iv. *Pet treatment.* Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA with the conclusion that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

D. Cumulative Effects

Imidacloprid is a chloronicotinyl insecticide. At this time, EPA has not made a determination that imidacloprid and other substances that may have a common mechanism of toxicity would have cumulative effects. Therefore, for these tolerance petitions, it is assumed that imidacloprid does not have a common mechanism of toxicity with other substances and only the potential risks of imidacloprid in its aggregate exposure are considered.

E. Safety Determination

1. *U.S. population.* EPA has considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. These studies are discussed under section A (Toxicology Profile) above. The developmental toxicity data demonstrated no increased sensitivity of rats or rabbits to *in utero* exposure to imidacloprid. In addition, the multi-generation reproductive toxicity study did not identify any increased sensitivity of rats to *in utero* or post-natal exposure. Parental NOAELs were lower or equivalent to developmental or offspring NOAELs. 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.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different MOS will be safe for infants and children. MOS are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors (UF) in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard UF (usually 100 for combined inter-species and intra-species variability) and not the additional tenfold MOE/UF when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/UF.

Although developmental toxicity studies showed no increased sensitivity in fetuses as compared to maternal animals following *in utero* exposures in rats and rabbits, no increased sensitivity in pups as compared to adults was seen in the 2-generation reproduction toxicity study in rats, and the toxicology data base is complete as to core requirements, EPA has determined that the additional safety factor for the protection of infants and children will be retained but reduced to 3x based on the following weight-of-the-evidence considerations relating to potential sensitivity and completeness of the data:

i. There is concern for structure activity relationship. Imidacloprid, a chloronicotinyl compound, is an analog to nicotine and studies in the published literature suggests that nicotine, when administered causes developmental toxicity, including functional deficits, in animals and/or humans that are exposed *in utero*.

ii. There is evidence that imidacloprid administration causes neurotoxicity following a single oral dose in the acute study and alterations in brain weight in rats in the 2–year carcinogenicity study.

iii. The concern for structure activity relationship along with the evidence of neurotoxicity dictates the need of a developmental neurotoxicity study for assessment of potential alterations on functional development. Because a developmental neurotoxicity study potentially relates to both acute and chronic effects in both the mother and the fetus, EPA has applied the additional UF for FQPA for all population subgroups, and in both acute and chronic risk assessments.

Based on the exposure assessments described above and on the completeness and reliability of the toxicity data, it can be concluded that the dietary exposure estimates from all label and pending uses of imidacloprid are 7.73% of the aPAD at the 99.9th percentile and 1.4% of the cPAD for the U.S. population. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children.* Based on the exposure assessments described above for the safety determination of the U.S. population and on the completeness and reliability of the toxicity data, it can be concluded that the dietary exposure estimates from all label and pending uses of imidacloprid are 16.42% of the aPAD at the 99.9th percentile and 3.0% of the cPAD for the most sensitive population subgroup, children 1–6 years. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

F. International Tolerances

No CODEX Maximum Residue Levels have been established for residues of imidacloprid on any crops pending in EPA's 2003 work plan.

[FR Doc. 03–5034 Filed 3–4–03; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0047; FRL–7294–5]

Trifloxystrobin; 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–2003–0047, must be received on or before April 4, 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: Sidney Jackson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–7610; e-mail address: *jackson.sidney@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. To determine whether you or your business may be affected by this action, you should carefully examine the applicability provisions in OPP–2003–0047. 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–2003–0047. 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–2003–0047. 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–2003–0047. 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–2003–0047.

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–2003–0047. 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: February 21, 2003.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner's 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. The Interregional Research Project No. 4 (IR–4) assembled and submitted the petition to EPA in behalf of Bayer CropScience, the registrant.

Interregional Research Project Number 4 and Bayer CropScience

PP 3E6522

EPA has received a pesticide petition (3E6522) from Interregional Research Project Number 4 (IR–4), 681 U.S. Highway #1 South, North Brunswick, NJ 08902–3390 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR 180.555 by establishing a tolerance for residues of trifloxystrobin in or on the raw agricultural commodities (RACs) vegetable, root, except sugar beet, subgroup 1B, except radish at 0.1 part per million (ppm) and leafy petiole subgroup 4B at 2.0 ppm. EPA has determined that the petition contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The metabolism of trifloxystrobin in plants (cucumbers, apples, wheat, sugar beets, and peanuts), is well understood. Identified metabolic pathways are substantially similar in plants and animals (goat, rat, and hen). EPA has determined that trifloxystrobin parent and its metabolite CGA–321113 are the residues of concern for tolerance setting purposes.

