groups associated with the use of fenthion who may be interested in participating in the risk assessment/risk management process, and will contact them individually to inform them that no Technical Briefing will be held. EPA is willing to meet with stakeholders to discuss the fenthion revised risk assessments. Minutes of all meetings will be docketed.

In addition, this notice starts a 60-day public participation period during which the public is encouraged to submit risk management proposals or otherwise comment on risk management for fenthion. The Agency is providing an opportunity, through this notice, for interested parties to provide written risk management proposals or ideas to the Agency on the pesticides specified in this notice. Such comments and proposals could address ideas about how to manage dietary, occupational, or ecological risks on specific fenthion use sites or crops across the United States or in a particular geographic region of the country. To address dietary risk, for example, commenters may choose to discuss the feasibility of lower application rates, increasing the time interval between application and harvest (''pre-harvest intervals''), modifications in use, or suggest alternative measures to reduce residues contributing to dietary exposure. For occupational risks, for example, commenters may suggest personal protective equipment or technologies to reduce exposure to workers and pesticide handlers. For ecological risks, commentors may suggest ways to reduce environmental exposure, e.g., exposure to birds, fish, mammals, and other non-target organisms. EPA will provide other opportunities for public participation and comment on issues associated with the organophosphate tolerance reassessment program. Failure to participate or comment as part of this opportunity will in no way prejudice or limit a commenter's opportunity to participate fully in later notice and comment processes. All comments and proposals must be received by EPA on or before December 13, 1999 at the addresses given under Unit I. of the ''SUPPLEMENTARY INFORMATION.'' Comments and proposals will become part of the Agency record for the organophosphate specified in this notice.

### List of Subjects

Environmental protection, Chemicals, Pesticides and pests.

Dated: October 7, 1999.

**Lois Rossi,**

*Director, Special Review and Reregistration Division, Office of Pesticide Programs.*

[FR Doc. 99–26807 Filed 10–13–99; 8:45 am]

**BILLING CODE 6560–50–F**

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–893; FRL–6382–7]**

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

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice.

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

**DATES:** Comments, identified by docket control number PF–893, must be received on or before November 15, 1999.

**ADDRESSES:** Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION'' section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–893 in the subject line on the first page of your response.

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

| Product Manager | Office location/telephone number/e-mail address | Address | Petition number(s) |
|---|---|---|---|
| Sidney Jackson .... | Rm. 272, CM #2, 703–305–7610, e-mail: jackson.sidney@epamail.epa.gov. | 1921 Jefferson Davis Hwy, Arlington, VA | PP 9E6035 |
| Mary L. Waller ..... | Rm. 249, CM #2, 703–308–9354, e-mail: waller.mary@epamail.epa.gov. | Do. | PP 9F5066, 9F6023, 7E4830 |

**SUPPLEMENTARY INFORMATION:**

### I. General Information

*A. Does this Action Apply to Me?*

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

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

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

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

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

2. *In person*. The Agency has established an official record for this action under docket control number PF–893. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

*C. How and to Whom Do I Submit Comments?*

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–893 in the subject line on the first page of your response.

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

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by E-mail to: ''*opp-docket@epa.gov*,'' or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–893. Electronic comments may also be filed online at many Federal Depository Libraries.

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

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

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

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

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

## II. What Action is the Agency Taking?

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

### List of Subjects

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

Dated: October 4, 1999.

**James Jones,**

*Director, Registration Division, Office of Pesticide Programs.*

### Summaries of Petitions

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

### 1. Interregional Research Project Number 4 (IR-4)

*PP 9E6035*

EPA has received a pesticide petition [9E6035] from the IR-4 New Jersey Agricultural Experiment Station, P.O. Box 231, Rutgers University, New Brunswick, NJ 08903 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 the insecticide, spinosad (Factor A and Factor D): Factor A is 2-[(6-deoxy-2,3,4-tri-O-methyl-alpha-L-manno-pyranosyl)oxy]-13-[5-(dimethylamino)-tetrahydro-6-methyl-2 H-pyran-2-yl]oxy]-9-ethyl-2,3,3a,5a,5b,6,9,10,11,12,13,14,16a,6b-tetradecahydro-14-methyl-1 H-as-Indaceno [3,2-d]oxacyclododecin-7,15-dione. Factor D is 2-[(6-deoxy-2,3,4-tri-O-methyl-alpha-L-manno-pyranosyl)oxy]-13-[[5-(dimethylamino)-tetrahydri-6-methyl-2H-pyran-2-yl]oxy]-9-ethyl-2,3,3a,5a,5b,6,9,10,11,12,13,14,16a,16b-tetradecahydro-4,14-dimethyl- 1H-as-Indaceno[3,2-d]oxacyclododecin-7,15-dione in or on the raw agricultural

commodities (RACs) barley, buckwheat, oats, and rye (grains) at 0.02 parts per million (ppm); pearl millet, proso millet, and grain Amaranth (grains) at 1 ppm; teosinte and popcorn (grains); grass, forage, fodder and hay (crop group 17); and animal feed, nongrass (crop group 18) at 0.02 ppm; turnip greens at 10 ppm; cilantro, and watercress at 8 ppm; tropical fruits (sugar apple, cherimoya, atemoya, custard apple, ilama, soursop, biriba, lychee, longan, spanish lime, rambutan, pulasan, papaya, star apple, black sapote, mango, sapodilla, canistel, mamey sapote, avocado, guava, feijoa, jaboticaba, wax jambu, starfruit, passion fruit, acerola, and white sapote) at 0.3 ppm; ti palm at 10 ppm. Additionally, IR-4 requested a tolerance for spinosad on pistachio at 0.02 ppm under conditional registration. Spinosad is manufactured by Dow AgroSciences LLC, 9330 Zionsville Road, Indianapolis, IN 46268.

