F. International Tolerances

To date, no international tolerances exist for indoxacarb.

[FR Doc. 03–16739 Filed 7–1–03; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0211; FRL–7312–8]

Dinotefuran; 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–2003–0211, must be received on or before August 1, 2003.

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: Rita Kumar, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–8291; e-mail address: *kumar.rita@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, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

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

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

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

1. *Docket.* EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0211. 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 EPA's 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–2003–0211. 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–2003–0211. 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–2003–0211.

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–2003–0211. 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 pesticide petitions 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 these petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions.

List of Subjects

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

Dated: June 20, 2003.

Debra Edwards,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

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

Mitsui Chemicals, Inc.

PP 2F6427 and 3F6566

EPA has received pesticide petitions (2F6427 and 3F6566) from Mitsui Chemicals, Inc., Chiyoda-ku, Tokyo, Japan, 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 dinotefuran, (*RS*)-1-methyl-2-nitro-3-(tetrahydro-3-furylmethyl)guanidine and its major metabolites, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine,

and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in or on fruiting vegetables, leafy vegetables, head and stem brassica vegetables, cotton, cucurbits, grapes, and potato. The tolerances are set at the following value: Fruiting vegetables, 0.7 part per million (ppm); leafy vegetables, 5.0 ppm; tomato paste, 1.0 ppm; cucurbits, 0.5 ppm; head and stem brassica vegetables, 1.4 ppm; grape, 0.8 ppm; raisin, 2.5 ppm; potato, 0.05 ppm; chips, 0.10 ppm; granules, 0.15 ppm; cotton seed undelinted at 0.2 ppm, and cotton gin byproducts at 7.0 ppm. Tolerances for meat, milk, and byproducts is set at 0.05 ppm. This new active ingredient has been accepted by EPA as a reduced risk chemical. EPA has determined that the petitions 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 petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The primary metabolic pathways of dinotefuran in plants (rice, apple, potato, oilseed, rape, and lettuce) were similar to those described for animals, with certain extensions of the pathway in plants. Parent compound, dinotefuran, and two metabolites, 1-methyl-3-(tetrahydro-3-furymethyl)guanidine and 1-methyl-3-(tetrahydro-3-furymethyl)-urea were major metabolites in all crops. The metabolism of dinotefuran in plants and animals is understood for the purposes of the proposed tolerances. Parent dinotefuran and the metabolites, 1-methyl-3-(tetrahydro-3-furymethyl)guanidine and 1-methyl-3-(tetrahydro-3-furymethyl)-urea are the residues of concern for tolerance setting purposes.

2. *Analytical method*. Mitsui Chemicals, Inc., has submitted practical analytical methodology for detecting and measuring levels of dinotefuran and its metabolites, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in or on raw agricultural commodities (RACs). The high performance liquid chromotography (HPLC) method was validated for determination of, dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea in or on tomatoes, peppers, cucurbits, brassica, grapes, potatoes, and lettuce for raw agricultural commodity matrices and in or on tomato paste, puree, grape juice, raisins, potato chips, granules, and wet peel for processed commodity matrices. After extraction with a water/acetonitrile mixture and clean up with hexane and extraction columns, concentrations of dinotefuran and its metabolites were quantified after HPLC separation by mass spectrometry/molecular size (MS/MS) detection. The limit of quantitation (LOQ) was 0.01 ppm for all matrices.

The HPLC method was validated for the determination of dinotefuran and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea in or on cotton (undelinted seed, gin trash, meal, hulls, refined oil), and leafy vegetables. After extraction with a water/acetonitrile mixture and clean up dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea were quantified after HPLC separation by MS/MS detection. For undelinted seed, gin trash, meal, and hulls, a LOQ of 0.05 milligram/kilogram (mg/kg) and a working range from 0.05 to 0.50 mg/kg were successfully validated for dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea. For refined oil, a LOQ of 0.01 mg/kg and a working range from 0.01 to 0.10 mg/kg were successfully validated for dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea. An HPLC method was validated for the determination of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in lettuce. After extraction with water/acetonitrile mixture and clean-up, dinotefuran was quantified after HPLC separation by ultraviolet ray (UV) detection, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea by MSD. A LOQ 0.010 mg/kg and a working range from 0.01 to 5.00 mg/kg were successfully validated from dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea. All of the above methods have been independently validated.

