EPA's level of concern for acute dietary exposure.

Regarding dietary cancer risk assessment, EPA's Cancer Peer Review Committee has classified 2,4-D as a Group D chemical (''not classifiable as to human carcinogenicity'') on the basis that, ''the evidence is inadequate and cannot be interpreted as showing either the presence or absence of a carcinogenic effect.''

2. *Infants and children*. The data base on 2,4-D relative to pre-and post-natal toxicity is complete with respect to current data requirements. Since the developmental NOELs for rats and rabbits are 25–fold greater and 90–fold greater, respectively, than the RfD NOEL of 1 mg/kg/day in the one–year oral toxicity study in dogs, an additional uncertainty factor to protect infants and children is not warranted.

Using conservative EPA calculations underlying the most recent final rule establishing tolerances for 2,4-D cited above, which included soybeans and all other existing uses, aggregate acute MOEs for exposure to 2,4-D from food are 214 for infants less than 1–year old and 399 for females 13 and older. The maximum estimated concentrations of 2,4-D in surface and ground water are less than EPA's Drinking Water Level of Comparison (DWLOC) figures for 2,4-D as a contribution to acute aggregate exposure. EPA concluded with reasonable certainty that residues of 2,4-D in drinking water do not contribute significantly to the aggregate acute human health risk.

Using the same conservative assumptions described earlier to estimate chronic risk from aggregate chronic exposure to 2,4-D from food, 11.4% of the reference dose (RfD) is utilized for nursing infants less than one year old up to 49.2% of the RfD for non-nursing infants less than one–year old. Further refinement using additional anticipated residue values in crops and percent crop-treated information would result in lower chronic dietary (food) exposure estimates, thus reducing the aggregate risk estimate. Despite the potential for exposure to 2,4-D in drinking water and from non-dietary, non-occupational exposure, EPA concluded that, it did not expect the aggregate exposure to exceed 100% of the RfD.

F. International Tolerances

There are no Codex, Canadian, or Mexican maximum residue limits (MRLs) for use of 2,4-D on hops.

[FR Doc. 05–7224 Filed 4–12–05; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2005–0047; FRL–7699–9]

Etoxazole; 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–2005–0047, must be received on or before May 13, 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: Kable Bo Davis, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 306–0415; e-mail address: *davis.kable@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you 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–2005–0047. 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, 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. 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–2005–0047. 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–2005–0047. 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–2005–0047.

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, 1801 S. Bell St., Arlington, VA, Attention: Docket ID number OPP–2005–0047. 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: April 1, 2005.

Lois Rossi,

Director, Registration Division, Office of PesticidePrograms.

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 Valent U.S.A. Corporation 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.

Valent U.S.A. Corporation

PP 3F6739

EPA has received a pesticide petition PP 3F6739 from Valent U.S.A. Corporation, 1333 North California Boulevard, Suite 600, Walnut Creek, CA 94596–8025 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 the chemical etoxazole, 2-(2,6-difluorophenyl)-4-[4-(1,1-dimethylethyl)-2-ethoxyphenyl]-4,5-dihydrooxazole, in or on the raw agricultural commodities nut, tree (Crop Group 14), including pistachios at 0.01 parts per million (ppm), almond, hulls at 2.0 ppm, grapes at 0.5 ppm, and raisins at 1.5 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 metabolism of etoxazole is adequately understood for the purpose of the proposed tolerances.

2. *Analytical methods.* Practical analytical methods for detecting and measuring levels of etoxazole have been developed and validated in/on all appropriate agricultural commodities and respective processing fractions. The extraction methodology has been validated using aged radiochemical residue samples from 14_C-metabolism studies. The enforcement methods have been validated in cottonseed, cotton gin trash, and in fresh mandarin oranges at independent laboratories. The LOQ of etoxazole in these methods is 0.01 ppm in grapes and nutmeats and 0.05 ppm in almond, hulls, which will allow monitoring of food with residues at the levels proposed for the tolerances.

