exposures are expected for the general population.

*D. Cumulative Effects*

EPA does not have, at this time, available data to determine whether tralkoxydim has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Tralkoxydim is structurally a cyclohexanedione. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, tralkoxydim does not appear to produce a toxic metabolite produced by other substances. For the purposes of these tolerances action, therefore, EPA has not assumed that tralkoxydim has a common mechanism of toxicity with other substances.

*E. Safety Determination*

1. *U.S. population*—i. *Acute risk*. The acute dietary analysis based on the NOAEL of 30 mg/kg/day from the rat developmental study using the DEEM computer program estimates that the distribution of single-day exposures utilizes 0.02% of acute RfD. The drinking water level of comparisons (DWLOCs) for acute exposure to tralkoxydim in drinking water calculated for females 13 + years old was 9,000 ppb. The estimated average concentration in surface water for tralkoxydim is 9 ppb. EPA's acute DWLOC is well above the estimated exposures for tralkoxydim in water for the subgroup of concern. For ground water, the estimated environmental concentrations (EEC's) using the SCI-GROW model were all less than 1 ppb.

ii. *Chronic risk*. A DEEM chronic exposure analysis showed that exposure from tolerance level residues in or on wheat, and barley for children 1 to 6 years old (the subgroup with the highest exposure) would be 1.4% of the RfD. The exposure for the general U.S. population would be less than 1% of the RfD. The DWLOCs for chronic exposure to tralkoxydim in drinking water calculated for U.S. population was 150 ppb and for children (1 to 6 years old) the DWLOC was 50 ppb. The estimated average concentration in surface water for tralkoxydim is 9 ppb. EPA's chronic DWLOC is above the estimated exposures for tralkoxydim in water for the U.S. population and the subgroup of concern. Conservative model estimates SCI-GROW of the concentrations of tralkoxydim in ground water indicate that exposure will be minimal.

iii. *Cancer risk*. A DWLOC for cancer was calculated as 1 ppb. The estimated concentration in surface water and ground water for tralkoxydim for chronic exposure are 0.9 ppb, 2.8 ppb, (the 56–day concentration)/3, and 0.1 ppb, respectively. The model exposure estimates are less than the cancer DWLOC. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to tralkoxydim residues.

2. *Infants and children*. The Agency concluded that an extra safety factor to protect infants and children is not needed based on the following considerations: The toxicology data base is complete for the assessment of special sensitivity of infants and children. The developmental and reproductive toxicity data do not indicate increased susceptibility of rats or rabbits to *in utero* and/or postnatal exposure. The NOAEL used in deriving the RfD is based on changes in liver function and morphology in male adult dogs (not developmental or neurotoxic effects) after chronic exposure and thus are not relevant for enhanced sensitivity to infants and children. Unrefined dietary exposure estimates (assuming all commodities contain tolerance level residues) overestimate dietary exposure. Model data used for ground water and surface water source drinking water exposure assessments result in estimates considered to be upper-bound concentrations. There are no registered uses for tralkoxydim that could result in residential exposures. EPA concludes that there is a reasonable certainty that no harm will result to children from aggregate exposure to tralkoxydim residues.

*F. International Tolerances*

There are no codex Alimentarius Commission (Codex) or Mexican maximum residue levels for tralkoxydim at this time.

[FR Doc. 03–6823 Filed 3–20–03; 8:45 am]

**BILLING CODE 6560–50–S**

## ENVIRONMENTAL PROTECTION AGENCY

**[OPP–2003–0057; FRL–7296–6]**

## Trifloxysulfuron-sodium; 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–2003–0057, must be received on or before April 21, 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:** James A. Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5697; e-mail address: tompkins.jim@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–2003–0057. 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 on 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–2003–0057. 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–2003–0057. 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–2003–0057.

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–2003–0057. 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**.

*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: March 10, 2003.

**Debra Edwards,**

*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 Syngenta Crop Protection, Inc, 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.

