FIFRA after June 28, 2004, except for the purposes of shipping such stocks for export consistent with section 17 of FIFRA or for proper disposal.

As of May 31, 2007, all sale and distribution by Bayer CropScience, the sole registrant of fenamiphos products (manufacturing-use and end-use products), shall be prohibited. Persons other than Bayer CropScience may sell and distribute such products until May 31, 2008.

*B. Retail and Other Distribution or Sale of Existing Stock of Products*

Persons other than Agriliance, LLC; Dow AgroSciences, LLC; and RiceCo, LLC may continue to sell or distribute the existing stocks of any propanil product listed in Table 2 that bears instructions for use on small grains until those stocks are depleted.

*C. Use of Existing Stocks*

EPA intends to permit the use of existing stocks of products listed in Table 1 or 2 until such stocks are exhausted, provided such use is in accordance with the existing labeling of that product.

**List of Subjects**

Environmental protection, Fenamiphos, Pesticides and pests, Propanil.

Dated: November 18, 2003.

**Betty Shackleford,**

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

[FR Doc. 03–30159 Filed 12–9–03; 8:45 a.m.]

**BILLING CODE 6560–50–S**

## ENVIRONMENTAL PROTECTION AGENCY

**[OPP–2003–0385; FRL–7337–6]**

## Spiroxamine; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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

**ACTION:** Notice.

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

**DATES:** Comments, identified by docket ID number OPP–2003–0385, must be received on or before January 8, 2004.

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

**FOR FURTHER INFORMATION CONTACT:** Mary L. Waller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–9354; e-mail address:*waller.mary@epa.gov*.

**SUPPLEMENTARY INFORMATION:**

### I. General Information

*A. Does this Action Apply to Me?*

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

- Crop production (NAICS 1111)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

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

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

1. *Docket.* EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0385. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

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

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

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

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

copyrighted material, will be available in the public docket.

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

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

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

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

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket/*, and follow the online instructions for submitting comments. Once in the system, select''search,'' and then key in docket ID number OPP–2003–0385. The system is an''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to opp-docket@epa.gov, Attention: Docket ID Number OPP–2003–0385. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket.

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

2. *By mail*. Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID Number OPP–2003–0385.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, Attention: Docket ID Number OPP–2003–0385. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

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

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

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

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

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

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

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

5. Provide specific examples to illustrate your concerns.

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

7. To ensure proper receipt by EPA, be sure to identify the docket ID number 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 a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in FFDCA section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

**List of Subjects**

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

Dated: November 26, 2003.

**Peter Caulkins,**

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

**Summary of Petition**

The petitioner summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the view of the petitioner. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

**Bayer CropScience**

*PP 3E6783*

EPA has received a pesticide petition (PP 3E6783) from Bayer CropScience, 2 T.W. Alexander Drive, P.O. Box 12014, Research Triangle Park, NC 27709roposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of Spiroxamine, 8-(1,1-Dimethylethyl)-*N*-ethyl-*N*-propyl-1,4-dioxaspiro[4,5]decane-2-methanamine in or on the raw agricultural commodity hop, dried cone - import at 50.0 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*. A hop plant metabolism study has been conducted, and the nature of the residue is adequately understood. An animal metabolism study is not required since the proposed crop to be treated with Spiroxamine is not fed to livestock.

2. *Analytical method*. A method to determine the total residues of Spiroxamine using gas chromatography has been submitted to the EPA. In addition, Spiroxamine has been evaluated using the multi-residue methodologies as published in the FDA Pesticide Analytical Manual, Volume I.

3. *Magnitude of residues*. Eight field trials were conducted on fields in typical hop-growing regions in Germany to assess the residue levels of Spiroxamine, 8-(1,1-dimethylethyl)-*N*-ethyl-*N*-propyl-1,4-dioxaspiro[4,5]decane-2-methanamine, in/on hops following foliar applications. A formulation containing Spiroxamine (KWG 4168 500 EC) was applied in two spray applications as an emulsifiable concentrate formulation containing 500 grams (g) active ingredient/liter(L), with an application rate of 1.5 liters/hectare (L/ha) and 3,000 L of water/ha. This corresponded to a total applied amount of the active ingredient of 0.750 kilograms (kg)/ha. The applications were carried out at growth stages 75–79 and 77–81, respectively (corresponding to a 9–10 day interval between applications).