3. *Magnitude of residues.* An extensive crop residue program has been conducted for etoxazole in all major growing regions of the United States for the following crops: Almond and pecans (representing nut, tree, Crop Group 14), and grapes. The results of these studies can be summarized as follows:

- For almonds, the maximum etoxazole residues from two applications at 0.135 pounds active ingredient/acre/treatment, was 0.005 ppm for nutmeats and 1.79 ppm for hulls harvested 28–days after application. Almond hulls were also analyzed for R–3, a metabolite of etoxazole. The maximum residue of R–3 was as 0.12 ppm.
- For pecans, no etoxazole residues were observed in nutmeats (LOD = 0.005 ppm) treated twice at 0.135 pounds active ingredient/acre/treatment and harvested 28–days after application.
- The maximum etoxazole residue in grapes harvested 28–days following the last of two treatments at 0.135 pounds active ingredient/acre/treatment was 0.33 ppm.
- The results of a grape processing study indicate that etoxazole residues concentrate in both grape juice and raisins. The concentration factor for grape juice was determined in this study to be 5.3X, which exceeds the theoretical concentration factor of 1.2X. Using this theoretical concentration factor to estimate the tolerance for juice, a tolerance of 0.32 ppm was calculated. Since this tolerance is less than the tolerance proposed for grapes, grape juice tolerances are not required. The concentration factor for raisins was determined in this study to be 3.5X. The theoretical concentration factor for raisins is, however, 4.7x. To be consistent with the grape juice calculations, this theoretical concentration factor was used to determine the proposed tolerance for raisins.

These field trial data are adequate to support proposed tolerances of 0.01 ppm for nut, tree (Crop Group 14); pistachios at 0.01 ppm; 2.0 ppm for almond, hull; 0.5 ppm for grapes; and 1.5 ppm for raisins.

Almond, hull is the only commodity under consideration that is a significant feed item for beef and dairy cattle. Tolerances of 0.03 ppm in the fat of animals and 0.04 ppm in milk fat, previously proposed and pending at the Agency, are adequate to support the use on almonds.

None of the commodities under consideration are used as poultry feed items. Additionally, the results of a hen metabolism study demonstrated very low potential for residues in feed to transfer to poultry tissues or eggs. Therefore, no hen residue feeding study was performed and tolerances are not proposed for secondary residues in poultry commodities.

B. Toxicological Profile

A full battery of toxicology testing, including studies of acute, chronic, oncogenicity, developmental, mutagenicity, and reproductive effects has been completed for etoxazole. The acute toxicity of etoxazole is low by all routes. Etoxazole is not a developmental or reproductive toxicant, and is not mutagenic or oncogenic. For the purpose of dietary risk analysis, Valent proposes 0.04 milligrams/kilogram body weight/day (mg/kg bwt/day) as the chronic Population Adjusted Dose (cPAD) and 2 mg/kg bwt/day as the acute Population Adjusted Dose (aPAD). The cPAD is based on a chronic endpoint of 4 mg/kg bwt/day no observed adverse effect level (NOAEL) for males from the rat chronic/oncogenicity feeding study and an uncertainty factor of 100. The aPAD is based on the 200 mg/kg bwt/day NOAEL from the rabbit developmental toxicity study and an uncertainty factor of 100. Valent is unable to identify toxicity endpoints of concern for acute, short-term or chronic human exposures by any route other than oral.

1. *Acute toxicity.* The acute toxicity of technical grade etoxazole is low by all routes. The battery of acute toxicity studies place etoxazole in Toxicity Category III. The oral LD_{50} in the rat was greater than 5 grams/kilogram (g/kg), the dermal LD_{50} was greater than 2.0 g/kg, and the inhalation LC_{50} in the rat was greater than 1.09 milligrams/liter (mg/L). Etoxazole technical was not an irritant to eyes or skin and was not a skin sensitizer.

