certainty that no harm will occur to infants and children from aggregate exposure to residues of buprofezin.

F. International Tolerances

Canada, Codex, and Mexico do not have maximum residue limits for residues of buprofezin in/on the proposed crops. Therefore, harmonization is not an issue.

[FR Doc. 04–27772 Filed 12–21–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0403; FRL–7689–6]

Pyriproxyfen: Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket identification (ID) number OPP–2004–0403, must be received on or before January 21, 2005.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Joseph Tavano, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6411; e-mail address: *tavano.joseph@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

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 this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to 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 Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket ID number OPP–2004–0403. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr/*.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at *http://www.epa.gov/edocket/* to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and to Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that

is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2004–0403. The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to *opp-docket@epa.gov*, Attention: Docket ID number OPP–2004–0403. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM*. You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID number OPP–2004–0403.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA, Attention: Docket ID number OPP–2004–0403. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI to the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is 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 docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

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 ID number as signed 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 a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in FFDCA section 408(d)(2); 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.

List of Subjects

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

Dated: December 9, 2004.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. 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.

Valent U.S.A. Corporation

PP 4F6847

EPA has received a pesticide petition 4F6847 from Valent U.S.A. Corporation, 1600 Riviera Ave., Suite 200, Walnut

Creek, California 94596–8025 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 pyriproxyfen, 2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine (CA), in or on the raw agricultural commodities (RAC) grass forage and hay (crop group 17). 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*. Metabolism of ^{14}C-pyriproxyfen labeled in the phenoxyphenyl ring and in the pyridyl ring has been studied in cotton, apples, tomatoes, lactating goats, and laying hens (and rats). The major metabolic pathways in plants is aryl hydroxylation and cleavage of the ether linkage, followed by further metabolism into more polar products by further oxidation and/or conjugation reactions. However, the bulk of the radio-chemical residue on RAC samples remained as parent. Comparing metabolites detected and quantified from cotton, apple, tomato, goat and hen (and rat) shows that there are no significant metabolites in plants which are not also present in the excreta or tissues of animals. Therefore, the residue of concern is best defined as the parent, pyriproxyfen.

Ruminant and poultry metabolism studies demonstrated that transfer of administered ^{14}C-residues to tissues was low. Total ^{14}C-residues in goat milk, muscle and tissues accounted for less than 2% of the administered dose, and were less than 1 part per million (ppm) in all cases. In poultry, total ^{14}C-residues in eggs, muscle and tissues accounted for about 2.7% of the administered dose, and were less than 1 ppm in all cases except for gizzard.

2. *Analytical method*. Practical analytical methods for detecting and measuring levels of pyriproxyfen (and relevant metabolites) have been developed and validated in/on all appropriate agricultural commodities, respective processing fractions, milk, animal tissues, and environmental samples. The extraction methodology has been validated using aged radio-chemical residue samples from metabolism studies. The methods have been validated in cotton seed, apples, soil, and oranges at independent laboratories. EPA has successfully validated the analytical methods for analysis of cotton seed, pome fruit, nutmeats, almond hulls, and fruiting vegetables. The limit of detection of pyriproxyfen in the methods is 0.01 ppm which will allow monitoring of food with residues at the levels proposed for the tolerances.

3. *Magnitude of residues*—i. *Grass, forage, fodder, and hay*. Twelve field trials in grass were conducted in 2002 and 2003. The analytical data show that the average measured residue in/on grass forage samples was 0.05 ppm (n = 24, σn-1 = 0.10 ppm) pyriproxyfen. Similarly, the analytical data show that the average measured residue in/on grass hay samples was 0.10 ppm (n = 24, σn-1 = 0.19 ppm). These data support a proposed tolerance for pyriproxyfen in/on grass forage of 0.5 ppm and grass hay of 1.0 ppm.

ii. *Secondary residues*. The proposed new use on grass represents an additional feed commodity. Using established and proposed tolerances to calculate the maximum feed exposure to feed animals, and using the very low potential for residue transfer demonstrated in the milk cow feeding residue study, detectable secondary residues in animal tissues, milk, and eggs are not expected. Therefore, no tolerances are required for these commodities.

iii. *Rotational crops*. The results of a confined rotational crops accumulation study indicate that no rotational crop planting restrictions or rotational crop tolerances are required.