**Syngenta Crop Protection, Inc.**

*PP 1F6280*

EPA has received a pesticide petition (1F6280) from Syngenta Crop Protection, Inc., Greensboro, NC 27419 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180, by establishing a tolerance for residues of trifloxysulfuron-sodium in or on the raw agricultural commodities sugarcane at 0.01 parts per million (ppm), cottonseed at 0.05 ppm, cotton by-products at 1.0 ppm, citrus at 0.01, almond hulls at 0.01 ppm, almond nut meat at 0.01 ppm, and tomatoes at 0.01 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

*A. Residue Chemistry*

1. *Plant metabolism.* The primary metabolic pathways of trifloxysulfuron-sodium in plants (cotton, sugarcane and citrus) were similar to those described for animals, with certain extensions of the pathway in plants. The metabolism of trifloxysulfuron-sodium is well characterized in plants and animals and the data is adequate for tolerance setting purposes.

The metabolism profile in plants and animals supports the use of an analytical enforcement method that accounts for parent trifloxysulfuron-sodium. The multiple other metabolites formed in plants and animals are considered of equal or lesser toxicity than parent compound.

2. *Analytical method.* Syngenta Crop Protection, Inc. has submitted practical analytical methodology for detecting and measuring levels of trifloxysulfuron-sodium in or on raw agricultural commodities. This method is based on crop specific cleanup procedures and determination by liquid chromatography with a ultraviolet (UV/Vis) detector. The limit of detection (LOD) for each analyte of this method is 2 nanograms of trifloxysulfuron-sodium. The limit of quantitation (LOQ), as demonstrated by acceptable recoveries from fortified control samples, is 0.01 ppm for each substrate.

3. *Magnitude of residues.* A residue program was performed with trifloxysulfuron-sodium on a full geography to support use on cotton, sugarcane, citrus, and almonds. Adequate residue trials were performed to support the proposed use on tomatoes.

*B. Toxicological Profile*

1. *Acute toxicity.* Trifloxysulfuron-sodium has low acute toxicity. The oral $LD_{50}$ in rats is >5,000 millgrams/kilogram (mg/kg) for males and females combined. The rat dermal $LD_{50}$ is >2,000 mg/kg and the rat inhalation $LC_{50}$ is >5.03 milligrams/liter (mg/L) air. Trifloxysulfuron-sodium is not a skin sensitizer in guinea pigs and is considered to have slight dermal or eye irritation in rabbits. End-use formulations of Trifloxysulfuron-sodium have similar low acute toxicity profiles.

2. *Genotoxicity.* Trifloxysulfuron-sodium has been tested for its potential to induce gene mutation and chromosomal changes in five different test systems. Trifloxysulfuron-sodium technical did not induce point mutations in bacteria (Ames assay in *Salmonella typhimurium* or *Escherichia coli*) or in cultured mammalian cells (Chinese hamster V79) and was not genotoxic in an *in-vitro* unscheduled DNA synthesis assay in rat hepatocytes. Chromosome aberrations were not observed in an *in-vitro* test using Chinese hamster ovary cells and there were no clastogenic or aneugenic effects on mouse bone marrow cell *in-vivo* in a mouse micronucleus test. These studies show that trifloxysulfuron-sodium is not genotoxic.

3. *Reproductive and developmental toxicity.* Data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat have been considered. In rabbit (0, 50, 100, 250, 500 mg/kg/day) and rat (0, 30, 300, 1,000 mg/kg/day) teratology studies there was no evidence of teratogenicity. Maternal toxicity was seen at 500 mg/kg/day and 250 mg/kg/day as evidenced by deaths and premature sacrifices. For the control (50, 100, and 250 mg/kg) groups, pre-implantation losses, number of implantation sites, and post-implantation losses were not affected by treatment. The findings after fetal post mortem examination and fetal visceral examination revealed no treatment related effects. Similarly, there were no skeletal malformations in this study and the incidence of anomalies and variations were not affected by treatment. In conclusion, the no observed adverse effect levels (NOAEL) for maternal toxicity was 100 mg/kg/day and the NOAEL for fetal toxicity was 250 mg/kg/day. There was no indication of embryotoxic, fetotoxic or teratogenic potential for trifloxysulfuron-sodium in rabbits.