3. *Magnitude of residues*. Crops in residue trials were treated at maximum label rates and harvested at the specified minimum treatment to harvest intervals. The residue method for dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in all components utilized HPLC separation with MS/MS detection.

For cucurbit vegetables (crop group 9), residue trials were conducted for each of the three representative crops, cucumbers, melons, and squash. The proposed tolerance in or on cucurbit vegetables for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, is 0.5 ppm. The maximum combined residue found for the representative cucurbit vegetable crops was 0.44 ppm for a melon sample.

For leafy vegetables (crop group 4), residue trials were conducted for each of the four representative crops, celery, leaf lettuce, head lettuce, and spinach, at six locations. The proposed tolerance in or on leafy vegetables for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, is 5.0 ppm. The maximum combined residue found for the representative leafy vegetable crops was 4.36 ppm for a spinach sample.

Residue trials for cotton were conducted at 13 locations and undelinted cotton seed samples were collected and analyzed. Cotton gin byproducts (gin trash) samples were obtained for 7 of the locations. Processing studies with analyses of cotton seed meal, hulls, and oil were performed with cotton seed harvested at two locations that were both treated with 5X the maximum label rate. The proposed tolerance for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in or on cotton seed undelinted is 0.2 ppm. All cotton seed residue samples had combined residues of less than 0.2 ppm. The proposed tolerance for cotton gin byproducts is 7.0 ppm for combined residues of dinotefuran and its two major metabolites. The maximum combined residues for cotton gin byproducts in these trials was 6.4 ppm. Processing studies established that residues of dinotefuran and its metabolites, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, did not concentrate in cotton seed meal, oil, or hulls. Therefore, tolerances are not proposed for these processing fractions.

Residue trials for grapes were conducted at 13 locations and 2 grape juice and raisin processing studies were performed with grapes from exaggerated treatment rate applications. The proposed tolerance for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in or on grapes is 0.8 ppm. The maximum combined residue for an individual grape residue sample was 0.73 ppm and the highest average field trial (HAFT) for grapes had

combined residues of 0.55 ppm. The proposed tolerance for raisins is 2.5 ppm for combined residues of dinotefuran and its two major metabolites based on the average concentration factor of 4.0 for processing grapes to raisins. The grape juice processing studies established an average concentration factor of 1.3 for residues of dinotefuran and its metabolites 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, because the product of multiplying the grape HAFT times the average concentration factor for processing grapes into juice is less than the proposed grape tolerance, a separate tolerance is not proposed for grape juice.

For potatoes, residue trials were performed at 17 locations and 2 studies processing potatoes into chips, granules, and wet peel were performed with potatoes that were treated with exaggerated application rates. The proposed tolerance for combined residues of dinotefuran and its metabolites 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, on potatoes is 0.05 ppm. The maximum combined residues found on potatoes were less than 0.05 ppm with maximum residues of dinotefuran less than 0.03 ppm. The HAFT result was 0.04 ppm of combined residues. The average concentration factors for processing potatoes into chips, granules, and wet peel were 2.2, 3.65, and less than 1 respectively. No separate tolerance is proposed for wet peel. Based on the average concentration factors and the HAFT, tolerances for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, are proposed for potato chips at 0.1 ppm and for potato granules at 0.15 ppm.

For fruiting vegetables (crop group 8) residue trials were conducted for the three representative commodities, tomatoes, bell pepper, and non-bell pepper. The proposed tolerance for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in or on fruiting vegetables is 0.7 ppm. The maximum combined residue for the representative fruiting vegetables was 0.58 ppm on peppers. The HAFT result for combined residues on tomatoes was 0.20 ppm. Three studies for processing tomatoes into tomato puree and tomato paste were performed with tomatoes that were treated at exaggerated application rates. The average concentration factors determined in these studies were 1.8 for processing tomatoes into puree and 4.8 for processing tomatoes into paste. Since the product of the average concentration factor for puree and the HAFT for tomatoes is less than the proposed tolerance for fruiting vegetables, no separate tolerance is proposed for tomato puree. A combined tolerance of 1.0 ppm is proposed for tomato paste, based on the average concentration factor for processing of 4.8 and the HAFT of 0.20 ppm for tomatoes.