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.

*A. Residue Chemistry*

1. *Plant metabolism*. The metabolism of spinosad in plants (apples, cabbage, cotton, tomato, and turnip) and animals (goats and poultry) is adequately understood for the purposes of these tolerances. A rotational crop study showed no carryover of measurable spinosad related residues in representative test crops.

2. *Analytical method*. There is a practical method (immunoassay) for detecting (0.005 ppm) and measuring (0.01 ppm) levels of spinosad in or on food with a limit of detection (LOD) that allows monitoring of food with residues at or above the level set for these tolerances. The method has had a successful method tryout in EPA's laboratories.

3. *Magnitude of residues*. No additional residue data are being submitted in support of the proposed residue tolerances. Previously submitted cereal grain crops residue data in support of a pending tolerance petition (PP 8F5002) are to be used for barley, buckwheat, oats, and rye (wheat residue studies); pearl millet, proso millet, and grain Amaranth (sorghum residue studies); and popcorn and teosinte (field corn residue studies). In the same petition, there is a pending tolerance of 1 ppm for forage, fodder, hay, and straw of cereal grains (crop group 16). Previously submitted residue data in support of the established residue tolerance on Brassica (cole) leafy vegetables, greens subgroup are to be used for turnip greens and ti palm. Previously submitted residue data in support of the established residue tolerance on leafy vegetables (except Brassica) are to be used for cilantro and watercress. Previously submitted residue data in support of almond are used for pistachio. Previously submitted residue data in support of established residue tolerances on citrus fruits and apples and a pending residue tolerance (PP 8F5002) on stone fruits are to be used for tropical fruits. The use pattern (low application rate and spot treatment nature) associated with the forage crops (crop groups 17 and 18) indicates that no residue data are needed to establish a limit of quantitation (LOQ) tolerance.

As a condition for registration of spinosad on pistachios, the Agency requires IR-4 to fulfill the guideline requirements of a total of five completed field rials on representative commodities for Crop Group 14, almonds and pecans.

*B. Toxicological Profile*

1. *Acute toxicity—Spinosad has low acute toxicity*. The rat oral lethal dose ($LD_{50}$) is 3,738 milligrams/kilograms (mg/kg) for males and > 5,000 mg/kg for females, whereas the mouse oral $LD_{50}$ is > 5,000 mg/kg. The rabbit dermal $LD_{50}$ is > 5,000 mg/kg and the rat inhalation lethal concentration ($LC_{50}$) is > 5.18 milligrams/liter (mg/L) air. In addition, spinosad is not a skin sensitizer in guinea pigs and does not produce significant dermal or ocular irritation in rabbits. End use formulations of spinosad that are water-based suspension concentrates have similar low acute toxicity profiles.

2. *Genotoxicity*. Short-term assays for genotoxicity consisting of a bacterial reverse mutation assay (Ames test), an *in vitro* assay for cytogenetic damage using the Chinese hamster ovary cells, an *in vitro* mammalian gene mutation assay using mouse lymphoma cells, an *in vitro* assay for DNA damage and repair in rat hepatocytes, and an *in vivo* cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with spinosad. These studies show that spinosad does not elicit a genotoxic response.

3. *Reproductive and developmental toxicity*. Spinosad caused decreased body weight (bwt) in maternal rats given 200 mg/kg/day by gavage, the highest dose tested (HDT). This was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The no observed adverse effect levels (NOAELs) for maternal toxicity and fetal toxicity in rats were 50 and 200 mg/kg/day, respectively. A teratology study in rabbits showed that spinosad caused decreased bwt gain and a few abortions in maternal rabbits given 50 mg/kg/day, the HDT. Maternal toxicity was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOAELs for maternal and fetal toxicity in rabbits were 10 and 50 mg/kg/day, respectively. In a 2-generation reproduction study in rats, parental toxicity was observed in both males and females given 100 mg/kg/day, the HDT. Perinatal effects (decreased litter size and pup weight) at 100 mg/kg/day were attributed to maternal toxicity. The NOAEL for maternal and pup effects was 10 mg/kg/day.