2. *Genotoxicity.* Etoxazole was evaluated and found to be negative in an Ames reverse mutation assay, a chromosome aberration assay, a micronucleus assay, and an unscheduled DNA synthesis (UDS) assay. Etoxazole produced a positive result in the mouse lymphoma gene mutation assay but only in the presence of metabolic activation. Etoxazole does not present a genetic hazard.

3. *Reproductive and developmental toxicity*—i. *Rat developmental study.* Etoxazole did not produce developmental toxicity in rats. Etoxazole technical was administered by oral gavage to pregnant rats at dosage levels of 40, 200, and 1,000 mg/kg/day on days 6 through 15 of gestation. There were no mortalities or treatment-related adverse effects in any dose group. Food consumption was slightly decreased in dams during the dosing period for the 1,000 mg/kg/day group. On cesarean section evaluation there was no differences in number of corpora lutea, number of live and dead fetuses, percent resorption, placental weight, fetal weight or sex ratio in the dams and no treatment-related external, visceral or skeletal malformations noted in any of the fetuses. It was concluded that, the maternal no observed adverse effect Level (NOAEL) was 200 mg/kg/day, based on decreased food consumption at 1,000 mg/kg/day. The developmental NOAEL was 1,000 mg/kg/day, the highest dose tested (HDT).

ii. *Rabbit developmental study.* Etoxazole did not produce developmental toxicity in rabbits. Etoxazole technical was administered by oral gavage to pregnant rabbits at dosage levels of 40, 200, and 1,000 mg/kg/day on days 6 through 18 of gestation. No treatment-related adverse effects were found on maternal rabbits in the 40 and 200 mg/kg/day groups. One high dose rabbit died but it is unclear whether this death was attributed to treatment. Decreased body weight, body weight gain, food consumption and enlarged liver were noted at 1,000 mg/kg/day. Cesarean section findings showed that there was no differences in number of corpora lutea, number of live and dead fetuses, percent resorptions, placental weight, fetal weight and sex ratio in the dams and showed no treatment-related malformations (external, visceral, skeletal) in any of the fetuses. A statistically significant increased incidence of 27 presacral vertebrae with 13th ribs was observed in fetuses at 1,000 mg/kg/day compared with controls. This finding was within historical control range for fetal incidence but above the historical control range for litter incidence. No dose response was evident and the variation is considered to be equivocally treatment related. The NOAEL for maternal and developmental toxicity was 200 mg/kg/day based on decreased body weight and body weight gain, decreased food consumption, and liver enlargement at 1,000 mg/kg/day. The NOAEL for developmental toxicity was 200 mg/kg/day based on statistically significant increased incidence of 27 presacral vertebrae with 13th ribs in fetuses at 1,000 mg/kg/day.

iii. *Rat reproduction study.* Etoxazole showed no effects on reproduction in a two-generation rat study. Etoxazole technical was fed to two generations of male and female Sprague Dawley rats at dietary concentrations of 80, 400, and 2,000 ppm. No treatment-related adverse effects were observed in the 80 and 400 ppm groups for any parameter. In the 2,000 ppm group, relative liver weights were increased in the F0 and F1 parental males. No adverse reproductive effects were noted at any dose level in the incidence of normal estrous cycle, mating index, fertility and gestation indices, the number of implantation sites, and duration of gestation in F0 and F1 parental animals. For the offspring, it was noted that at 2,000 ppm, the viability index on lactation Day 4 was significantly lower in the F1 pups and body weights were lowered in pups during the latter half of the lactation period. For the F0 and F1 pups of the 80 and 400 ppm groups, there were no treatment-related adverse effects observed for any parameter, i.e. mean number of pups delivered, sex ratio, viability indices on lactation days 0, 4 and 21, clinical signs, body weights and gross pathological findings. The parental NOAEL was 400 ppm (17.0 mg/kg/day) based on the effects on relative liver weight in males at 2,000 ppm. The pup NOAEL was 400 ppm (37.9 mg/kg/day) based on decreased viability on lactation Day 4 and decreased body weight at 2,000 ppm in the F1 pups. The reproductive NOAEL was 2,000 ppm (86.4 mg/kg/day), the (HDT).