For vegetables, brassica head, and stem crop subgroup (crop subgroup 5–A), residue trials were conducted with three representative crops, broccoli, cauliflower, and cabbage. The proposed tolerance for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, on stem and head brassica vegetables is 1.4 ppm. The maximum combined residue in field trials was 1.25 ppm on broccoli.

Metabolism studies in livestock and poultry (nature of residue studies with goats and hens), established that dinotefuran was rapidly metabolized and excreted and that there was very little transmittal of residues of dinotefuran and its metabolites to meat, milk, or eggs. For goats fed 10 ppm of radiolabeled dinotefuran, the total radioactive residues (TRR) in meat and milk were less than 0.05 ppm.

The maximum livestock dietary burden from feeding cotton commodities and potatoes (which all contain residues at the proposed tolerance levels) was 1.9 ppm for beef cattle and 1.9 ppm for dairy cattle. To provide for the possible transmittal of the residues of dinotefuran and its metabolites, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in cattle and other livestock, tolerances are proposed for combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in milk at 0.05 ppm, in meat (from cattle, goats, hogs, horses, and sheep) at 0.05 ppm and in meat byproducts, including fat, liver, and kidney, (from cattle, goats, hogs, horses, and sheep) at 0.05 ppm. These proposed tolerances are based on the results of a cow feeding study where dairy cows received dosages of combined residues of dinotefuran and its metabolites and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, representing 5ppm (1X), 15 ppm (3X), and 50 ppm (10X) in the daily diet. The dosages contained dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, in a 3:1:1 ratio, thus, the 5 ppm level contained 3 ppm of dinotefuran, 1 ppm of 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1 ppm of 1-methyl-3-(tetrahydro-3-furylmethyl)-urea. The dosing period was 29 to 30 days, whole milk, skim milk, and cream were analyzed through the collection period and meat, fat, and edible tissues were analyzed at conclusion of the dosing period.

There were only low levels of residues transmitted to milk, meat, fat, and edible tissues in the study. No dinotefuran residues (<0.01 ppm) were measured in milk from 5 ppm dosage cows. Maximum residues of dinotefuran in milk were 0.012 ppm in the 3X level cows and 0.032 ppm in the 10X level cows. No detectable residues of parent dinotefuran were found in muscle, fat, or edible tissues from cows at any dosage level. Milk, muscle, fat, and edible tissues were also analyzed for 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea, the two dinotefuran metabolites included in the combined residues in the proposed tolerance expression. Transmittal of quantifiable residues of 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine was found at the 1X dosage level with maximum residues of 0.013 ppm of 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine in milk and at the 10X level with 0.011 ppm of 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine in milk and 0.02 ppm of 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine in muscle, liver, and kidney. Quantifiable residues of 1-methyl-3-(tetrahydro-3-furylmethyl)-urea were found in the 1X dosage level, with 1-methyl-3-(tetrahydro-3-furylmethyl)-urea residues up to 0.02 ppm in whole milk and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea residues of 0.011 to 0.012 in muscle, liver, and kidney. The 1-methyl-3-(tetrahydro-3-furylmethyl)-urea residues increased proportional to dosage with the 10X level having 1-methyl-3-(tetrahydro-3-furylmethyl)-urea residues of up to 0.24 ppm in milk, 0.13 ppm in muscle, 0.07 ppm in fat, 0.12 ppm in liver, and 0.18 ppm in kidney. In the cow feeding study at the 1X dosage level comprising combined residues of dinotefuran, 1-methyl-3-(tetrahydro-3-furylmethyl)guanidine, and 1-methyl-3-(tetrahydro-3-furylmethyl)-urea of 5 ppm of diet, the total combined residues for milk, muscle, fat, liver, and kidney were each less than 0.05 ppm. Since the maximum theoretical combined

residues from the proposed uses of dinotefuran on cotton and potatoes would be 1.9 ppm, for dairy and beef cattle, the proposed tolerances in milk, meat, and meat byproducts, would be sufficient to provide for potential transmittal of residues from livestock diets containing residues of dinotefuran and its metabolites.

