibit the mobility or persistence characteristics of pesticides that are normally found in ground water. As a worst-case screen, GMJA specialties used EPA's GENEEC model to estimate drinking water risk, although GENEEC is an inappropriate model for the purpose because it was designed to estimate surface water runoff for ecological risk assessment purposes and greatly overestimates likely residues in surface water. Nevertheless, it is the model EPA currently is using to estimate drinking water exposure in order to assess aggregate risk.

Based on the results of the generic expected environmental concentration (GENEEC) model, the 56–day chronic EEC (calculated from the lowest K_{oc} value measured for PT807-HCl) is 0.315 µg/L. Using the standard drinking water consumption scenarios of 2 liters per day for a 70 kg adult and 1 liter per day for a 10 kg child, the calculated consumption of PT807-HCl in drinking water is 0.009 µg/kg/day for an adult and 0.032 µg/kg/day for a child. These consumption values correspond to 0.07% of the RfD for adults and 0.26% of the RfD for children ages 1 to 6 years old. As discussed above, drinking water concentrations calculated by the GENEEC procedure represent very conservative screening level assessments of drinking water exposure.

2. *Non-dietary exposure*. There are currently no registered uses for PT807-HCl, and therefore, there is no anticipated non-occupational exposure to the chemical.

D. Cumulative Effects

GMJA Specialities/Tropicana Products, Inc. is not aware of any currently registered products that are structurally similar to PT-807-HCl or that would be likely to share a common mechanism of action. Therefore, no cummulative exposures are considered in the PT807-HCl dietary risk assessment.

E. Safety Determination

1. *U.S. population*. The RfD was 0.0125 mg/kg/day based on a no observed adverse effect level (NOAEL) of 12.5 mg/kg/day and an uncertainty factor of 1,000. Although we do not believe there were any findings of concern in the toxicology studies that warrant a 1,000-fold safety factor, we used it as a very consecutive, worst-case screening value. NOAEL was obtained from the results of the rat reproduction study that showed developmental delay and decreased weight gain in pups at levels that were also toxic to adult rats.

2. *Infants and children*. The chronic dietary exposure from the consumption of oranges and its processed products treated with PT807-HCl is very low. The exposure is only 0.5% of the RfD (0.000063 mg/kg/day) for the most highly exposed sub-population, children 1 to 6 years old. The dietary exposure is only 0.17% of the RfD (0.000021 mg/kg/day) for the U.S. population.

F. International Tolerance

There are not Codex Maximum Residue Levels (MRLs) established for PT807-HCl.

2. Interregional Project Number 4

PP 9E3810, 9E3813, 0E3912, 9E5075, and 9E6061

EPA has received pesticide petitions (9E3810, 9E3813, 0E3912, 9E5075, and 9E6061) from the Interregional Project Number 4, Center for Minor Crop, Pest Management, Technology Centre of New Jersey, Rutgers University, 681 U.S. Highway No. 1 South, North Brunswick, NJ 08902-3390 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of esfenvalerate,(S)-cyano-(3-phenoxyphenyl)methyl(S)-4-chloro-alpha-(1-methylethyl) benzeneacetate in or on the raw agricultural commodities (RAC) as follows:

1. PP 9E3810 proposes the establishment of a tolerance for bok choy at 1.0 ppm. Registration will be limited to areas east of the Mississippi River based on the geographical representation of the residue data submitted to EPA.

2. PP 9E3813 proposes the establishment of a tolerance for sweet potatoes at 0.05 ppm.

3. PP 0E3912 proposes the establishment of a tolerance for cardoon at 1.0 ppm. Registration will be limited to California based on the geographical representation of the residue data submitted to EPA.

4. PP 9E5075 proposes the establishment of a tolerance for canola seed at 0.3 ppm.

5. PP 9E6061 proposes the establishment of a tolerance for brussels

sprout at 0.2 ppm for regional registration only.

Fenvalerate is a racemic mixture of four isomers (S,S; R,S; S,R; and R,R). Technical Asana (esfenvalerate) is enriched in the insecticidally active S,S-isomer (84%). Tolerance expressions are proposed for esfenvalerate based on the sum of all isomers.

EPA has determined that the petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions. This notice includes a summary of the petitions prepared by E.I. du Pont Nemours and Company, Agricultural Products, Wilmington, Delaware 19898.

A. Residue Chemistry

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

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

3. *Magnitude of residues*. The following tolerances have been proposed: cardoon at 1.0 ppm, bok choy at 1.0 ppm, sweet potatoes at 0.05 ppm, canola at 0.3 ppm, and brussels sprout at 0.2 ppm. Magnitude of residue studies support the proposed tolerances.

