Dated: October 7, 1999.

Harold Zenick,

Acting Deputy Assistant Administrator for Science, Office of Research and Development.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–891; FRL–6099–6]

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–891, 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–891 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)
Ann Sibold	Rm. 212, CM #2, 703–305–6502, e-mail: sibold.ann@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA	PP 6H5743
William Sproat	Rm. 6044, CM #2, 703–308–8587, e-mail: sproat.william@epamail.epa.gov.	Do.	PP 9F6043

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–891. 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–891 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 5.1/6.1 or ASCII file format. All comments in electronic form must be identified by docket control number PF–891. 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 7, 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. AgrEvo Environmental Health

PP 6H5743

EPA has received a pesticide petition (PP 6H5743) from AgrEvo Environmental Health, 95 Chestnut Ridge Road, Montvale, NJ 07645 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of esbiothrin and S-bioallethrin in or on food/feed items as a result of applications in food/feed handling establishments at 1.0 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residues of esbiothrin and S-bioallethrin in plants relevant to the establishment of a food/feed additive tolerance is adequately understood. Metabolism data have been generated on tomatoes, wheat and lettuce as well as samples of these stored commodities. All degradates found from the metabolism samples had structures consistent with photoproducts of allethrin. Only very minor amounts of cleavage products were found, indicating that metabolic or abiotic cleavage was not occurring to any great extent. In view of the known rapid photodegradation of allethrin and related compounds, it is most likely that these products arose from photolysis, rather than metabolism. No metabolites of toxicological concern were identified. Therefore, the only residue of concern is allethrin.

2. *Analytical method*. Analytical methods for determining residues of allethrin in a variety of food commodities have been developed and submitted to the Agency. These methods use gas chromatography (GC) with quantitation by an electron capture detector (ECD) for determination of total allethrin residues. These methods have been validated and are appropriate for the determination of allethrin residues in a variety of food commodities after application in food/feed handling establishments.

3. *Magnitude of residues*. The magnitude of the residue study demonstrated that residues of esbiothrin and S-bioallethrin are not expected to exceed the proposed tolerance level of 1.0 ppm as a result of the use of these compounds in food/feed handling establishments.

B. Toxicological Profile

1. *Acute toxicity*—i. *S-bioallethrin*. The acute rat oral LD_{50} of S-bioallethrin was 574 milligrams/kilograms (mg/kg) (males) and 413 mg/kg (females) when administered in PEG 200 and 607 mg/kg (males) and 497 mg/kg (females) when administered in corn oil. The acute rabbit dermal LD_{50} was greater than 2,000 mg/kg. The acute rat inhalation LC_{50} was 1.26 milligrams per liter (mg/L). S-bioallethrin was found to be slightly irritating to rabbit eyes, non-irritating to rabbit skin, and did not elicit a sensitizing response in guinea pigs.

ii. *Esbiothrin*. The acute oral LD_{50} of esbiothrin in rats was 432.3 mg/kg (males) and 378 mg/kg (females). The acute dermal LD_{50} in rabbits was greater than 2,000 mg/kg. The acute inhalation LC_{50} in rats was 2.59 mg/L. Esbiothrin

was found to be non-irritating to rabbit eyes, slightly irritating to rabbit skin, and did not elicit a sensitizing response in guinea pigs.

2. *Genotoxicity*. No indication of genotoxicity was noted in a battery of *in vivo* and *in vitro* studies conducted with either S-bioallethrin or esbiothrin.

3. *Reproductive and developmental toxicity*—i. *S-bioallethrin*. In a rat developmental toxicity study, animals were administered S-bioallethrin at 0, 5, 20, and 80 mg/kg/day during gestation days 6-15. Maternal mortality, tremors, piloerection and body weight (bwt) changes were observed. No evidence of developmental toxicity was observed. The maternal no observed adverse effect levels (NOAEL) was 20 mg/kg/day. The developmental NOAEL was 80 mg/kg/day.

In a rabbit developmental toxicity study, animals were administered S-bioallethrin at 0, 5, 50, or 200 mg/kg/day during gestation days 6-19. Tremors and reduced bwts and food consumption were reported. The maternal NOAEL was 50 mg/kg/day. Some evidence of slight developmental delay and an associated increased incidence of extra ribs and vertebrae were noted at the 200 mg/kg/day level. However these findings were only observed at the maternally toxic dose. The developmental NOAEL was 50 mg/kg/day.

ii. *Esbiothrin*. In a developmental toxicity study, rats were administered 0, 5, 25, and 125 mg/kg/day esbiothrin during gestation days 6-15. The maternal NOAEL was 25 mg/kg/day based on mortality and excess salivation, urine staining of the abdominal fur, tremors, body jerks and hypersensitivity to sound. There were no indications of developmental toxicity. The developmental NOAEL was 125 mg/kg/day.