4. *Subchronic toxicity*. Spinosad was evaluated in 13–week dietary studies and showed NOAELs of 4.89 and 5.38 mg/kg/day, respectively in male and female dogs; 6 and 8 mg/kg/day, respectively in male and female mice; and 33.9 and 38.8 mg/kg/day, respectively in male and female rats. No dermal irritation or systemic toxicity occurred in a 21–day repeated dose dermal toxicity study in rabbits given 1,000 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with spinosad in the dog and the rat, the EPA has set a chronic population adjusted dose (cPAD) of 0.027 mg/kg/day for spinosad. The cPAD has incorporated a 100-fold uncertainty factor to the NOAELs found in the chronic dog study to account for interspecies and intraspecies variation. cPAD is equivalent to the reference dose (RfD) divided by the Food Quality Protection Act (FQPA) safety factor (SF). For spinosad, EPA has determined that the additional 10x SF to account for enhanced sensitivity of infants and children be reduced to 1x, i.e., removed. Thus, the cPAD of 0.027 mg/kg/day is equivalent to the chronic RfD. The NOAELs shown in the dog chronic study were 2.68 and 2.72 mg/kg/day, respectively for male and female dogs. The NOAELs (systemic) shown in the rat chronic/carcinogenicity/ neurotoxicity studies were 9.5 and 12.0 mg/kg/day, respectively for male and female rats. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), it is proposed that spinosad be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18–month mouse feeding study and a 24–month rat feeding study at all dosages tested. The

NOAELs shown in the mouse carcinogenicity study were 11.4 and 13.8 mg/kg/day, respectively for male and female mice. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive mortality. Thus, the petitioner believes that the doses tested are adequate for identifying a cancer risk and that a cancer risk assessment is not needed.

6. *Animal metabolism*. There were no major differences in the bioavailability, routes or rates of excretion, or metabolism of spinosyn A and spinosyn D following oral administration in rats. Urine and fecal excretions were almost completed in 48 hours post-dosing. In addition, the routes and rates of excretion were not affected by repeated administration.

7. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent material (spinosyn A and spinosyn D). Thus, there is no need to address metabolite toxicity.

8. *Endocrine disruption*. There is no evidence to suggest that spinosad has an effect on any endocrine system.

*C Aggregate Exposure*

1. *Dietary exposure*—i. *Food*. For purposes of assessing the potential dietary exposure from use of spinosad on the RACs listed in this notice as well as from other existing and pending spinosad crop uses, a conservative estimate of aggregate exposure is determined by basing the Theoretical Maximum Residue Contribution (TMRC) on the proposed tolerance level for spinosad and assuming that 100% of these proposed new crops and other pending and existing (registered for use) crops grown in the United States were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. The use of a tolerance level and existing and pending spinosad crop uses, a conservative estimate of aggregate exposure is determined by basing the TMRC on the proposed tolerance level for spinosad and assuming that 100% of these proposed new crops and other pending and existing (registered for use) crops grown in the United States were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. The use of a tolerance level and 100% of crop treated clearly results in an overestimate of human exposure and a safety determination for the use of spinosad on crops cited in this summary that is based on a conservative exposure assessment.

ii. *Drinking water*. Another potential source of dietary exposure to pesticides is residues in drinking water. Based on the available environmental studies conducted with spinosad wherein its properties show little or no mobility in soil, there is no anticipated exposure to residues of spinosad in drinking water. In addition, there is no established maximum concentration level for residues of spinosad in drinking water.

2. *Non-dietary exposure*. Spinosad is currently registered for use on a number of crops including cotton, fruits, and vegetables in the agriculture environment. Spinosad is also currently registered for outdoor use on turf and ornamentals at low rates of application (0.04 to 0.54 pounds of active ingredient per acre (lbs a.i./ per acre) and indoor use for drywood termite control (extremely low application rates used with no occupant exposure expected). Thus, the potential for non-dietary exposure to the general population is considered negligible.

*D. Cumulative Effects*

The potential for cumulative effects of spinosad and other substances that have a common mechanism of toxicity is also considered. In terms of insect control, spinosad causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotinic acetylcholine receptors by a mechanism that is clearly novel and unique among known insecticidal compounds. Spinosad also has effects on the gamma aminobatopic acid (GABA) receptor function that may contribute further to its insecticidal activity. Based on results found in tests with various mammalian species, spinosad appears to have a mechanism of toxicity like that of many amphophilic cationic compounds. There is no reliable information to indicate that toxic effects produced by spinosad would be cumulative with those of any other pesticide chemical. Thus it is appropriate to consider only the potential risks of spinosad in an aggregate exposure assessment.

*E. Safety Determination*

1. *U.S. population*. Using the conservative exposure assumptions and the cPAD, the aggregate exposure to spinosad use on other pending and existing crop uses will utilize 25.5% of the cPAD for the U.S. population. A more realistic estimate of dietary exposure and risk relative to a chronic toxicity endpoint is obtained if average anticipated residue values from field trials are used. Inserting the average residue values in place of tolerance residue levels produces a more realistic, but still conservative risk assessment. Based on average anticipated residue levels in a dietary risk analysis, the use of spinosad on other pending and existing crop uses will utilize 4.1% of the cPAD for the U.S. population. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. The new crop uses proposed in this notice are minor uses. The petitioner expects these uses to contribute only a negligible impact to the cPAD, and also believes that there is reasonable certainty that no harm will result from aggregate exposure to spinosad residues on existing and all pending crop uses including the ones listed in this notice.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of spinosad, data from developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability and potential systemic toxicity of mating animals and on various parameters associated with the well-being of pups.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. Based on the current toxicological data requirements, the data base for spinosad relative to prenatal and postnatal effects for children is complete. Further, for spinosad, the NOAELs in the dog chronic feeding study which were used to calculate the cPAD (0.027 mg/kg/day) are already lower than the NOAELs from the developmental studies in rats and rabbits by a factor of more than 10-fold.