Duplicate composite samples of hop cones were collected at a 10–day preharvest interval (PHI) from each plot. In addition, single samples were collected on day 0 and, in four of the trials, on days 6 and 13 after treatment. Hop cones were analyzed both fresh and, on days 10 and 13, after kiln drying. The highest total residue value of Spiroxamine (defined as parent and metabolites converted to aminodiol equivalents) in dried hop cones was 30 ppm (highest individual value)/24.5 ppm (highest average value [HAFT] from two samples in a single trial) at a 10–day PHI. The total Spiroxamine residues in hop cones appeared to decline with time. Note: The residue trials submitted with this petition are being submitted as a national submission in Germany in the European Union (EU). The European procedure for calculating a maximum residue limit (MRL) differs from the procedure used in the USA. Although no final decision has yet been made by the European authorities at the present time, an evaluation of the dried hop cone data according to EU principles leads to an MRL proposal equivalent to 50 ppm total residues of Spiroxamine. In order to avoid possible trade conflicts, it is proposed that the U.S. tolerances be harmonized with the expected EU-MRL.

Samples from two of the above field trials were also processed into beer (four individual processing trials). Average total residues for the dried hop cones prior to processing in these trials were 9.5 and 16 ppm, respectively. In beer, the total residues of Spiroxamine were below the level of detection (i.e., <0.05 ppm) in all four trials.

*B. Toxicological Profile*

1. *Acute toxicity—KWG 4168 (Spiroxamine) Technical*. The acute oral $LD_{50}$ in male rats was 595 milligrams/kilogram (mg/kg) and in female rats was >500 but <560 mg/kg. The acute dermal $LD_{50}$ in rats was >1,600 and 1,068 mg/kg for males and females, respectively. The 4–hour inhalation $LC_{50}$ in rats was 2.772 and 1.982 milligrams/liter (mg/L) for males and females, respectively. Irritation studies in rabbits revealed Spiroxamine was severely irritating to the skin while not irritating to the eye. Spiroxamine exhibited a skin-sensitizing potential in guinea pigs in both the Magnusson/Kligman maximization test and the Buehler patch test.

2. *Genotoxicty*. The genotoxic action of Spiroxamine was studied in bacteria and mammalian cells with the aid of various *in vitro* test systems (*Salmonella* microsome test, forward mutation assay, cytogenetic study with Chinese hamster ovary cells and unscheduled DNA synthesis test) and in one *in vivo* test (micronucleus test). None of the tests revealed any evidence of a mutagenic or genotoxic potential of Spiroxamine. The compound did not induce point mutations, DNA damage or chromosome aberrations.

3.*Reproductive and developmental toxicity*. In a reproduction study using rats, Spiroxamine was administered for two generations at dietary concentrations of 20, 80 or 300 ppm. Reproductive effects such as reduced litter size at birth and clinical signs of toxicity occurred at the high dose in conjunction with maternal toxicity. The parental and reproductive NOAELs were 20 ppm (equal to 2.13 milligrams per kilogram of bodyweight per day (mg/kg bw/day)) and 80 ppm (equal to 9.19 mg/kg bw/day), respectively.

In a developmental toxicity study in rats, Spiroxamine was administered by oral gavage at dose levels of 0, 10 and 25 mg/kg bw/day and in a supplemental study at doses of 0 and 150 mg/kg bw/day. Severe maternal toxicity occurred at 150 mg/kg bw/day resulting in the deaths of 21 of 25 animals. Embryotoxicity (palatoschisis and omphalocele) was observed at the high dose in conjunction with the severe maternal toxicity. The two lower dose levels did not reveal any maternal or developmental toxicity. The results of these studies showed that the dose of 150 mg/kg bw/day was too high to obtain unequivocal results with respect to embryotoxicity and teratogenicity.