4. *Subchronic toxicity.* Subchronic toxicity studies conducted with etoxazole technical in the rat (oral and dermal), mouse and dog indicate a low level of toxicity. Effects observed at high dose levels consisted primarily of anemia and histological changes in the adrenal gland, liver and kidneys.

i. *Rat feeding study.* A 90–day subchronic toxicity study was conducted in rats, with dietary intake levels of 100, 300, 1,000 and 3,000 ppm etoxazole technical. The NOAEL was 100 ppm for males and 300 ppm for females based on increased incidence of hepatocellular swelling at 1,000 ppm and 3,000 ppm.

ii. *Mouse feeding study.* A 90–day subchronic toxicity study was conducted in mice, with dietary intake levels of 100, 400, 1,600, and 6,400 ppm etoxazole technical. The NOAEL was 400 ppm for males and 1,600 ppm for females based on increased alkaline phosphatase activity, increased liver weights, and increased incidence of hepatocellular swelling at 6,400 ppm (both sexes) and at 1,600 ppm in males and enlarged livers in females at 6,400 ppm.

iii. *Dog feeding study.* Etoxazole technical was fed to male and female Beagle dogs for 13 weeks at dietary concentrations of 200, 2,000, and 10,000 ppm. The NOAEL was 200 ppm (5.3 mg/kg/day) based on clinical signs, clinical pathology changes, liver weight effects and histopathological changes at 2,000 and 10,000 ppm.

iv. *Repeated dose dermal study.* A 28–day dermal toxicity study was conducted in rats at dose levels of 30, 100, and 1,000 mg/kg. There were no treatment related changes in any of the parameters monitored. The NOAEL was 1,000 mg/kg, the (HDT).

5. *Chronic toxicity.* Etoxazole technical has been tested in chronic studies with dogs, rats and mice. Valent proposes a chronic oral endpoint of 4 mg/kg bwt/day, based on the NOAEL for male rats in a 2–year chronic toxicity oncogenicity feeding study.

i. *Dog chronic feeding study.* Etoxazole technical was fed to male and female beagle dogs for one year at dietary concentrations of 200, 1,000, and 5,000 ppm. The NOAEL was 200 ppm (4.6 mg/kg/day for males and 4.79 mg/kg/day for females) based on increased absolute and relative liver weights with corresponding histopathological changes in the liver at 1,000 and 5,000 ppm.

ii. *Rat chronic feeding/oncogenicity study.* Etoxazole was not oncogenic in rats in either of two chronic feeding studies conducted. In the first study, etoxazole technical was fed to male and female Sprague Dawley rats for 2–years at dietary concentrations of 4, 16, and 64 mg/kg/day. A trend toward decreased

body weight gain for males at 64 mg/kg/day in the latter half of the study was observed. Hemotology and clinical chemistry changes, increased liver weights and hepatic enlargement at 16 mg/kg/day or above were observed. Testicular masses, centrilobular hepatocellular swelling and testicular interstitial (Leydig) cell tumors occurred at or above 16 mg/kg/day. The interstitial (Leydig) cell tumors were believed to be incidental. The NOAEL was 4 mg/kg/day for males and 16 mg/kg/day for females. Because an MTD level was not achieved in this study, a second study was conducted in which etoxazole technical was fed to male and female Sprague Dawley rats for 2–years at dietary concentrations of 50, 5,000, and 10,000 ppm. In this study, decreased mortality, body weight and food consumption/ efficiency (females) at 10,000 ppm was observed. Hematological, clinical, and histopathological changes of the incisors, and increased liver weights occurred in both sexes at 5,000 and 10,000 ppm. Centrilobular hepatocellular hypertrophy was observed in both sexes at 10,000 ppm. The interstitial (Leydig) cell tumors observed in the first study, were not observed in the repeat study. The NOAEL in the repeat study was 50 ppm (1.8 mg/kg/day).