Concerning the reproduction study in rats, the pup effects shown at the HDT were attributed to maternal toxicity. Therefore, the petitioner concludes that an additional uncertainty factor is not needed and that the cPAD at 0.027 mg/

kg/day is appropriate for assessing risk to infants and children.

In addition, EPA has determined that the 10x factor to account for enhanced sensitivity of infants and children is not needed for spinosad because: (i) The data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or postnatal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and 2-generation reproduction in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity, (ii) no neurotoxic signs have been observed in any of the standard required studies conducted, and (iii) the toxicology data base is complete and there are no data gaps.

Using the conservative exposure assumptions previously described as tolerance level residues, the percent cPAD utilized by the aggregate exposure to residues of spinosad on other pending and existing crop uses is 51.2% for children 1 to 6 years old, the most sensitive population subgroup. If average or anticipated residues are used in the dietary risk analysis, the use of spinosad on these crops will utilize 9.4% of the cPAD for children 1 to 6 years old. The new crop uses proposed in this notice are minor ones and are expected to contribute only a negligible impact to the cPAD. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, the petitioner concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues on the above proposed uses including other pending and existing crop uses.

*F. International Tolerances*

There are no Codex maximum residue levels established for residues of spinosad on barley, buckwheat, oats, rye, pearl millet, proso millet, grain Amaranth, teosinte, popcorn, turnip greens, cilantro, watercress, tropical fruit, ti palm, grass forage, fodder, and hay (crop group 17), and nongrass animal feeds (crop group 18) or any other food or feed crop.

**2. Sipcam Agro USA, Inc.**

*PP 9F5066, 9F6023, and 7E4830*

EPA has received three pesticide petitions [9F5066, 9F6023, and 7E4830] from Sipcam Agro USA, Inc., 70 Mansell Court, Suite 230, Rosewell, GA 30076 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of 1-2(2,4-dichlorophenyl)-3-(1,1,2,2-tetrafluoroethoxy)propyl-1H-1,2,4-triazole (Tetraconazole) in or on the RAC of beets, sugar at 0.01 ppm; beets, sugar, roots at 0.1 ppm; beets, sugar, tops at 7.0 ppm; beets, sugar, pulp, dried at 0.3 ppm; and beets, sugar, molasses at 0.3 ppm (9F5066), peanuts meat (hulls removed) at 0.03 ppm, peanuts meal at 0.03 ppm, and peanuts oil at 0.1 ppm (9F6023), and imported bananas at 0.2 ppm (7E4830) and in animal commodities of milk at 0.02 ppm; cattle, meat at 0.01 ppm; cattle meat byproducts at 2.0 ppm and cattle fat at 0.1 ppm (9F5066). EPA has determined that the petitions contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

*A. Residue Chemistry*

1. *Plant metabolism*. The nature of the residue of tetraconazole in plants was studied extensively in wheat, grapes and sugar beets. The principal compounds found in all three plant species were unchanged tetraconazole and the degradation product triazole. Evidence was found for more extensive metabolism in plant tissues to form bound residues that were incorporated into the structural matrices (cellulose and lignin) surrounding plant cells.

2. *Analytical method*. An analytical residue method utilizing gas chromatography with electron capture detection is available for enforcement purposes, which has been validated among all banana, sugar beet, and peanut raw and processed matrices, as well as for milk, meat, and meat byproduct matrices. This method is described within the magnitude of residue studies provided to EPA in support of the petitions for tolerances pertaining to bananas, sugar beets, and peanut matrices.

3. *Magnitude of residues*—i. *Banana*. Residue data from a study conducted with tetraconazole applied in the field to banana plants at 12 locations in the field throughout Latin America to support establishment of a tolerance of 0.2 ppm (unbagged, whole fruit basis) for residues of tetraconazole on bananas. The magnitude of residues on the edible pulp portion of the fruit grown under typical banana cultivation practices was less than 0.02 ppm, which is the maximum anticipated residue to be used for dietary exposure risk assessment.

ii. *Sugar beets*. Residue data from a study conducted with tetraconazole applied to sugar beets in the field at 11 locations in the United States in the manner proposed for registration, and a further study among the products of sugar beet processing, support the establishment of tolerances for residues of tetraconazole on sugar beet roots at a level of 0.1 ppm, on sugar beet tops at 7 ppm, in sugar beet pulp (dried) and in (sugar beet) molasses at 0.3 ppm, and in refined (sugar beet) sugar at 0.01 ppm. A magnitude of residue study conducted with lactating dairy cows fed tetraconazole for a duration of 28 days, followed by terminal sacrifice and analysis of tissues, supports the establishment of tolerances for residues of tetraconazole in milk at 0.02 ppm, in cattle meat at 0.01 ppm, in cattle meat byproducts at 2 ppm, and in cattle fat at 0.1 ppm.

iii. *Peanuts*. Residue data from a study conducted with tetraconazole applied to peanuts in the field at 12 locations in the United States in the manner proposed for registration, and a further study among the products of peanut processing, support the establishment of tolerances for residues of tetraconazole on peanuts (nutmeats) at a level of 0.03 ppm, and in processed peanut meal and oil at 0.03 ppm and 0.1 ppm, respectively.

