were observed in the mid-high and highest dose group test with a slight progression of severity to the highest dose group tested; (d) a higher incidence of splenomegaly was observed only in the male mice of the highest dose group; (e) histopathological examinations revealed an ectopic proliferation of the mucosal and glandular epithelium in the submucosal layer of the glandular stomach in male and female mice in the highest dose group tested, these changes were assessed to represent heteroplastic, ectopic proliferative changes accompanied by lumen dilatation and cytological degeneration; (f) a higher incidence of hyperkeratosis of the forestomach was observed in both male and female mice and hyperplasia of the squamous epithelium of the forestomach of female male mice was observed in the highest dose group tested; (g) vacuolic changes in the exocrine pancreas of the high dose female was observed; (h) no increased incidence of neoplasms occurred at any dose levels tested in this study.

iv. *Carcinogenicity*. Prohexadione calcium was shown to be non-carcinogenic in mice, rats, and dogs. Therefore, based on the results of the carcinogenicity studies in mice, rats, and dogs and the results of genotoxicity testing, the threshold approach to regulating prohexadione calcium is appropriate

5. *Animal metabolism*. The metabolism in animals (goats and poultry) is adequately understood.

6. *Endocrine disruption*. No specific tests have been conducted with prohexadione calcium to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there were no significant findings in other relevant toxicity studies, *i.e.*, teratology and multi-generation reproductive studies, which would suggest that prohexadione calcium produces endocrine related effects.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure, K-I has estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from the proposed tolerance for prohexadione calcium in/on peanut nutmeat at 0.8 ppm. The TMRC is a ''worse case'' estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are established are treated and that pesticide residues are always found at the tolerance levels. Dietary exposure to residues of prohexadione calcium in or on food will be limited to residues on peanut nutmeat. Peanut hay and meal are fed to animals; thus exposure of humans to residues in peanut hay and meal might result if such residues carry through to meat, milk, poultry, or eggs. However, K-I has concluded that there is no reasonable expectation that measurable residues of prohexadione calcium will occur in meat, milk, poultry, or eggs from this use. There are no other established U.S. tolerances for prohexadione calcium, and there are no currently registered uses for prohexadione calcium on food or feed crops in the U.S.

Dietary exposure to residues of prohexadione calcium from the proposed tolerances on peanuts would account for less than 0.14% of the RfD (0.20 mg/kg/day) for the general population of the US and all subpopulation groups. The most highly exposed group in the subpopulation groups would be non-nursing infants (< 1 year old), which uses 0.39% of the RfD.

2. *Drinking water*. Other potential sources of exposure to prohexadione calcium for the general population are residues in drinking water and exposure from non-occupational sources. Exposure to residues of prohexadione calcium in drinking water is not anticipated. There is no established Maximum Concentration Level (MCL) or Health Advisory Level (HAL) for prohexadione calcium under the Safe Drinking Water Act (SDWA).

3. *Non-dietary exposure*. Prohexadione calcium is not currently registered for any nonagricultural use. The potential for non-occupational exposure to the general population is therefore not present.

D. Cumulative Effects

The potential for cumulative effects of prohexadione calcium and other substances that have a common mechanism of toxicity has been considered. No evidence or information exists to suggest that toxic effects produced by prohexadione calcium would be cumulative with those of any other chemical compound.

E. Safety Determination

1. *U.S. population— Reference dose (RfD)*. Using the conservative exposure assumptions described above and based on the completeness and the reliability of the toxicity data, it has estimated that aggregate exposure to prohexadione calcium will utilize 0.14% of the RfD for the U.S. population. K-I concludes that there is a reasonable certainty that no harm will result from the aggregate exposure to residues of prohexadione calcium, including anticipated dietary exposure and non-occupational exposures.

2. *Infants and children*. Since developmental and reproductive toxicity occurs at levels at or above the levels shown to exhibit parental toxicity and since these levels are significantly higher than those used to calculate the RfD, K-I believes the RfD of 0.20 mg/kg/day is an appropriate measure of safety for infants and children.

Using the conservative exposure assumptions described above, it is concluded that the portion of the RfD that will be utilized by aggregate exposure to residues of prohexadione calcium resulting from the proposed tolerances will be less than 0.14% for all populations of infants and children. The most highly exposed group in the subpopulation groups would be non-nursing infants (< 1 year old) which uses 0.39% of the RfD. 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 the residues of prohexadione calcium, including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

A maximum residue level has not been established for prohexadione calcium by the Codex Alimentarius Commission.

[FR Doc. 98–20768 Filed 8–4–98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–818; FRL–6017–1]

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–818, must be received on or before September 4, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division

(7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

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

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 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
Sidney Jackson	Rm. 268, CM #2, 703–305–7610, e-mail:jackson.sidney@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Beth Edwards	Rm. 206, CM #2, 703–305–5400, e-mail: edwards.beth@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–818] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:
opp-docket@epamail.epa.gov

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

List of Subjects

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

Dated: July 23, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

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

1. Interregional Research Project 4 (IR-4)

PP 6E4667

EPA has received a pesticide petition (PP 6E4667) from the Interregional Research Project 4(IR4), 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 pyridate, 0-(6-chloro-3-phenyl-4-pyridazinyl)-S-octyl carbonothioate and its metabolite 6-chloro-3-phenyl-pyridazine-4-ol (known as SAN 1367), and conjugates of SAN 1367 in or on the raw agricultural commodity garbanzo beans (also known as chick peas) at 0.1 ppm.

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

2. Novartis Crop Protection, Inc.

PP 6F4754

EPA has received a pesticide petition (PP 6F4754) from Novartis Crop Protection, Inc, 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 pyridate, 0-(6-chloro-3-phenyl-4-pyridazinyl)-S-octyl carbonothioate and its metabolite 6-chloro-3-phenyl-pyridazine-4-ol (known as SAN 1367), and conjugates of SAN 1367 in or on the raw agricultural commodities head and stem Brassica Subgroup 5A at 0.3 parts per million (ppm). (Sidney Jackson)

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of pyridate in plants is well understood based on studies with broccoli, corn, and peanut. Pyridate is rapidly broken down by hydrolysis to its major degradation product, SAN 1367. The SAN 1367 metabolite is further conjugated to glucoside and degraded.