In a rabbit developmental toxicity study, animals were administered esbiothrin at 0, 30, 100, and 300 mg/kg/day during gestation days 6-18. The maternal NOAEL was 100 mg/kg/day based on deaths, tremors, decreased motor activity, and ataxia. There were no indications of developmental toxicity. The developmental NOAEL was 300 mg/kg/day.

In a 2-generation reproduction study, esbiothrin was administered to rats at dietary concentrations of 0, 70, 200, 600, and 1,800 ppm. Decreased body weights (bwts) and mortality were observed in F1 parental animals. Slight decreases in pup viability and pup weights were observed only in the F1 generation and were confined to four litters in the high dose group. The reproductive NOAEL was 600 ppm or 50.4 mg/kg/day.

4. *Subchronic toxicity*—i. *S-bioallethrin*. A 28–day dermal toxicity study was conducted with S-bioallethrin applied to the backs of rats at 0, 10, 100, or 1,000 mg/kg/day for 6 hours/exposure 5 days/week for a total of 28 exposures. There were no treatment-related effects observed. The NOAEL was 1,000 mg/kg/day.

A 28–day rat inhalation study was conducted with S-bioallethrin at analytical concentrations of 0 (air only), 0.0051, 0.025, and 0.073 mg/L. Animals were exposed for 6 hours/day, 5 days/week for a total of 4 weeks. Intermittent limb tremors, walking on ''tip toes,'' hunched posture, aggressive behavior and vocalizing when handled were observed at 0.025 and 0.073 mg/L. The NOAEL was 0.0051 mg/L.

In a 90–day feeding study, rats were administered S-bioallethrin at dietary concentrations of 0, 250, 500, 2,000, and 8,000 ppm. Reduced bwt gain, food and water consumption, and increased absolute and relative liver and thyroid weights were observed at 2,000 ppm and higher. Various microscopic findings were reported for liver, kidneys and the thyroid. The NOAEL was 250 ppm or 18.5 mg/kg/day.

In a 90–day feeding study, beagle dogs were administered S-bioallethrin at dietary concentrations of 0, 400, 1,000, and 2,250 ppm. Decreased bwt gains, muscle tremors, wasted body condition, and intermittent incidences of decreased activity, hunched posture, diarrhea, and increased absolute and relative liver weights were observed. Histopathologic examination of the liver revealed centrilobular hepatocyte enlargement. The NOAEL was 1,000 ppm (38.54 mg/kg/day).

ii. *Esbiothrin*. In a 21–day dermal toxicity study, rabbits were exposed to 0, 40, 200 and 1,000 mg/kg esbiothrin for 6 hours/day for 5 days/week for 3 weeks. There were no treatment-related systemic effects. Dermal effects were noted at all dose levels. The NOAEL for systemic toxicity was 1,000 mg/kg/day highest dose tested (HDT).

5. *Chronic toxicity*. In a 2–year toxicity/oncogenicity study, rats were administered 0, 100, 500, 1,500, or 4,500 ppm esbiothrin in the diet. Decreased bwt gain, increased liver enzymes and cholesterol levels, increased liver weights, hepatocellular hypertrophy and hepatic cell degeneration and necrosis were observed. There was no evidence of oncogenicity. The NOAEL was 500 ppm (27 mg/kg/day).

A 2–year toxicity/oncogenicity study was conducted with esbiothrin in mice at dietary concentrations of 0, 50, 250, or 1,250 ppm esbiothrin. Increased absolute and relative liver weights were observed. There was no evidence of oncogenicity. The NOAEL was 1,250 ppm (214.3 mg/kg/day).

In a 1–year feeding study, beagle dogs were administered dietary concentrations of 0, 80, 400, and 2,000 ppm esbiothrin. There were no toxicologically significant effects observed. The NOAEL for this study was 2,000 ppm (69.9 mg/kg/day).

6. *Animal metabolism*. It appears that absorption of the allethrins is dependent upon the vehicle and route of administration. However, once absorbed, the allethrins are readily excreted. The dermal absorption determined from a rat dermal absorption study was approximately 25% when administered in an aromatic hydrocarbon vehicle.

7. *Endocrine disruption*. No special studies have been conducted to investigate the potential of esbiothrin or S-bioallethrin to induce estrogenic or other endocrine effects. However, the standard battery of required toxicity studies has been completed. The studies include an evaluation of the potential effects on reproduction and development and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure. These studies are generally considered to be sufficient to detect any endocrine effects, yet no such effects were detected. Thus, the potential for esbiothrin or S-bioallethrin to produce any significant endocrine effects is considered to be minimal.

C. Aggregate Exposure

Esbiothrin and S-bioallethrin are broad-spectrum insecticides used to control various pests in domestic indoor and outdoor areas (including use on pets), commercial and industrial food use areas and on ornamental plants. Thus, aggregate non-occupational exposure would include exposures resulting from non-food uses in addition to consumption of potential residues in food and water.