iii. *Mouse oncogenicity study.* Etoxazole was not oncogenic in either of 2 mouse oncogenicity studies conducted. In the first study, etoxazole technical was fed to male and female CD–1 mice for 18–months at dietary concentrations of 15, 60, and 240 mg/kg/day. Increased liver weights occurred in females at the highest dose tested. Histopathology parameters were altered for males at 240 mg/kg/day. No neoplastic lesions were observed at any dose level. The NOAEL was 60 mg/kg/day. Since the toxicity in this study was minimal and did not meet the definition of MTD, a second study was conducted at dose levels of 2,250 and 4,500 ppm etoxazole. There were no effects in any group on clinical observations, mortality, body weight, food consumption or hematology. Females showed a significant elevation in relative liver weight after 52–weeks of treatment at 4,500 ppm. In histopathology, a significantly higher incidence of centrilobular hepatocellular fatty change was observed in males in the 4,500 ppm group necropsied after 78–weeks of treatment. There were no treatment-related changes in either sex in the 2,250 ppm dose group. No increase in neoplastic lesions were observed in any treated group of either sex. Therefore, it was concluded that, the NOAEL is 2,250 ppm (242 mg/kg/day for the males and 243 mg/kg/day for the females).

6. *Animal metabolism.* The absorption, tissue distribution, metabolism and excretion of etoxazole were studied in rats after single oral doses of 5 or 500 mg/kg, and after 14 daily oral doses at 5 mg/kg. Etoxazole, labeled in both the t-butylphenyl ring and the oxazole ring were used in this study. For both single dose groups, most (94–97%) of the administered radiolabel was excreted in the urine and feces within 7–days after dosing. Most of this excretion occurred in the first 48 hours after dosing. Maximum plasma concentrations occurred 2–4 hours after dosing, with half-lives ranging from 53–89 hours at the low dose and 7–44 hours at the high dose. Plasma levels were significantly lower in females. Concentrations of radioactivity were significantly higher in the tissues of male rats compared to females. The highest concentrations occurred at 3 hours after dosing and were greatest in the gastrointestinal tract and tissues such as liver and kidneys, which are responsible for metabolism and excretion. By 168 hours, the concentration in most tissues was below the concentration in the corresponding plasma, with only the liver and fat having significant levels of radioactivity. After multiple doses, peak concentrations of radioactivity in tissues occurred 2 hours after dosing and then declined. The distribution of radioactivity showed a similar profile to those found after single oral doses but were significantly higher, indicating some accumulation. Etoxazole was extensively metabolized by rats. The main metabolic reactions in rats were postulated to be hydroxylation of the 4,5-hydrooxazole ring followed by cleavage of the molecule and hydroxylation of the t-butyl side chain.

7. *Metabolite toxicology.* In an oral toxicity limit test in rats, the oral LD_{50} of metabolite R–3 was estimated to be greater than 5 g/kg for both male and female rats. No treatment related body weight changes and no treatment related macroscopic abnormalities were observed in this study. In another test, the oral toxicity of metabolite R–7 (as the HCl salt) was assessed. The oral LD_{50} of this metabolite was also estimated to be greater than 5 g/kg for both male and female rats. No treatment related macroscopic abnormalities were observed in this test, although, some clinical signs were observed within 6–minutes of dosing. Mutagenicity screens were performed with metabolite R–3 and metabolite R–7 (as the HCl salt). Neither metabolite was mutagenic when tested with multiple strains of two bacterial cultures (*salmonella typhimurium and e coli*).

8. *Endocrine disruption.* No special studies to investigate the potential for estrogenic or other endocrine effects of etoxazole have been performed. However, as summarized above, a large and detailed toxicology data base exists for the compound including studies in all required categories. These studies include acute, sub-chronic, chronic, developmental, and reproductive toxicology studies including detailed histology and histopathology of numerous tissues, including endocrine organs, following repeated or long term exposures. These studies are considered capable of revealing endocrine effects. The results of all of these studies show no evidence of any endocrine-mediated effects and no pathology of the endocrine organs. Consequently, it is concluded that etoxazole does not possess estrogenic or endocrine disrupting properties.