The maximum theoretical poultry dietary burden from feeding cotton commodities containing residues of dinotefuran and its metabolites at the proposed tolerance levels was calculated to be 0.09 ppm. Since the TRR in meat and eggs from hens fed 10 ppm of radiolabeled dinotefuran in the poultry metabolism study was less than 0.05 ppm it can be concluded that there is no reasonable expectation of transmittal of finite residues of dinotefuran and its metabolites to meat and eggs, for poultry fed cotton commodities treated with dinotefuran. Therefore no tolerances are proposed for combined residues of dinotefuran and its metabolites in poultry or eggs.

B. Toxicological Profile

1. *Acute toxicity*. Dinotefuran has low acute oral, dermal, and inhalation toxicity. The oral lethal dose $(LD)_{50}$ in rats is 2,450 mg/kg, the dermal LD_{50} is >2,000 mg/kg and the inhalation 4-hour lethal concentration $(LC)_{50}$ is >4.09 milligrams/Liter (mg/L) air. Dinotefuran is not a skin sensitizer in guinea pigs, but is slightly irritating to the skin and eyes of rabbits. End-use formulations of dinotefuran have similar low acute toxicity profiles.

2. *Genotoxicity*. Dinotefuran and its metabolites do not induce gene mutations in bacterial and mammalian cells, chromosome aberrations in mammalian cells or deoxyribonucleic acid (DNA) damage in bacterial cells in *in vitro* test systems. Similarly, it does not exhibit a clastogenic effect *in vivo* in the mouse micronucleus test. Therefore, there is no evidence to suggest a genotoxic hazard at any of the three main levels of genetic organization.

3. *Reproductive and developmental toxicity*. In rat and rabbit developmental toxicity studies with dinotefuran, there was no evidence of teratogenicity or other embryotoxic effects at the highest dose levels, although maternal toxicity was evident. There were no treatment-related effects on litter parameters at any dose level in either species. In rats, 1,000 mg/kg produced decreased food consumption, body weight gain, and increased water intake. In rabbits, 300 mg/kg produced hypoactivity, prone position, panting, flushing of the nose and ears, tremors, reduced weight gain, food consumption, and water intake. Necropsy revealed pale brown discoloration of liver and gray/white plaques in the stomach at 125 and 300 mg/kg. The no adverse effect level (NOAEL) values in maternal rats and rabbits were 300 and 52 mg/kg/day, respectively. The NOAEL values in rats and rabbits for embryonic development and teratogenicity were the highest dose levels administered, 1,000 and 300 mg/kg/day, respectively. In a 2-generation study, parental animals of both sexes and both generations showed reduced body weight gain and food consumption at the highest dose level evaluated (10,000 ppm), but there was no effect of treatment at any dose level in either generation on reproductive performance indicators. There were no treatment-related effects at any dose level on the histopathological appearance of the reproductive organs of either sex. Similarly, there were no effects at any dose level in either generation on quantitative ovarian histopathology or on sperm counts, motility and morphology. Reduced spleen weight in probit dose extrapolation model (P) generation animals and reduced thyroid weight in F1 generation parental females were apparent at 10,000 ppm. F1 pup behavioral and sexual development was unaffected by treatment at all dose levels but pup weight gain during lactation was reduced at 10,000 ppm in both generations. Furthermore, the spleen weight of F1 generation progeny was reduced at 10,000 ppm. Based on reduced weight gain and food consumption in parental animals at 10,000 ppm and reduced pre-weaning weight gain in the offspring, the NOAEL value for parental animals and offspring is 241 mg/kg.