B. Toxicological Profile

1. *Acute toxicity*. The acute toxicity of technical grade pyriproxyfen is low by all routes. The compound is classified as Category III for acute dermal and inhalation toxicity, and Category IV for acute oral toxicity, and skin/eye irritation. Pyriproxyfen is not a skin sensitizing agent.

2. *Genotoxicity*. Pyriproxyfen does not present a genetic hazard. Pyriproxyfen was negative in the following tests for mutagenicity: Ames assay with and without S9, *in vitro* unscheduled DNA synthesis in HeLa S3 cells, *in vitro* gene mutation in V79 Chinese hamster cells, and *in vitro* chromosomal aberration with and without S9 in Chinese hamster ovary cells.

3. *Reproductive and developmental toxicity*. Pyriproxyfen is not a developmental or reproductive toxicant. Developmental toxicity studies have been performed in rats and rabbits, and multigenerational effects on reproduction were tested in rats. These studies have been reviewed and found to be acceptable to the Agency.

In the developmental toxicity study conducted with rats, technical pyriproxyfen was administered by gavage at levels of 0, 100, 300, and 1,000 milligrams/kilogram/bodyweight day (mg/kg/bwt day) during gestation days 7–17. Maternal toxicity (mortality, decreased bwt gain and food consumption, and clinical signs of toxicity) was observed at doses of 300 mg/kg/bwt day and greater. The maternal no observed adverse effect level (NOAEL) was 100 mg/kg/bwt day. A transient increase in skeletal variations was observed in rat fetuses from females exposed to 300 mg/kg/bwt day and greater. These effects were not present in animals examined at the end of the postnatal period, therefore, the NOAEL for prenatal developmental toxicity was 100 mg/kg/bwt day. An increased incidence of visceral and skeletal variations was observed postnatally at 1,000 mg/kg/bwt day. The NOAEL for postnatal developmental toxicity was 300 mg/kg/bwt day.

In the developmental toxicity study conducted with rabbits, technical pyriproxyfen was administered by gavage at levels of 0, 100, 300, and 1,000 mg/kg/bwt day during gestation days 6–18. Maternal toxicity (clinical signs of toxicity including one death, decreased bwt gain and food consumption, and abortions or premature deliveries) was observed at oral doses of 300 mg/kg/bwt day or higher. The maternal NOAEL was 100 mg/kg/bwt day. No developmental effects were observed in the rabbit fetuses. The NOAEL for developmental toxicity in rabbits was 1,000 mg/kg/bwt day.

In the rat reproduction study, pyriproxyfen was administered in the diet at levels of 0, 200, 1,000, and 5,000 ppm through two generations of rats. Adult systemic toxicity (reduced bwts, liver and kidney histopathology, and increased liver weight) was produced at the 5,000 ppm dose (453 mg/kg/bwt day in males, 498 mg/kg/bwt day in females) during the pre-mating period. The systemic NOAEL was 1,000 ppm (87 mg/kg/bwt day in males, 96 mg/kg/bwt day in females). No effects on reproduction were produced at 5,000 ppm, the higest dose tested (HDT).