In the rat teratology study, 300 and 1,000 mg/kg/day caused maternal toxicity consisting of reduced body weight and food consumption. Developmental toxicity was secondary to maternal toxicity and consisted of slightly reduced fetal body weights and an increase in minor skeletal anomalies and variations. The NOAELs for maternal and developmental toxicity were both 30 mg/kg/day. Trifloxysulfuron-sodium was not embryotoxic, fetotoxic or teratogenic in rats when tested under the conditions of this study.

In a rat multigeneration study, trifloxysulfuron-sodium technical was administered in feed at concentrations of 0, 500, 1,000, 8,000 or 12,000 ppm. The dose in mg/kg/day spans a wide range over the duration of the study as animals gain weight and go through gestation and lactation. The ranges are 24–70, 48–137, 400–1,133, 608–1,755 for males and 32–100, 60–199, 500–1,557, 792–2,374 for females at the 500, 1,000, 8,000, and 12,000 ppm dietary level, respectively.

Trifloxysulfuron-sodium had no effect on reproductive parameters. Parental body weight gain and food consumption were reduced at 12,000 ppm in both sexes and at 8,000 ppm in males only. In addition, there was an increased relative liver weight and an increased incidence of hepatocellular hypertrophy at 12,000 ppm in both sexes of adults and at 8,000 ppm in adult males only. Offspring body weights were reduced in males and females greater than or equal to 8,000 ppm.

In conclusion, the NOAEL for systemic toxicity in both sexes and both generations was 1,000 ppm. The mean dose in mg/kg/day for all weekly means for both sexes, both generations, all time points at this dietary level was 83.4 mg/kg/day. There were no effects on the reproductive parameters and the NOAEL for reproductive toxicity was >12,000 ppm. Offspring effects were observed only at dose levels that produced parental toxicity. Thus, there is no evidence that developing offspring are more sensitive than adults to the effects of trifloxysulfuron-sodium, and it is concluded, that trifloxysulfuron-sodium does not cause developmental or reproductive toxicity.

4. *Subchronic toxicity.* Trifloxysulfuron-sodium technical was evaluated in a number of subchronic studies. In a 3–month rat feeding study the NOAEL was 65.7 mg/kg with hematologic and liver effects noted. In a 3–month mouse feeding study, the NOAEL was 67.9 mg/kg. Effects seen were adaptive liver effects. In a 3–month feeding study in dogs the NOAEL was 19.6 mg/kg and hematopoietic and liver effects were seen. In a 28–day dermal (rat) study, the NOAEL was 100 mg/kg. In this study only body weight effects were noted, and only occurred at 1,000 mg/kg.

5. *Chronic toxicity.* Trifloxysulfuron-sodium technical was not oncogenic in rats or mice. In a 12–month feeding study in dogs fed diets containing trifloxysulfuron-sodium that resulted in average (sexes combined) daily test substance intakes of 0, 1.67, 6.71, 15.0, 48.2 or 122 mg/kg/day, all animals survived. In life observations, food consumption, eye and neurological examinations, and urine profiles were not affected by treatment. Macroscopic and microscopic examinations revealed no findings that were considered to be treatment related and indicative of systemic toxicity.

The body weight gain was decreased by 16% in males at 122 mg/kg/day. The 33% decrease at 48.2 mg/kg/day was mainly due to one male that gained significantly less weight than the other animals of this group. There was a tendency for a decrease in the erythrocyte count, hemoglobin concentration and hematocrit for both sexes at 122 mg/kg/day at the end of treatment, and for males throughout the treatment period. In female dogs treated with 48.2 and 122 mg/kg/day, the mean absolute and relative liver weights were increased, and a tendency for an increase in relative liver weight was noted for males at the same dose levels.

The maximum tolerance dose (MTD) was achieved at 122 mg/kg/day based on the decrease in the body weight gain in males at 48.2 and 122 mg/kg/day. Administration of trifloxysulfuron-sodium to dogs for 12 months caused a tendency for decrease in red blood cell parameters in both sexes at 122 mg/kg/day. There was neither histopathological nor functional evidence for compound related neurotoxicity. Based on the effects at 48.2 and 122 mg/kg/day, the NOAEL was established at 15.0 mg/kg/day for males and 14.9 mg/kg/day for females.