*B. Toxicological Profile*

1. *Acute toxicity*. Acute toxicity studies with technical grade tetraconazole include: an acute oral dose study in the rat which demonstrated an average (both sexes) $LD_{50}$ level of 1,140 mg/kg bwt; an acute dermal dose toxicity study on the rat which indicated an $LD_{50} > 2,000$ mg/kg; a 4–hour inhalation study in the rat which found the $LD_{50}$ to be greater than 3.66 mg/L of air (MMAD 1.1 microns); a primary eye irritation study with rabbit, indicating that tetraconazole may be a slight eye irritant; a primary dermal irritation study in rabbit showing tetraconazole to be non-irritating; and a dermal sensitization study on guinea pig which demonstrated that tetraconazole was not a skin sensitizer.

2. *Genotoxicty*. The mutagenic potential of tetraconazole has been evaluated in five studies including: a reverse gene mutation assay in *Salmonella typhimurium* cells; a cell mutation assay in mouse lymphoma L5178Y cells *in vitro*, with and without metabolic activation; a chromosomal aberration assay in Chinese hamster ovary cells *in vitro*, with and without metabolic activation; a mouse bone marrow micronucleus assay *in vivo*; and an unscheduled DNA synthesis assay in

HeLa epithelioid cells. All studies were negative for genotoxicity and/or mutagenic potential.

3. *Reproductive and developmental toxicity*. A developmental toxicity study with rats given oral gavage doses of 5, 22.5, and 100 mg/kg/day from days 6 through 15 of gestation resulted in a NOAEL for maternal toxicity of 5 mg/kg/day based upon bwt reduction, reduced food intake and post-dose salivation at the two higher doses, as compared with zero-dose controls. The developmental NOAEL was 22.5 mg/kg/day. Among the highest dose group there was evidence of minimal increase in the incidence of supernumerary ribs among the fetuses.

A developmental toxicity study in rabbits given oral gavage doses of 7.5, 15, and 30 mg/kg/day on days 6 through 18 of gestation resulted in a maternal NOAEL of 15 mg/kg/day. Effects observed in the dams in the high-dose group were decreased bwt gain and reduced food consumption as compared with zero-dose controls. There were no developmental effects observed in this study.

A 2-generation reproduction study in rats fed diets containing 10, 70, and 490 ppm resulted in a reproductive NOAEL of 10 ppm (0.6 mg/kg/day) based upon toxicity to the dam, slightly retarded growth rate in offspring at the higher two doses, and slightly increased liver weights in offspring at the highest dose, as compared with zero-dose controls.

4. *Subchronic toxicity*. A 90–day oral subchronic toxicity study was conducted with technical grade tetraconazole in rats at 10, 60, and 360 ppm in the diet. Treatment related increased liver weights and centrilobular hepatocyte enlargement were observed at the two highest dose levels. The NOAEL was 10 ppm (0.8 mg/kg/day), by comparison with data from the zero-dose control group.

A 90–day oral subchronic toxicity study was conducted in mice with dietary concentrations of technical grade tetraconazole at 5, 25, 125, and 625 ppm. The two highest dosages resulted in liver enlargement, accentuated lobular markings and liver pallor. Microscopic tissue alterations related to tetraconazole were liver enlargement at the three highest doses and single cell necrosis/degeneration and/or areas of necrosis at the two highest doses. The NOAEL was 5 ppm (1 mg/kg/day).

5. *Chronic toxicity*. A 12–month chronic oral toxicity study in Beagle dogs was conducted with technical tetraconazole at dose levels of 0.7, 2.8, and 5.6 mg/kg/day (22.5, 90, and 360 ppm dietary concentrations, respectively). At the highest dose, liver and kidney weights and cholesterol levels were elevated, and liver injury occurred based upon increased levels of GPT, δ-GT and OCT. The no effect level was 0.7 mg/kg/day, as compared with zero-dose control animals.

A chronic (full-lifetime) feeding/carcinogenicity study was conducted with Crl:CD(SD)BR rats fed tetraconazole at dietary levels of 10, 80, 640, and 1,280 ppm for 104 weeks in males and 10, 80, and 640 ppm for 104 weeks in females. In the liver, changes such as hepatocyte enlargement and increased incidence of eosinophilic hepatocytes, seen at doses of 80, 640, or 1,280 ppm were associated with hepatic enzyme induction.

The class of compounds (triazoles) to which tetraconazole belongs is known to induce liver microsomal enzymes. The follicular cell hypertrophy and cystic follicular hyperplasia of the thyroid seen in male rats at 1,280 ppm are also likely to be linked to the hepatic changes. Compounds such as phenobarbital are also known to induce thyroid changes in rats due to increased hepatic clearance of thyroxin, mediated by hepatic enzyme induction.

A special mechanistic study was conducted in order to more fully determine the potential role of microsomal enzyme induction by tetraconazole administered in the diet upon the histopathologic findings in rat. Dietary administration of tetraconazole to rats for 4 weeks resulted in the induction of cytochrome P450, including those of the CYP2B and 3A subfamilies, and of UDP-glucuronyl transferase.