In another oral developmental toxicity study in rats, Spiroxamine was administered by gavage during gestation

at doses of 0, 10, 30 or 100 mg/kg bw/day. Developmental toxicity occurred in conjunction with distinct maternal toxicity at the highest dose tested. The maternal NOAEL was 30 mg/kg bw/day based on reduced body weight gain and feed intake at 100 mg/kg bw/day. The NOAEL for developmental toxicity was 30 mg/kg bw/day based on delayed ossification, slightly reduced fetal weights and three cases of palatoschisis at 100 mg/kg bw/day.

In oral developmental toxicity studies in rabbits, Spiroxamine was administered by gavage during gestation at doses of 0, 5, 20 or 80 mg/kg bw/day and in a supplemental study at doses of 0 and 80 mg/kg bw/day. The maternal NOAEL was 20 mg/kg bw/day based on clinical findings, reduced body weight gain, reduced food intake and lethality at 80 mg/kg bw/day. The NOAEL for developmental toxicity was 20 mg/kg bw/day based on marginal developmental toxicity (reduced fetal weight and a slight increased rate of spontaneous malformations) at the highest dose level.

In a dermal developmental toxicity study in rats, Spiroxamine was administered for 6 hours/day during gestation at doses of 0, 5, 20 or 80 mg/kg. Reduced body weight gain occurred in dams at 20 mg/kg and greater. Dose-related skin reactions were observed at all treated doses. Developmental toxicity, such as wavy ribs, occurred in conjunction with maternal toxicity at the highest dose tested. The NOAELs for systemic and local maternal toxicity were 5 and <5 mg/kg, respectively. The NOAEL for developmental toxicity was 20 mg/kg. Spiroxamine did not reveal any teratogenic potential associated with dermal application.

4. *Subchronic toxicity.*In subacute dermal toxicity studies, rabbits were treated with Spiroxamine at doses ranging from 0.05 to 5 mg/kg bw/day for 6 hours/day over a period of 3 weeks. Systemic effects were not observed in these studies. Local irritation, increased skin fold thickness, and histopathological findings of the skin occurred in these studies. The overall NOAELs for local and systemic effects were 0.2 and 5 mg/kg bw/day, respectively.

In a 90-day feeding study, mice were administered Spiroxamine at dietary concentrations of 0, 20, 80, 320 or 1,280 ppm. Effects observed included clinical signs of toxicity, decreased body weight and food consumption, changes in hematological parameters, hyperplastic changes in the epidermis of the auricles and/or tail, and effects on the liver, kidney, and urinary bladder. The NOAEL was 20 ppm (equal to 6.2 mg/kg bw/day) for male mice based on marginally reduced body weight development at 80 ppm. The NOAEL for female mice was 80 ppm (equal to 28.5 mg/kg bw/day) based on slight morphological findings in the liver at 320 ppm.

In another subchronic mouse study, Spiroxamine was administered by oral gavage at doses of 0, 60, 180 or 240 mg/kg. Effects observed included clinical signs of toxicity, and effects of the liver, urinary bladder and hyperplastic changes in the epidermis of the auricles and tails. Evidence of liver enzyme induction was seen in all treatment groups. The NOAEL was <60 mg/kg bw/day for both males and females.

Spiroxamine was administered to rats in a subchronic feeding study at dietary concentrations of 0, 25, 125 or 625 ppm over a period of 13 weeks. Effects included clinical signs of toxicity, reduced body weight gains, changes in hematological parameters, and effects on the liver, urinary bladder, esophagus and forestomach. The NOAEL for both male and female was 25 ppm (equal to 1.9 and 2.7 mg/kg bw/day, respectively) based on histopathological findings in the esophagus and forestomach at 125 ppm.

In two subchronic feeding studies in dogs, Spiroxamine was administered at dietary concentrations of 0, 25, 750 or 1,500 ppm and at 0, 150, 250 or 500 ppm over a period of 13 weeks. Toxicological effects included changes in clinical chemistries, increased relative liver weights, and histopathological findings in the liver. The overall NOAELs from these studies were 500 ppm (equal to 16.9 mg/kg bw/day) and 750 ppm (equal to 21.29 mg/kg bw/day) for males and females, respectively, based on liver effects.