4. *Subchronic toxicity*. Subchronic oral toxicity studies conducted with pyriproxyfen technical in the rat, mouse, and dog indicate a low level of toxicity. Effects observed at high dose levels consisted primarily of decreased bwt gain; increased liver weights; histopathological changes in the liver and kidney; decreased red blood cell counts, hemoglobin and hematocrit;

altered blood chemistry parameters; and, at 5,000 and 10,000 ppm in mice, adecrease in survival rates. The NOAELs from these studies were 400 ppm (23.5 mg/kg/bwt day for males, 27.7 mg/kg/bwt day for females) in rats, 1,000 ppm (149.4 mg/kg/bwt day for males, 196.5 mg/kg/bwt day for females) in mice, and 100 mg/kg/bwt day in dogs. In a four week inhalation study of pyriproxyfen technical in rats, decreased bwt and increased water consumption were observed at 1,000 mg/m^3. The NOAEL in this study was 482 mg/m^3.

A 21-day dermal toxicity study in rats with pyriproxyfen technical did not produce any signs of dermal or systemic toxicity at 1,000 mg/kg/bwt day, the HDT. In a 21-day dermal study conducted with KNACK®. Insect Growth Regulator, the test material produced a NOAEL of 1,000 mg/kg/bwt day HDT for systemic effects, and a NOAEL for skin irritation of 100 mg/kg/bwt day.

5. *Chronic toxicity*. Pyriproxyfen technical has been tested in chronic studies with dogs, rats, and mice. EPA has established a reference dose (RfD) for pyriproxyfen of 0.35 mg/kg/bwt day, based on the NOAEL in female rats from the 2-year chronic/oncogenicity study. Effects cited by EPA in the RfD Tracking Report include negative trend in mean red blood cell volume, increased hepatocyte cytoplasm and cytoplasm: Nucleus ratios, and decreased sinusoidal spaces.

Pyriproxyfen is not a carcinogen. Studies with pyriproxyfen have shown that repeated high dose exposures produced changes in the liver, kidney, and red blood cells, but did not produce cancerin test animals. No oncogenic response was observed in a rat 2-year chronic feeding/oncogenicity study or in a seventy-eight week study on mice. The oncogenicity classification of pyriproxyfen is ''E'' (no evidence of carcinogenicity for humans).

Pyriproxyfen technical was administered to dogs in capsules at doses of 0, 30, 100, 300, and 1,000 mg/kg/bwt day for 1-year. Dogs exposed to dose levels of 300 mg/kg/bwt day or higher showed overt clinical signs of toxicity, elevated levels of blood enzymes and liver damage. The NOAEL in this study was 100 mg/kg/bwt day.

Pyriproxyfen technical was administered to mice at doses of 0, 120, 600, and 3,000 ppm in diet for 78–weeks. The NOAEL for systemic effects in this study was 600 ppm (84 mg/kg/bwt day in males, 109.5 mg/kg/bwt day in females), and a LOAEL of 3,000 ppm (420 mg/kg/bwt day in males, 547 mg/kg/bwt day in females) was established based on an increase in kidney lesions.

In a 2-year study in rats, pyriproxyfen technical was administered in the diet at levels of 0, 120, 600, and 3,000 ppm. The NOAEL for systemic effects in this study was 600 ppm (27.31 mg/kg/bwt day in males, 35.1 mg/kg/bwt day in females). A lowest observed adverse effect level (LOAEL) of 3,000 ppm (138 mg/kg/bwt day inmales, 182.7 mg/kg/bwt day in females) was established based on a depression in bwt gain in females.

6. *Animal metabolism*. The absorption, tissue distribution, metabolism and excretion of ^{14}C-labeled pyriproxyfen were studied in rats after single oral doses of 2 or 1,000 mg/kg/bwt (phenoxyphenyl and pyridyl label), and after a single oral dose of 2 mg/kg/bwt (phenoxyphenyl label only) following 14 daily oral doses at 2 mg/kg/bwt of unlabelled material. For all dose groups, most (88–96%) of the administered radio-label was excreted in the urine and feces within 2–days after radio- labeled test material dosing, and 92–98% of the administered dose was excreted within 7–days. Seven–days after dosing, tissue residues were generally low, accounting for no more than 0.3% of the dosed ^{14}C. Radiocarbon concentrations in fat were the higher than in other tissues analyzed. Recovery in tissues over time indicates that the potential for bioaccumulation is minimal. There were no significant sex or dose-related differences in excretion or metabolism.