B. Toxicological Profile

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

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

3. *Reproductive and developmental toxicity*. Esfenvalerate was administered to pregnant female rats by gavage in a pilot developmental study at doses of 0, 1, 2, 3, 4, 5, or 20 mg/kg/day and a main study at 0, 2.5, 5, 10, or 20 mg/kg/day. Maternal clinical signs (abnormal gait and mobility) were observed at 2.5 mg/kg/day and above. A no observed adverse effect level (NOAEL) of 2 mg/kg/day was established for the pilot study. The developmental NOAEL was > 20 mg/kg/day.

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

A 2-generation feeding study with esfenvalerate was conducted in the rat at dietary levels of 0, 75, 100, or 300 ppm. Skin lesions and minimal (non-biologically significant) parental body weight effects occurred at 75 ppm. The NOAEL for reproductive toxicity was 75 ppm (4.2-7.5 mg/kg/day) based on decreased pup weights at 100 ppm.

4. *Subchronic toxicity*. Two 90–day feeding studies with esfenvalerate were conducted in rats, one at 50, 150, 300, or 500 ppm esfenvalerate, and a second at 0, 75, 100, 125, or 300 ppm to provide additional dose levels. The NOAEL was 125 ppm (6.3 mg/kg/day) based on clinical signs (jerky leg movements) observed at 150 ppm (7.5 mg/kg/day) and above.

A 90–day feeding study in mice was conducted at 0, 50, 150, or 500 ppm esfenvalerate with a NOAEL of 150 ppm (30.5 mg/kg) based on clinical signs of toxicity at 500 ppm (106 mg/kg).

A 21–day dermal study in rabbits with fenvalerate conducted at 100, 300, or 1,000 mg/kg/day with a NOAEL of 1,000 mg/kg/day.

5. *Chronic toxicity*. In a 1–year study, dogs were fed 0, 25, 50, or 200 ppm esfenvalerate with no treatment related effects at any dietary level. The NOAEL was 200 ppm (5 mg/kg/day). An effect level for dietary administration of esfenvalerate for dogs of 300 ppm had been established earlier in a 3–week pilot study used to select dose levels for the chronic dog study.

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

In an 18–month study, mice were fed 0, 35, 150, or 350 ppm esfenvalerate. Mice fed 350 ppm were sacrificed within the first 2 months of the study after excessive self-trauma related to skin stimulation and data collected were not used in the evaluation of the carcinogenic potential of esfenvalerate. The NOAEL was 35 ppm (4.29 and 5.75 mg/kg/day for males and females, respectively) based on lower body weight and body weight gain at 150 ppm. Esfenvalerate did not produce carcinogenicity.

In a 2–year feeding study, mice were administered 0, 10, 50, 250, or 1,250 ppm fenvalerate in the diet. The NOAEL was 10 ppm (1.5 mg/kg/day) based on granulomatous changes (related to fenvalerate only, not esfenvalerate) at 50 ppm (7.5 mg/kg/day). Fenvalerate did not produce carcinogenicity.

In an 18–month feeding study, mice were fed 0, 100, 300, 1,000, or 3,000 ppm fenvalerate in the diet. The NOAEL is 100 ppm (15.0 mg/kg/day) based on fenvalerate-related microgranulomatous changes at 300 ppm (45 mg/kg/day). No compound related carcinogenicity occurred.

Mice were fed 0, 10, 30, 100, or 300 ppm fenvalerate for 20 months. The NOAEL was 30 ppm (3.5 mg/kg/day) based on red blood cell effects and granulomatous changes at 100 ppm (15 mg/kg/day). Fenvalerate was not carcinogenic at any concentration tested.

In a 2–year study, rats were fed 1, 5, 25, or 250 ppm fenvalerate. A 1,000 ppm group was added in a supplemental study to establish an effect level. The NOAEL was 250 ppm (12.5 mg/kg/day). At 1,000 ppm (50 mg/kg/day), hind limb weakness, lower body weight, and higher organ-to-body

weight ratios were observed. Fenvalerate was not carcinogenic at any concentration.

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

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

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

8. *Endocrine disruption*. Estrogenic effects have not been observed in any studies conducted on fenvalerate or esfenvalerate. In subchronic or chronic studies there were no lesions in reproductive systems of males or females. In the recent reproduction study with esfenvalerate, full histopathological examination of the pituitary and the reproductive systems of males and females was conducted. There were no compound-related gross or histopathological effects. There were also no compound-related changes in any measures of reproductive performance including mating, fertility, or gestation indices or gestation length in either generation. There have been no effects on offspring in developmental toxicity studies.