5.*Chronic toxicity*. In a chronic dog study, Spiroxamine was administered at dietary concentrations of 0, 25, 75, 1,000 or 2,000 ppm for a period of 52 weeks. Effects included opthalmological findings, changes in clinical chemistries, mild anemia, and histopathological findings (eye and liver). The NOAEL for both sexes was 75 ppm (equal to 2.47 and 2.48 mg/kg bw/day for males and females, respectively) based on eye and liver effects.

Rats were administered Spiroxamine for 2 years at dietary concentrations of 0, 10, 70 or 490 ppm. Effects included reduced body weight gains, a slight increase in mortality and histopathological findings in the esophagus and urinary bladder. The NOAEL for both sexes was 70 ppm (equal to 4.22 and 5.67 mg/kg bw/day for males and females, respectively) based on esophagus and urinary bladder effects.

The carcinogenicity potential of Spiroxamine was investigated in rats and mice at maximum dietary concentrations of 490 ppm (equal to 32.81 mg/kg bw/day) and 600 ppm (equal to 149.8 mg/kg bw/day), respectively. No evidence of an oncogenic potential of Spiroxamine was found in the long-term studies in rats and mice.

6. *Animal metabolism*. Rats were gavaged with 1 or 100 mg/kg radio-labeled technical Spiroxamine. Seventy percent of the oral low dose was absorbed. Within 48 hours of dosing, over 97% of the dose was excreted in urine and feces. At sacrifice (48 hours post dosing), the radioactivity remaining in the body was below 1% in the low dose groups and approximately 1% and 2% in the male and female rats, respectively, from the high dose group. Concentrations found in tissues and organs were relatively low: i.e., they do not exceed 0.04 micrograms/gram (µg/g). The highest concentrations were found in liver, thymus and adrenals. Slightly smaller concentrations were observed in the thyroid, spleen, fat, ovaries and uterus. The main metabolite in all dose groups is Spiroxamine oxidized to the carboxylic acid in the t-butyl-moiety. The identification rate was approximately 77% of the recovered radioactivity in all dose groups.

7. *Metabolite toxicology*. Toxicological studies have been conducted on KWG 4168 N-oxide, a plant and animal metabolite of Spiroxamine. In an acute oral toxicity study on KWG 4168 N-oxide using female rats, the $LD_{50}$ was 707 mg/kg. In a subacute toxicity study, rats were administered KWG 4168 N-oxide at dietary concentrations of 0, 30, 150 and 1,000 ppm. The highest concentration resulted in treatment-related effects. The main targets were the epithelia of the digestive tract and the urinary bladder. A mild liver enzyme induction was observed without any correlating gross- or micropathological findings. In a subchronic study, rats were administered KWG 4168 N-oxide at dietary concentrations of 0, 25, 125 and 625 ppm, and KWG 4168 at 625 ppm. Toxic effects were observed at 625 ppm for both test substances. Similar effects included delayed body weight development, changes in clinical chemistries and micropathological findings of the esophagus and stomach. The effects were less pronounced for KWG 4168 N-oxide when compared to KWG 4168 (parent). Effects noted only in animals treated with KWG 4168 included changes in hematological

parameters and micropathological findings of the urinary bladder (females). The mutagenic potential of KWG 4168 N-oxide was studied *in vitro* in bacteria and mammalian cells. It did not cause mutations *in vitro* in the Ames assay, the V–79–HPRT gene mutation assay, or produce clastogenicity in the chromosome aberration assay with or without metabolic activation.

8. *Endocrine disruption*. The toxicology data base for Spiroxamine is current and complete. Studies in this data base include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short- or long-term exposure. These studies revealed no primary endocrine effects due to Spiroxamine.

*C. Aggregate Exposure*

1. *Dietary exposure*. An aggregate risk assessment was conducted for all pending uses (grape, hop (domestic and imported) and banana (imported)) to assess the potential acute and chronic dietary exposure resulting from applications of Spiroxamine to these crops. Novigen Sciences, Inc.'s Dietary Exposure Evaluation Model (DEEM®) was used to estimate the chronic and acute dietary exposure.