7. *Metabolite toxicology*. Metabolism studies of pyriproxyfen in rats, goats, and hens, as well as the fish bioaccumulation study demonstrate that the parent is very rapidly metabolized and eliminated. In the rat, most (88–96%) of the administered radiolabel was excreted in the urine and feces within 2–days of dosing, and 92–98% of the administered dose was excreted within 7–days. Tissue residues were low 7–days after dosing, accounting for no more than 0.3% of the dosed ^{14}C. Because parent and metabolites are not retained in the body, the potentil for acute toxicity from *in situ* formed metabolites is low. The potential for chronic toxicity is adequately tested by chronic exposure to the parent at the maximum tolerance dose and consequent chronic exposure to the internally formed metabolites.

Seven metabolites of pyriproxyfen, 4'-OH-pyriproxyfen, 5'3'-OH-pyriproxyfen, desphenyl-pyriproxyfen, POPA, PYPAC, 2-OH-pyridine and 2,5-diOH-pyridine, have been tested for mutagenicity (Ames) and acute oral toxicity to mice. All seven metabolites were tested in the Ames assay with and without S9 at doses up to 5,000 micro-grams per plate or up to the growth inhibitory dose. The metabolites did not induce any significant increases in revertant colonies in any of the test strains. Positive control chemicals showed marked increases in revertant colonies. The acute toxicity to mice of 4'-OH-pyriproxyfen, 5'3'-OH-pyriproxyfen, desphenyl-pyriproxyfen, POPA, and PYPAC did not appear to markedly differ from pyriproxyfen, with all metabolites having acute oral LD_{50} values greater than 2,000 mg/kg/bwt. The two pyridines, 2-OH-pyridine and 2,5-diOH-pyridine, gave acute oral LD_{50} values of 124 (male), and 166 (female) mg/kg/bwt, and 1,105 (male) and 1,000 (female) mg/kg/bwt, respectively.

8. *Endocrine disruption*. Pyriproxyfen is specifically designed to be an insect growth regulator and is known to produce juvenoid effects on arthropod development. However, this mechanism-of-action in target insects and other some arthropods has no relevance to any mammalian endocrine system. While specific tests, uniquely designed to evaluate the potential effects of pyriproxyfen on mammalian endocrine systems have not been conducted, the toxicology of pyriproxyfen has been extensively evaluated in acute, sub-chronic, chronic, developmental, and reproductive toxicology studies including detailed histopathology of numerous tissues. The results of these studies show no evidence of any endocrine-mediated effects and no pathology of the endocrine organs. Consequently, it is concluded that pyriproxyfen does not possess estrogenic or endocrine disrupting properties applicable to mammals.

C. Aggregate Exposure

1. *Dietary exposure*. An evaluation of chronic dietary exposure to including both food and drinking water has been performed for the U.S. population and various sub-populations including infants and children. No acute dietary endpoint and dose was identified in the toxicology data base for pyriproxyfen, therefore, the Agency has concluded that there is a reasonable certainty of no harm from acute dietary exposure.

i. *Food*. Chronic dietary exposure to pyriproxyfen residues was calculated for the U.S.population and 16 population subgroups assuming tolerance level residues, processing factors from residue studies, and 100 percent of the crop treated (PCT). The analyses included residue data for all existing uses, pending uses, and proposed new uses. The results from

several representative subgroups are listed below. Chronic exposure to the overall U.S. population is estimated to be 0.0008 mg/kg/bwt day, representing 0.24% of the RfD. For the most highly exposed sub-population, children 1 to 2 years of age, exposure is calculated to be 0.0009 mg/kg/bwt day, or 0.26% of the RfD in the following Table 1. Generally speaking, the Agency has no cause for concern if total residue contribution for established and proposed tolerances is less than 100% of the RfD.