C. Aggregate Exposure

1. *Dietary exposure*. Tolerances have been established for the residues of fenvalerate/esfenvalerate, in or on a variety of agricultural commodities. For purposes of assessing dietary exposure, chronic and acute dietary assessments have been conducted using all existing and pending tolerances for esfenvalerate. EPA recently reviewed the existing toxicology data base for esfenvalerate and selected the following toxicological endpoints. For acute toxicity, EPA established a NOAEL of 2.0 mg/kg/day from rat and rabbit developmental studies based on maternal clinical signs at higher concentrations. A margin of exposure (MOE) of 100 was required for chronic toxicity. EPA established the chronic population adjusted dose (cPAD) for esfenvalerate at 0.02 mg/kg/day. This cPAD was also based on the NOAEL of 2.0 mg/kg/day in the rat developmental study with an uncertainty factor of 100. Esfenvalerate is classified as a Group E carcinogen - no evidence of carcinogenicity in either rats or mice. Therefore, a carcinogenicity risk analysis for humans is not required.

i. *Food*. A chronic dietary exposure assessment was conducted using Novigen's Dietary Exposure Estimate Model (DEEM). Anticipated residues and adjustment for percent crop treated were used in the chronic dietary risk assessment. The percentages of the cPAD utilized by the most sensitive sub-population, children 1–6 years old, was 4.6% based on a daily dietary exposure of 0.000911 mg/kg/day. Chronic exposure for the overall U.S. population was 1.9% of the cPAD based on a dietary exposure of 0.000376 mg/kg/day. Results of the chronic dietary risk assessment adding cardoon, bok choy, sweet potatoes, canola, and brussels sprout had no significant effect on chronic dietary exposure when compared to the previous chronic dietary risk assessment. EPA has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

Potential acute exposures from food commodities were estimated using a Tier 3 (Monte Carlo) Analysis and appropriate processing factors for processed food and distribution analysis. This analysis used field trial data to estimate exposure, and federal and market survey information to derive the percent of crop treated. Regional consumption information was taken into account. The MOEs for the most sensitive sub-population (children 1–6 years old) were 202 and 103 at the 99th, and 99.9th percentile of exposure, respectively, based on daily exposures of 0.009914 and 0.019390 mg/kg/day. The MOEs for the general population are 355 and 171 at the 99th and 99.9th percentile of exposure, respectively, based on daily exposure estimates of 0.005638 and 0.011710 mg/kg/day. The registrant has stated there is no cause for concern if total acute exposure calculated for the 99.9th percentile yields an MOE of 100 or larger. This acute dietary exposure estimate is considered conservative and EPA considered the MOEs adequate in a recent Final Rule (62 FR 63019) (FRL 5754-6) November 26, 1997.

ii. *Drinking water*. Esfenvalerate is immobile in soil and will not leach into ground water. Due to the insolubility and lipophilic nature of esfenvalerate, any residues in surface water will rapidly and tightly bind to soil particles and remain with sediment, therefore, not contributing to potential dietary exposure from drinking water.

A screening evaluation of leaching potential of a typical pyrethroid was conducted using EPA's Pesticide Root Zone Model (PRZM). Based on this screening assessment, the potential concentrations of a pyrethroid in ground water at depths of 1 and 2 meters are essentially zero (much less than 0.001 parts per billion) (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 be treated before consumption.

Chronic drinking water exposure was estimated to be 0.000001 mg/kg/day for both the United States general population and for non-nursing infants. Less than 0.1% of the cPAD was occupied by both population groups.

Using these values, the contribution of water to the acute dietary risk estimate was estimated for the U.S. population to be 0.000019 mg/kg/day at the 99th percentile and 0.000039 mg/kg/day at the 99.9th percentile resulting in MOEs of 105,874 and 51,757, respectively. For the most sensitive subpopulation, non-nursing infants less than 1–year old, the exposure is 0.000050 mg/kg/day and 0.000074 mg/kg/day at the 99th and 99.9th percentile, respectively, resulting in MOEs of 39,652 and 27,042, respectively.

Therefore, the registrant concludes that there is reasonable certainty of no harm from drinking water.

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

To assess risk from (nonfood) short- and intermediate-term exposure, the registrant selected a toxicological endpoint of 2.0 mg/kg/day, the NOAEL from the rat and rabbit developmental studies. For dermal penetration/

absorption, the registrant selected 25% dermal absorption based on the weight-of-evidence available for structurally related pyrethroids. For inhalation exposure, the registrant used the oral NOAEL of 2.0 mg/kg/day and assumed 100% absorption by inhalation.

Individual non-dietary risk exposure analyses were conducted using a flea infestation scenario that included pet spray, carpet and room treatment, and lawn care, respectively. The total potential short- and intermediate-term aggregate non-dietary exposure including lawn, carpet, and pet uses are: 0.000023 mg/kg/day for adults, 0.00129 mg/kg/day for children 1-6 years old and 0.00138 mg/kg/day for infants less than 1–year old.

EPA concluded November 26, 1997 (62 FR 63019)(FRL 5754–6) that the potential non-dietary exposure for esfenvalerate are associated with substantial margins of safety.