2. *Analytical method*. The proposed analytical method is ''Method of analysis of determination of residues of pyridate and its metabolites CL 9673

and conjugated CL 9673 in plant materials'', Report No. 758e, March 1992, Agrolinz Agrarchemikalien Ges.m.b.H. (V/6).

3. *Magnitude of residues*. Residue trials have been conducted with pyridate on the additional crops requested in the pending petitions. The proposed tolerances are recommended at the limit of determination for the method, which is the maximum expected residue from the geographically representative field trial data.

Pyridate strongly adsorbs to soil and was shown to be immobile in soil column leaching studies. Pyridate has a short half-life, low water solubility, and low volatility. Due to its solubility and hydrolysis characteristics, pyridate will not persist in the environment.

San 1367 is further degraded and mineralized to volatile CO2 and bound metabolites. It is susceptible to photolysis. Column leaching studies and field dissipation studies indicated that SAN 1367 tends to degrade faster than it is translocated below the 0-15 cm layer. Therefore under typical agricultural conditions and labeled uses, leaching of SAN 1367 is not an issue of concern.

B. Toxicological Profile

Data are summarized below concerning the mammalian toxicity of pyridate. According to Novartis' interpretation of these data, pyridate is not a carcinogen or a mutagen, has low oral and dermal toxicity to mammals, and causes no reproductive or developmental effects.

1. *Acute toxicity*. Results of a rat acute oral study showed a lethal dose LD_{50} of 4,690 milligram/kilogram (mg/kg) body weight/day (bwt/day) (5,993 mg/kg in males and 3,544 mg/kg in females). In a rat acute dermal study, the LD_{50} was shown to be >2,000 mg/kg. A rat acute inhalation study yielded a LD_{50} >4.37 mg/milliliter (ml).

Results of a primary eye irritation study in the rabbit indicated that pyridate is a mild irritant. A primary dermal irritation study showed pyridate to be a moderate skin irritant, whereas, a dermal sensitization study indicated it is a sensitizer.

2. *Genotoxicity*. Pyridate was tested in the Ames test, mouse micronucleus assay, chromosome aberration assay with Chinese hamster ovary cells, the REC assay, and rat hepatocyte unscheduled DNA synthesis assay. Results were negative for mutagenicity and chromosome aberrations.

3. *Reproductive and developmental toxicity*. A developmental toxicity study in the rat dosed at 0, 55, 165, 400, and 495 milligram/kilograms/day (mg/kg/day) showed maternal toxicant no-observed effect level (NOEL) of 165 mg/kg/day, and developmental NOEL >495 mg/kg/day.

A developmental toxicity study in the rabbit with doses of 0, 150, 300, and 600 mg/kg/day showed a maternal toxicant NOEL of 300 mg/kg/day and developmental NOEL >600 mg/kg/day.

Results of a multi-generational reproduction study with rats dosed at 0, 2.2, 10.8, and 67.5 mg/kg/day showed a NOEL of 10.8 mg/kg/day for maternal and developmental toxicity.

4. *Subchronic toxicity*. Results of a 21-day dermal study showed a NOEL >1,000 mg/kg. A 90-day feeding study in rat dosed at 0, 62.5, 177, and 500 mg/kg/day showed a NOEL of 62.5 mg/kg/day. No neuropathological effects were found. A 90-day feeding study in dogs with doses of 0, 20, 60 and 200 mg/kg/day showed a NOEL of 20 mg/kg/day. Slight degenerative myelopathy in the peripheral nerves was observed at the highest dose level (HDL), which is much higher than the NOEL and the expected exposure from field use.

5. *Chronic toxicity*. A 1-year feeding study in dogs was conducted with doses of 0, 5, 20 and 60 mg/kg/day for 34-weeks. After week 34, the doses were increased to 30, 100, and 150 mg/kg/day because no toxic effects were evident at the lower doses. The final results showed a systemic NOEL of 20 mg/kg/day.

A lifespan (121 week) chronic/carcinogenicity study in rats treated with analytical levels of 0, 2.2, 10.8, and 67.5 mg/kg/day (equivalent to 0, 48, 240, and 1,500 ppm) showed a systemic NOEL of 10.8 mg/kg/day (240 ppm) based on body weight depression. No carcinogenic potential was observed.

In an 18- month carcinogenicity study, mice were fed doses of 0, 400, 800, 1,600 and 7,000 ppm of pyridate. In males, dose levels were approximately 0, 47.7; 97.1; 169.5, and 882.6 mg/kg bwt/day; in females, dose levels were approximately 0, 54.5, 114.6, 204.3, and 1,044.6 mg/kg bwt/day. NOEL was 800 ppm (97.1 mg/kg in males and 114.6 mg/kg in females). Results showed no evidence of carcinogenicity.

i. *Chronic effects*. The Reference Dose (RfD) has been established based on the chronic toxicity database. The RfD = 0.11 mg/kg bwt/day based on the NOEL of 10.8 mg/kg bwt/day from the lifespan rat oncogenicity study due to body weight depression in males, and assuming a safety factor of 100.

ii. *Acute effects*. Acute dietary analysis compared the daily dietary exposure to the lowest NOEL for subchronic studies. EPA's current policy for Tier I analysis uses the conservation assumption that all residues are at a high end estimate or maximum, typically taken as the tolerance value. Acute dietary assessment for pyridate was generated by comparing the ratio of exposure and the NOEL from the 90-day feeding study in dogs of 20 mg/kg bwt/day to determine a margin of exposure (MOE). The exposure estimate includes all current and pending tolerances from Novartis Agro, Inc. and IR-4. MOE of 100 or more are considered acceptable. For all subgroups evaluated, the MOE is greater than 140,000.

iii. *Carcinogenicity*. Existing data demonstrate that there is no evidence of carcinogenicity in rats at 1,500 ppm (67.5 mg/kg/day) or mice at 7,000 ppm (883 mg/kg bwt/day in males, and 1,044.6 mg/kg bwt/day in females). These data have been obtained at dosing in excess of any dietary exposure.

6. *Animal metabolism*. Pyridate has been tested in rats, dogs, cattle,goats, and hens. In every study, pyridate was hydrolyzed to SAN 1367 and rapidly excreted, primarily through the urine as SAN 1367 or its glucoside or glucuronide conjugates.

