Country	Crop	MRL (ppm)
Argentina	Soybean Sunflower	0.015 0.02
Brazil	Soybean	0.05
France	Grape	0.05
Paraguay	Soybean	0.015
South Africa	Soybean Groundnut	0.02 0.02
Spain	Soybean Peanut	0.05 0.05

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2005–0016; FRL–7703–7]

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

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket identification (ID) number OPP–2005–0016, must be received on or before May 9, 2005.

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

FOR FURTHER INFORMATION CONTACT: Mary 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, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

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

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

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

1. *Docket*. EPA has established an official public docket for this action under docket ID number OPP–2005–0016. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

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

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

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. 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–2005–0016. 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–2005–0016. 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–2005–0016.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 S. Bell St., Arlington, VA, Attention: Docket ID Number OPP–2005–0016. 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 record keeping requirements.

Dated: March 28, 2005.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

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

Kureha Chemical Industry Co., Ltd

PP 9E5052

EPA has received a pesticide petition (PP 9E5052) from Kureha Chemical Industry Co., Ltd, c/o Company Agent, BASF Corporation, P.O. Box 13528, Research Triangle Park, NC, 27704-3528 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 metconazole in or on the raw agricultural commodity bananas at 0.05 parts per million. 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 qualitative nature of the residues of metconazole in bananas is adequately understood. The metabolism of metconazole in bananas is characterized by a significant amount (greater than 85%) of unchanged parent compound. In addition to the parent compound, many other minor residue components (each less than 2% of the total recovered radioactivity in the whole fruit) were detected. Metconazole is the only residue of toxicological concern in bananas.

2.*Analytical method*. A practical analytical method for detecting and measuring the level of metconazole residues in whole bananas and banana pulp is submitted to EPA with this petition. Quantitation of residues of metconazole in bananas is by gas chromatography with a nitrogen-phosphorus detector. This independently validated method is appropriate for the enforcement purposes of this petition.

3.*Magnitude of residues*. Residue field trials were conducted in representative countries exporting the commodities of this petition to the United States. Twelve field trials were conducted with bagged and unbagged bananas, with three sites located in each of four countries, Ecuador, Honduras, Costa Rica, and Mexico. The residue values reported from these field trials were all less than the proposed tolerance of 0.05 ppm for whole bananas. No processing study is included with this petition as bananas have no processed commodities according to the EPA Residue Chemistry Test Guidelines.

B. Toxicological Profile

A complete, valid and reliable database of mammalian and genetic toxicology studies supports the proposed tolerance for metconazole on bananas. Two geometric isomers of metconazole exist, with the fungicidal activity being associated primarily with the cis isomer. The technical material that is manufactured for use on bananas is a mixture of cis and trans isomers in an 85 to 15 ratio (85:15). Toxicology studies submitted in support of this petition were conducted on the technical material composed of either the 85:15 isomer mixture (AC 900768) or a more purified (greater than 95%) sample of the cis isomer (WL 136184).

1.*Acute toxicity*. AC 900768 technical is considered to be slightly toxic (Toxicity Category III) to the rat by the oral route of exposure. In an acute oral study in rats, the LD_{50} value of AC 900768 technical was 727 milligrams per kilogram of body weight (mg/kg b.w.) for males and 595 mg/kg b.w. for females. The oral LD_{50} for combined sexes was 660 mg/kg b.w. An oral LD_{50} study in rats conducted with WL 136184 technical also supports the classification of metconazole as slightly toxic by the oral route of exposure. The oral LD_{50} values of WL 136184 technical were 1,626 mg/kg b.w. for males and 1,312 mg/kg b.w. for females, with an LD_{50} value for combined sexes of 1,459 mg/kg b.w. Since this petition is for an import tolerance, anticipated exposure is only via the oral route. As such, oral toxicity data sufficiently assess risk of acute exposure.

2.*Genotoxicty*. AC 900768 technical (the 85:15 isomer mixture) and WL 136184 technical (greater than 95% cis isomer) were tested in an extensive battery of *in vitro* and *in vivo* genotoxicity assays measuring several different endpoints of potential genotoxicity. Collective results from these studies indicate that metconazole does not pose a genotoxic risk, and therefore, is not likely to be a genotoxic carcinogen.