C. Aggregate Exposure

1. *Dietary exposure.* A full battery of toxicology testing including studies of acute, chronic, oncogenicity, developmental, mutagenicity, and reproductive effects is available for etoxazole. In these risk assessments, Valent proposes as the chronic oral toxic endpoint the NOAEL for males from the rat chronic/oncogenicity feeding study, 4 mg/kg/day. To assess the chronic risk to the U.S. population from exposure to etoxazole, the daily chronic exposures were compared against an estimated chronic population adjusted dose (cPAD) of 0.04 mg/kg bwt/day. This endpoint is derived from the NOAEL from the 2–year chronic rat study by applying an uncertainty factor of 100 to account for intraspecies and interspecies variations. There is no evidence that any additional safety factors are needed to further protect vulnerable subpopulations. The proposed acute oral toxic endpoint is the NOAEL from the rabbit oral developmental toxicity study, 200 mg/kg/day. To assess the acute risk to the U.S. population from exposure to etoxazole, acute exposures were compared against an estimated acute population adjusted dose (aPAD) of 2 mg/kg bwt/day. This endpoint is derived from the NOAEL from the rabbit oral developmental toxicity study by applying an uncertainty factor of 100 to account for intraspecies and interspecies variations. Based on dietary, drinking water, and non-occupational exposure assessments, there is reasonable certainty of no harm

to the U.S. population, any population subgroup, or infants and children from short-term or chronic exposure to etoxazole.

i. *Food.* Dietary exposure was estimated using the Cumulative and Aggregate Risk Evaluation System (CARES). Acute dietary exposure was estimated for the overall U.S. population and 16 population subgroups using proposed tolerances and conservative estimates of the percentages of crop treated. The results demonstrate that estimated exposure is less than 1% of the estimated aPAD (at the 99.9th percentile) for all population groups examined. Acute dietary exposure for the overall U.S. population was estimated to be 0.006 mg/kg bwt/day at the 99.9th percentile of exposure (0.29% of the aPAD). Chronic dietary exposure was estimated for the overall U.S. population and 16 population subgroups. Annual exposure for the overall U.S. population was estimated to be 0.00014 mg/kg bwt/day, representing 0.36% of the estimated cPAD. Annual exposure for the most highly exposed population subgroup, children 1–2 years of age, was estimated to be 0.00065 mg/kg bwt/day, or 1.62% of the estimated cPAD.

ii. *Drinking water.* Since etoxazole is applied outdoors to growing agricultural crops, the potential exists for the parent or its metabolites to reach ground water or surface water that may be used for drinking water. But, because of the physical properties of etoxazole, it is unlikely that etoxazole or its metabolites can leach to potable ground water. Although, relatively stable to hydrolysis, etoxazole undergoes fairly rapid photolysis, degrades fairly readily in soil and is immobile in all soil types examined. To quantify potential exposure from drinking water, FIRST and SCI-GROW models were used to estimate surface water and ground water residues. Estimated surface water residues were much higher than estimated ground water residues and therefore, the surface residues were used as the Drinking Water Environmental Concentration (DWEC). The peak (acute) concentration predicted in the simulated pond water was estimated to be 2.47 ppb and the annual average (chronic) concentration predicted in the simulated pond water was estimated to be 1.93 ppb. To assess the contribution to the dietary risk from exposure to drinking water containing residues of etoxazole, these DWEC's are compared to drinking water levels of comparison (DWLOC's), the maximum drinking water concentration allowed before combined water, dietary, and other exposures will exceed the population adjusted doses. If the DWLOC is greater than the DWEC, then overall exposure will not exceed the population adjusted doses and combined exposure from water and food is considered to be acceptable. Acute DWLOC's for etoxazole range from 19,900 to 69,910 ppb and chronic DWLOC's range from 377 to 1,380 ppb for all U.S. population subgroups examined. Since these DWLOC's exceed the modeled acute and chronic DWEC surface water residues by a wide margin, it can be concluded that, exposure to potential residues in drinking water is negligible and that aggregate (food and water) exposure to etoxazole residues will be acceptable.