For the acute dietary analysis, the proposed acute reference dose (aRfD) of 0.1 mg/kg/day was used. This aRfD is based on NOELs of 10 mg/kg from an acute oral toxicity and an acute neurotoxicity screening study and applying a 100–fold uncertainty factor.

For the chronic dietary analysis, the proposed chronic reference dose (cRfD) of 0.02 mg/kg/day was used. This cRfD is based on a parental toxicity NOEL of 2.13 mg/kg/day from the two-generation reproduction study and the application of a 100–fold uncertainty factor.

Results from the acute and chronic dietary exposure analyses described below demonstrate a reasonable certainty that no harm to the overall U.S. population or any population subgroup will result from the use of Spiroxamine on grape, hop and banana.

i. *Food*. An acute, Tier 1 dietary (food) risk assessment was conducted using the highest residue values and 100% crop treated. The estimated percent of the aRfD for the overall U.S. population (all seasons) at the 95 percentile is 8.5%. The most highly exposed population subgroup, non-nursing infants, had an exposure equal to 33.3% of the aRfD at the 95 percentile. These exposure estimates in are within EPA's criteria of acceptability.

A chronic, Tier 1 dietary (food) risk assessment was conducted using average residue values and 100% crop treated. The estimated percent of the cRfD for the overall U.S. population (all seasons) is 9.1%. For the most highly exposed population subgroup, children 1 to 6) years old, the exposure consumed 30.6% of the cRfD. These exposure estimates are within EPA's criteria of acceptability.

ii. *Drinking water*. No monitoring data are available for residues of Spiroxamine in ground water, and EPA has established no health advisory levels or maximum contaminant levels for residues of Spiroxamine in drinking water.

Studies show low to no soil mobility for Spiroxamine and its primary metabolites. In addition, field studies show that Spiroxamine and its degradates do not leach below the 6–inch depth level, and show very low potential to leach into ground water. Therefore, it can be concluded with reasonable certainty that no harm will result from acute or chronic aggregate exposure to Spiroxamine residues in drinking water.

2. *Non-dietary exposure*. Spiroxamine is not registered nor are registrations pending for uses that would result in non-dietary exposure.

*D. Cumulative Effects*

Spiroxamine belongs to a new class of chemistry know as spiroketalamines. Therefore, for this tolerance petition, it is assumed that Spiroxamine does not have a common mechanism of toxicity with other substances and only the potential risks of Spiroxamine in its aggregate exposure are considered.

*E. Safety Determination*

1. *U.S. population*. Based on the above aggregate food exposure estimates for the overall U.S. population (8.5% of the aRfD and 9.1% of the cRfD), the low potential for Spiroxamine and its degradates to leach into ground water, and the completeness of the toxicity data base, there is reasonable certainty that no harm to the U.S. population will result from aggregate exposure to Spiroxamine.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of Spiroxamine, data from developmental toxicity studies in mice, rats, rabbits and a two-generation reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

Based on the above, aggregate food exposure estimates for the most highly exposed population subgroups, i.e., non-nursing infants and children (1–6 years old), consumed 33.3% and 30.6% of the aRfD and cRfD, respectively. This, in combination with the low potential for Spiroxamine and its degradates to leach into ground water, and on the completeness of the toxicity data base, there is reasonable certainty that no harm to infants and children will result from aggregate exposure to Spiroxamine.

*F. International Tolerances*

There are no established Codex, Canadian or Mexican MRLs for Spiroxamine.

[FR Doc. E3–00489 Filed 12–8–03; 8:45 am]

**BILLING CODE 6560–50–S**

## ENVIRONMENTAL PROTECTION AGENCY

**[OPP–2003–0362; FRL–7335–5]**

## Alkyl ($C_{10}$–$C_{16}$) Polyglycosides; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

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

**ACTION:** Notice.

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

**DATES:** Comments, identified by docket ID number OPP–2003–0362, must be received on or before January 9, 2004.

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

**FOR FURTHER INFORMATION CONTACT:** James Parker, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–0731; e-mail address:*parker.james@epa.gov*.

**SUPPLEMENTARY INFORMATION:**

### I. General Information

*A. Does this Action Apply to Me?*

You may be potentially affected by this action if you are an agricultural