3.*Reproductive and developmental toxicity*. Developmental toxicity studies in rats conducted with AC 900768 technical and WL 136184 technical showed no evidence of teratogenic effects in fetuses, and no evidence of developmental toxicity in the absence of maternal toxicity. Thus, metconazole is neither a selective developmental toxicant nor a teratogen in the rat. In the rat developmental toxicity study with AC 900768 technical, the no-observable-adverse-effect-level (NOAEL) for maternal toxicity was 12 mg/kg b.w./day, based on decreased body weight gain at 30 mg/kg b.w./day, the next highest dose tested, and the NOAEL for developmental toxicity was also 12 mg/kg b.w./day, based on decreased fetal body weights and an increased incidence of skeletal ossification variations at 30 mg/kg b.w./day. In the rat developmental toxicity study conducted with WL 136184 technical, the NOAEL for maternal toxicity was 24 mg/kg b.w./day based on decreased body weight gain at 60 mg/kg b.w./day, the highest dose tested, and the NOAEL for developmental toxicity was also 24 mg/kg b.w./day, based on an increase in the total number of resorptions, reductions in fetal body weights and an increased incidence of skeletal ossification variations at 60 mg/kg b.w./day.

Results from a developmental toxicity study in rabbits with AC 900768 also indicated no evidence of teratogenicity or developmental toxicity in the absence of maternal toxicity. Thus, metconazole technical is neither a selective developmental toxicant nor a teratogen in the rabbit. In this rabbit developmental study, the NOAEL for maternal toxicity was 20 mg/kg b.w./day based on decreased food consumption and body weight gain, reductions in hemoglobin, hematocrit and corpuscular volume, increases in platelet counts and alkaline phosphatase activity, and increased absolute and relative liver weights at 40 mg/kg b.w./day (the highest dose tested). The NOAEL for developmental toxicity was also 20 mg/kg b.w./day, based on an increase in the total number and mean number of resorptions and decreased fetal body weight at 40 mg/kg b.w./day.

A 2-generation reproductive toxicity study in rats conducted with WL 136184 technical (greater than 95% cis isomer) is submitted in support of this tolerance petition. The results of the two-generation reproduction study with WL 136184 technical are sufficiently conservative for evaluating the potential reproductive toxicity of the 85:15 isomer mixture of metconazole technical. The results from the reproductive toxicity study with WL 136184 technical support a NOAEL for parental toxicity of 8 mg/kg b.w./day, based on increased ovarian weight and increased gestation length at the next highest dose tested (32 mg/kg b.w./day). The NOAEL for growth and development of the offspring is also 8 mg/kg b.w./day, based on reductions in live litter size for F_2 litters at 32 mg/kg b.w./day. The NOAEL for reproductive performance and fertility was 48 mg/kg b.w./day (the highest dose tested).

Results of the pilot and definitive reproduction studies and developmental toxicity studies conducted with AC 900768 technical and/or WL 136184 technical show no increased sensitivity to developing offspring as compared to parental animals, as comparable NOAELs were obtained for parental toxicity and growth and development of offspring.

4.*Subchronic toxicity*. Short-term (28-day) dietary toxicity studies in rats were conducted with AC 900768 and WL 136184 technical materials. In the 28-day study with AC 900768, the NOAEL was 100 ppm (approximately 9.6 mg/kg b.w./day), based on reductions in body weight, body weight gain, food consumption, and hemoglobin concentration for males, as well as increased absolute and relative liver weights, and increased incidences of hepatic fatty vacuolation and parenchymal hypertrophy for males and females at 1,000 ppm (the next highest concentration tested). Similar results were observed in the study conducted with WL 136184 technical. Based on these results, the NOAEL for WL 136184 is 300 ppm (approximately 28.5 mg/kg b.w./day), supported by decreased body weights and body weight gains and increased incidences of hepatic fatty vacuolation for males and females, increased absolute and adjusted liver weights for females, and decreased food consumption for males at 1,000 ppm (the next highest concentration tested).

In a 28-day dietary study in dogs conducted with AC 900768 technical (85:15 isomer mixture), the NOAEL was a dietary concentration of 1,000 ppm (approximately 38.6 mg/kg b.w./day), based on decreased food consumption, body weight losses, increased alkaline phosphatase activity, increased spleen and liver weights, and urinalysis changes for males and females, and increased absolute and relative thyroid gland weights for females at 7,000 ppm, the highest concentration tested.

Subchronic (90-day) dietary studies in rats were conducted with AC 900768 technical and WL 136184 technical. In the study conducted with AC 900768, the NOAEL was 100 ppm (approximately 6.8 mg/kg b.w./day) based on hepatic fatty vacuolation in males at 300 ppm, the next highest concentration tested. The NOAEL from the study conducted with WL 136184 technical was 450 ppm (approximately 30.9 mg/kg b.w./day) based on decreased food consumption, body weights, and body weight gains, clinical chemistry changes, increased absolute and adjusted liver weights, and histopathological changes in the liver and/or stomach for males and females, and decreased red blood cell parameters for females at 1,350 ppm, the highest concentration tested.

In a 90-day dietary study in mice conducted with AC 900768, the NOAEL was 30 ppm (approximately 5.5 mg/kg b.w./day), based on increased aspartate and alanine aminotransferase activities in males, increased absolute and relative weights of the liver and spleen of females, and increased incidences of hepatocelluar vacuolation and hypertrophy for males and females at 300 ppm, the next highest concentration tested.

