toxicological endpoint for short-term dermal risks is the NOEL of 100 mg/kg/day from a 21–day dermal toxicity study in rabbits. No acute oral hazard has been identified following an acute exposure to S-metolachlor and, therefore, no nondietary assessment is needed.

The short-term dermal post-application risks for adults and children are acceptable, ranging from 520 to 870. These risk estimates exceed the EPA's level of concern for S-metolachlor (all MOEs are greater than 100).

Aggregate exposure. (Drinking Water and Dietary Exposure). Using the total MOE equation for the determination of aggregate chronic exposure (food and drinking water only) resulted in an aggregate MOET of >4,000 for the most sensitive subpopulation, non-nursing infants. For this particular subpopulation, there are no non-dietary exposure contributions to the MOET aggregate value.

D. Cumulative Effects

EPA has examined the common mechanism potential for S-metolachlor and has concluded that S-metolachlor should not be included with some pesticides that comprise the class of chloroacetanilides included in a ''Common Mechanism Group.'' Therefore, a cumulative assessment is not necessary for S-metolachlor.

E. Safety Determination

1. *U.S. population*. Based on the aggregate assessment described above and the completeness and reliability of the toxicity data, it is concluded that aggregate exposure to S-metolachlor (including the proposed uses) in food will utilize less than 0.1 percent of the cRfD for the U.S. population. EPA generally has no concern for exposures below 100 percent of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to S-metolachlor in drinking water and from non-dietary, non-occupational exposures, the assessment presented above demonstrates that the high levels of safety exist for current and proposed uses of S-metolachlor; it is not expected that aggregate exposure from all sources will exceed 100% of the RfD. Therefore, one can conclude there is a reasonable certainty that no harm will result from aggregate exposure to S-metolachlor.

2. *Infants and children*. [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 pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. A full consideration of the available reproductive toxicity data supporting S-metolachlor demonstrates no increased sensitivity to infants and children. Therefore, it is concluded that an additional uncertainty factor is not warranted to protect the health of infants and children and that the cRfD at 0.1 mg/kg/day is appropriate for assessing aggregate risk to infants and children from use of S-metolachlor.

Based on the aggregate assessment described above, the percent of the cRfD that will be utilized by aggregate exposure to residues of S-metolachlor is less than 0.7 percent for all children subpopulations. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to S-metolachlor in drinking water and from non-dietary, non-occupational exposure, the assessment described above demonstrates that it is not expected that aggregate exposure from all sources provides for a large margin of safety and will exceed 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the exposure assessment, it is concluded there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to S-metolachlor residues.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) maximum residue levels (MRL's) established for residues of S-metolachlor in or on raw agricultural commodities.

[FR Doc. 03–20643 Filed 8–12–03; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0271; FRL–7322–6]

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 ID number OPP–2003–0271, must be received on or before September 12, 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: Daniel Kenny, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–7546; e-mail address: *kenny.dan@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–2003–0271. 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–2003–0271. 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–2003–0271. 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–2003–0271.

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–2003–0271. 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: August 6, 2003.

Debra Edwards,

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 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 2F6420

EPA has received a pesticide petition (2F6420) from Valent U.S.A. Corporation, 1333 North California Blvd., Suite 600, Walnut Creek, CA 94596 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 etoxazole in or on the raw agricultural commodity pome fruit (Crop Group 11) at 0.2 parts per million (ppm), apple wet pomace at 1.0 ppm, strawberry at 0.5 ppm, cottonseed at 0.05 ppm, cotton, gin byproducts (gin trash) at 1.0 ppm, and oranges at 0.10 ppm (to support the importation of mandarin oranges into the U.S.). As residues in processed commodities fed to animals may be transferred to milk and edible tissue of ruminants, tolerances are also proposed for animal fat at 0.03 ppm and milk fat at 0.04 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Metabolism of 14C-etoxazole labeled in the t-butylphenyl, difluorophenyl, or oxazole rings has been studied in apples, cotton, oranges, and eggplants. Etoxazole was rapidly and extensively metabolized to many metabolites in all plants. Even with exaggerated treatment, individual metabolites and parent were only found at very low concentrations. Comparisons of metabolites detected and quantified from plants and animals show that there are no significant aglycones in plants which are not also present in the excreta or tissues of animals. The residue of concern is best defined as the parent etoxazole.

2. *Analytical method*. 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 mandarin oranges and cottonseed and 0.2 ppm in cotton gin trash, which will allow monitoring of food with residues at the levels proposed for the tolerances. Methods have also been developed and validated for determining etoxazole in animal samples. The LOQ of etoxazole in these methods is 0.02 ppm in milk fat and beef fat.

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: apples and pears (representing pome fruits), strawberries, and cotton. Residue trials have also been conducted for etoxazole in Europe to support the importation of mandarin oranges into the U.S. The results of these studies can be summarized as follows:

- For pome fruit, the maximum etoxazole residues from two applications at 0.135 lbs. active ingredient/acre/treatment, are 0.07 ppm for apples and 0.11 for pears harvested 28 days after application.
- The results of an apple processing study indicate that etoxazole residues do not concentrate in apple juice, but do concentrate in wet apple pomace with an average concentration factor of 5.7x.
- The maximum etoxazole residue in strawberries harvested one day following the last of two treatments at

0.135 lbs. active ingredient/acre/treatment is 0.32 ppm.