In an 18–month oncogenicity study, mice were fed diets containing trifloxysulfuron-sodium that resulted in average (sexes combined) daily test substance intakes of 0, 5.84, 24.3, 116, and 836 mg/kg/day. Treatment had no adverse effect on appearance or behavior. Survival in treated animals was comparable to controls. There were no effects on organ weights, and there were no macroscopic or microscopic findings indicative of treatment-related systemic toxicity. Trifloxysulfuron-sodium was not carcinogenic in the mouse. Body weight gain in females at 836 mg/kg/day was decreased by 21% compared to controls after 3 months and 16% after 18 months. Food consumption was decreased in this group by 8%. The MTD was achieved at 836 mg/kg/day based on a decrease in body weight gain of greater than 15% throughout the study. Trifloxysulfuron-sodium was not carcinogenic in the mouse. Based on the findings at 836 mg/kg/day, the NOAEL for chronic toxicity was established at 121 mg/kg/day for males and 112 mg/kg/day for females.

In a 2–year chronic toxicity and carcinogenicity study, rats were fed diets containing trifloxysulfuron-sodium that resulted in average (sexes combined) daily test substance intakes of 0, 2.08, 22.0, 91.0 or 464 mg/kg/day. Clinical signs, survival, eye examinations, blood chemistry, urinalysis, and water consumption were not adversely affected by treatment. Survival in high dose females was greater than 80%, than in controls of 60%. There were no treatment-related findings at the 12–month interim or terminal necropsy.

A treatment-related decrease in body weight gain (17% decrease compared to controls) was seen in both females and males at 464 mg/kg/day (10,000 ppm), which was considered to be the maximum tolerated dose (MTD). Overall food consumption was decreased by 6% in males or 9% in females at 464 mg/kg/day. At the interim and terminal

sacrifices, mean carcass weights were lower in males (9% and 13%, respectively) and females (17% and 12%, respectively) for the 464 mg/kg/day group. At terminal sacrifice, the testes to body weight ratio was increased by 19% in the 464 mg/kg/day group.

Microscopical examination revealed a non-dose responsive increase in the incidence of kidney tubular atrophy in the two top dose groups of female rats, and an increase in Leydig cell hyperplasia in high dose males only. Both treatment-related lesions occurred late in age/treatment, and were not seen in animals sacrificed in the initial year of the study. Neither lesion showed an increase in severity (only incidence) or a progression of the lesion. Both lesions are commonly seen in high incidence in aged control rats; 26% of control females showed renal tubular atrophy, and 22% of control males showed Leydig cell hyperplasia. The control incidence in 10 studies was less than 10%, suggesting that the animals in this study were particularly susceptible to this lesion. There were no data from other measured parameters in this study that suggest kidney or testis as target organs, therefore indicating that these lesions are high-dose, long-term effects.

In conclusion, the MTD was reached or exceeded at 464 mg/kg/day for the 2–year rat feeding study. The NOAEL in males was 82.6 mg/kg/day based on the increased incidence of Leydig cell hyperplasia, and 23.7 mg/kg/day in females based on the increased incidence of kidney tubular atrophy. There was no evidence of a carcinogenic effect after 2 years of treatment with trifloxysulfuron-sodium in rats.

6. *Animal metabolism*. Metabolism in rats proceeded primarily via three concurrent metabolic pathways (typical sulfonylurea chemistry: Oxidative o-demethylation, hydroxylation of the pyrimidine ring and Smiles rearrangement of the sulfonylurea. Hydrolysis of the sulfonylurea and oxidative O-demethylation are minor pathways in the rat. Parent compound was the major residue in the rat. The metabolite pattern in urine and feces extracts of dogs is similar to that of rats. Trifloxysulfuron-sodium was the major component detected in extracts of urine and feces for dogs, as in the rats. In hens and goats, the metabolite profile was very similar to that observed in the rat.