Both mixtures possess similar qualitative toxicologic profiles, but the overall weight of evidence indicates that the d-trans of d isomer is the most toxicologically significant isomer in these mixtures. Consequently, after converting into S-bioallethrin equivalents from esbiothrin data, or vice versa, based on the relative proportions of d-trans of d, the toxicity data for these mixtures can be used interchangeably.

1. *Dietary exposure—Food*. Since there are no agricultural uses with these active ingredients, an acute dietary exposure was not evaluated. According to EPA guidelines, food handling establishment uses should only be

evaluated for chronic dietary exposure. Potential chronic dietary exposures from food commodities under the proposed food and feed additive tolerance for esbiothrin and S-bioallethrin were estimated using the Exposure 1 software system (TAS, Inc.) and the 1977-78 USDA consumption data. Dietary risk assessment was conducted in a tiered approach whereby three scenarios were evaluated. The first scenario assumed 100% of all food and feed handling establishments (FHE) are treated with S-bioallethrin or esbiothrin and that all residues from these treatments are at the proposed tolerance level (1 ppm). The second scenario assumes that 100% of the FHE are treated and all residues are at the proposed tolerance level except where actual residue data are available. The third scenario assumes that, more realistically, only 25% of the FHE are treated and all residues are at the proposed tolerance level except for where actual residue data exist.

2. *Drinking water*. Exposure via drinking water is expected to be negligible since esbiothrin and S-bioallethrin are neither persistent in the environment nor likely to leach. As is characteristic of pyrethroids, the allethrins bind strongly to soil and will not be leached out by water. Further, this pyrethroid is rapidly degraded under environmental conditions (in soil, water and in the presence of sunlight). The half-life of esbiothrin and S-bioallethrin is approximately 7-15 minutes in sunlight and no more than 2 hours in total darkness. Due to these properties, no residues in drinking water are expected to be present.

3. *Non-dietary exposure*. As noted above, esbiothrin and S-bioallethrin are broad-spectrum insecticides developed for use in non-agricultural applications including indoor foggers, insect mats and coils, household commercial and institutional insect killers; food and feed handling applications, commercial non-food/feed sites, pet applications and greenhouse/ornamental applications. To evaluate non-dietary exposure, the ''flea infestation control,'' scenario was chosen to represent a plausible but worst-case non-dietary (indoor and outdoor) non-occupational exposure. This scenario provides a situation where S-bioallethrin and/or esbiothrin is commonly used and one in which both can be used concurrently for a multitude of uses, e.g. spot treatment of infested indoor surfaces such as carpets and rugs, treatment of pets and treatment of animal housing. This hypothetical situation provides a very conservative, upper bound estimate of potential non-dietary exposures. Consequently, if health risks are acceptable under these conditions, the potential risks associated with other more likely scenarios would also be acceptable.

Aggregate short-term risk was calculated by combining the risk calculated for the ''flea infestation'' scenario (non-dietary risk) with the chronic dietary risk analyses. As indicated previously, S-bioallethrin and esbiothrin possess similar qualitative toxicity profiles. Due to their isomeric mixtures, the product toxicity data for either product can be converted to the other after the appropriate conversions have been made based on relative proportions of the d-trans of d isomer content. For risk assessment purposes, S-bioallethrin will be used to assess the risk of S-bioallethrin and esbiothrin since it contains a greater proportion of the more toxicologically significant isomer, d-trans of d. As a result of using the data in this manner, a conservative, worst-case evaluation can be made.

D. Cumulative Effects

At the present time, there are insufficient data available to allow AgrEvo to properly evaluate the potential for cumulative effects from the various pyrethroids now being used, or from any other chemicals that may have similar mechanisms of toxicity. Furthermore, because of the need to utilize data from multiple registrants, such an analysis cannot be conducted by a single registrant. AgrEvo is currently participating in a joint industry effort to evaluate the potential aggregate risks from exposure to all pyrethroids but the results from this evaluation are not yet available.

As an interim measure, AgrEvo has evaluated the potential cumulative risks associated with exposure to three products in the allethrin series: bioallethrin, esbiothrin, and S-bioallethrin. These products contain varying proportions of d-trans chrysanthemate ester of d- and l-allethrolone (d-trans d and d-trans l). The uses for these products are very similar except that no food uses are being proposed for bioallethrin. The use rates for the three products differ based on relative efficacy which appears to be related to the percentage of the most active isomer (d-trans d). The risk assessments conducted in support of this petition were based on the worst-case assumption that all residues were from S-bioallethrin, the product with the highest percentage of the most active isomer. Therefore, the potential cumulative risks associated with a combination of all three of these products would actually be lower than those presented here.