Chronic dietary administration of tetraconazole to rats did not induce a carcinogenic response. No increase in tumors was noted at the high dose groups among males or females. The liver was the target organ. There was a marginal increase in benign liver cell tumors among male rats fed 640 ppm but these were not statistically significant and not dose-related, and the benign tumors did not progress to malignant liver cell tumors. There were some changes in the liver at 80 ppm, whereas 10 ppm (approximately 0.6 mg/kg/day) was observed to be the NOAEL.

The incidence of foci or areas of basophilic hepatocytes was greater in male rats given 10, 80, or 640 ppm than in zero-dose controls. This is a common spontaneous age-related change which showed no dose relationship in this study and is considered unlikely to be of toxicological importance.

A chronic feeding/carcinogenicity study was conducted with tetraconazole in Crl:CD-l (ICR)BR mice at dietary levels of 10, 90, 800, and 1,250 ppm for 80 weeks. Treatment-related non-neoplastic changes were also seen at 1,250 ppm in the lungs, kidneys, testes, epididymides, ovaries and bone, particularly the cranium; a compression of the brain was noted in a number of mice reflecting the extent of cranial bone changes and an increased thymic involution was seen in male mice that died on test. The 1,250 ppm dietary level for tetraconazole, because of the substantial bwt gain changes and increased mortality (more in males), appeared to be above the maximum tolerated dose (MTD). At 800 ppm, there were increases in non neoplastic changes in lungs, kidneys, testes, epididymides, ovaries and bone. In addition, there was substantial reduction in weight gain as compared with zero-dose control animals, but the mortality rate was unaffected. Eight hundred ppm appeared to be a reasonable estimate of the MTD for mouse.

At 90 ppm, non-neoplastic changes were detected in bone and the epididymides in addition to liver changes. No treatment-related findings were seen in mice treated at 10 ppm (approximately 1.5 mg/kg/day), and this dose level was defined as the NOAEL.

In this same study, an increased incidence of benign liver cell tumors was observed in males and females fed 800 ppm, and an increased incidence of benign and malignant liver cell tumors in males and females given 1,250 ppm. These tumors were associated with increased signs of hepatotoxicity including hepatocyte vacuolation and fat deposition at 90, 800, and 1,250 ppm; granulomatous inflammation, pigmented macrophages, bile duct hyperplasia and pericholangitis in mice given 800 and 1,250 ppm. In addition, there was evidence of treatment-related hepatocellular enlargement and increased numbers of altered foci of eosinophilic and basophilic hepatocytes in both sexes given 800 and 1,250 ppm; eosinophilic hepatocytes were noted in male (only) mice receiving 90 ppm.

Tetraconazole is a triazole, and this class of compounds is known to induce liver microsomal enzymes. A special mechanistic study was conducted in order to more fully determine the potential role of microsomal enzyme induction by tetraconazole administered in the diet upon the formation of tumors in mouse. Dietary administration of tetraconazole to mice for 4 weeks results in the induction of cytochrome P450-related activities, as well as the concentrations of microsomal protein and cytochrome P450, and of the phase II activity, and p-nitrophenol UDP-

glucuronyl transferase activity. The effects of tetraconazole on the cytochrome P450-dependent MFO system were somewhat different from those of phenobarbital. Many of these enzymes have not been as well-characterized in mice compared to rats. However, the phase II enzyme activity increases were similar to those of phenobarbital. It is concluded from these studies that prolonged induction of liver microsomal enzymes and/or production of sustained liver injury can lead to the formation of liver tumors in mice.

6. *Animal metabolism*. Four metabolism studies (rat and goat triazole- and phenyl-labeled) were conducted in animals with $^{14}C$ labeled tetraconazole. In the rat the initial metabolism proceeded through cleavage of the tetrafluoroethyl ether moiety, followed by a 2-step oxidation to tetraconazole-acid. In the goat the initial oxidation step formed tetraconazole-difluoroacetic acid, followed by ether cleavage to tetraconazole-alcohol, then further oxidation to tetraconazole-acid. In both the rat and the goat, the tetraconazole-acid functional group was enzymatically displaced, and the resulting thioether was oxidized to tetraconazole-acid-methyl-sulfoxide. An alternative pathway for tetraconazole-alcohol degradation was to form either glucuronide derivatives of tetraconazole-alcohol, or enzymatic triazole displacement to form dichlorophenyl-acetyl-cysteine. The nature of the residue in the goat is adequately understood for the purpose of regulating dietary exposure to residues. The liver retained the highest radioactivity, and muscle contained the lowest radioactivity. Tetraconazole was found to be the major residue in the liver and fat, and triazole was the major residue in milk, muscle and kidney.

7. *Endocrine disruption*. Based upon the findings from all of the full-lifetime and chronic toxicology studies, teratogenicity, mutagenicity and multi-generational reproductive studies conducted with tetraconazole, it is concluded that there were no indications of any potential to cause disruption or modification of endocrine function among any of the four animal species that have been studied (rat, mouse, rabbit and dog). Among the studies conducted with these four species there were no behavioral, reproductive or teratogenic effects, or histopathological changes in endocrine sensitive tissues such as the uterus, ovaries, mammary glands, or the testes.