7. *Metabolite toxicology*. The metabolism profile for trifloxysulfuron-sodium supports the use of an analytical enforcement method that accounts for parent trifloxysulfuron-sodium. Other metabolites are considered of equal or lesser toxicity than parent compound.

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

9. *Neurotoxicity*. In an acute range finding neurotoxicity study in which rats received a single oral dose of 2,000 or 3,500 mg/kg trifloxysulfuron-sodium, there were no effects on clinical signs, body weight and food consumption, or parameters in an abbreviated functional observational battery (FOB). Therefore, the time to peak effect for FOB and motor activity testing was based on a blood kinetic study. In this study, trifloxysulfuron-sodium induced peak plasma levels at 1–2 hours post-dose, and levels were almost zero at 24 hours.

In an acute neurotoxicity study in rats, trifloxysulfuron-sodium was administered by gavage at 0 or 2,000 mg/kg. Mortality, body weight development and food consumption were not affected by treatment. Neither clinical signs nor changes in observation and functional test conducted as part of the FOB were observed. Reduced horizontal and vertical motor activity were observed in males and females only at the time of peak effect (1–2 hours post-dosing). There were no persistent signs of toxicity and no histopathological evidence of neurotoxicity.

In a second acute neurotoxicity study in rats, trifloxysulfuron- sodium was administered by gavage at 0, 200, 600 and 2,000 mg/kg. Mortality, body weight development and food consumption were not affected by treatment. There were no effects on clinical signs or on parameters in the FOB. During the peak plasma period (1–2 hours post-dosing), motor activity parameters of the males were comparable to the control while females tended to be slightly less active. Based on the results of this study, Trifloxysulfuron-sodium was devoid of neurotoxic effects. Due to the slightly reduced motor activity in top dose females, the NOAEL was established at 600 mg/kg.

In a 90–day subchronic neurotoxicity study in rats, trifloxysulfuron-sodium was not neurotoxic when administered in the diet for 13 weeks at concentrations resulting in average daily test substance intakes of 0, 112, 472, or 967 mg/kg/day for males or 0, 134, 553 or 1,128 mg/kg/day for females. There were no treatment-related deaths or clinical signs. Effects on body weight development and food consumption indicated systemic toxicity in males at doses 472 mg/kg/day and in females at 1,128 mg/kg/day. There were no treatment-related neurobehavioral or motor activity effects, no macroscopic findings, and no microscopic findings in central or peripheral nervous tissue.

In the absence of any functional or morphological changes in the nervous system at any of the dose levels tested, trifloxysulfuron-sodium is considered devoid of neurotoxic potential when administered to rats for 90 days. Based on body weight effects, the NOAEL was established at 112 mg/kg/day for male rats and 553 mg/kg/day for female rats.

*C. Aggregate Exposure*

1. *Dietary exposure.* Dietary exposure from trifloxysulfuron-sodium potentially exists through both food commodities and drinking water. Each exposure pathway is addressed below.

i. *Food*. Chronic dietary exposure to trifloxysulfuron-sodium was estimated based on proposed tolerance-based residue values and the assumption that 100% of all planted acres were treated. The assessment included cotton, processed cotton fractions, sugarcane and associated processed commodities, citrus, almonds and tomatoes. Chronic exposure for all populations was compared to a reference dose (RfD) of 0.15 milligrams/kilogram/body weight/day (mg/kg/bwt/day) based on a no observed adverse effect level (NOAEL) of 14.9 mg/kg/bwt/day from a 1–year study in dogs and a 100X uncertainty factor. The analysis was conducted using the dietary exposure evaluation model (DEEM™) and the USDA's 1994–96 Continuing Survey of Food Intake by Individuals (CSFII). Secondary residues in animal commodities were not considered in this evaluation since calculations showed that transfer from livestock and poultry was minimal and would result in residue levels significantly below current analytical method capabilities. Chronic exposure to trifloxysulfuron-sodium was found to be essentially zero with less than 0.1% of the RfD utilized for all populations. These exposure calculations are conservative in that 100% of the crop was assumed as treated and tolerance-based residue levels were entered into the dietary model.