E. Safety Determination

1. *U.S. population*. The combined toxicity and residue data base for esbiothrin and S-bioallethrin is considered to be valid, reliable and essentially complete. No evidence of oncogenicity has been observed. In accordance with EPA's ''Toxicology Endpoint Selection Process'' Guidance Document, the toxicology endpoint from the S-bioallethrin acute neurotoxicity study, 30 mg/kg, was used to evaluate acute non-dietary risk. According to current EPA policy, residues from Food Handling Establishment uses are only evaluated for potential chronic dietary risk. AgrEvo is proposing a RfD of 0.226 mg/kg bwt/day to evaluate chronic dietary risk for S-bioallethrin and esbiothrin. This RfD is based on the NOAEL from the esbiothrin rat chronic toxicity/oncogenicity study with a 100-fold safety factor to account for interspecies extrapolation and intraspecies variation. The S-bioallethrin NOAEL served as a worst-case scenario because it contains the largest amount of d-trans of d isomer by weight.

The potential chronic dietary exposure for the overall U.S. population under the three scenarios as described in section D utilize the following portions of the RfD: 10.73% for scenario 1 (100% FHE treated and all residues at the proposed tolerance level); 5.28% for the second scenario (100% FHE treated and all residues at proposed tolerance level except where actual data exist) and 1.32% of the third scenario (treatment of only 25% of FHE and residues at proposed tolerance except where actual data exist). There is generally no concern for chronic exposures below 100% of the RfD since it represents the level at or below which no appreciable risks to human health is posed.

Using an upper bound estimate of potential non-dietary exposure from a worst-case scenario (flea treatment) results in a margin of exposure (MOE) of approximately 610,000 for adults with S-bioallethrin and approximately 510,000 for esbiothrin.

Utilizing the scenario of chronic dietary exposure with an upper bound estimate of potential non-dietary exposure from a worst-case scenario (flea treatment), the resulting MOE for aggregate exposure to S-bioallethrin is 9,800 for the adult population and 8,100 for esbiothrin for the same population group.

There is generally no concern for MOEs greater than 100 or utilization of less than 100% RfD. Therefore, there is

reasonable certainty that no harm will result to the U.S. population in general from aggregate exposure to S-bioallethrin or esbiothrin.

2. *Infants and children*. Data from developmental toxicity studies in rats and rabbits and multi-generation reproduction studies in rats are generally used to assess the potential for increased sensitivity of infants and children. 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 reproductive and other effects on adults and offspring from prenatal and postnatal exposure to the pesticide. None of the studies conducted with S-bioallethrin or esbiothrin indicated evidence of developmental or reproductive effects resulting from exposure to either material at non-maternally toxic doses.

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 relative to prenatal and postnatal effects in children is complete. No indication of increased susceptibility to younger animals was noted in the developmental or reproduction studies at non-maternally toxic doses or in the majority of studies with other pyrethroids. Therefore, use of the S-bioallethrin acute neurotoxicity NOAEL of 30 mg/kg for short-term risk, and the proposed RfD of 0.226 mg/kg/day for assessing chronic aggregate risk to infants and children is appropriate and an additional uncertainty factor is not warranted.

Using the dietary exposure assumptions described above in section D, the first scenario utilizes 41.98% of RfD for non-nursing infants (< 1–year) and 26.14% of RfD for children 1-6 years. The second scenario utilizes 11.96% of the RfD for non-nursing infants < 1–year and 11.54% of RfD for children 1-6 years. The third scenario utilizes 2.96% of RfD for non-nursing infants < 1–year and 2.88% of the RfD for children 1-6 years. There is generally no concern for chronic exposures below 100% of the RfD since it represents the level at or below which no appreciable risks to human health is posed.

Using an upper bound estimate of potential non-dietary exposures for a worst case scenario (flea infestation) results in a MOE of 2,300 for infants less than 1–year old for S-bioallethrin and 1,900 for esbiothrin. A MOE of 2,400 for children 1-6 years was noted for S-bioallethrin and a MOE of 2,000 for esbiothrin.

Utilizing the scenario of chronic dietary exposure with an upper bound estimate of potential non-dietary exposure from a worst case scenario (flea infestation), it can be seen that for aggregate exposure to S-bioallethrin and esbiothrin, the MOE for infants less than 1–year is 1,500 for S-bioallethrin and 1,200 for esbiothrin. For children 1–6 years, the MOE's are 1,600 for S-bioallethrin and 1,300 for esbiothrin.

As noted for the U.S. population, these compounds have a very short half-life in light and in darkness. These products are metabolized rapidly from the body and based on general practices, are applied not more than once per month. Based on these properties and use patterns, real-life exposures would be acute in nature and at much lower levels than used in this assessment.

There is generally no concern for MOE's greater than 100, or less than 100% utilization of RfD. Therefore, there is reasonable certainty that no harm will result to the most sensitive population subgroup, described as non-nursing infants less than 1–year and children 1–6 years, from aggregate exposure to esbiothrin and S-bioallethrin.

F. International Tolerances

Esbiothrin and S-bioallethrin are broad spectrum insecticides used throughout the world to control pests of ornamental plants, household, commercial and industrial areas (indoor and outdoor). There are currently no maximum residue limits (MRLs) for esbiothrin or S-bioallethrin.