4. *Subchronic toxicity*. Dinotefuran was evaluated in a 13-week oral (diet) toxicity studies in rats, mice, and dogs. No specific target organs were identified in any species. In the rat study, a NOAEL of 500 ppm (34/38 mg/kg/day for males and females) was established, based on minimal growth retardation in females and adrenal cortical vacuolation in males. A NOAEL was established at 5,000 ppm (336/384 mg/kg/day for males/females) based on marked growth retardation at 25,000 ppm (adrenal cortical vacuolation not adverse). In the mouse study, a NOAEL of 25,000 ppm (4,442/5,414 mg/kg/day for males/females) was established based on growth retardation at 50,000 ppm. In the dog study, a NOAEL of 8,000 ppm (307/323 mg/kg/day in males/females) was established based on growth retardation. Dinotefuran was also evaluated for dermal and inhalation toxicity for 4 weeks in rats. Daily inhalation exposure of rats for 6 hours/day for 4 weeks did not elicit toxicologically significant effects at any exposure concentration up to and including the highest technically achievable concentration (2.08 mg/L) with a low mass median aerodynamic diameter) (MMAD≤±GSM of 2.03 µm±3.60. Dinotefuran was well tolerated and there were no treatment-related effects on clinical condition, hematology, and clinical chemistry profiles, organ weights, macroscopic, and microscopic pathology. Dermal application for 4 weeks at dose levels up to 1,000 mg/kg/day did not elicit any local or systemic effects on any of the parameters examined. Therefore, no target organs were identified in the rat either by dermal or inhalation exposure.

5. *Neurotoxicity*. Dinotefuran did not produce any functional or histomorphological evidence of neurotoxicity in acute (gavage) and 13–week (dietary) neurotoxicity studies in rats. The NOAEL for neurotoxicity in the acute study was 1,500 mg/kg, the highest dose level administered. The NOAEL for neurotoxicity in the 13–week dietary study was 50,000 ppm (3,413/3,806 mg/kg/day for males and females). The NOAEL for all effects in this study was 5,000 ppm (327/400 mg/kg/day for males and females) based on reduced body weight gain and food consumption.

6. *Chronic toxicity*. Chronic toxicity studies with dinotefuran have been conducted in rats, mice, and dogs. In common with the subchronic studies in these species, no specific target organs could be identified. In the 52-week dog study, a NOAEL of 559/512 mg/kg/day for males/females was established based on decreased weight gain in both sexes and decreased food consumption in females. In the 78-week mouse study, a NOAEL of 345/441 mg/kg/day for males/females was established, based on decreased weight gain and a decrease in circulating platelet counts. In the 104-week rat study, a NOAEL of 991/127 mg/kg/day for males/females was established. This was based on a decrease in weight gain in females.

7. *Carcinogenicity*. The carcinogenic potential of dinotefuran has been evaluated in rats and mice. Survival incidences in the oncogenicity studies were unaffected by treatment at all dose levels. There were no treatment-related effects on the nature and incidence of neoplastic and adverse non-neoplastic histomorphological findings in either species at any dose level. Therefore, the NOAEL values for all effects, 991/127 mg/kg/day (male/female rats) and 345/441 mg/kg/day (male/female mice) are based on reduced weight gain, and also

on reduced numbers of platelets in mice.

8. *Animal metabolism*. In the rat, dinotefuran is rapidly and almost completely absorbed from the gastrointestinal tract into the general circulation, and is widely distributed throughout the tissues and fluids of the body. Elimination is rapid, predominantly by urinary excretion and almost complete within 7 days of administration. There is no evidence for tissue accumulation. Dinotefuran is rapidly transferred to maternal milk and widely distributed into fetal tissues but rapidly eliminated from them. More than 90% of orally and intravenously administered dinotefuran is eliminated as unchanged parent molecule, which is also the major radioactive component in plasma, milk, bile, and most tissues. The major route of metabolism is an initial enzymatic hydroxylation of the tetrahydrofuran ring to form isomers of 6-hydroxy-5-(2-hydroxyethyl)-1-methyl-1,3-diazinane-2-ylidine-*N*-nitroamine, followed by further oxidation, reduction and acetylation of 6-hydroxy-5-(2-hydroxyethyl)-1-methyl-1,3-diazinane-2-ylidine-*N*-nitroamine, to produce possible isomers of 1-methyl-2-nitro-3-(2-oxotetrahydro-3-furylmethyl)guanidine, 1-[4-hydroxy-2-(hydroxymethyl)butyl]-3-methyl-2-nitroguanidine, 6-hydroxy-5-(2-hydroxyethyl)-1-methyl-1, 3-diazinane-2-ylidene-*N*-nitroamine acetyl conjugate and 3-hydroxymethyl-4- (3-methyl-2-nitroguanidine) butyric acid. Several minor pathways of metabolism of dinotefuran were identified in animals. The absorption, distribution, metabolism and elimination of dinotefuran is unaffected by sex and treatment regimen. In hens and goats, the metabolite profile was similar as in plant metabolism.