Data from bovine metabolism and feeding studies established that the uses proposed do not yield secondary residues in meat and milk above the limit of detection. Novartis believes that data from metabolism and feeding studies in poultry established that at the maximum expected dietary burden from crops treated with pyridate will not result in quantifiable residues above the limits of the analytical method. Pyridate and its metabolites are not persistent and do not accumulate in animal systems.

7. *Metabolite toxicology*. Pyridate has been tested in rats, dogs, cattle, goats, and hens. In every study, pyridate was hydrolyzed to SAN 1367 and rapidly excreted, primarily through the urine as SAN 1367 or its glucoside or glucuronide conjugates. Pyridate and its metabolites are not persistent and do not accumulate in animal systems.

C. Aggregate Exposure

Based on environmental fate data, pyridate is not expected to be found in drinking water. There are no non-crop uses for pyridate, and no non-occupational exposure for residential use. Exposure would be limited to dietary exposure described below. Novartis Agro has no information that would indicate that pyridate would have a mechanism of toxicity common to any other registered pesticide.

1. *Dietary exposure—food*. Pyridate is registered for use in corn, peanut, and cabbage. The pending petitions add the use in grain sorghum, collards, and the stem and head Brassica subgroup. The potential dietary exposure of the population to residues of pyridate or its metabolites is calculated based on Theoretical Maximum Residue Contribution (TMRC) for all crops with pyridate use. The TMRC is a worst case estimate of dietary exposure since it assumes that 100% of all crops for which tolerances are established are treated with pyridate, and that pesticide residues are present at the tolerance levels. Novartis maintains that this method of calculation result in an overestimation of the exposure and is considered conservative. Dietary exposure is not expected in meat, milk, poultry, or eggs, based on cow and hen feeding studies, animal metabolism studies, and the fact the residue studies indicate that residues are not present in crops fed to animals above the limit of detection.

2. *Drinking water*. Drinking water is not expected to be a means of exposure to pyridate. Environmental studies indicate that pyridate binds to the soil and is rapidly hydrolyzed into its metabolites. The metabolites are then photolyzed and further degraded and finally mineralized to CO_2. Leaching studies and lysimeter studies indicate that under typical agricultural conditions, neither pyridate nor its metabolites were detected below 30 cm. Groundwater monitoring studies conducted in Europe have not confirmed any detection of pyridate or metabolites. Therefore significant movement of pyridate is not likely and is not a considerable factor in assessing human health risk.

3. *Non-dietary exposure*. There are no registered uses for pyridate on residential or recreational turf. Therefore, non-dietary exposure of pyridate is not likely and not a factor in assessing human health risk.

D. Cumulative Effects

Pyridate belongs to the pyridazine group of herbicidal compounds and has a unique mode of action in plants. Novartis does not have data to indicate a common mechanism of toxicity to other compounds in humans. Therefore, Novartis concludes that cumulative effects from common mechanisms of action are unlikely.

E. Safety Determination

1. *U.S. population*. The RfD is calculated to be 0.11 mg/kg bwt/day. The estimates of exposure are based on conservative assumptions that all crops with a tolerance for pyridate are treated and that all residues found are at the maximum or tolerance level. The dietary exposure to the U.S. population for the current uses plus the garbanzo beans and Brassica uses is estimated at most to be 0.000019 mg/kg/day, which is 0.017% of the RfD. Therefore Novartis concludes that there is reasonable certainty of no harm from aggregate exposure of residues of pyridate or its metabolites including all dietary and other non-occupational exposures.

2. *Infants and children*. Pyridate is not a reproductive or developmental toxicant. Therefore no specific effects on infants and children are expected. Based on the weight of evidence of the toxicity studies, Novartis concludes that an additional safety factor is not warranted.

Using the same assumptions as above, the exposure to infants and children is presented as a percent of RfD. The dietary exposure for the current uses plus the garbanzo beans and Brassica uses for non-nursing infants is estimated at 0.000045 mg/kg/day, which is 0.041% of the RfD. For children age 1-6, the estimated exposure is 0.000057 mg/kg/day, 0.052% of the RfD, and exposure to children age 7-12 is estimated to be 0.000044 mg/kg/day, which is 0.040% of the RfD. Therefore, Novartis concludes that there is reasonable certainty of no harm from aggregate exposure of residues of pyridate or its metabolites including all dietary and other non-occupational exposures.

F. International Tolerances

No international tolerances have been established by CODEX Alimentarius Commission (Sidney Jackson).

3. Valent U.S.A. Company

PP 7F3485, 1F3949, 6F4648

EPA has received a request from Valent U.S.A. Company, 1333 North California Boulevard, Suite 600, Walnut Creek, CA 94596-8025 proposing pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR 180.466 by establishing tolerances for residues of fenpropathrin, alpha-cyano-3-phenoxybenzyl 2,2,3,3-tetramethylcyclopropanecarboxylate, in or on the raw agricultural commodities pome fruit (crop group 11) and grapes at 5.0 ppm, head and stem brassica (crop group 5A) at 3.0 ppm, citrus fruit (crop group 10) at 2.0 ppm, melons (crop group 9A) at 0.5 ppm, and in the processed products citrus oil at 50 ppm, raisins at 10 ppm, and dried citrus pulp at 4.0 ppm. The tolerances were first proposed in response to pesticide petitions PP 7F3485, 1F3949, and 6F4648. EPA has determined that the request contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

Background Information and Use Profile

Fenpropathrin is the active ingredient in DANITOL 2.4 EC Spray (EPA Reg. No. 59639-35) and TAME 2.4 EC Spray (EPA Reg. No. 59639-77). To support DANITOL use, tolerances have been established on cottonseed; cottonseed oil; meat, meat byproducts, and fat of cattle, goats, hogs, horses, sheep and poultry; eggs; milkfat; peanuts; peanut hay; strawberries; and tomatoes. A time limited tolerance on red currants has been established to support a Section 18 in the state of Washington with an expiration date of December 31, 1998. The pending tolerances that are the subject of this notice of filing are on grapes and on the crop groups pome fruits (11), citrus (10), head and stem brassica (5A), and melons (9A), with associated processing products citrus oil, raisins, and dried citrus pulp.

Fenpropathrin is a pyrethroid insecticide with broad spectrum activity on insects and mites. When formulated as the product DANITOL 2.4 EC Spray the product is registered for agricultural use on outdoor terrestrial food crops. A separate fenpropathrin product, TAME 2.4 EC Spray, is registered for commercial, professional non-food use on indoor and outdoor ornamental and nursery stock. There are no uses registered for professional indoor pest control, termite prevention, homeowner use, or turf application.