2. *Analytical method.* A practical analytical methodology for detecting and measuring levels of trifloxystrobin in or on RACs has been submitted. The limit of detection (LOD) for each analyte of this method is 0.08 nanogram (ng) injected, and the limit of quantitation (LOQ) is 0.02 ppm. The method is based on crop specific cleanup procedures and determination by gas chromatography (GC) with nitrogen-phosphorus detection.

3. *Magnitude of residues*—i. *Vegetable root, except sugar beet, subgroup 1B, except radish.* Interregional Research Project Number 4 received a request from Michigan for the use of trifloxystrobin on carrots. Interregional Research Project Number 4 performed

10 field trials to support the requested tolerance of 0.1 ppm.

ii. *Leaf petiole subgroup 4B.* Interregional Research Project Number 4 received a request from Florida, Michigan, Oregon, California, and Ohio, for the use of trifloxystrobin on celery. Interregional Research Project Number 4 performed nine field trials to support the requested tolerance of 2.0 ppm.

B. Toxicological Profile

1. *Acute toxicity.* Studies conducted with the technical material of trifloxystrobin:

- *Rat.* Acute oral toxicity study with a lethal dose $(LD)_{50}$ >5,000 milligrams/kilogram (mg/kg).
- *Mouse.* Acute oral toxicity study with a LD_{50} >5,000 mg/kg.
- *Rabbit.* Acute dermal toxicity study with a LD_{50} >2,000 mg/kg.
- *Rat.* Acute dermal toxicity study with a LD_{50} >2,000 mg/kg.
- *Rat.* Acute inhalation toxicity study with a lethal concentration $(LC)_{50}$ >4.65 milligrams/liter (mg/L).
- *Rabbit.* Eye irritation study showing slight irritation (toxicity category III).
- *Rabbit.* Dermal irritation study showing slight irritation (toxicity category IV).
- *Guinea pig.* Dermal sensitization study with the Buehler's method showing negative findings.
- *Guinea pig.* Dermal sensitization study with the maximization method showing some positive findings.

2. *Genotoxicity.* No genotoxicity activity is expected of trifloxystrobin under *in vivo* or physiological conditions. The compound has been tested for its potential to induce gene mutation and chromosomal changes in five different test systems. The only positive finding was seen in the *in vitro* test system (chinese hamster V79 cells) as a slight increase in mutant frequency at a very narrow range (250 – 278 micrograms/milliliter (µg/ml) of cytologic and precipitating concentrations (compound solubility in water was reported to be 0.61 µg/ml; precipitate was visually noted in culture medium at 150 µg/ml). The chemical was found to be non-mutagenic in the *in vivo* system or all other *in vitro* systems. Consequently, the limited gene mutation activity in the V79 cell line is considered a nonspecific effect under non-physiological *in vitro* conditions and not indicative of a real mutagenic hazard.

3. *Reproductive and developmental toxicity.* FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. Based on the current toxicological data requirements, the data base on trifloxystrobin relative to prenatal and postnatal effects for children is complete.

In assessing the potential for additional sensitivity of infants and children to residues of trifloxystrobin, data were considered from teratogenicity studies in the rat, rabbit, and a 2–generation reproduction studies in the rat. The teratogenicity studies are designed to evaluate adverse effects on the developing embryo as a result of chemical exposure during the period of organogenesis. Reproduction studies provide information on effects from chemical exposure on the reproductive capability of mating animals and systemic and developmental toxicity from *in utero* exposure.

In the rat teratology study, reductions in body weight gain and food consumption were observed in the dam at ≥100 mg/kg. No teratogenic effects or any other effects were seen on pregnancy or fetal parameters except for the increased incidence of enlarged thymus, which is a type of variation, at 1,000 mg/kg. The developmental no observed adverse effect level (NOAEL) was 100 mg/kg.

In the rabbit teratology study, body weight loss and dramatically reduced food consumption were observed in the dam at ≥250 mg/kg. No teratogenic effects or any other effects were seen on pregnancy or fetal parameters except for the increase in skeletal anomaly of fused sternebrae–3 and –4 at the top dose level of 500 mg/kg. This finding is regarded as a marginal effect on skeletal development that could have resulted from the 40–65% lower food intake during treatment at this dose level. The developmental NOAEL was 250 mg/kg.