Acute dietary assessments were conducted for trifloxysulfuron-sodium using proposed tolerance-based residue values and the assumption that 100% of all planted acres were treated. The assessment included cotton, processed cotton fractions, sugarcane and

associated processed commodities, citrus, almonds and tomatoes. Acute exposure to the female population (13–50 years old) was compared to a RfD of 0.30 mg/kg/bwt/day based on a NOAEL of 30 mg/kg/bwt/day from a rat teratology study and a 100X uncertainty factor. Acute exposure to the general population and all other population subgroups (including infants and children) was compared to a RfD of 6.0 mg/kg/bwt/day based on a NOAEL of 600 mg/kg/bwt/day from an acute neurotoxicity study in rats and a 100X uncertainty factor. The analyses were conducted using the Dietary Exposure Evaluation Model (DEEM™) from Novigen Sciences and the USDA's 1994–96 CSFII. Secondary residues in animal commodities were not considered in this evaluation since calculations showed that transfer from livestock and poultry was minimal and would result in residue levels significantly below current analytical method capabilities. The acute exposures are presented at the 99.9th percentile of exposure although the Agency accepts the 95th percentile when conservative Tier I estimates are made (tolerance-based residues and 100% crop treated assumptions). Even at the 99.9th percentile, exposure and subsequent risk was found to be 0.2% of the acute reference dose (aRfD) for the female population (13–19 years not pregnant or nursing) and essentially zero with less than 0.1% of the aRfD utilized for all other populations. These exposure calculations are conservative in that 100% of the crop was assumed as treated, and tolerance-based residue levels were entered into the dietary model.

ii. *Drinking water*. For chronic exposure in water, the estimated maximum concentrations of trifloxysulfuron-sodium in surface water at day 56/3 was 0.35 parts per billion (ppb) generic expected environmental concentration (GENEEC) (sugarcane) and 0.051 ppb in ground water (SCI-GROW) (turf). The chronic drinking water levels of concern (DWLOC) values were calculated and compared to these estimated water concentrations. From the chronic dietary exposure analysis, an exposure estimate of 0.000015 mg/kg/day was determined for the U.S. population and less than or equal to 0.000037 mg/kg/day for all subgroups. Using this information, chronic drinking water levels of concern ($DWLOC_{chronic}$) were calculated for trifloxysulfuron-sodium. The trifloxysulfuron-sodium estimated ground water (0.051 ppb) and surface water (0.35 ppb) concentrations do not exceed the calculated chronic DWLOC values (µg/L): 1,500 to 5,250). Therefore, trifloxysulfuron-sodium exposures would not exceed the exposure allowable by the chronic risk cup.

The estimated maximum proposed rates for the ''worst case'' estimation of the proposed use concentrations of trifloxysulfuron-sodium in surface water at Peak Day–0 was 2.56 ppb GENEEC (sugarcane) and 0.051 ppb in ground water (SCI-GROW) (turf). The acute DWLOC values were calculated and compared to these estimated water concentrations.

From the acute dietary exposure analysis, the lowest margin of exposure (MOE) from the use of trifloxysulfuron-sodium was at the 95th percentile for the U.S. population and all population subgroups. This indicates a food exposure of less than or equal to 0.00016 mg/kg/day for all populations. Based on the EPA's ''Interim Guidance for Conducting Drinking Water Exposure and Risk Assessments'' document (draft 12/2/97), acute drinking water levels of concern ($DWLOC_{acute}$) were calculated for trifloxysulfuron-sodium. The lowest acceptable MOE for any pesticide is 100. This value was used in the DWLOC calculations. Based on this analysis, trifloxysulfuron-sodium estimated surface water (2.56 ppb) and ground water (0.051 ppb) concentrations, for sugarcane, do not exceed the calculated acute DWLOC values (µg/L: 8997 to 209,965). Therefore, trifloxysulfuron-sodium exposures would not exceed the exposure allowable by the risk cup.