9. *Metabolite toxicology*. The metabolism profile for dinotefuran supports the use of an analytical enforcement method that accounts for parent dinotefuran, and 1-methyl-3-(tetrahydro-3-furymethyl)guanidine and 1-methyl-3-(tetrahydro-3-furymethyl)-urea. Other metabolites are considered of equal or lesser toxicity than parent compound.

10. *Endocrine disruption*. Dinotefuran does not belong to a class of chemicals known or suspected of having adverse effects on the endocrine system. There is no evidence that dinotefuran has any effect on endocrine function in developmental or reproduction studies. Furthermore, histological investigation of endocrine organs in chronic dog, rat, and mouse studies did not indicate that the endocrine system is targeted by dinotefuran.

C. Aggregate Exposure

1. *Dietary exposure*. Chronic dietary exposure assessments were conducted using a Tier I approach. This Tier I assessment incorporated tolerance level residues and 100% crop-treated in the EXP estimated dietary intake trends evaluation system (EXPedite™ system, Version 4.1). EXPedite™ utilized the food consumption data derived from the 1994–1996 U.S. Department of Agriculture (USDA) Continuing Surveys of Food Intake by Individuals (CSFII) with the 1998 supplemental children's survey. The resulting exposures were compared to a RfD of 1.27 mg/kg/day, which was based on the female NOAEL of 127 mg/kg/day from the 104-week rat study and a 100–fold uncertainty factor. Chronic dietary exposure estimates for the overall U.S. population and 25 population subgroups are well below the chronic RfD. Results of these analyses are summarized below.

TABLE 1.—CHRONIC DIETARY RISK (DEEM™) ANALYSIS OF DINOTEFURAN

Population Subgroup	Mg/Kg Bwt/Day	%RfD
U.S. population	0.004109	0.32%
All infants (<1–year old)	0.002815	0.22%
Non-nursing infants	0.003438	0.27%
Children (1 to 6)	0.007247	0.57%
Children (7 to 12)	0.004348	0.34%
Females (13 to 50)	0.003350	0.26%
Males 13+ years	0.003173	0.25%

There are no acute toxicity concerns with dinotefuran as there is no toxicological endpoint attributable to a *single* exposure in the dinotefuran toxicology data base, including the rat and rabbit developmental studies. Therefore, only chronic dietary exposures have been assessed.

2. *Non-dietary exposure*. Mitsui also requests registrations for the use of dinotefuran on cats, turf, ornamentals, indoor foggers, and ready to use sprays. Mitsui has considered potential non-dietary and aggregate (non-dietary + dietary) exposures to adults, adult females, and toddlers (1 to 3 years of age) for these uses.

Applicator and post-application exposures can result from dermal and inhalation routes for both adults and toddlers. Additionally, toddlers can be exposed through the post-application incidental ingestion route via hand-to-mouth behavior. Based on the label instructions and typical use patterns of these product types, only short-term and intermediate-term exposure scenarios should be considered for dinotefuran products. However, since there are no toxicological endpoints attributable to a single or possible multiple exposures in a very short duration, as in a short-term scenario, only the intermediate-term exposure scenario has been evaluated for this document.

Dermal exposures for applicator and post-application activities were not assessed because the very high dermal NOAEL (>1,000 mg/kg/day) for dinotefuran indicates that dermal exposures are not of concern. Short-term oral (e.g., incidental ingestion) exposures for toddlers, as mentioned above, were not assessed because there are no toxicological endpoints attributable to a single exposure or multiple exposures during a very short-term time frame in the dinotefuran toxicology data base. Since the oral endpoint is used to calculate inhalation risks, short-term inhalation exposures for toddlers and adults were also not evaluated since there is no toxicological endpoint attributable to a short-term endpoint. Intermediate-term inhalation exposures for applicator and post-application activities also were not assessed because the very high inhalation NOAEL (>7,000 mg/kg/day) for dinotefuran indicates that inhalation exposures are not of concern. Therefore, only intermediate-term oral (incidental ingestion) exposures for toddlers were assessed. These exposures were assessed for each individual dinotefuran product, as well as for the aggregation of all products. In the aggregate assessment, it was assumed that the toddlers would be exposed to residues resulting from the agricultural uses (chronic dietary), all within 1-day.