In the 2–generation rat reproduction study, body weight gain and food consumption were decreased at ≥750 ppm, especially in females during lactation. Consequently, the reduced pup weight gain during lactation (≥750 ppm) and the slight delay in eye opening (1,500 ppm) are judged to be a secondary effect of maternal toxicity. No other fetal effects or any reproductive changes were noted. The low developmental NOAEL, 50 ppm (5 mg/kg), seen in this study was probably due to the lack of intermediate dose levels between 50 and 750 ppm. Based on an evaluation of the dose-response relationship for pup weight at 750 ppm and 1,500 ppm, the NOAEL should have been nearly ten-fold higher if such a dose was available.

Based on all these teratology and reproduction studies, the lowest NOAEL for developmental toxicity is 5 mg/kg while the lowest NOAEL in the subchronic and chronic studies is 2.5 mg/kg/day (from the rat chronic study). Therefore, no additional sensitivity for infants and children to trifloxystrobin is suggested by the data base.

4. *Subchronic toxicity.* In subchronic studies, several mortality related changes were reported for the top dose in dogs (500 mg/kg) and rats (800 mg/kg). At these dose levels, excessive toxicity has resulted in body weight loss and mortality with the associated and nonspecific changes in several organs (such as atrophy in the thymus, pancreas, bone marrow, lymph node, and spleen) which are not considered specific target organs for the test compound.

In the dog, specific effects were limited to hepatocellular hypertrophy at ≥150 mg/kg and hyperplasia of the epithelium of the gall bladder at 500 mg/kg. Target organ effects in the rat were noted as hepatocellular hypertrophy (≥200 mg/kg) and the related liver weight increase (≥50 mg/kg). In the mouse, target organ effects included single cell necrosis (≥300 mg/kg) and hypertrophy (1,050 mg/kg) in the liver and extramedullary hematopoiesis (≥300 mg/kg) and hemosiderosis in the spleen (1,050 mg/kg).

In general, definitive target organ toxicity, mostly in the liver, was seen at high feeding levels of over 100 mg/kg for an extended treatment period. At the lowest observed adverse effect level (LOAEL), no serious toxicity was observed other than mostly non-specific effects including a reduction in body weight and food consumption or liver hypertrophy.

5. *Chronic toxicity.* The liver appears to be the major primary target organ based on the chronic studies conducted in mice, rats, and dogs. It was identified as a target organ in both the mouse and the dog studies with trifloxystrobin. However, no liver effect was seen in the chronic rat study which produced the lowest NOAEL of 2.5 mg/kg based on reduced body weight gain and food consumption seen at higher dose levels.

The compound did not cause any treatment-related increase in general tumor incidence, any elevated incidence of rare tumors, or shortened time to the development of palpable or rapidly lethal tumors in the 18–month mouse and the 24–month rat studies. Dosages in both studies were sufficient for identifying a cancer risk. In the absence of carcinogenicity, a reference dose

(RfD) approach is appropriate for quantitation of human risks.

6. *Animal metabolism*. Trifloxystrobin is moderately absorbed from the gastrointestinal tract of rats and is rapidly distributed. Subsequent to a single oral dose, the half life of elimination is about 2 days and excretion is primarily via bile. Trifloxystrobin is extensively metabolized by the rat into about 35 metabolites, but the primary actions are on the methyl ester (hydrolysis into an acid), the methoxyimino group (O-demethylation), and the methyl side chain (oxidation to a primary alcohol). Metabolism is dose dependent as it was almost complete at low doses but only about 60% complete at high doses.

In the goat, elimination of orally administered trifloxystrobin is primarily via the feces. The major residues were the parent compound and the acid metabolite (CGA–321113) plus its conjugates. In the hen, trifloxystrobin is found as the major compound in tissues and in the excreta, but hydroxylation of the trifluormethyl-phenyl moiety and other transformations, including methyl ester hydrolysis and demethylation of the methoxyimino group, are also seen. In conclusion, the major pathways of metabolism in the rat, goat, and hen are the same.

7. *Metabolite toxicology*. Metabolism of trifloxystrobin has been well characterized in plants, soil, and animals. In plants and soil, photolytically induced isomerization results in a few minor metabolites not seen in the rat; however, most of the applied materials remained as parent compound as shown in the apple and cucumber studies. All quantitatively major plant and/or soil metabolites were also seen in the rat. The toxicity of the major acid metabolite, CGA–321113 (formed by hydrolysis of the methyl ester), has been evaluated in cultured rat hepatocytes and found to be 20 times less cytologic than the parent compound. Additional toxicity studies were conducted for several minor metabolites seen uniquely in plants and/or soil. The studies indicate that these metabolites, including CGA–357261, CGA–373466, and NOA–414412, are not mutagenic to bacteria and are of low acute toxicity (LD_{50} >2,000 mg/kg).