2. *Non-dietary exposure*. The acute MOE for children ingesting pesticide-treated turf exceeds 190 million. The risk estimate does not exceed the level of concern (MOE = 100), indicating there are no oral exposure concerns for children ingesting trifloxysulfuron-sodium-treated turf.

*D. Cumulative Effects*

The potential for cumulative effects of trifloxysulfuron-sodium and other substances that have a common mechanism of toxicity has also been considered. Trifloxysulfuron-sodium is a member of the class of herbicides designated as sulfonylureas. There is no reliable information to indicate that toxic effects produced by trifloxysulfuron-sodium would be cumulative with those of any other chemical including another pesticide. Therefore, Syngenta believes it is appropriate to consider only the potential risks of trifloxysulfuron-sodium in an aggregate risk assessment.

*E. Safety Determination*

1. *U.S. population*. In assessing the potential for additional sensitivity of infants and children to residues of trifloxysulfuron-sodium, data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat have been considered.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of trifloxysulfuron-sodium, data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat have been considered. In rabbit (0, 50, 100, 250, 500 mg/kg/day) and rat (0, 30, 300, 1,000 mg/kg/day) teratology studies there was no evidence of teratogenicity. Delayed fetal development was apparent only at maternally toxic doses of trifloxysulfuron-sodium technical in rats. In rabbits, 500 mg/kg/day was clearly toxic to does and at 250 mg/kg/day,lesser toxicity was seen. For the control (50, 100, and 250 mg/kg) groups, pre-implantation losses, number of implantation sites, and post-implantation losses were not affected by treatment. The findings after fetal post mortem examination and fetal visceral examination revealed no treatment related effects. Similarly, there were no skeletal malformations in this study and the incidence of anomalies and variations were not affected by treatment. The no observed adverse effect levels (NOAEL) for maternal toxicity was 100 mg/kg/day and the NOAEL for fetal toxicity was 250 mg/kg/day. There was no indication of embryotoxic, fetotoxic, or teratogenic potential for trifloxysulfuron-sodium in rabbits.

In the rat teratology study developmental toxicity was secondary to maternal toxicity and consisted of slightly reduced fetal body weights and an increase in minor skeletal anomalies and variations. The NOAELs for maternal and developmental toxicity were both 30 mg/kg/day. Trifloxysulfuron-sodium was not embryotoxic, fetotoxic, or teratogenic in rats when tested under the conditions of this study.

In a rat multigeneration study, trifloxysulfuron-sodium had no effect on reproductive parameters. The NOAEL for systemic toxicity in both sexes and both generations was 1,000 ppm. The mean dose in mg/kg/day for all weekly means for both sexes, both generations, all time points at this dietary level was 83.4 mg/kg/day. There were no effects on the reproductive parameters and the NOAEL for

reproductive toxicity was > 12,000 ppm. Offspring effects were not observed at dose levels that did not produce parental toxicity. There is no evidence that developing offspring are more sensitive than adults to the effects of trifloxysulfuron-sodium.

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 requirements, the data base for trifloxysulfuron-sodium relative to prenatal and postnatal effects for children is complete. Further, for trifloxysulfuron-sodium, the developmental studies showed no increased sensitivity in fetuses as compared to maternal animals following *in-utero* exposures in rats and rabbits, and no increased sensitivity in pups as compared to the adults in the multi-generation reproductive toxicity study. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that a RfD of 0.15 mg/kg/day is appropriate for assessing aggregate risk to infants and children of trifloxysulfuron-sodium.

Assuming tolerance level residues and 100% of crops treated, less than 0.1% of the trifloxysulfuron-sodium chronic RfD is utilized in the population subgroup all infants (>1 year old). Therefore, based on the completeness and reliability of the toxicity data base, Syngenta concludes that there is reasonable certainty that no harm will result to infants and children from aggregate exposure to trifloxysulfuron-sodium residues.

*F. International Tolerances*

There are no Codex MRLs established for residues of trifloxysulfuron-sodium on cottonseed, cotton byproducts, citrus, almonds, sugarcane or tomatoes.