A 90-day dietary study in beagle dogs with AC 900768 technical supports a NOAEL of 60 ppm (approximately 2.5 mg/kg b.w./day) based on decreased body weight gain and food consumption for females, and a slight increase in reticulocyte count for males at 600 ppm, the next highest concentration tested.

5.*Chronic toxicity*. Findings similar to those observed in the short-term subchronic studies were also apparent in the long-term dietary toxicity studies conducted in rats, dogs and mice. Long-term (104-weeks) administration of AC 900768 (85:15 isomer mixture) to rats supported a NOAEL for systemic toxicity of 100 ppm (approximately 4.8 mg/kg b.w./day), based on increased adjusted liver weight, and increased incidences of hepatocellular lipid vacuolation and centrilobular hypertrophy at interim sacrifice for males at 300 ppm, the next highest concentration tested. In a one-year dietary study in beagle dogs, the NOAEL was 300 ppm (approximately 11.1 mg/kg b.w./day), based on decreased body weight gain for males during weeks 1 to 13 and increased alkaline phosphatase activity for males and females at 1,000 ppm, the next highest concentration tested.

In a 104-week carcinogenicity study in rats conducted with AC 900768, the NOAEL for carcinogenicity was 1,000 ppm (approximately 50 mg/kg b.w./day), the highest concentration tested. In this study the NOAEL for chronic systemic toxicity was 100 ppm (approximately 5.6 mg/kg b.w./day), based on increased incidences of centrilobular hypertrophy and pigment disposition in the liver, and increased incidences of cortical vacuolation in the adrenal in males at 300 ppm, the next highest concentration tested.

A 91-week carcinogenicity study in mice with AC 900768 supports a NOAEL for non-neoplastic effects of 30 ppm (approximately 4.8 mg/kg b.w./day), based on increased white blood cell count for males, increased aspartate and alanine aminotransferase activities and increased absolute and adjusted liver weight for females, and microscopic changes in the liver, spleen and adrenal gland for males and females at 300 ppm (the next highest concentration tested). The NOAEL for carcinogenicity was 300 ppm (approximately 48.3 mg/kg b.w./day) based on increased incidences of hepatocellular adenomas in males and females and hepatocellular carcinomas in females at 1,000 ppm, the highest concentration tested. The increased incidences of hepatic adenomas and carcinomas at the highest concentration tested are considered to occur through promotional and non-genotoxic secondary mechanisms following toxicity and induction of mixed function oxidase in mice. Consequently, metconazole is not likely to be oncogenic in humans at the insignificant levels of exposure resulting from its use as a fungicide.

AC 900768 technical and WL 136184 technical are not genotoxic carcinogens, as supported by a battery of *in vitro* and *in vivo* mutagenicity tests, which cover all major genetic endpoints.

6.*Animal metabolism*. The rat metabolism studies indicate that the qualitative nature of the residues of metconazole in animals is adequately understood. In studies conducted with radiolabeled AC 900768 (85:15 isomer mixture) or radiolabeled WL 136184 (greater than 95% cis isomer) radioactivity was rapidly eliminated in urine and feces with 48 hours of dosing. Biliary excretion was shown to be a prominent route of elimination. At both high and low doses of AC 900768, male rats generally excreted statistically significantly lower amounts of radioactivity in the urine, and greater amounts of radioactivity in the feces,

compared to females. The pattern of metabolites detected was similar at high and low doses, and little or no parent compound was found in the feces or urine. Five days following oral dosing of AC 900768 at the higher level, low levels of radioactivity were detected in the majority of tissues analyzed; however higher concentrations of radioactivity were found in the adrenal glands, gastro-intestinal tract and liver. A comparison of radioactivity levels in the adrenal glands following oral administration of low and high doses indicates that uptake in the adrenal may be saturable. No differences in tissue levels were noted between males and females. Hen and goat metabolism studies are not required, because bananas are not used as significant feedstuff for poultry or cattle.

7.*Metabolite toxicology*. The metabolite CL 382390 was identified in the banana metabolism study at levels of less than 0.02 ppm or less than 2% of the total radioactive residue in whole bananas. This specific monohydroxylated metabolite was not confirmed in the rat metabolism studies; however, other monohydroxylated metabolites, including its stereo isomer were identified. In addition, CL 382390 was shown to have a low order of acute toxicity via the oral route with an LD_{50} value of greater than 5,000 mg/kg b.w. Another metabolite not identified in the rat metabolism studies, triazolylalanine, was found in the triazole-3,5-14C CL 900768 treated banana at less than 0.02 ppm or less than 2% of the total radioactive residue in whole bananas. Triazolylalanine has been shown to have a low order of acute toxicity by the oral route with an oral LD_{50} value of greater than 5,000 mg/kg [WHO/FAO Joint Meeting on Pesticide Residues (JMPR) review, 1989]. Thus, the parent metconazole is considered to be the only toxicologically significant residue in bananas.