In conclusion, the metabolism and toxicity profiles support the use of an analytical enforcement method that accounts for parent trifloxystrobin.

8. *Endocrine disruption*. CGA–279202 does not belong to a class of chemicals known for having adverse effects on the endocrine system. Developmental toxicity studies in rats, rabbits, and reproduction study in rats, gave no indication that CGA–279202 might have any effects on endocrine function related to development and reproduction. The subchronic and chronic studies also showed no evidence of a long-term effect related to the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. Assessments were conducted to evaluate potential risks due to chronic and acute dietary exposure of the U.S. population and selected population subgroups to residues of trifloxystrobin. These analyses cover all registered crops plus the crops of vegetable, root, except sugar beet, subgoup1B, except radish; and the leafy petiole subgroup 4B.

The dietary exposure evaluation model ((DEEM™) v.7.76 software) was used to estimate the chronic and acute dietary exposure. This software uses the food consumption data from the United States Department of Agriculture (USDA) Continuing Surveys of Food Intake by Individuals CSFII 1994–1998.

EPA established an acute population adjusted dose (aPAD) of 2.5 milligrams/kilogram/day (mg/kg/day) for acute dietary risk assessments based on a NOAEL of 250 milligrams/kilogram of body weight/day from a rabbit developmental toxicity study and an uncertainty factor (UF) of 100. For chronic dietary analyses, EPA established a chronic population adjusted dose (cPAD) of 0.038 based on a NOAEL of 3.8 from a rat reproductive toxicity study and UF of 100.

Bayer CropScience believes that 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 trifloxystrobin on currently registered uses plus the pending uses on vegetable root crops, except sugar beets, subgoup1B, except radish; and the leafy petiole subgroup 4B.

i. *Food*. Acute and chronic dietary exposure assessments were performed using tolerance values for all crops and assuming 100% crop treated. Acute exposure, expressed at the 95th percentile of exposure, was 0.59% of the aPAD for females 13 to 50 years old (only population subgroup of concern). The chronic exposure was 17.3% cPAD for the total U.S. population and 51.5% cPAD for the most sensitive population, children 1 to 6 years old.

ii. *Drinking water*. Using DEEM software, acute and chronic drinking water levels of concern (DWLOC) were calculated. The acute DWLOC was 74,560 and the chronic DWLOC was 1,100 for the total U.S. population and 184 for the most sensitive population subgroup, children 1 to 6 years old. These values are above the estimated concentrations of trifloxystrobin and its metabolites in drinking water as published in the **Federal Register** of May 22, 2002, (67 FR 35915–35924) (FRL–7178–6). Therefore, Bayer CropScience believe that there is reasonable certainty that exposure from trifloxystrobin will not result in harm to the adult U.S. population or infants and children.

2. *Non-dietary exposure*. As published in the **Federal Register** of May 22, 2002, (67 FR 35915–35924) (FRL–7178–6), EPA calculated post application exposure estimates and risk estimates for adults and children resulting from the use of trifloxystrobin on turf and recreational use sites.

The margin of exposure (MOE) that resulted were above 100 and all risks were considered below EPA's level of concern (LOC).

D. Cumulative Effects

EPA has determined that unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, trifloxystrobin does not appear to produce a toxic metabolite produced by other substances. Therefore, EPA has not assumed, as published in the **Federal Register** of May 22, 2002, (67 FR 35915–35924) (FRL–7178–6), that trifloxystrobin has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. Based on the information supplied under aggregate exposure described above, Bayer CropScience believe that there is reasonable certainty that exposure from trifloxystrobin will not result in harm to the adult U.S. population.

2. *Infants and children*. Based on the information supplied under aggregate exposure described above, Bayer CropScience believes that there is reasonable certainty that exposure from trifloxystrobin will not result in harm to infants and children.

F. International Tolerances

There are no codex, Canadian, or Mexican maximum residue limits (MRLs) established for trifloxystrobin.

[FR Doc. 03–5193 Filed 3–4–03; 8:45 am]

BILLING CODE 6560–50–S