TABLE 1.—CALCULATED CHRONIC DIETARY EXPOSURES TO THE TOTAL U.S. POPULATION AND SELECTED SUB-POPULATIONS TO PYRIPROXYFEN RESIDUES IN FOOD

Population Subgroup	Exposure (mg/kg/bwt day)	Percent of RfD
Total U.S. population	0.00083	0.24
Children (1–2 years)	0.00091	0.26
Non-nursing infants(<1 year old)	0.00049	0.14
All infants (<1 year old)	0.00051	0.15
Children (6–12 years)	0.00044	0.13
Females (13–49 years)	0.00042	0.12
Nursing Infants (<1 year old)	0.00023	0.07

ii. *Drinking water*. Since pyriproxyfen is applied outdoors to growing agricultural crops, the potential exists for pyriproxyfen or its metabolites to reach ground surface or surface water that may be used for drinking water. Because of the physical properties of pyriproxyfen, it is unlikely that pyriproxyfen or its metabolites can leach to potable ground water. To quantify potential exposure from drinking water, surface water concentrations for pyriproxyfen were estimated using generic expected environmental concentration (GENEEC). The peak predicted concentration in drinking water was 0.86 part per billion (ppb). Using standard assumptions about bwt and water consumption, the chronic exposure to pyriproxyfen from this drinking water would be 0.00009 mg/kg/day for infants, the most sensitive subpopulation. This represents 0.025% of the RfD (0.35 mg/kg/day) for infants. Based on this worse case analysis, the contribution of water to the dietary risk is negligible.

2. *Non-dietary exposure*. Pyriproxyfen is currently registered for use on residential non-food sites. Pyriproxyfen is the active ingredient (a.i.) in numerous registered products for flea and tick control. Formulations include foggers, aerosol sprays, emulsifiable concentrates, and impregnated materials (pet collars). With the exception of the pet collar uses, consumer use of pyriproxyfen typically results in acute and short-term intermittent exposures. No acute dermal, or inhalation dose or endpoint was identified in the toxicity data for pyriproxyfen. Similarly, doses and endpoints were not identified for short and intermediate term dermal or inhalation exposure to pyriproxyfen. The Agency has concluded that there are reasonable certainties of no harm from acute, short term, and intermediate term dermal and inhalation occupational and residential exposures due to the lack of significant toxicological effects observed.

Chronic residential post-application exposure and risk assessments were conducted to estimate the potential risks from pet collar uses. The risk assessment was conducted using the following assumptions: Application rate of 0.58 mg/a.i. day (product label), average bwt for a 1-6 year old child of 10 kg, the a.i. dissipates uniformly through 365 days (the label instruct to change collar once a year), 1% of the a.i. is available for dermal and inhalation exposure per day (assumption from Draft EPA Standard Operating Procedures (SOPs) for Residential Exposure Assessments, December 18, 1997). The assessment also assumes an absorption rate of 100%. This is a conservative assumption since the dermal absorption was estimated to be 10%. The estimated chronic term margin of exposure (MOE) was 61,000 for children, and 430,000 for adults. The risk estimates indicate that potential risks from pet collar uses do not exceed the Agency's level of concern.

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 no other pesticidal compounds that are structurally related

to pyriproxyfen and have similar effects on animals. In consideration of potential cumulative effects of pyriproxyfen 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 pyriproxyfen would be cumulative with those of other chemical compounds. Thus, only the potential risks of pyriproxyfen have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of pyriproxyfen consistent with the schedule established by EPA **Federal Register** of August 4, 1997 (62 FR 42020) (FRL–5734–6) and other subsequent EPA publications pursuant to the Food Quality Protection Act(FQPA).

