TABLE 1.—REGISTRATIONS WITH REQUESTS FOR AMENDMENTS TO DELETE USES IN CERTAIN PESTICIDE REGISTRATIONS—Continued

Registration No.	Product Name	Active Ingredient	Delete From Label
062719–00100	Balan Technical	Benfluralin	Peanuts
068156–00001	Technical Benefin	Benfluralin	Peanuts

Users of these products who desire continued use on crops or sites being deleted should contact the applicable registrant before dates indicated in **DATES** section of this notice to discuss withdrawal of the application for amendment. This 30–day or 180–day period will also permit interested members of the public to intercede with registrants prior to the Agency's approval of the deletion.

Table 2 of this unit includes the names and addresses of record for all registrants of the products in Table 1 of this unit, in ascending sequence by EPA company number.

TABLE 2.—REGISTRANTS REQUESTING VOLUNTARY CANCELLATION

EPA Company No.	Company Name and Address
001270	Zep Manufacturing Company Agent for: Zep Manufacturing Company, 1310 Seaboard Industrial Blvd., NW, Atlanta, GA 30318
001812	Griffin L.L.C., P.O. Box 1847, Valdosta, GA 31603
002393	Haco Inc, P.O. Box 7190, Madison, WI 53707
019713	Drexel Chemical Co, 1700 Channel Avenue, P.O. Box 13327, Memphis, TN 38113
040322	Equine Chemical Co. Inc., P.O. Box 771, Skiatook, OK 74070
042750	Pyxis Regulatory Consulting Agent for: Albaugh Inc., 11324 17th Avenue Court NW, Gig Harbor, WA 98332
062719	Dow AgroSciences LLC, 9330 Zionsville Rd 308/2E225, Indianapolis, IN 46268
068156	Dintec AgriChemicals, 9330 Zionsville Rd, Indianapolis, IN 46268

III. What is the Agency Authority for Taking this Action?

Section 6(f)(1) of FIFRA provides that a registrant of a pesticide product may at any time request that any of its pesticide registrations be amended to delete one or more uses. The Act further provides that, before acting on the request, EPA must publish a notice of receipt of any such request in the **Federal Register**. Thereafter, the Administrator may approve such a request.

IV. Procedures for Withdrawal of Request

Registrants who choose to withdraw a request for use deletion must submit the withdrawal in writing to James A. Hollins using the instructions in Unit I.C. The Agency will consider written withdrawal requests postmarked on or before dates indicated in **DATES** section of this notice.

V. Provisions for Disposition of Existing Stocks

The Agency has authorized the registrants to sell or distribute products under the previously approved labeling for a period of 18 months after approval of the revision, unless other restrictions have been imposed, as in Special Review actions.

List of Subjects

Environmental protection, Pesticides and pests.

Dated: June 11, 2003.

Arnold E. Layne,

Director, Information Resources and Services Division, Office of Pesticide Programs.

[FR Doc. 03–16031 Filed 6–24–03; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0166; FRL–7307–8]

Flufenpyr-ethyl; 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–2003–0166, must be received on or before July 25, 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: Joanne I. Miller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6224; e-mail address: *Miller.Joanne@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. Potential affected entities may include, but are not limited to:

- Crop production (NAICS)
- 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 identification (ID) number OPP–2003–0166. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

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

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

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

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at *http://www.epa.gov/edocket*, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2003–0166. 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–2003–0166. 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–2003–0166.

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–2003–0166. 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: June 16, 2003.

Debra Edwards,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

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

Valent U.S.A. Corporation

PP 0F6164

EPA has received a request from Valent U.S.A. Corporation at 1333 North California Boulevard, Suite 600, Walnut Creek, California 94596–8025 pursuant to section 408(d) of FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 to establish tolerances for residues of the herbicide chemical flufenpyr-ethyl, ethyl [2-chloro-4-fluoro-5-(5-methyl-6-oxo-4-trifluoromethyl-1,6-dihydropyridazin-1-yl)phenoxy]acetate, in or on the raw agricultural commodities corn, field, grain; soybean, seed; and sugarcane, cane at 0.01 parts per million (ppm) and for the combined residues of the herbicide chemical flufenpyr-ethyl, and its metabolite, *S*-3153-acid-4-*OH*, [2-chloro-4-hydroxy-5-(5-methyl-6-oxo-4-trifluoromethyl-1,6-dihydropyridazin-1-yl)phenoxy]acetic acid, free and conjugated, in or on the raw agricultural commodities corn, field, forage and corn, field, stover at 0.05 ppm. EPA has determined that the request contains data or information consistent with 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 petitions. Additional data may be needed before EPA rules on the request.