The products are applied as dilute emulsions in water directly to plants to control harmful insects and mites. In agriculture, depending on the crop and pest, the use rates vary from 0.15 to 0.4 pounds of active ingredient per acre (lb. ai./a), with a maximum total use on all crops of 0.8 lb. ai./a per season. Pre-harvest intervals (phi) range from 21-days on cotton to 1-day on citrus. Plant metabolism studies have shown that the plant and animal residues are best defined as parent fenpropathrin. Because of the mode of application and short phi, finite residues of fenpropathrin are often found on treated agricultural commodities requiring tolerances above the 0.01 ppm limit of quantitation of the residue analytical methodology. However, analyses of RAC samples from plants treated at the

maximum application rates, and minimum retreatment intervals and phi demonstrate that anticipated residues are much below tolerance levels. In addition, it has been demonstrated that fenpropathrin is not plant systemic and that residues occur only on plant parts that have been directly treated.

A. Residue Chemistry

Summary. An extensive plant and animal metabolism data base demonstrates that the appropriate definition of aged fenpropathrin residue is parent. Ruminant and poultry metabolism followed by feeding studies have shown that the ratios of residues in feed to secondary residues in animal products are very low in most commodities, with higher (but still relatively low) ratios in body fat and milk fat. This section will describe metabolism and field residue data supporting the establishment of tolerances for residues of fenpropathrin in or on the raw agricultural commodities pome fruit (crop group 11) and grapes at 5.0 ppm, head and stem brassica (crop group 5A) at 3.0 ppm, citrus fruit (crop group 10) at 2.0 ppm, melons (crop group 9A) at 0.5 ppm, and in the processed products citrus oil at 50 ppm, raisins at 10 ppm, and dried citrus pulp at 4.0 ppm The approved analytical method is capillary gas-liquid chromatography with flame ionization detection.

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

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

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

3. *Magnitude of residues*—i. *Pome fruit (Crop Group 11).* The proposed section 408 tolerance for fenpropathrin in/on Pome Fruit (Crop Group 11) is 5.0 ppm. The proposed tolerance will permit finite residues of fenpropathrin on pome fruit -- apple, pear, oriental pear, crabapple, and related fruits -- as a result of application of DANITOL 2.4 EC Spray to orchards. The field residue data to support a fenpropathrin tolerance on the pome fruit crop grouping includes data on apples from 26 sites and pears from 18 sites providing data from 44 sites across the U.S. The mean residue from all samples is 1.83 ppm. In the subset of samples that exactly fit the proposed use pattern the average residue is 0.83 ppm (n = 16, $\sigma n -1 = 0.55$ ppm) with a maximum value of 1.8 ppm.

ii. *Apples*. The residue data base from apples that supports the proposed crop group tolerance includes all samples from field residue studies that were treated two or more times at 0.4 lb. ai./a with a 14-day phi. These experiments were performed over 5- years at 26 sites in 10 states. There were 38 separate treatments yielding 73 separate, treated samples for analysis. The average residue was 2.15 ppm (n = 73,$\sigma n -1 =$ 1.37 ppm). These data do not include supporting information at higher or lower rates, and harvested at different phi. In the 38-treatment data base there are only four treatments with only two applications that are completely consistent with the proposed use pattern that is limited to a maximum single application rate of 0.4 lb. ai./a, a seasonal maximum of 0.8 lb. ai./a, and a 14 phi. The highest average residue (HAR) found in these crop field trials for fenpropathrin on apples was 1.13 ppm. The average residue was 0.77 ppm (n = 8, $\sigma n -1 = 0.40$). Data obtained by separate analyses of peelings and pulp demonstrated that the bulk of fenpropathrin residues were located on the peeling of the apples.

Five apple processing studies were performed. These studies demonstrated that fenpropathrin residues did not concentrate in apple juice (concentration factor all <<1, average = 0.06), but did concentrate in wet pomace (average concentration factor = 3.05). No additional tolerance for the processed product wet apple pomace is needed because the HAR times the average concentration factor for wet pomace is less than the proposed tolerance of 5 ppm (1.13 ppm x 3.05 = 3.45 ppm).

iii. *Pears*. The residue data base from pears that supports the proposed crop group tolerance includes all samples from field residue studies that were treated two or more times at 0.4 lb. ai./a with a 14-day phi. These experiments were performed over 4-years at 18 sites in 5 states. There were 30 separate treatments yielding 60 separate, treated samples for analysis. The average residue was 1.44 ppm (n = 60, $\sigma n -1 =$ 1.01). This does not include supporting information at higher or lower rates, and harvested at different phi. In the 30-treatment data base there are only four treatments with only 2 applications that are completely consistent with the proposed use pattern, which is the same as in apples, and is limited to a maximum single application rate of 0.4 lb. ai./a, a seasonal maximum of 0.8 lb. ai./a, and a 14-day phi. The HAR found in these crop field trials for fenpropathrin on pears was 1.8 ppm. The average residue was 0.88 ppm (n = 8, $\sigma n -1 = 0.69$).

iv. *Grapes*. The proposed section 408 tolerance for fenpropathrin on grapes is 5 ppm. The residue data base that supports the tolerance includes all samples from field residue studies that were treated 4- times at 0.2 lb. ai./a with a 21-day phi. Excluded from the calculation of the tolerance, and the chronic and acute exposure analyses is data from one site that were demonstrated to be outliers (The analytical determinations were very high, more than six sigma above the mean of the other determinations). These experiments were performed over 4-years at 14 sites in 4 states. There were 14 separate treatments yielding 28 separate, treated samples for analysis. The average residue was 1.06 ppm (n = 28, $\sigma n -1 = 0.71$). This does not include supporting information at higher or lower rates, different numbers of applications, or different phi. The HAR

found in crop field trials for fenpropathrin on grapes was 3.1 ppm.