[FR Doc. 03–6822 Filed 3–20–03; 8:45 am]

**BILLING CODE 6560–50–S**

## ENVIRONMENTAL PROTECTION AGENCY

**[FRL–7471–2]**

### Strategic Plan for North American Cooperation in the Conservation of Biodiversity—Draft for Public Review

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

**ACTION:** Notice of availability.

**SUMMARY:** Comments are requested on the final draft of the *Strategic Plan for North American Cooperation in the Conservation of Biodiversity* (Strategic Plan). The Strategic Plan has been prepared by the Secretariat of the Commission for Environmental Cooperation (CEC), under the North American Agreement on Environmental Cooperation, in coordination with representatives from Canada, Mexico, and the United States. The Strategic Plan will be used to guide the CEC Council, its Biodiversity Conservation Working Group, and the CEC Secretariat in their work with stakeholders in cooperatively defining and implementing mutually beneficial biodiversity conservation activities in North America. Comments will be categorized and responses will be developed for each category. Responses to comment categories will be published in the **Federal Register** within 45 days of the closing date for comments. Changes to the final draft of the Strategic Plan, to be made in response to comments, will be discussed with representatives from Canada, Mexico and the CEC Secretariat.

**DATES:** Written comments will be accepted for 30 calendar days. Please submit or postmark written comments on the final draft document by April 21, 2003.

**ADDRESSES:** Comments should be sent to Patrick Cotter, Office of International Affairs (2260R), U.S. Environmental Protection Agency, and 1300 Pennsylvania Avenue, NW., Washington, DC 20004. Faxed comments should be sent to Patrick Cotter at (202) 565–2409. Comments can also be sent by email to *Cotter.Patrick@epa.gov.*

*Access to the Document:* The complete text of the final draft document, in English, is available through a link on the EPA Office of International Affair's Web site at: *http://www.epa.gov/international/trade/index.html,* or you may access the document directly on the CEC's Web site at: *http://www.cec.org/pubs_docs/documents/index.cfm?varlan=english&ID=1088.* Copies of the final draft document can be obtained in electronic or hard copy format by request from Patrick Cotter at the above mailing address, email address or by calling (202) 564–6414.

**FOR FURTHER INFORMATION CONTACT:** Patrick Cotter by telephone at (202) 564–6414 or by email at *Cotter.Patrick@epa.gov.*

Dated: March 17, 2003.

**Dona M. Harris,**

*Acting Director, Office of Management Operations, Office of International Affairs.*

[FR Doc. 03–6818 Filed 3–20–03; 8:45 am]

**BILLING CODE 6560–50–P**

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### Food and Drug Administration

**[Docket No. 02N–0281]**

### Agency Information Collection Activities; Announcement of OMB Approval; General Administrative Procedures: Citizen Petitions; Petition for Reconsideration or Stay of Action; Advisory Opinions

**AGENCY:** Food and Drug Administration, HHS.

**ACTION:** Notice.

**SUMMARY:** The Food and Drug Administration (FDA) is announcing that a collection of information entitled ''General Administrative Procedures: Citizen Petitions; Petition for Reconsideration or Stay of Action; Advisory Opinions'' has been approved by the Office of Management and Budget (OMB) under the Paperwork Reduction Act of 1995.

**FOR FURTHER INFORMATION CONTACT:** JonnaLynn P. Capezzuto, Office of Information Resources Management (HFA–250), Food and Drug Administration, 5600 Fishers Lane, Rockville, MD 20857, 301–827–4659.

**SUPPLEMENTARY INFORMATION:** In the **Federal Register** of December 18, 2002 (67 FR 77498), the agency announced that the proposed information collection had been submitted to OMB for review and clearance under 44 U.S.C. 3507. An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. OMB has now approved the information collection and has assigned OMB control number 0910–0183. The approval expires on March 31, 2006. A copy of the supporting statement for this information collection is available on the Internet at http://www.fda.gov/ohrms/dockets.

Dated: March 14, 2003.

**William K. Hubbard,**

*Associate Commissioner for Policy and Planning.*

[FR Doc. 03–6739 Filed 3–20–03; 8:45 am]

**BILLING CODE 4160–01–S**