A. Residue Chemistry

Plant and animal metabolism studies with ^{14}C-flufenpyr-ethyl have demonstrated that the residue of concern is adequately understood for the purposes of these tolerances. Practical, validated residue methodology is available to analyze all appropriate matrices for flufenpyr-ethyl residues with an LOQ (limit of quantitation) of 0.01 ppm, and for *S*-3153-acid-4-*OH* metabolite with an LOQ of 0.02 ppm, adequate to enforce all proposed tolerances. The potential for residues of flufenpyr-ethyl has been evaluated in field corn grain, forage, and stover; soybeans; sugarcane; feed items; in appropriate processed products; and animals. These studies are adequate to support appropriate tolerances and dietary risk analyses.

1. *Plant metabolism*. Metabolism of flufenpyr-ethyl radiolabeled with ^{14}C in the phenyl and in the pyridazinyl rings has been studied in corn and soybean plants and in lactating goats, laying hens, and rats. The major metabolic pathway in plants is hydrolysis of the ethyl ester, followed by further metabolism into more polar products by formation of phenolic glyclones. At the proposed pre-harvest intervals total radiocarbon residue in grain samples was very low and adequately represented by parent. However, in plant material and forage samples, a conjugated carboxylic acid phenolic metabolite was present, the aglycone of which, *S*-3153-acid-4-*OH*, was not detected as an animal metabolite. This metabolite was not detected in any raw

agricultural commodity (RAC) grain sample or in sugarcane. The residue of concern in grain and sugarcane is best defined as the parent, flufenpyr-ethyl. However, consistent with plant metabolism studies, finite residues of *S*-3153-acid-4-*OH* were detected in field corn forage and stover, and tolerances are proposed.

2. *Ruminant and poultry metabolism*. Metabolism studies in goats and hens demonstrated that transfer of administered ^{14}C-flufenpyr-ethyl residues to tissues was low. Total ^{14}C-residues in goat milk, muscle and tissues accounted for less than 1% of the administered dose. Total radiocarbon residues (parent equivalent) were less than 0.01 ppm in all cases except for approximately 0.15 ppm in kidney and 0.04 ppm in liver. Residues were identified in excreta and all appropriate tissues. In milk (0.002 to 0.008 ppm), kidney, and liver approximately 70 to 90 percent of the residues was identified as the ester hydrolysis product, *S*-3153-acid. In poultry, total ^{14}C residues (parent equivalent) in eggs, muscle and tissues accounted for about 0.1% of the administered dose, and were less than 0.01 ppm in all cases except for approximately 0.02 ppm in liver. More than half of the liver residue was *S*-3153-acid.

3. *Analytical method*. Practical analytical method for detecting and measuring levels of flufenpyr-ethyl and validated in/on all appropriate agricultural commodities and respective processing fractions. Methodology that converts the *S*-3153-acid-4-*O*-glucoside to the corresponding aglycone, *S*-3153-acid-4-*OH*, was developed and validated with an LOQ of 0.02 ppm. The extraction methodologies have been validated using plant samples containing aged radiochemical residue samples from ^{14}C-metabolism studies. The methods have been validated in soybean seed, corn grain, and corn stover at an independent laboratory. The LOQ for flufenpyr-ethyl is 0.01 ppm and the LOQ for *S*-3153-acid-4-*OH* is 0.02 ppm which will allow monitoring of food with residues at the levels proposed for the tolerances. Both flufenpyr-ethyl and *S*-3153-acid-4-*OH* have been evaluated using the FDA multiresidue method protocol.

4. *Magnitude of residues*— i. *Soybean seed*. Twenty-two field trials in soybeans were conducted in 1997 and 1998 in 15 states representing approximately 99% of the soybean acreage in the U.S. (EPA Regions II, IV, and V). Analysis of duplicate samples from these trials showed that at the proposed maximum application rate (24.5 grams active ingredient (a.i.)/acre), or at 5 times the proposed maximum application rate, there were no measurable residues of flufenpyr-ethyl in soybean seed (<0.005 ppm). The analytical LOQ was 0.01 ppm. A processing study in soybean seed treated at the 5-fold application rate demonstrated that flufenpyr-ethyl was undetectable in all processed commodities. All these data support a proposed tolerance of 0.01 ppm for flufenpyr-ethyl in/on soybean seed. No additional tolerances are necessary for processed commodities.