- The maximum expected etoxazole residues in cottonseed and cotton gin trash from two applications at 0.045 lbs. active ingredient/acre/treatment applied 28 days before harvest are 0.02 ppm and 0.60 ppm, respectively. Cotton gin trash was also analyzed for metabolite R–3 but was detected in samples from only one of seven test sites at a level only slightly above the 0.10 ppm limit of detection (LOD) (mean residue was 0.13 ppm). Therefore, no tolerance is being proposed for metabolite R–3 in cotton gin trash.
- The results of a cotton processing study indicate that etoxazole does not concentrate in hulls, meal, or oil.
- Following a single application to mandarin oranges of 0.05 lbs. active ingredient/acre, the maximum etoxazole residues in whole fruit harvested 14 days after application is 0.05 ppm.
- No processing of mandarins was required because only fresh or canned mandarins will be imported for direct consumption. Separate analysis of mandarin peel and pulp from residue field trials demonstrated that etoxazole residues are confined to the peel. Canned mandarins that contain only mandarin pulp would therefore not be expected to contain detectable residues of etoxazole.

These field trial data are adequate to support proposed tolerances of 0.2 ppm for pome fruit; 1.0 ppm for wet apple pomace; 0.5 ppm for strawberries; 0.05 ppm for cottonseed; 1.0 ppm in cotton gin trash; and 0.1 ppm in oranges.

Apple pomace and all cotton commodities are significant feed items for beef and dairy cattle and results of a goat metabolism study suggest the possibility that etoxazole residues in feed may transfer to edible tissues and milk. Therefore, a cow feeding study was conducted with etoxazole to determine the level of secondary residues and the need for corresponding tolerances. Etoxazole was detected in fat and cream only and Valent is therefore proposing tolerances of 0.03 ppm in the fat of animals and 0.04 ppm in milk fat. Cotton meal is the only commodity under consideration that is used as a poultry feed item and the results of the cotton processing study indicate that etoxazole residues in this commodity are very low. 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. The toxicology reports for etoxazole have not yet been reviewed by EPA and thus, the Agency has not yet established toxic endpoints of concern, specifically chronic and acute oral toxicity endpoints for the compound. For the purpose of dietary risk analysis, Valent proposes 0.04 mg/kg bwt/day as the chronic Population Adjusted Dose (cPAD) and an acute Population Adjusted Dose (aPAD) of 2 mg/kg bwt/day. The cPAD is based on a chronic endpoint of 4 mg/kg bw/day NOEL 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 NOEL 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. *Genotoxicty*. 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.

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 13^{th} 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 highest dose tested.

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 highest dose tested.

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 two-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 two 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 two years at dietary concentrations of 50, 5,000, and 10,000 ppm. In this study, decreased mortality, bodyweight 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 two 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 of 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 no observed effect level 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 seven 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 six 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 *Escherichia 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. EPA has not had the opportunity to review all of the toxicity studies on etoxazole and has not established toxic endpoints. Thus, 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 DEEM™, proposed tolerances, and assuming 100% crop treated. Results of the acute analysis demonstrate that estimated exposure is 0.5% or less of the estimated aPAD (at the 95th percentile) for all population groups examined. Acute dietary exposure for the overall U.S. population was estimated to be 0.002572 mg/kg bwt/day at the 95th percentile of exposure (0.13% of the aPAD). Chronic dietary exposure was estimated for the overall U.S. population and 25 population sub groups. Daily exposure for the overall U.S. population was estimated to be 0.000574 mg/kg bwt/day, representing 1.4% of the estimated cPAD. Daily exposure for the most highly exposed population subgroup, children 1–6 years of age, was estimated to be 0.002293 mg/kg bwt/day, or 5.7% 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 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 groundwater. 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 and groundwater residues. Estimated surface water residues were much higher than estimated groundwater 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 to1380 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, Valent concludes 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 **Federal Register** 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 0.06 to 0.13 % 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, Valent concludes 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 0.7% to 1.9% 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, Valent concludes 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 0.16 to 0.50 % 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, Valent concludes 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 2.1 to 5.7% 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, Valent concludes 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 sub-populations 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. Valent concludes 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 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 to allow the following uses of 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.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–7543–8]

Proposed CERCLA Section 122(h) Administrative Agreement for Recovery of Response Costs for the Amenia Town Landfill Superfund Site, Town of Amenia, Dutchess County, NY

AGENCY: Environmental Protection Agency.

ACTION: Notice; request for public comment.

SUMMARY: In accordance with Section 122(i) of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended (''CERCLA''), 42 U.S.C. 9622(i), notice is hereby given by the U.S. Environmental Protection Agency (''EPA''), Region II, of a proposed administrative agreement pursuant to Section 122(h) of CERCLA, 42 U.S.C. 9622(h), for recovery of response costs concerning the Amenia Town Landfill Superfund Site (''Site'') located in the Town of Amenia, Dutchess County, New York. The settlement requires the settling parties, Town of Amenia, New York; Ashland, Inc.; BP America Inc.; Curtiss-Wright Corporation; International Business Machines Corporation; Alastair B. Martin; Estate of Edith Martin; Metal Improvement Company, Inc.; Town of Sharon, Connecticut; Syngenta Crop Protection, Inc.; TBG Services, Inc.; Unisys Corporation; and Weyerhaeuser Company to pay $361,873.17 in reimbursement of EPA's response costs at the Site. The settlement includes a covenant not to sue the settling parties pursuant to Section 107(a) of CERCLA, 42 U.S.C. 9607(a), in exchange for their