2. ZENECA Ag Products

PP 9F6043

EPA has received a pesticide petition [9F6043] from ZENECA Ag Products, 1800 Concord Pike, Wilmington, DE 19850 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for combined residues of pirimicarb 2-(dimethylamino)-5,6-dimethyl-4-pyrimidinyl dimethylcarbamate (9Cl) and its two carbamate metabolites: desmethyl pirimicarb and desmethylformamido pirimicarb, expressed as desmethyl pirimicarb in or on the raw agricultural commodities (RAC): potatoes and pre-blossom apples at 0.01 ppm, head lettuce at 0.3 ppm, leaf lettuce at 2.0 ppm, and endive (curly and escarole) at 2.0 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Studies of the nature of residues in three diverse crops, potatoes, apples, and lettuce, have demonstrated that pirimicarb undergoes very extensive metabolism, with the residues of concern in primary crops being both pirimicarb and its carbamate metabolites. Zeneca proposes that combined residues of pirimicarb, 2-(dimethylamino)-5,6-dimethyl-4-pyrimidinyl dimethylcarbamate (9Cl), and its two carbamate metabolites (desmethyl pirimicarb and desmethylformamido pirimicarb) expressed as desmethyl pirimicarb are to be included in the tolerance.

2. *Analytical method*. The analytical enforcement method uses Gas Chromatography (GC) equipped with a thermionic nitrogen specific detector. Crop samples are macerated with methanol and then filtered. After filtration, the methanol is evaporated and the samples resuspended and partitioned with hexane and hydrochloric acid. The samples are left overnight to allow conversion of the desmethylforamido pirimicarb metabolite to the desmethyl pirimicarb metabolite. The hexane layer is discarded and the acidic aqueous layer is further partitioned with ethyl acetate. Sodium hydroxide is added to the aqueous layer and pirimicarb and its carbamate metabolites are extracted with dichloromethane. This method has been validated by an independent laboratory, with a LOD of 0.01 ppm.

3. *Magnitude of residues*. Residue trials were conducted on potatoes, pre-blossom apples, and lettuce in the major crop growing areas of the United States. Sixteen residue trials were done on potatoes at the maximum label rate. At time of harvest, there were no detectable residues of either pirimicarb or its carbamate metabolites at the LOD of 0.01 ppm. A processing study on potatoes at 5x the maximum label rate also demonstrated that there are no detectable residues of pirimicarb or its carbamate metabolites at the LOD of 0.01 ppm on potatoes, potato peel, or any of the processed fractions (flakes and chips).

Sixteen residue trials were conducted on apples at the pre-blossom stage, using one application at the maximum label rate. At time of harvest, there were

no detectable residues of pirimicarb or its carbamate metabolites at the LOD of 0.01 ppm. An apple processing study at 5x the maximum label rate also demonstrated that there were no detectable residues of pirimicarb or its carbamate metabolites at the LOD of 0.01 ppm on apples, or any of the processed fractions (pomace, juice).

Six residue trials were completed on head lettuce at the maximum label rate. Mature lettuce leaves were analyzed for pirimicarb and its carbamate metabolites. Maximum residues of 0.24 ppm were detected for the combined residues of pirimicarb and its carbamate metabolites.

Six residue trials were completed on leaf lettuce at the maximum label rate. Mature lettuce leaves were analyzed for pirimicarb and its carbamate metabolites. Maximum residues of 1.73 ppm were detected for the combined residues of pirimicarb and its carbamate metabolites. ZENECA requests that the Agency also use these leaf lettuce residue trials as surrogate data for the commodity endive (curly and escarole).

B. Toxicological Profile

1. *Acute toxicity*. In common with other carbamate insecticides, pirimicarb induces toxic signs characteristic of cholinesterase inhibition. These effects are rapidly reversed on the cessation of treatment and recovery is usually full and complete.

Formulated pirimicarb (PIRIMOR DF) is classed as Category II toxicity based on the highest hazard for either the technical or formulated product.

PIRIMICARB TOXICITY SUMMARY

Toxicity test	Results	Toxicity category
Acute oral rat	LD_{50} 152 mg/kg (m),142 mg/kg (f)	II
Acute dermal rat ..	LD_{50} >1,000 mg/kg (f)	III
Acute inhalation rat.	0.95 mg/L (m); 0.86 mg/L (f)	III
Eye irritation rabbit	Non-irritant	IV
Skin irritation rabbit.	Slight irritant	IV
Skin sensitization	Moderate	May cause allergic reaction

FORMULATED MATERIAL (PIRIMOR DF) TOXICITY SUMMARY

Toxicity test	Results	Toxicity category
Acute oral rat	LD_{50} 87 mg/kg	II
Acute dermal rat ..	LD_{50} > 2,000 mg/kg	III
Acute inhalation rat.	1.7 mg/L (f)	III
Eye irritation rabbit	Moderate irritant	II
Skin irritation rabbit.	Slight irritant	IV
Skin sensitization	Not a sensitizer	- -

2. *Genotoxicity*. Pirimicarb has been evaluated for genotoxicity and mutagenicity. Pirimicarb does not induce gene mutation in either prokaryotic or non-mammalian eukaryotic cells.