*C. Aggregate Exposure*

1. *Dietary exposure*. Tolerances have been proposed to accompany uses proposed for tetraconazole products on bananas, sugar beets and peanuts. Tolerance-level residues may be utilized to conduct dietary exposure risk assessments, except that for bananas, the anticipated residue would be only 10% of the tolerance level because more than 90% of the residue on a whole-fruit basis remained on the peel.

*Drinking water*. A drinking water exposure assessment was performed for surface water with the screening model generic expected environmental concentration (GENEEC), using the input parameters represented by the environmental fate data obtained for tetraconazole in guideline-compliant studies. The model SCI-GROW was utilized to perform a ground water exposure assessment. The combined predicted levels of exposure in drinking water from surface and ground water, without any mitigation by means of filtration or other treatments typically applied to human drinking water, were 0.32 micrograms/kg/day for the highest-exposure age cohort nursing and non-nursing infants (> 1 year), or 5.3% of the chronic reference dose (RfD). The level of exposure to infants through drinking water, coupled with the maximum dietary exposure for non-nursing infants, thereby resulted in a maximum combined potential exposure of 0.90 micrograms/kg/day, or 15.1% of the RfD.

2. *Non-dietary exposure*. Tetraconazole products are not yet registered for any uses in the United States, however there is a pending registration for usage on turf grass which would permit applications to golf courses, commercial turf grass and sod farms. Tetraconazole products will be labeled so as to prohibit applications on residential turf grass. Tetraconazole products are not intended for registration or utilization in any setting which would contribute to human exposure in households or residential vicinities.

*D. Cumulative Effects*

Tetraconazole is a member of a class of compounds with structures containing 1,2,4-triazole substituents. Data are not yet available to determine whether tetraconazole has a common mechanism of toxicity in mammalian systems with other substances, or how to include this pesticide in a cumulative risk assessment.

*E. Safety Determination*

1. *U.S. population*. The lowest dietary NOAEL for tetraconazole in chronic or subchronic studies, expressed in terms of bwt dose on a daily basis, was confirmed in two studies to be 0.6 mg/kg/day. These two studies were the chronic/oncogenicity (full-lifetime) study in rat, and the 2-generation reproduction study in rat. Therefore the chronic RfD to be used for human exposure risk assessment should be 0.006 mg/kg/day by incorporation of both a 10-fold interspecies safety factor and a 10-fold intraspecies safety factor. A chronic dietary exposure analysis dietary risk evaluation system (DRES) was conducted for tetraconazole, conservatively assuming tolerance-level residues in/on bananas, sugar beets, and peanuts, including all secondary processed commodity tolerances associated with these crops plus milk, meat and meat byproducts. The maximum potential dietary exposure of tetraconazole to the U.S. population was calculated to be 0.223 micrograms/kg/day, or 4.5% of the chronic RfD.

For acute effects, the lowest NOAEL for tetraconazole was observed for maternal effects in the rat developmental study at 5 mg/kg/day, wherein decreased maternal bwt and food consumption were observed at the lowest observed adverse levels (LOAELs) of 22.5 mg/kg/day; therefore, the acute RfD for human exposure risk assessments is 0.05 mg/kg/day. An acute dietary exposure analysis was performed, focusing upon females aged 13 to 50 years, based upon the acute RfD. The dietary exposure model EXPedite predicted a maximum (99.9th percentile) potential dietary exposure level of 1.06 micrograms/kg/day for females of childbearing age, which represents 2.1% of the acute RfD.

2. *Infants and children*. There is a complete data base for tetraconazole which includes prenatal and postnatal developmental and reproduction toxicity data. In a 2-generation reproduction study with rats, all reproductive parameters investigated showed no treatment-related effects except slightly retarded growth rate and slightly increased liver weight at weaning in the offspring at the highest dose of 35.8 mg/kg/day. The NOAEL for reproductive effects in offspring was 4.8 mg/kg/day, which was 12 times higher than the NOAEL for toxicity effects in the dams. Thus the available evidence suggests that mammalian offspring would be less sensitive to potential toxicological effects from tetraconazole than would adults.

In the developmental toxicity (teratology) study conducted in the rat, tetraconazole did not cause any developmental effects in fetuses at 22.5 mg/kg/day even when maternal toxicity

was observed. In the rabbit a dose level of 30 mg/kg/day caused maternal toxicity, but there were no developmental effects.

The extensive data base that is available for tetraconazole contains no indication that tetraconazole would represent any unusual or disproportionate hazard to infants or children. Therefore there is no need to impose additional safety factors above the 10x interspecific uncertainty factor, coupled with the 10x intraspecific uncertainty factor, for conducting risk assessments pertaining to infants or children.

A chronic DRES was conducted for tetraconazole, conservatively assuming tolerance-level residues in/on bananas, sugar beets, and peanuts, including all secondary processed commodity tolerances associated with these crops plus milk, meat, and meat byproducts. The highest potential dietary exposures to non-nursing infants less than l–year old and children 1 to 6 years old were 0.552 micrograms/kg/day and 0.527 micrograms/kg/day, or 11% and 10.5% of the chronic RfD, respectively. These were the two age cohorts which represented the highest proportionate utilization of the chronic reference dose.