2. *Non-dietary exposure.* Etoxazole is proposed only for agricultural uses and no homeowner or turf uses. Thus, no non-dietary risk assessment is needed.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that the Agency must consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances'' that have a common mechanism of toxicity. Available information in this context include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although, the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way.

In consideration of potential cumulative effects of etoxazole and other substances that may have a common mechanism of toxicity, there are currently no available data or other reliable information indicating that any toxic effects produced by etoxazole would be cumulative with those of other chemical compounds. Thus, only the potential risks of etoxazole have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of etoxazole consistent with the schedule established by EPA at (62 FR 42020) (Aug. 4, 1997) and other subsequent EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

1. *U.S. population*—i. *Acute risk.* The potential acute exposure from food to the U.S. population and various non-child/infant population subgroups are estimated to be 0.15 to 0.30% of the proposed aPAD. Exposure to potential acute residues in drinking water is expected to be negligible, as acute DWLOC's are substantially higher than modeled acute DWEC's. Based on this assessment, it can be concluded that, there is a reasonable certainty that no harm to the U.S. population or any population subgroup will result from acute exposure to etoxazole.

ii. *Chronic risk.* The potential chronic exposure from food to the U.S. population and various non-child/infant population subgroups are estimated to be 0.24 to 1.59% of the proposed cPAD. Chronic exposure to potential residues in drinking water is also expected to be negligible, as chronic DWLOC's are substantially higher than modeled chronic DWEC's. Based on this assessment, it can be concluded that there is a reasonable certainty that no harm to the U.S. population or any population subgroup will result from chronic exposure to etoxazole.

2. *Infants and children*—i. *Safety factor for infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of etoxazole, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to ten–fold, for added protection for infants and children in the case of threshold effects unless EPA determines that a different margin of safety will be safe for infants and children. The toxicological data base for evaluating prenatal and postnatal toxicity for etoxazole is complete with respect to current data requirements. There are no special prenatal or postnatal toxicity concerns for infants and children, based on the results of the rat and rabbit developmental toxicity studies or the 2-generation reproductive toxicity study in rats. Valent has concluded, that reliable data support use of the standard 100–fold uncertainty factor and that an additional uncertainty factor is not needed for etoxazole to be further protective of infants and children.

ii. *Acute risk.* The potential acute exposure from food to infants and children are estimated to be 0.28 to 0.97% of the proposed aPAD. Exposure to potential acute residues in drinking water is expected to be negligible, as acute DWLOC's are substantially higher than modeled acute DWEC's. Based on this assessment, it can be concluded that, there is a reasonable certainty that

no harm to infants and children will result from acute exposure to etoxazole.

iii. *Chronic risk.* The potential chronic exposure from food to infants and children are estimated to be 0.64 to 1.62% of the proposed cPAD. Chronic exposure to potential residues in drinking water is expected to be negligible, as chronic DWLOC's are substantially higher than modeled DWEC's. Based on this assessment, it can be concluded that, there is a reasonable certainty that no harm to infants and children will result from chronic exposure to etoxazole.

3. *Safety determination summary.* Aggregate acute or chronic dietary exposure to various subpopulations of children and adults demonstrate acceptable risk. Acute and chronic dietary exposures to etoxazole occupy considerably less than 100% of the appropriate PAD. EPA generally has no concern for exposures below 100% of the acute and chronic PAD's because these represent levels at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Chronic and acute dietary risk to children from etoxazole should not be of concern. Further, etoxazole has only agricultural uses and no other uses, such as indoor pest control, homeowner or turf, that could lead to unique, enhanced exposures to vulnerable sub-groups of the population. It can be concluded that, there is a reasonable certainty that no harm will result to the U.S. population or to any sub-group of the U.S. population, including infants and children, from aggregate chronic or aggregate acute exposures to etoxazole residues resulting from the proposed uses.