3. *Reproductive and developmental toxicity*. Pirimicarb was not teratogenic to rats when tested in a study using oral gavage dose levels of 0, 10, 25, and 75 mg/kg/day. Fetotoxicity in the presence of maternal toxicity was observed at 75 mg/kg/day, but there were no effects on mother or fetus at a dose level of 25 mg/kg/day. The overall NOAELs for fetotoxicity was therefore, 25 mg/kg/day in the rat.

Pirimicarb was not teratogenic in the rabbit when tested in a study using oral gavage dose levels of 0, 2, 10, or 60 mg/kg/day. Maternal toxicity was observed at 60 mg/kg/day, but there were no effects on the fetus at any dose level. There was no evidence of fetotoxicity or teratogenicity in the rabbit at doses up to and including a maternally toxic dose of 60 mg/kg/day.

Neither study showed effects on the fetus in the absence of effects on the mother, and thus there was no evidence of enhanced fetal susceptibility to pirimicarb.

Pirimicarb showed no evidence of reproductive toxicity to rats in a 2–generation reproductive toxicity study using dose levels of 0, 50, 200, or 750 ppm. There were no effects on reproductive parameters at 750 ppm (88 mg/kg/day), the highest dose tested (HDT).

4. *Subchronic toxicity*—i. *Ninety–day rat feeding*. In an number of repeat dose studies, male and female rats were fed diets containing 0, 175, 250, or 750 ppm of pirimicarb for a period of 56-90 days. There were no adverse clinical, hematological, or pathological effects. The only effect was a reduction in body weight gain, which was clearly evident at 750 ppm in 2 studies, and one study showed slight effects at 250 ppm. The NOAEL for subchronic toxicity in the rat was concluded to be 175 ppm (17.5 mg/kg/day).

a. *Ninety–day dog feeding*. Groups of four male and four female beagle dogs were dosed with pirimicarb by capsule at 0, 0.4 or 1.8 mg/kg/day as an oral dose for a period of at least 90 days; a further group received pirimicarb at 4 mg/kg/day for 180 days. There were no adverse clinical or pathological effects, but the animals receiving 4 mg/kg/day showed evidence of increased erythropoetic activity on the bone marrow. The NOAEL in this study was 1.8 mg/kg/day.

b. *Twenty-one–day dermal study*. Pirimicarb was assessed for its sub-acute dermal toxicity. Groups of five male and five female rats were given 15, 6-hour dermal applications of 40, 200, or 1,000 mg/kg pirimicarb as a paste in deionized water over a period of 21 days. There was no signs of skin irritation and no indications of systemic toxicity. A small reduction in brain cholinesterase was found at 1,000 mg/kg. The NOAEL was 200 mg/kg.

5. *Neurotoxicity*-i. *Acute neurotoxicity*. In an acute neurotoxicity study, pirimicarb was administered as a single dose at levels of 0, 10, 40, or 110 mg/kg body weight. The animals were observed up to 14 days. A neurotoxicity screening battery of tests including a functional observational battery and quantitative measurement of motor activity was evaluated 1–week prior to the study, and on days 1, 8, and 15. Administration of 110 mg/kg resulted in early mortalities and adverse clinical signs. Brain neurotoxic esterase activity was not affected by treatment. Changes at the 40 mg/kg dose were transient and not accompanied by biologically significant reductions in brain or erythrocyte cholinesterase activity. It is concluded that pirimicarb shows reversible clinical signs of neurotoxicity following administration of a single oral dose of 110 mg/kg. The NOAEL for clinical signs of transient acute neurotoxicity is 40 mg/kg/day. The NOAEL for this study is 10 mg/kg/day.

ii. *Subchronic neurotoxicity*. A subchronic rat neurotoxicity study was performed. Pirimicarb was fed to rats at levels of 0, 75, 250, and 1,000 ppm for 90 days. A neurotoxicity screening battery of tests, including functional observational battery and quantitative assessment of motor activity was evaluated in week -1, 5, 9, and 14. Histopathological assessment and neurotoxic esterase activity in the brain was performed after 90 days. Reduced growth and food consumption/utilization were observed at 250 and

1,000 ppm. There were no treatment-related effects on the functional observational battery, motor activity, cholinesterase and neurotoxic esterase activities and neuropathology. The NOAEL for subchronic neurotoxicity was 1,000 ppm (approximately 81 mg/kg/day).

6. *Chronic toxicity*. In two chronic dog studies, dogs were dosed at levels up to 25 mg/kg/day for either 1 or 2 years. Pirimicarb produced hemolytic anemia or related hematological changes in a very small proportion of dogs. This effect was shown to require prolonged administration of pirimicarb and was reversible on cessation of exposure to pirimicarb. It was not observed in toxicity studies in the rat and mouse. A clear NOAEL of 3.5 mg/kg/day was established based on hematological changes in all of the available studies.