Four processing studies yielding raisins and juice, and 5 additional studies yielding grape juice only (total of 9), were performed. These studies demonstrated that fenpropathrin residues were greatly reduced in grape juice (concentration factor all <<1, average = 0.06), but did concentrate in raisins (average concentration factor = 1.76). An additional tolerance for the processed product raisins is needed because the HAR times the average concentration factor for raisins is greater than the proposed tolerance of 5 ppm (3.1 ppm x 1.76 = 5.55 ppm). A Section 408 tolerance for fenpropathrin on raisins of 10 ppm is proposed.

v. *Citrus*. The proposed Section 408 tolerance for fenpropathrin on citrus fruit (Crop Group 10) is 2 ppm. The residue data base from citrus that supports the tolerance includes all samples from field residue studies that were completely consistent with the proposed use pattern of 2 applications at 0.4 lb. ai./a with a 1-day phi. In oranges, the experiments were performed over 5-years at 13 sites in 4 states. There were 13 separate treatments yielding 24 separate, treated samples for analysis. The average residue in oranges was 0.39 ppm (n = 24, σn -1 = 0.35 ppm). In grapefruit, the experiments were performed in a single year at 7 sites in 3 states. There were 7 separate treatments yielding 14 separate, treated samples for analysis. The average residue in grapefruit was 0.29 ppm (n = 14, σn -1 = 0.13 ppm). In lemons, the experiments were performed in a single year at 3 sites in 2 states. There were 3 separate treatments yielding 6 separate, treated samples for analysis. The average residue in lemons was 0.52 ppm (n = 6, σn -1 = 0.06 ppm).

For the overall crop grouping citrus fruits the average residue was 0.37 ppm (n = 44, σn -1 = 0.28 ppm). The HAR found in all citrus crop field trials meeting the proposed use pattern for fenpropathrin on citrus was 1.2 ppm. These overall citrus data only include data from samples that are consistent with the proposed use pattern, and do not include supporting information at higher or lower rates, and harvested at different phi. Data obtained by separate analyses of peelings and pulp from oranges demonstrated that the bulk of fenpropathrin residues were located on the peeling, exterior, of the oranges.

There are two processing studies performed in citrus (oranges) with processing to juice, dried citrus pulp, and citrus oil. The studies demonstrated that fenpropathrin did not concentrate in juice (concentration factor all <<1), but did concentrate in dried citrus pulp (average concentration factor = 2.6), and in citrus oil (average concentration factor = 40.5). Thus it can be calculated from the HAR that residues of 3.12 ppm (1.2 x 2.6) could occur in dried citrus pulp and 48.6 ppm (1.2 x 40.5) could occur in citrus oil. Since residues could be present in the not ''ready to eat'' commodities at levels (3.12, 48.6 ppm) appreciably higher than the proposed RAC tolerance of 2 ppm, tolerances are being proposed. After rounding, the proposed tolerances are 4.0 ppm for dried citrus pulp, and 50.0 ppm for citrus oil.

vi. *Melons (Cantaloupe)*. The proposed Section 408 tolerance for fenpropathrin in/on melons (crop group 9A) is 0.5 ppm. The field residue data that support this proposal come from 10 locations in 7 states. At these ten locations there was a total of 14 separate trials, yielding 36 separate, treated samples for analysis. Samples from treatments that were consistent with the proposed maximum use pattern -- 0.2 lb. ai./a, 4 applications, 7-day spray interval, 7-day pre-harvest interval -- gave 20 separate samples for analysis. The mean of the 20 determinations is 0.175 ppm (n = 20, σn-1 = 0.077 ppm) and a maximum value of 0.31 ppm. Separate analyses of pulp and rind demonstrated that the bulk of the residues were present on the rind.

vii. *Head and Stem Brassica*. A proposed Section 408 tolerance of 3.0 ppm is proposed for fenpropathrin in/on Head and stem brassica (crop group 5A) -- cabbage, cauliflower, broccoli, brussels sprouts, and related non-leafy brassica. The field residue data to support a fenpropathrin tolerance on the crop grouping head and stem brassica includes data on broccoli from 7 sitesand cabbage from six sites providing data from 13 sites across the U.S. Samples from trials that were consistent with the proposed maximum use pattern for the crop group -- the first application at 0.2 lb. ai./a and 2 additional applications at 0.3 lb. ai./a (a total application of 0.8 lb. ai./a), 7-day spray interval, 7-day pre-harvest interval -- gave a mean residue of 0.62 ppm(n = 26, σn-1 = 0.69) with a maximum value of 2.8 ppm.

viii. *Broccoli*. Field residue data come from 7 locations in 4 states. At these locations there were a total of 8 separate trials yielding 28 separate, treated samples for analysis. Samples from trials that were consistent with the proposed maximum use pattern gave 14 separate samples for analysis. The mean of the 14 determinations is 0.369 ppm (n = 14, σn-1 = 0.157 ppm)and a maximum value of 0.58 ppm.

ix. *Cabbage*. Field residue data come from 6 locations in 6 states. At these six locations there was a total of 7 separate trials yielding 26 separate, treated samples for analysis. Trials that were consistent with the proposed maximum use pattern gave 12 separate samples for analysis. The mean of the determinations is 0.92 ppm (n = 12, σn-1 = 0.93 ppm) and a maximum value of 2.8 ppm. Analyses of cabbage heads with wrapper leaves removed demonstrated that the bulk of the residue was on the exterior of the cabbages with a mean residue of 0.04 ppm (n = 12, σn-1 = 0.05 ppm) and a maximum value of 0.19 ppm.

x. *Secondary residues*. Residues in animal feed may transfer to animal products, meat, milk, and eggs, used in human food. The existing tolerances on meat and meat by-products of cattle, goats, hogs, horses and sheep at 0.1 ppm, fat of cattle, goats, hogs, horses and sheep at 1.0 ppm, milk fat (reflecting 0.08 ppm in whole milk) at 2.0 ppm, and poultry meat, fat, meat by-products and eggs at 0.05 ppm are, adequate to allow the addition of the proposed uses. Both chronic and acute dietary assessments show very low residue contribution from secondary residues in animal products to all population sub-groups.

B. Toxicological Profile

Summary. The existing registrations and tolerances of fenpropathrin are supported at EPA by a complete toxicology data base. Toxicity endpoints of concern have been identified by the Agency's Health Effects Division, Hazard Identification Assessment Review Committee (Meeting July 17, 1997; Revised Memorandum November 14, 1997). The identified endpoints are an acute dietary of 6.0 mg/kg/day (systemic) and a chronic dietary of 2.5 mg/kg/day (RfD = 0.025 mg/kg/day, UF = 100). No endpoints of concern were identified by the Committee for occupational or residential, dermal or inhalation exposures of any duration. Further, in the Revised Memorandum of November 14, 1997, the Committee concluded that an additional safety factor, beyond 100 was not needed to account for special sensitivity of infants and children to fenpropathrin. In a separate action, fenpropathrin has been evaluated for carcinogenicity by the HED RfD/Peer Review Committee. In a Memorandum from Dr. G. Z. Ghali to Mr. G. La Rocca dated March 18, 1993, it was concluded that in valid studies with adequate doses that the compound ''did not alter the spontaneous tumor

profile in both rats and mice''. Fenpropathrin was classified as Group E.