These non-dietary assessments were conducted using equations and default parameters from EPA's Residential Standard Operation Procedures (SOPs) (EPA, 1997 and 2001) and maximum application rates. Although these exposures are based on the intermediate-term time frame, the residue on the day of application was used in the SOP equations in order to maintain an extra level of conservatism. This assumption implies that the toddlers are exposed to residue levels, which are equivalent to levels resulting on the day of application, every day over an intermediate-term time frame. The resulting oral and aggregate exposures were compared to the NOAEL

of 307 mg/kg/day observed in the 13-week dog study. These risk estimates (margin of exposures (MOE)) for toddlers (1 to 3 years of age) are summarized below. From the results below, Mitsui concludes there is reasonable certainty of no harm associated with the aggregate (dietary + non-dietary) exposure to dinotefuran.

TABLE 2.—INTERMEDIATE-TERM AGGREGATE MOES

Exposure Routes	Dietary	RTU Spray	Fogger	Turf	Cat	Aggregate
Toddlers (1 to 3 years old)						
Dietary	184,163	NA	NA	NA	NA	184,163
Incidental Ingestion	NA	23,356	11,431	80,050	1,850	1,410
Total						1,410

3. *Drinking water exposure*. EPA uses the drinking water level of comparison (DWLOC) as a theoretical upper limit on a pesticide's concentration in drinking water when considering total aggregate exposure to a pesticide in food, drinking water, and residential uses. DWLOCs are not regulatory standards for drinking water; however, EPA uses DWLOCs in the risk assessment process as a surrogate measure of potential exposure from drinking water. In the absence of monitoring data for pesticides, it is used as a point of comparison against conservative model estimates of a pesticides concentration in water.

An estimate of the drinking water environmental concentration (DWEC) in ground water and surface water for dinotefuran has been made for this notice of filing. The DWEC of dinotefuran in ground water was estimated to be 0.94 part per billion (ppb) using screening concentration in ground water (SCI–GROW) (the screening model for ground water), and the DWEC for surface water was estimated to be 6.24 ppb (for chronic and intermediate-term aggregate assessments) using FQPA Index Reservoir Screening Tool (FIRST).

To calculate the DWLOC for chronic aggregate exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure from EXPedite™, as addressed above, was subtracted from the reference dose (RfD) to obtain the acceptable chronic exposure to dinotefuran in drinking water. DWLOCs, as presented below, were then calculated using default body weights and drinking water consumption figures.

TABLE 3.—CHRONIC AGGREGATE DRINKING WATER ASSESSMENT

Population Subgroup	Dietary Mg/Kg Bwt/Day	Maximum Water Exposure Mg/Kg Bwt/Day	Kg Bwt	SCI–GROW (ppb)	FIRST (ppb)	DWLOC (ppb)
U.S. population	0.004109	1.265891	70	0.94	6.24	44,306
All infants (<1–year old)	0.002815	1.267185	10	0.94	6.24	12,672
Non-nursing infants	0.003438	1.266562	10	0.94	6.24	12,666
Children (1 to 6)	0.007247	1.262753	20	0.94	6.24	25,255
Children (7 to 12)	0.004348	1.265652	40	0.94	6.24	50,626
Females (13 to 50)	0.003350	1.266650	60	0.94	6.24	38,000
Males (13+ years)	0.003173	1.266827	70	0.94	6.24	44,339

Chronic RfD used in assessments - 1.27 mg/kg bwt/day

The estimated average concentration of dinotefuran in surface water is 6.24 ppb. This value is less than the lowest DWLOC for dinotefuran as a contribution to chronic aggregate exposure (12,666 ppb for non-nursing infants, the most highly exposed population group for the chronic scenario). Therefore, taking into account the proposed uses, it can be concluded with reasonable certainty that residues of dinotefuran in food and drinking water will not result in unacceptable levels of human health risk.