E. Safety Determination

1. *U.S. population*—i. *Chronic dietary exposure and risk s adult sub-populations.* The results of the chronic dietary exposure assessment described above demonstrate that estimates of chronic dietary exposure for all existing, pending and proposed uses of pyriproxyfen are well below the chronic RfD of 0.35 mg/kg/bwt day. The estimated chronic dietary exposure from food for the overall U.S. population and many non-child/infant subgroups is from 0.00014 to 0.00042 mg/kg/bwt day, 0.04 to 0.12% of the RfD. Addition of the small but worse case potential chronic exposure from drinking water (calculated above) increases exposure by only 0.00002 mg/kg/bwt day and does not change the maximum occupancy of the RfD significantly. 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 or any non-child/infant subgroups from aggregate, chronic dietary exposure to pyriproxyfen residues.

ii. *Acute dietary exposure and risk s adult sub-populations.* No acute dietary endpoint and dose were identified in the toxicology data base for pyriproxyfen; therefore, it can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. Population or any non-child/infant subgroups from aggregate, acute dietary exposure to pyriproxyfen residues.

iii. *Non-dietary exposure and aggregate risk s adult sub-populations.* Acute, short term,and intermediate term dermal and inhalation risk assessments for residential exposure are not required due to the lack of significant toxicological effects observed. The results of a chronic residential post-application exposure and risk assessment for pet collar uses demonstrate that potential risks from pet collar uses do not exceed the Agency's level of concern. The estimated chronic term MOE for adults was 430,000.

2. *Infants and children*—i. *Safety factor for infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of pyriproxyfen, 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 prenatal and postnatal toxicity for pyriproxyfen is complete with respect to current data requirements. There are no special prenatal or postnatal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies or the 2-generation reproductive toxicity study in rats. Valent concludes that reliable data support use of the standard 100-fold uncertainty factor and that an additional uncertainty factor is not needed for pyriproxyfen to be further protective of infants and children.

ii. *Chronic dietary exposure and risks infants and children.* Using the conservative exposure assumptions described above, the percentage of the RfD that will be utilized by chronic dietary (food only) exposure to residues of pyriproxyfen ranges from 0.00023 mg/kg/bwt day for nursing infants, up to 0.00091 mg/kg/bwt day for children (1 to 2 years of age), 0.07 to 0.26% of the RfD, respectively. Adding the worse case potential incremental exposure to infants and children from pyriproxyfen in drinking water (0.00009 mg/kg/bwt day) does not materially increase the aggregate, chronic dietary exposure and only increases the occupancy of the RfD by 0.009% to 0.010% for children (1 to 2 years of age). 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 dietary exposure to pyriproxyfen residues.

iii. *Acute dietary exposure and risk s infants and children.*No acute dietary endpoint and dose were identified in the toxicology data base for pyriproxyfen; therefore, it can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, acute dietary exposure to pyriproxyfen residues.

iv. *Non-dietary exposure and aggregate risk s infants and children.* Acute, short term, and intermediate term dermal and inhalation risk assessments for residential exposure are not required due to the lack of significant toxicological effects observed. The results of a chronic residential post-application exposure and risk assessment for pet collar uses demonstrate that potential risks from pet collar uses do not exceed the Agency's level of concern. The estimated chronic term MOE for children was 61,000.

F. International Tolerances

There are no presently existing Codex MRLs for pyriproxyfen.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0350; FRL–7684–8]

Pesticide Emergency Exemptions; Agency Decisions and State and Federal Agency Crisis Declarations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has granted or denied emergency exemptions under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) for use of pesticides as listed in this notice. The exemptions or denials were granted during the period July 1, 2004 to September 30, 2004 to control unforseen pest outbreaks.

FOR FURTHER INFORMATION CONTACT: See each emergency exemption or denial for the name of a contact person. The following information applies to all contact persons: Branch Chief, Emergency Response Team, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–9366.

SUPPLEMENTARY INFORMATION: EPA has granted or denied emergency exemptions to the following State and Federal agencies. The emergency exemptions may take the following