1. *Acute toxicity*. Oral LD_{50} in the rat is 54.0 milligram/kilogram (mg/kg) for males and 48.5 mg/kg for females - Toxicity Category I; dermal LD_{50} is 1,600 mg/kg for males and 870 mg/kg for females - Category II; acute inhalation (impossible to generate sufficient test article vapor or aerosol to elicit toxicity) - Category IV; primary eye irritation (no corneal involvement, mild iris and conjunctival irritation) - Category III; and primary dermal irritation (no irritation) - Category IV. Fenpropathrin is not a sensitizer.

2. *Genotoxicity*. Studies on gene mutation and other genotoxic effects: An Ames Assay was negative for *Salmonella* TA98, TA100, TA1535, TA1537, and TA1538; and E coli WP2uvrA (trp-) with or without metabolic activation. Sister Chromosome Exchange in CHO-K1 Cells - there were no increases in sister chromatid exchanges seen in the CHO-K1 cells treated with S-33206 or the DMSO vehicle. Cytogenetics *in vitro* (CHO/CA) - negative for chromosome aberrations (CA) in Chinese hamster ovary (CHO) cells exposed *in vitro* to toxic doses (> 30 nanogram) without activation; and to limit of solubility (1,000 nanogram) with activation. *In Vitro* Assay in Mammalian Cells - equivocal results - of no concern. DNA Damage/Repair in Bacillus subtilis - not mutagenic or showing evidence of DNA damage at > 5,000 nanogram/paper disk.

3. *Reproductive and developmental toxicity*. In a developmental toxicity study in rats, pregnant female rats were dosed by gavage on gestation days 6-15 at 0 (corn oil control) 0.4, 1.5, 2.0, 3.0, 6.0, or 10.0 mg/kg/day. The maternal no observed adverse effect level (NOAEL) is 6 mg/kg/day; maternal LEL is 10 mg/kg/day based on death, moribundity, ataxia, sensitivity to external stimuli, spastic jumping, tremors, prostration, convulsions, hunched posture, squinted eyes, chromodacryorrhea, and lacrimation; developmental NOAEL is > 10 mg/kg/day.

In a developmental toxicity study in rabbits, pregnant female New Zealand rabbits were dosed by gavage on gestation days 7 through 19 at 0, 4, 12, or 36 mg/kg/day. Maternal NOEL is 4 mg/kg/day; maternal LEL is 12 mg/kg/day based on grooming, anorexia, flicking of the forepaws; developmental NOEL is > 36 mg/kg/day (HDT).

A 3-generation reproduction study was performed in rats. Rats were dosed with fenpropathrin at concentrations of 0, 40, 120, or 360 ppm (0, 3.0, 8.9, or 26.9 mg/kg/day in males; 0, 3.4, 10.1, or 32.0 mg/kg/day in females, respectively). Parents (male/female): Systemic NOEL = 40 ppm (3.0/3.4 mg/kg/day). Systemic LEL = 120 ppm (8.9/10.1 mg/kg/day) based on body tremors with spasmodic muscle twitches, increased sensitivity and maternal lethality; reproductive NOEL = 120 ppm (8.9/10.1 mg/kg/day). Reproductive LEL = 360 ppm (26.9/32.0 mg/kg/day) based on decrease mean F1B pup weight, increased F2B loss. Pups (male/female): Developmental NOEL = 40 ppm (3.0/3.4 mg/kg/day). Developmental LEL = 120 ppm (8.9/10.1 mg/kg/day) based on body tremors, increased mortality.

4. *Subchronic toxicity*. In a subchronic oral toxicity study, rats were dosed at concentrations of 0, 3, 30, 100, 300, or 600 ppm in the diet. The lowest effect level (LEL) is 600 ppm (30 mg/kg/day) based on body weight (bwt) reduction (female), body tremors, and increased brain (female) and kidney (male) weights. The NOEL is 300 ppm (15 mg/kg/day).

In a subchronic oral toxicity study, dogs were dosed at concentrations of 0, 250, 500, or 1,000 ppm in the diet. A 1,000 ppm dog was sacrificed moribund during the third week after having tremors and showing other signs of poisoning caused by the test article. Because of this death, the dose for this group was reduced to 750 ppm for the remainder of the study. The LOEL is 250 ppm (7.25 mg/kg/day) based on signs of GI tract disturbance. There was no NOEL -- note dog chronic, below)

In a 21-day dermal toxicity study, rabbits were dosed 5-days/week for 3 weeks on abraded or unabraded skin at doses of 0, 500, 1,200, or 3,000 mg/kg/day. There were no dose-related effects on bwt, food consumption, clinical pathology, gross pathology, or organ weights. Trace or mild inflammatory cell infiltration was seen in the intact and abraded skin in all groups, including controls, and was attributed to the test article. The systemic NOEL is > 3,000 mg/kg/day. Local irritation only.

Although a 21-day dermal toxicity study in rabbits is available the Agency has determined that rats are the most sensitive species to ascertain the dermal toxicity potential of pyrethroid insecticides. Although these data are lacking, EPA has sufficient toxicity data to support these tolerances and these additional studies are not expected to significantly change the risk assessment.

5. *Chronic toxicity*. In a 1-year feeding study, dogs were dosed at 0, 100, 250, or 750 ppm in the diet. The systemic LEL is 250 ppm (6.25 mg/kg/day) based on tremors in all dogs. The neurologic NOEL is 100 ppm (2.5 mg/kg/day); the systemic NOEL is 100 ppm (2.5 mg/kg/day).