*F. International Tolerances*

There are no established Codex, Canadian, or Mexican tolerances (MRLs) established for tetraconazole. No MRLs for tetraconazole have been established under the EU uniform code for pesticide registrations. The following MRLs (expressed in ppm) have been established for tetraconazole residues on sugarbeet roots; Belgium, France, Portugal, Spain (0.05); Hungary (0.1); and Italy (0.2). In addition to sugar beets, the following MRLs (in ppm) for tetraconazole have also been established in the following countries for several RACs; apples, and/or pome fruits (Israel, Spain 0.2, France 0.3, Italy, Portugal, Poland 0.5); grapes (Israel, Jordan, France, Portugal, Spain 0.2, Italy 0.5); stone fruits (Italy, Spain 0.2); cucumbers (Italy, Poland, Egypt, Jordan 0.2); melons (Egypt, Jordan, Italy 0.05, Israel 0.2); peaches and/or stone fruits (Italy, Spain 0.2); wheat grain (Morocco, Belgium, France, Hungary, Poland, Italy, Portugal, United Kingdom 0.05); oat grain (United Kingdom 0.1); barley grain (Italy 0.1, United Kingdom 0.2); tomatoes (Egypt, Israel, Jordan 0.2); and mango (Israel 0.2).

[FR Doc. 99–26861 Filed 10–13–99; 8:45 am]

BILLING CODE 6560–50–F

## ENVIRONMENTAL PROTECTION AGENCY

**[FRL–6458]**

### Notice of Proposed Prospective Purchaser Agreement Pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as Amended by the Superfund Amendments and Reauthorization Act, Wellington Neighborhood Property, French Gulch/ Wellington-Oro Site

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice; request for public comment.

**SUMMARY:** Notification is hereby given of a Proposed Prospective Purchaser Agreement (PPA) associated with the Wellington Neighborhood Property near the French Gulch/Wellington-Oro Site, Summit County, Colorado. This Agreement is subject to final approval after the comment period. The Prospective Purchaser Agreement would resolve certain potential EPA claims under sections 106 and 107 of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 as amended by the Superfund Amendments and Reauthorization Act of 1986 (CERCLA), against Brynn Grey V LLC. and Wellington Neighborhood, LLC., the prospective purchasers (the purchasers).

The settlement would require the purchasers to cover and maintain areas of the property containing elevated levels of metals. The purchasers intend to develop the property for deed restricted affordable housing consistent with a master plan approved by local authorities. The purchasers will regrade areas disturbed by historical placer mining, will provide EPA with access to the property, will allow the use of a motion of the property for construction of response actions, if necessary, and will deposit funds for the purchase of the property into an EPA special account.

For seven (7) days following the date of publication of this document, the Agency will receive written comments relating to the proposed settlement. The Agency's response to any comments received will be available for public inspection at the Superfund Records Center at the U.S. Environmental Protection Agency, Region VIII, 999 18th Street, Denver, Colorado, 80202.

**DATES:** Comments must be submitted within seven (7) days from the date of this publication.

**AVAILABILITY:** The proposed settlement is available for public inspection at the U.S. Environmental Protection Agency, Region VIII, 999 18th Street, Denver, Colorado, 80202. A copy of the proposed Agreement may be obtained from the Superfund Records Center, U.S. Environmental Protection Agency, Region VIII, 999 18th Street, Suite 500, Denver, Colorado, 80202, 301/312–6473. Comments should reference the Wellington Neighborhood Property and should be forwarded to Andy Lensink, Enforcement Attorney, at the U.S. Environmental Protection Agency, Region VIII, 8ENF–T, 999 18th Street, Denver, Colorado, 80202.

**FOR FURTHER INFORMATION CONTACT:** Andy Lensink, U.S. Environmental Protection Agency, Region VIII, 8ENF–T, 999 18th Street, Denver, Colorado, 80202. (303) 312–6908.

It is so agreed:

**Max H. Dodson,**

*Assistant Regional Administrator, Office of Ecosystems Protection & Remediation, U.S. Environmental Protection Agency, Region VIII.*

[FR Doc. 99–26808 Filed 10–13–99; 8:45 am]

BILLING CODE 6560–50–M

## EQUAL EMPLOYMENT OPPORTUNITY COMMISSION

### Agency Information Collection Activities: Extension of Existing Collection; Comment Request

**AGENCY:** Equal Employment Opportunity Commission.

**ACTION:** Notice of Information Collection Under Review; Local Union Report (EEO–3).

**SUMMARY:** In accordance with section 3506(c)(2)(A) of the Paperwork Reduction Act of 1995, the Equal Employment Opportunity Commission (EEOC) announces that it intends to submit to the Office of Management and Budget (OMB) a request for an extension of the existing information collection listed below.

**DATES:** Written comments on this notice must be submitted on or before December 13, 1999.

**ADDRESSES:** Comments should be submitted to Frances M. Hart, Executive Officer, Executive Secretariat, Equal Employment Opportunity Commission, 10th Floor, 1801 L Street, NW, Washington, DC 20507. As a convenience to commentators, the Executive Secretariat will accept comments transmitted by facsimile (''FAX'') machine. The telephone number of the FAX receiver is (202) 663–4114. (This is not a toll-free number.) Only comments of six or fewer