In a 2–year rat combined chronic toxicity and oncogenicity study, pirimicarb was fed for up to 2 years at 0, 75, 250, and 750 ppm. The maximum tolerated dose was 750 ppm, with no carcinogenic response over 2 years. A NOAEL was established at 3.7 mg/kg/day.

In an 80–week mouse carcinogenicity study, the mice were given pirimicarb at 0, 6.7, 26.6, and 93.5 mg/kg/day (0, 50 ppm, 200 ppm, and 700 ppm). It was concluded that there was an increase of incidence of benign lung tumors in female at the top dose of 700 ppm, only. These tumors are benign and demonstrate a clear threshold for induction, leading to the conclusion that pirimicarb is not carcinogenic in the mouse. This conclusion is further supported by evidence that pirimicarb is non-genotoxic. A NOAEL of 26.6 mg/kg/day was established.

7. *Animal metabolism*. Radiolabeled studies in the rat and dog have demonstrated that following oral administration, pirimicarb is well absorbed, extensively metabolized, and the metabolites are rapidly eliminated. Metabolism following a single oral dose is quantitatively similar in rats and dogs and there is no evidence of bioaccumulation.

8. *Metabolite toxicology*. Pirimicarb and the carbamate metabolites are associated with acute effects in cholinesterase inhibition.

9. *Endocrine disruption*. Pirimicarb shows no evidence of hormonal effects, therefore there is no evidence of endocrine disruption. There are no toxicity endpoints involving reproductive organs in either male or female animals in any of these studies.

C. Aggregate Exposure

1. *Dietary exposure*. Pirimicarb is registered for non-food use on seed alfalfa. The current request is to register pirimicarb on endive (curly and escarole). An acute RfD of 0.1 mg/kg/day is proposed, based on clinical signs of systemic toxicity seen at 40 mg/kg/day in the rat acute neurotoxicity study and application of a standard 100-fold uncertainty factor to the NOAEL of 10 mg/kg. There is no indication of sensitivity to children and infants, and therefore, no requirement for additional FQPA safety factor. The chronic RfD is 0.035 mg/kg/day, based on hematological effects noted in the chronic dog studies at 4 mg/kg/day and application of a standard 100-fold uncertainty factor to the NOAEL of 3.5 mg/kg/day.

i. *Food*—a. *Acute risk*. An acute dietary (food) risk assessment (Dietary Exposure Evaluation Model, Novigen Sciences Inc., 1997; USDA Continuing Survey of Food Intake by Individuals (CSFII) 1994-96) was conducted using tolerance level residues for raw agricultural commodities (RACs) and average field residues with percent crop treated for blended commodities (apple juice and dried potatoes). Resulting exposure values and percent of the acute RfD utilized are shown below:

ACUTE DIETARY (FOOD ONLY) EXPOSURE AND RISK FOR PIRIMICARB

Population subgroup	Exposure @ 99.9th Percentile (mg/kg/day)	Percent Acute RfD
U.S. population (48 States).	0.005044	5.04%
Non-nursing infants (<1 year).	0.000252	0.25%
Children (1-6 years).	0.003217	3.22%
Females (13-50) ...	0.005924	5.92%

For pirimicarb, an acceptable acute dietary exposure (food plus water) of 100% or less of the acute RfD for all population subgroups is needed to protect the safety of all population subgroups. The estimated exposure for all population subgroups at the 99.9th percentile utilized less than 100% of the acute RfD, and does not exceed EPA's level of concern.

b. *Chronic risk*. Chronic dietary risk assessments (Dietary Exposure Evaluation Model, Novigen Sciences Inc., 1997; USDA Continuing Survey of Food Intake by Individuals (CSFII) 1994-96) were conducted for pirimicarb using two approaches: (1) using tolerance level residues and assuming 100% crop treated, and (2) using anticipated residue concentration levels adjusted for percent crop treated and limit of detection residues. The Theoretical Maximum Residue Contribution (TMRC) and Anticipated Residue Contribution (ARC) from these two scenarios represents 0.3% and 0.1%, respectively, of the RfD for the U.S. population as a whole. The subgroup with the greatest chronic exposure is children ages one to six for which the TMRC and ARC estimates represented 0.4% and 0.1%, respectively of the RfD. The chronic dietary risks from these uses do not exceed EPA's level of concern.

ii. *Drinking water*. Other potential sources of exposure of the general population are residues in drinking water. Laboratory data on pirimicarb indicate that its potential soil mobility ranges between low and very high, depending on a number of factors including pH. However field dissipation data on both the parent and its metabolites indicate that under agricultural conditions, degradation is so rapid (half-lives < 21 days) that significant leaching does not occur. In a 1995-96 field dissipation study conducted using ^{14}C labeled material, the half-life of pirimicarb was found to average 3.1 days, and no radioactive residue (pirimicarb and/or metabolites) of greater than 0.01 ppm was found below 6 inches in depth. This study conducted in 1995-96 confirms previous laboratory and field dissipation studies.