In a chronic feeding/carcinogenicity study, rats were dosed at 0, 50, 150, 450, or 600 ppm in the diet (0, 1.93, 5.71, 17.06, or 22.80 mg/kg/day in males, and 0, 2.43, 7.23, 19.45, or 23.98 mg/kg/day in females). There was no evidence of carcinogenicity at any dose up to and including 600 ppm. The systemic NOEL (male) is 450 ppm (17.06 mg/kg/day). The systemic NOEL (female) is 150 ppm (7.23 mg/kg/day). Systemic LEL (male) is 600 ppm highest dose tested (HDT) based on increased mortality, body tremors, increased pituitary, kidney, and adrenal weights. The systemic LEL (female) is 450 ppm (19.45 mg/kg/day) based on increased mortality and body tremors.

In a chronic feeding/carcinogenicity study, mice were dosed at 0, 40, 150, or 600 ppm in the feed (0, 3.9, 13.7, or 56.0 mg/kg/day in males, and 0, 4.2, 16.2, or 65.2 mg/kg/day in females). Mortality was highest during the final quarter of the study, but the incidence was similar in all dosed and control groups. No other indications of toxicity or carcinogenicity were seen. The systemic NOEL is ≤ 600 ppm (HDT; male/female, 56.0/65.2 mg/kg/day). text.

6. *Animal metabolism*. In a metabolism study in rats, animals were dosed with radiolabelled fenpropathrin radiolabelled in either the alcohol or acid portion of the molecule. Rats received 14 daily oral low-doses of 2.5 mg/kg/day of unlabelled fenpropathrin followed by a 15th dose of either the alcohol or acid radiolabelled fenpropathrin. Groups of rats received a single dose of either of the 2 radiolabelled test articles at 2.5 mg/kg or 25 mg/kg. No clinical signs were seen in any rats.

The major biotransformations included oxidation at the methyl group of the acid moiety, hydroxylation at the 4'-position of the alcohol moiety, cleavage of the ester linkage, and conjugation with sulfuric acid or glucuronic acid.

Four metabolites were found in the urine of rats dosed with alcohol labeled fenpropathrin. The major metabolites were the sulfate conjugate of 3-(4'-hydroxyphenoxy)benzoic acid and 3-phenoxybenzoic acid (22-44% and 3-9% of the administered dose, respectively). The major urinary metabolites of the acid-labeled fenpropathrin were TMPA-glucuronic acid and TMPA-CH2OH (11-26% and 6-10% of the administered dose, respectively). None of the parent chemical was found in urine.

The major elimination products in the feces included the parent chemical (13-34% of the administered dose) and four

metabolites. The fecal metabolites (and the percentage of administered dose) included CH2OH-fenpropathrin (9 20%), 4'-OH-fenpropathrin (4-11%), COOH-fenpropathrin (2-7%), and 4'-OH-CH2OH-fenpropathrin (2-7%).

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

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

7. *Endocrine disruption*. No special studies to investigate the potential for estrogenic or other endocrine effects of fenpropathrin have been performed. However, as summarized above, a large and detailed toxicology data base exists for the compound including studies acceptable to the Agency in all required categories. These studies include evaluations of reproduction and reproductive toxicity and detailed pathology and histology of endocrine organs following repeated or long term exposure. These studies are considered capable of revealing endocrine effects and no such effects were observed.

C. Aggregate Exposure

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

2. *Food*. Chronic and acute dietary exposure analyses were performed for fenpropathrin using anticipated residues and accounting for proportion of the crop treated. The crops included in the analyses are the raw agricultural commodities cottonseed, currants, peanuts, strawberries, tomatoes, pome fruits, citrus, grapes, head and stem brassica, and melons; processed products from these crops; and the resulting secondary residues in meat, milk, and eggs. A report along with a supplemental report of these exposure/risk analyses has been submitted to the Agency including a detailed description of the methodology and assumptions used.

Chronic dietary exposure was calculated for the U.S. population and 26 population subgroups. The results from several representative subgroups are listed below. Chronic dietary exposure was at or below 1.7 % of the reference dose with grapes and apples the commodities contributing the most to chronic exposure. Generally speaking, the Agency has no cause for concern if total residue contribution for published and proposed tolerances is less than 100% of the RfD.

SUMMARY OF CHRONIC DIETARY (FOOD) EXPOSURES TO FENPROPATHRIN RESIDUES

Population Subgroup	Exposure(mg/kg bw/day)	Percent ofRfD
Total U.S. Population (all seasons)	0.000165	0.7
Females (13+/Nursing)	0.000285	1.1
Non-Hispanic other than B/W	0.000246	1.0
Children (1-6 Years)	0.000435	1.7
All Infants (<1 Year Old)	0.000193	0.8
Non-Nursing Infants (<1 Year Old)	0.000127	0.5
Nursing Infants (<1 Year Old)	0.000351	1.4

Acute dietary exposure was calculated for the U.S. population, Females (13+/Pregnant/Not Nursing), and five children subgroups. The subpopulation, Females (13+/Pregnant/Not Nursing), was included because the toxicity endpoint for acute dietary exposure identified by the Agency is based on clinical signs of toxicity in the dams from the rat developmental toxicity study. The calculated exposures and margins of exposure (MOE) for the higher exposed proportions of the subgroups are listed below. In all cases, margins of exposure exceed one-hundred.

CALCULATED ACUTE DIETARY EXPOSURES TO FENPROPATHRIN RESIDUES IN FOOD (PER-CAPITA DAYS)

Population Subgroup	99th Percentile		99.9th Percentile	
	Exposure(mg/kg bw/day)	MOE	Exposure(mg/kg bw/day)	MOE
U.S. Polulation	0.003296	1,821	0.010173	590
Females (13+/Pregnant/NotNursing)	0.002737	2192	0.005595	1072
Children 1-6	0.008461	709	0.020678	290
Children 7-12	0.005322	1,127	0.012195	492
All Infants	0.002963	2,025	0.029691	202
Nursing Infants (<1)	0.007142	840	0.050337	119
Non-Nursing Infants (<1)	0.001874	3,202	0.004863	1,234

It should be noted that the numbers of individuals in the dietary survey of some population subgroups is small. These ''under represented'' subgroups are weighted to account for their proportions in the total U.S. Population and in various geographic and ethnic subpopulations. If in these under represented subgroups there are individuals with unusual dietary consumption patterns anomalous Monti Carlo selected diets will occur at the lower probability exposures (e.g. 99th and 99.9th percentiles) often times leading to unrealistically high calculated exposures. Such is the case for Nursing Infants (<1). Two of these babies were reported to be fed raw grapes. In one case, one nursing infant was reported to consume 310 grams of raw grapes in a single day. This is a very unusual diet for any infant. Because of this dietary anomaly, and the weighting factor for this population subgroup, the

MOE for nursing infants approaches 100.