8.*Endocrine disruption*. Collective organ weight data and histopathological findings from the two-generation rat reproductive study, as well as from the subchronic and chronic toxicity studies in three different animal species, demonstrate no apparent estrogenic effects or treatment-related effects of metconazole on the endocrine system.

C. Aggregate Exposure

1.*Dietary exposure*. The potential dietary exposure to metconazole has been calculated from the proposed tolerance for bananas. The very conservative chronic dietary exposure estimates for this crop assumes that 100 percent of all bananas were treated with metconazole and that all treated bananas contain metconazole residues at the tolerance level of 0.05 ppm.

2.*Food*. Using the assumptions discussed above, the Theoretical Maximum Residue Concentration (TMRC) values of metconazole were calculated for the U.S. general population and subgroups. Based on the proposed tolerance, the TMRC values for each group are:

- 0.0000142 mg/kg b.w./day for the general population;
- 0.0000461 mg/kg b.w./day for all infants;
- 0.0000473 mg/kg b.w./day for non-nursing infants;
- 0.0000407 mg/kg b.w./day for children 1 to 6 years of age; and
- 0.0000156 mg/kg b.w./day for children 7 to 12 years of age.

Potential exposure to residues of metconazole in food will be restricted to intake of bananas, dried bananas, and banana nectar.

3.*Drinking water*. The tolerance proposed in this petition is for a raw agricultural commodity imported into the United States. There are no approved uses for metconazole in the United States; therefore, the potential exposure to metconazole in drinking water is not relevant to this petition.

4.*Non-dietary exposure*. This petition is for a tolerance on an imported commodity. There is no approved use of metconazole in the United States. and none is being sought; therefore, the potential for non-dietary exposure to metconazole is not pertinent to this petition.

D. Cumulative Effects

Metconazole is a member of the triazole class of fungicides. Other members of this class are registered for use in the United States. Although metconazole and other triazoles may have similar fungicidal modes of action, there are no available data to determine whether metconazole has a common mechanism of mammalian toxicity with other triazoles or information on how to include this pesticide in a cumulative risk assessment. Therefore, for the purposes of this tolerance petition no assumption has been made with regard to cumulative exposure with other compounds having a common mode of action.

E. Safety Determination

1.*U.S. population*. The Reference Dose (RfD) represents the level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. The chronic toxicity studies in rats and mice are the most appropriate studies to assess chronic dietary risk. These studies support a NOAEL of 4.8 mg/kg b.w./day, as the most sensitive dose for the estimation of the RfD for metconazole in humans. Based on the presence of a complete database for reproductive and developmental toxicity, and in the absence of teratogenicity or selective developmental toxicity, the use of a 100-fold safety factor is warranted for this compound. Applying a safety factor of 100 to this NOAEL results in the RfD of 0.048 mg/kg b.w./day. The chronic dietary exposure of 0.0000142 mg/kg b.w./day for the general U.S. population will utilize only 0.03% of the RfD of 0.048 mg/kg b.w./day. EPA generally has no concern for exposures below 100% of the RfD. The complete and reliable toxicity data and the conservative chronic dietary exposure assumptions support the conclusion that there is a ''reasonable certainty of no harm'' from potential dietary exposure to residues of metconazole in bananas.

2.*Infants and children*. The conservative dietary exposure estimates previously presented will utilize 0.1 percent of the RfD for all infants and as well as for the non-nursing infant group, which is the most highly exposed population subgroup. The chronic dietary exposures for children 1 to 6 years of age will utilize only 0.08% of the RfD, while for children ages 7 to 12 the estimated exposure will utilize only 0.03% of the RfD. Results from the two-generation reproduction study in rats with WL 136184 (greater than 95% cis isomer) and the developmental toxicity studies with AC 900768 in rats and rabbits indicate no increased sensitivity to developing offspring when compared to parental toxicity. For both the rat and rabbit developmental toxicity studies, embryotoxicity was only observed at maternally toxic doses. These results indicate that metconazole is neither a selective developmental toxicant nor a teratogen in either the rat or rabbit. Therefore, an additional safety factor is not warranted, and the RfD of 0.048 mg/kg b.w./day, which utilizes a 100-fold safety factor is appropriate to ensure a reasonable certainty of no harm to infants and children.

F. International Tolerances

There are no Codex maximum residue levels established or proposed for residues of metconazole in bananas.

[FR Doc. 05–7064 Filed 4–7–05; 8:45 am]

BILLING CODE 6560–50–S