D. Cumulative Effects

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

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

E. Safety Determination

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

1. *U.S. population*. A chronic dietary exposure assessment using anticipated residues, monitoring information, and percent crop treated indicated the percentage of the cPAD utilized by the general population to be 1.9%. There is generally no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

For acute exposure, a MOE greater than 100 is considered adequate. A Tier 3 acute dietary exposure assessment found the general population to have MOEs of 355 and 171 at the 99th and 99.9th percentile of exposure, respectively. These values were generated using actual field trial residues and market share data for percentage of crop treated. These results depict an accurate exposure pattern at an exaggerated daily dietary exposure rate.

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

2. *Infants and children*. FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children unless EPA determines that a different margin of safety will be safe for infants and children. EPA has stated that reliable data supports the use of the standard MOE and uncertainty factor (100 for combined interspecies and intraspecies variability), and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor. In a recent final rule (62 FR 63019), EPA concluded that reliable data support use of the standard 100-fold uncertainty factor for esfenvalerate, and that an additional uncertainty factor is not needed to protect the safety of infants and children. This decision was based on no evidence of developmental toxicity at doses up to 20 mg/kg/day (10 times the maternal NOAEL) in prenatal developmental toxicity studies in both rats and rabbits; offspring toxicity only at dietary levels which were also found to be toxic to parental animals in the 2–generation reproduction study; and no evidence of additional sensitivity to young rats or rabbits following prenatal or postnatal exposure to esfenvalerate.

A chronic dietary exposure assessment found the percentages of the cPAD utilized by the most sensitive sub-population to be 4.6% for children 1-6 years old based on a dietary exposure of 0.000911 mg/kg/day. The percent cPAD for nursing and non-nursing infants was 1.1% and 2.7%, respectively. The registrant has no cause for concern if cPADs are below 100%.

The most sensitive sub-population, children 1-6 years old, had acute dietary MOEs of 202 and 103 at the 99th and 99.9th percentile of exposure, respectively. Nursing infants had MOEs of 198 and 146 at the 99th and 99.9th percentile of exposure, respectively. Non-nursing infants had MOEs of 300 and 156 at the 99th and 99.9th percentile of exposure, respectively. The registrant has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger.

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

F. International Tolerances

There are no Codex MRL values established for fenvalerate on cardoon, bok choy, sweet potatoes, canola, brussels sprout, and rapeseed; therefore, no harmonization is required.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–00625; FRL–6388–8]

Pesticides; Policy Issues Related to the Food Quality Protection Act

AGENCY: Environmental Protection Agency (EPA).
ACTION: Notice of availability.

SUMMARY: To assure that EPA's policies related to implementing the Food Quality Protection Act are transparent and open to public participation, EPA is soliciting comments on the pesticide draft science policy paper entitled ''Guidance for Performing Aggregate Exposure and Risk Assessments.'' This notice is the thirteenth in a series concerning science policy papers related to Food Quality Protection Act and the Tolerance Reassessment Advisory Committee.
DATES: Comments for the draft science policy paper, identified by docket control number OPP–00625, must be received on or before January 10, 2000.
ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, it is imperative that you identify docket control number OPP–00625 in the subject line on the first page of your response.
FOR FURTHER INFORMATION CONTACT: Carol Christensen, Environmental Protection Agency (7505C), 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–6230; fax: (703) 305–7147; e-mail: christensen.carol@epa.gov.
SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you manufacture or formulate pesticides. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS	Examples of potentially affected entities
Pesticide producers	32532	Pesticide manufacturers Pesticide formulators

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action affects certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under ''FOR FURTHER INFORMATION CONTACT.''

B. How Can I Get Additional Information, Including Copies of this Document or Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, the draft science policy paper, and certain other related documents that might be available from the Office of Pesticide Programs' Home Page at http://www.epa.gov/pesticides/. On the Office of Pesticide Programs' Home Page select ''FQPA'' and then look up the entry for this document under ''Science Policies.'' You can also go directly to the listings at the EPA Home Page at http://www.epa.gov/. On the Home Page select ''Laws and Regulations'' and then look up the entry for this document under ''**Federal Register**--Environmental Documents.'' You can go directly to the **Federal Register** listings http://www.epa.gov/fedrgstr/.

2. *Fax on demand*. You may request a faxed copy of the draft science policy paper, as well as supporting information, by using a faxphone to call (202) 401–0527. Select item 6043 for the paper entitled ''Guidance for Performing Aggregate Exposure and Risk Assessments.'' You may also follow the automated menu.

3. *In person*. The Agency has established an official record for this action under docket control number OPP–00625. In addition, the documents referenced in the framework notice, which published in the **Federal Register** on October 29, 1998 (63 FR 58038) (FRL–6041–5) have also been inserted in the docket under docket control number OPP–00557. The official record consists of the documents specifically referenced in this action, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number OPP–00625 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: Public Information