To calculate the DWLOC for the intermediate-term aggregate exposure relative to a sub-chronic toxicity endpoint, the chronic dietary food exposure from EXPedite™ plus the intermediate-term non-dietary exposures were subtracted from the NOAEL, divided by the target MOE (100), to obtain the acceptable intermediate-term exposure to dinotefuran in drinking water. DWLOCs, as presented below, were then calculated using default body weights and drinking water consumption figures.

TABLE 4.—INTERMEDIATE-TERM AGGREGATE DRINKING WATER ASSESSMENT

Population Subgroup	NOAEL/MOE Mg/Kg/Day	Aggregate Exposure Mg/Kg/Day	Maximum Water Exposure mg/kg/day	SCI–GROW (ppb)	FIRST (ppb)	DWLOC (ppb)
Toddlers (1 to 3)[1]	0.307	0.217	2.852	0.94	6.24	42,785

[1] Assume 70kg bodyweight

The estimated average concentration of dinotefuran in surface water is 6.24 ppb. This value is less than the DWLOC for dinotefuran as a contribution to intermediate-term aggregate exposure (42,785 ppb). Therefore, taking into account the proposed uses, it can be concluded with reasonable certainty that residues of dinotefuran in residential environments and in food and drinking water will not result in unacceptable levels of human health risk.

D. Cumulative Effects

The potential for cumulative effects of dinotefuran and other substances that have a common mechanism of toxicity has also been considered. Dinotefuran belongs to a pesticide chemical class known as the neonicotinoids and subclass nitroguanadines. There is no reliable information to indicate that toxic effects produced by dinotefuran would be cumulative with those of any other chemical including another pesticide. Therefore, Mitsui believes it is appropriate to consider only the potential risks of dinotefuran in an aggregate risk assessment.

E. Safety Determinations

1. *U.S. population*. Using the chronic exposure assumptions and the proposed RfD described above, the dietary exposure to dinotefuran for the U.S. population (48 states, all seasons) was calculated to be 0.32% of the RfD of 1.27 mg/kg/day. The resulting DWLOC, 44,306 ppb, is much greater than the estimated average concentration of dinotefuran in surface water, 6.24 ppb. Therefore, taking into account the proposed uses, it can be concluded with reasonable certainty that residues of dinotefuran in residential environments and in food and drinking water will not result in unacceptable levels of human health risk.

2. *Infants and children*. FFDCA section 407 provides that EPA shall apply an additional safety factor for infants and children to account for prenatal and postnatal toxicity and the completeness of the data base. Only when there is no indication of increased sensitivity of infants and children and when the data base is complete, may the extra safety factor be removed. In the case of dinotefuran, the toxicology data base is complete. There is no indication of increased sensitivity in the data base overall, and specifically, there is no indication of increased sensitivity in the developmental and multi-generation reproductive toxicity studies. Therefore, Mitsui concludes that there is no need for an additional safety factor; the RfD of 1.27 mg/kg/day and sub-chronic NOAEL of 307 mg/kg/day are protective of infants and children.

Using the chronic exposure assumptions and the proposed RfD described above, the dietary exposure to dinotefuran for infants and children (1 to 6 years) was calculated to be 0.57% of the reference dose of 1.27 mg/kg bwt/day. The resulting DWLOC for non-nursing infants, 12,666 ppb, is much greater than the estimated average concentration of dinotefuran in surface water, 6.24 ppb.

Using the intermediate-term exposure assumptions and the proposed NOAEL described above, the intermediate-term aggregate exposure to dinotefuran for the toddlers (1 to 3 years) resulted in an MOE of 1,410. The resulting DWLOC, 42,785 ppb, is much greater than the estimated average concentration of dinotefuran in surface water, 6.24 ppb. Therefore, taking into account the proposed uses, it can be concluded with reasonable certainty that residues of dinotefuran in residential environments and in food and drinking water will not result in unacceptable levels of human health risk.

F. International Tolerances

No codex maximum residue levels have been established for residues of dinotefuran on any crops at this time.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0226; FRL–7315–2]

Copper Hydroxide; Notice of Filing of 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–2003–0226, must be received on or before August 1, 2003.

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: Kathryn Boyle, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6304; e-mail address: *boyle.kathryn@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, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Crop production (NAICS code 111)
- Animal production (NAICS code 112)
- Food manufacturing (NAICS code 311)
- Pesticide manufacturing (NAICS code 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**.