3. *Drinking water.* Since fenpropathrin is applied outdoors to growing agricultural crops, the potential exists for fenpropathrin or its metabolites to reach ground or surface water that may be used for drinking water. Because of the physical properties of fenpropathrin, the Agency has determined that it is unlikely that fenpropathrin or its metabolites can leach to potable groundwater.

To further quantify potential exposure from drinking water, surface water concentrations for fenpropathrin were estimated using GENEEC 1.2. The average 56-day concentration predicted in the simulated pond water was 0.22 ppb. The residence time of fenpropathrin in surface water has been measured and is short. In pond studies, fenpropathrin half-lives in the water column were less than 1.5 days, thus this 56-day modeled half-life probably considerably overestimates any real surface water concentration. Using standard assumptions about bwt and water consumption, the chronic exposure from this drinking water would be 6.3 x 10-6 and 2.2 x 10 -5 mg/kg bw/day for adults and children, respectively; less than 0.09 % of the RfD for children. Based on this worse case analysis, the contribution of water to the dietary risk is negligible.

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

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' Available information in this context include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way.

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

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

E. Safety Determination

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

1. *U.S. population*—i *Chronic exposure.* Using the dietary exposure assessment procedures described above for fenpropathrin, calculated chronic dietary exposure resulting from residue exposure from existing and proposed uses of fenpropathrin is minimal. The estimated chronic dietary exposure from food for the overall U.S. population and many non-child/infant subgroups is 1.1 [Females (13+/Nursing), 0.000285 mg/kg bw/day] to 0.4 % of the RfD. Addition of the small but worse case potential chronic exposure from drinking water (calculated above) increases exposure by only 6.3 x 10-6 mg/kg bw/day, and the maximum occupancy of the RfD from 1.14 % to 1.16 %. Generally, the Agency has no cause for concern if total residue contribution is less than 100 % of the RfD. It can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. Population and many non-child/infant subgroups from aggregate, chronic exposure to fenpropathrin residues.

ii. *Acute.* The potential acute exposure from food to the U.S. population and various non-child/infant population subgroups (shown above) provide MOE values greatly exceeding 100. Addition of the worse case, but very small ''background'' dietary exposure from water is not sufficient to change the MOE values significantly (see table below). In a conservative policy, the Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger. It can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. Population and many non-child/infant subgroups from aggregate, acute exposure to fenpropathrin residues.

AGGREGATE U.S.POULATION ACUTE DIETARY EXPOSURE

Source of Exposure	Exposure(mg/kgbw/day)	99.9th Percentile Margin ofExposure
Chronic Water ...	0.000006	-
99.9th Percentile Acute Exposure -- Food ...	0.010173	589.8
99.9th Percentile Aggregate Acute Exposure Food + Water	0.010179	589.4

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

The toxicological data base for evaluating pre- and post-natal toxicity for fenpropathrin is complete with respect to current data requirements. There are no special pre- or post-natal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies or the 3-generation reproductive toxicity study in rats. EPA HED Hazard

ID Committee (Revised Memorandum, November 14,1997) has concluded that reliable data support use of the standard 100-fold uncertainty factor and that an additional uncertainty factor is not needed for fenpropathrin to be further protective of infants and children.

3. *Chronic risk*. Using the conservative exposure assumptions described above, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of fenpropathrin ranges from 0.5 % for Non-Nursing Infants (<1 year old), up to 1.7 % for Children (1 - 6 years). Addingthe worse case potential incremental exposure to infants and children from fenpropathrin in drinking water (2.2 x 10 -5 mg/kg bw/day) to the chronic dietary exposure from food (0.000435 mg/kg bw/day) does not materially increase the aggregate, chronic dietary exposure and only increases the occupancy of the RfD by 0.09% to 1.8 % for Children (1 - 6 years). EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. It can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, chronic exposure to fenpropathrin residues.

4. *Acute*. The potential acute exposure from food to the various child and infant population subgroups (shown above) provide MOE values exceeding 100. Addition of the worse case, but very small ''background'' dietary exposure from water (2.2 x 10 -5 mg/kg bw/day) is not sufficient to change the MOE values significantly (see table below). In a conservative policy, the Agency has no cause for concern if total acute exposure calculated for the 99.9th percentile yields a MOE of 100 or larger. It can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, acute exposure to fenpropathrin residues.

AGGREGATE NURSING INFANTS (> 1 YEAR) ACUTE DIETARY EXPOSURE

Source of Exposure	Exposure (mg/kg bw/day)	99.9th Percentile Margin of Exposure
Chronic Water ...	0.000022	-
99.9th Percentile Acute Exposure - Food	0.050337	119.2
99.9th Percentile Aggregate Acute Exposure Food + Water	0.050359	119.1

F. Safety Determination Summary

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

G. International Tolerances

Codex Maximum Residue Limits

186 -- FENPROPATHRIN

Main uses -- 8 -- INSECTISCIDE/ ACARACIDE

JMPR -- 83

ADI -- 0.03 mg/jg body weight (1993)

RESIDUE -- Fenpropathrin (fat soluble)

Commodity					
Code	Name	MRL (mg/kg)	Step	JMPR	CCPR
MM 0812	Cattle meat	0.5 (fat)	6	93	.....
ML 0812	Cattle milk	0.1 F	6	93	.....
MO 0812	Cattle, Edible offal of	0.05	CXL		(1995)
SO 0691	Cotton seed	1	CXL		(1995)
OC 0691	Cotton seed oil, Crude	3	CXL	.....	(1995)
VO 0440	Egg plant	0.2	6	93	.....
PE 0112	Eggs	0.01 (*)	CXL	.....	(1995)
VC 0425	Gherkin	0.2	CXL	D (1995)	
FB 0269	Grapes	5	6	93	.....
VO 0445	Peppers, Sweet	1	CXL	.....	(1995)
FP 0009	Pome fruits	5	CXL	.....	(1995)
PM 0110	Poultry meat	0.02 (fat)	CXL	.....	(1995)
PO 0111	Poultry, Edible offal of	0.01 (*)	CXL	.....	(1995)
V0 0448	Tomato	1	CXL	.....	(1995)

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

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