F. International Tolerances

Etoxazole has not been evaluated by the JMPR and there are no codex maximum residue limits (MRL) for etoxazole. MRL values have been established for etoxazole in the following countries: Turkey, Israel, South Africa, Japan, France, Taiwan, and Korea. The use pattern and MRL's are similar to those proposed for the U.S.

[FR Doc. 05–7223 Filed 4–12–05; 8:45 am]

BILLING CODE 6560–50–S

EQUAL EMPLOYMENT OPPORTUNITY COMMISSION

Meeting, Sunshine Act

DATE AND TIME: Thursday, April 21, 2005, a.m. eastern time.

PLACE: Clarence M. Mitchell, Jr. Conference Room on the Ninth Floor of the EEOC Office Building, 1801 ''L'' Street, NW., Washington, DC 20507.

STATUS: Part of the meeting will be open to the public and part of the meeting will be closed.

MATTERS TO BE CONSIDERED:

Open Session

1. Announcement of Notation Votes.
2. Renewal of LexisNexis Subscription Services.
3. Renewal of Westlaw and West Publishing Subscriptions.
4. Oracle License Maintenance Agreement.
5. Competitive Lease Contract for New Mail Machine Systems.

Closed Session

Litigation Authorization: General Counsel Recommendations.

Note: In accordance with the Sunshine Act, the open session of the meeting will be open to public observation of the Commission's deliberations and voting. (In addition to publishing notices on EEOC Commission meetings in the **Federal Register**, the Commission also provides a recorded announcement a full week in advance on future Commission sessions.)

Please telephone (202) 663–7100 (voice) and (202) 663–4074 (TTY) at any time for information on these meetings.

CONTACT PERSON FOR MORE INFORMATION: Stephen Llewellyn, Acting Executive Officer on (202) 663–4070.

This notice issued April 11, 2005.

Stephen Llewellyn,

Acting Executive Officer, Executive Secretariat.

[FR Doc. 05–7537 Filed 4–11–05; 8:45 am]

BILLING CODE 6750–06–M

FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) being Reviewed by the Federal Communications Commission for Extension Under Delegated Authority

April 4, 2005.

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act (PRA) of 1995, Public Law 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the Paperwork Reduction Act (PRA) that does not display a valid control number. Comments are requested concerning (a) whether the proposed collection of information is necessary for the proper performance of the functions of the Commission, including whether the information shall have practical utility; (b) the accuracy of the Commission's burden estimate; (c) ways to enhance the quality, utility and clarity of the information collected; and (d) ways to minimize the burden of the collection of information on the respondents, including the use of automated collection techniques or other forms of information technology.

DATES: Written Paperwork Reduction (PRA) comments should be submitted on or before June 13, 2005. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of time allowed by this notice, you should advise the contact listed below as soon as possible.

ADDRESSES: Direct all Paperwork Reduction Act (PRA) comments to Cathy Williams, Federal Communications Commission, Room 1–C823, 445 12th Street, SW., Washington, DC 20554 or via the Internet to *Cathy.Williams@fcc.gov*.

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collection(s), contact Cathy Williams at 202–418–2918 or via the Internet at Cathy.Williams@fcc.gov.

SUPPLEMENTARY INFORMATION: OMB Control Number: 3060–0386.

Title: Section 73.1635, Special Temporary Authorizations (STA).

Form Number: Not applicable.

Type of Review: Extension of a currently approved collection.

Respondents: Business or other for-profit entities; not-for-profit institutions.

Number of Respondents: 1,550.

Estimated Time per Response: 1–4 hours.

Frequency of Response: On occasion reporting requirement.

Total Annual Burden: 2,000 hours.

Total Annual Cost: $939,950.

Privacy Impact Assessment: No impact(s).

Needs and Uses: 47 CFR 73.1635 allows licensees/permittees of broadcast stations to file for special temporary authority to operate broadcast stations at specified variances from station authorization not to exceed 180 days. Data is used by FCC staff to ensure that such operations will not cause interference to other stations.