Pirimicarb is rapidly dissipated under field conditions by both photolysis and microbial metabolism leading to significantly less persistence than demonstrated under conditions of laboratory soil degradation studies. This rapid dissipation under field conditions is independent of soil pH. Pirimicarb, therefore, does not leach and is unlikely to enter surface water under the conditions of the recommended label use patterns.

Drinking water levels of comparison (DWLOC) were calculated for pirimicarb for adults and children for both acute and chronic exposures, in accordance with EPA's Standard Operating Procedure (SOP) for Drinking Water Exposure and Risk Assessments (November 20, 1997). Drinking water exposure from surface and ground water for pirimicarb was estimated using Tier II model EPA's pesticide root zone model (PRZM)/EXAMS and Tier I model SCI-GROW, respectively. The exposure estimates and DWLOCs are summarized below:

DRINKING WATER LEVELS OF COMPARISON AND ACUTE EXPOSURE ESTIMATES FOR PIRIMICARB

Population subgroup	SCI-GROW (ug/L)[1]	PRZM/EXAMS (ug/L)[2]	Acute DWLOC (ug/L)
Adult - U.S. population.	0.25	4.66	3323
Children	0.25	4.66	968

[1] SCI-GROW estimate based on highest water estimate from all crop uses.

[2] PRZM/EXAMS based on instantaneous concentration for total carbamate residues (parent + metabolites)

DRINKING WATER LEVELS OF COMPARISON AND CHRONIC EXPOSURE ESTIMATES FOR PIRIMICARB

Population subgroup	SCI-GROW (ug/L)[1]	PRZM/EXAMS (ug/L)[2]	Chronic DWLOC (ug/L)
Adult - U.S. population	0.25	0.88	1224
Children	0.25	0.88	350

[1] SCI-GROW estimate based on highest water estimate from all crop uses.
[2] PRZM/EXAMS based on annualized average value for total carbamate residues (parent + metabolites).

Based on the estimated dietary and water exposures for pirimicarb, Zeneca has concluded that there is a reasonable certainty of no harm to infants, children and adults resulting from potential acute or chronic aggregate exposure to pirimicarb.

2. *Non-dietary exposure*. Pirimicarb is not registered for either indoor or outdoor residential uses. There are no non-occupational exposures to pirimicarb. Non-food uses for alfalfa grown for seed and small seeded vegetable seeds are occupational exposures. These exposures are represented in inhalation, oral and dermal estimates contained in the acute toxicology summaries, as well as the dermal penetration studies.

D. Cumulative Effects

Pirimicarb, as a carbamate insecticide, exerts its insecticidal effect through inhibition of acetyl-cholinesterase. At this time, methodologies and mechanistic data are not available to resolve this complex issue of cumulative effects concerning common mechanisms of toxicity. At this time, there are no available data to determine whether pirimicarb has a common mechanism of toxicity with other substances, or how to include this pesticide in a cumulative risk assessment.

E. Safety Determination

1. *U.S. population*. Based on the available toxicity data, a chronic RfD is set for pirimicarb at 0.035 mg/kg/day. This RfD is based on chronic dog studies with a NOAEL of 3.5 mg/kg/day and an uncertainty factor of 100. The acute RfD is 0.01 mg/kg/day, based on clinical signs of toxicity at 40 mg/kg/day in the rat acute neurotoxicity study. No additional uncertainty factors are necessary.

2. *Infants and children*. Developmental toxicity and reproductive toxicity studies have not shown fetal effects other than mild fetotoxicity in the rat (reduced fetus/litter weight and indications of delayed development) at doses which were also toxic to the mother. There was no evidence in these studies of any extra susceptibility of the fetus. Neither has there been any indication of any particular susceptibility of juvenile animals. Based on the data base, there is no reason to consider human infants and children to be inherently more at risk of toxicity from pirimicarb than adults.

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 relative to prenatal and postnatal effects for children is complete. No additional FQPA safety factor is required for pirimicarb.

F. International Tolerances

The CODEX maximum residue levels for pirimicarb and its carbamate metabolites (desmethyl and desmethyl formamido pirimicarb) are: potatoes 0.05 ppm, lettuce 1.0 ppm, and apples (pome fruit) 1.0 ppm.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–181070; FRL–6389–6]

1, 3 Dichloropropene; Receipt of Application for Emergency Exemption, Solicitation of Public Comment

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: EPA has received a specific exemption request from the State of California Department of Pesticide Regulation to use the pesticide 1,3 dichloropropene (CAS No. 542–75–6) to treat up to 50,000 acres of wine grapes to control Grape phylloxera and nematodes. The Applicant proposes the use of a chemical which is or has been the subject of a Special Review by the EPA. EPA is soliciting public comment before making the decision whether or not to grant the exemption.

DATES: Comments, identified by docket control number OPP–181070, must be received on or before November 1, 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. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, it is imperative that you identify docket control number OPP–181070 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: Barbara Madden, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–6463; fax