ii. *Sugarcane cane*. Nine field trials in sugarcane were conducted in 1998 in 4 states representing approximately 100% of the sugarcane acreage in the U.S. (EPA Regions II, IV and V). Analysis of duplicate samples from these trials showed that at the proposed seasonal maximum application rate (24.5 grams a.i./acre), or at five times the proposed maximum application rate, there were no measurable residues of flufenpyr-ethyl in sugarcane cane (<0.005 ppm). The analytical LOQ was 0.01 ppm. Because sugarcane is the vegetative portion of the plant, it is possible that residues of the carboxylic acid phenol metabolite (*S*-3153-acid-4-*OH*) might be present. With an LOQ of 0.02 ppm, there was no detected metabolite in any sugarcane sample. Samples of processed commodities from the sugarcane processing studies treated at 5-fold were not analyzed because of the absence of finite residues in any of the cane RAC samples. All these data support a proposed tolerance of 0.01 ppm for flufenpyr-ethyl in/on sugarcane cane. No additional tolerances are necessary for processed commodities.

5. *Field corn*. Twenty-four field trials in field corn were conducted in 1997 (10) and 1998 (14) in 16 states representing approximately 97% of the field corn acreage in the U.S. (EPA Regions I, II, V, VI, VII, VIII, and X). Field plots were treated at the V10 crop stage. Forage was sampled 32 to 65 days after treatment at late R4 to early R5 crop stage. Grain and stover were sampled at dry maturity 58 to 115 days after application.

i. *Field corn grain*. Analysis of duplicate samples of grain from these trials showed that at the proposed maximum application rate (24.5 grams a.i./acre), at half the proposed maximum application rate (12 grams a.i./acre), or from two field plots treated at five times the proposed maximum application rate (121 grams a.i./acre) there were no measurable residues of flufenpyr-ethyl (<0.005ppm). The analytical LOQ was 0.01 ppm. A processing study in field corn grain treated at the five times the normal application rate demonstrated that flufenpyr-ethyl was undetectable in all processed commodities. All these data support a proposed tolerance of 0.01 ppm for flufenpyr-ethyl in/on field corn grain. No additional tolerances are necessary for processed commodities.

ii. *Field corn forage and stover*. Analysis of duplicate samples of forage and stover from these trials showed that at the proposed maximum application rate (24.5 grams a.i./acre), and at half the proposed maximum application rate (12 grams a.i./acre) there were no measurable residues of flufenpyr-ethyl (<0.005 ppm). In forage and stover from plots treated at 5 times the proposed application rate (121 grams a.i./acre) finite residue of flufenpyr-ethyl were detected (0.015 to 0.008 ppm). Forage and stover samples were also analyzed for *S*-3153-acid-4-*OH*. Finite residues of the metabolite were detected in 28 of 52 forage samples, and 11 of 54 stover samples from plots treated at 24.5 grams a.i./acre. Maximum residue values in the two feed commodities were 0.03 ppm. Forage and stover samples from the two plots treated at the 5-fold rate showed maximum residue values of 0.05 ppm. All these data support proposed tolerances of 0.01 ppm for flufenpyr-ethyl and 0.04 ppm *S*-3153-acid-4-*OH* in/on field corn forage and stover.

6. *Secondary residues*. Using proposed tolerances, or for field corn forage and stover the sum of the tolerances for parent and metabolite, to calculate the maximum feed exposure to fed animals, and using the very low potential for residue transfer demonstrated in the lactating goat and laying hen metabolism studies, detectable secondary residues in animal tissues, milk, and eggs are not expected. Therefore, tolerances are not proposed for these commodities.

7. *Rotational crops*. The results of a confined rotational crops accumulation study with ^{14}C-flufenpyr-ethyl indicate that no rotational crop planting restrictions or rotational crop tolerances are required.

B. Toxicological Profile

A full battery of toxicology testing including studies of acute, chronic, oncogenicity, developmental, mutagenicity, and reproductive effects is available for flufenpyr-ethyl. The acute toxicity of flufenpyr-ethyl is low by all routes. Subchronic and chronic toxicity studies exhibit no observable adverse effect level (NOAEL) values from a low of 40 milligrams/kilogram/day (mg/kg/day) (male mouse 18-month oncogenicity) to greater than 1,000 mg/kg/day. Flufenpyr-ethyl is not oncogenic

or mutagenic, and it is not a reproductive or developmental toxicant when tested in standard toxicity studies. Animal metabolism and excretion is rapid; there appear to be no special toxicity concerns for a unique plant metabolite; and there is no evidence for endocrine effects. The kidney and liver appear to be the target organs of flufenpyr-ethyl. EPA has not had the opportunity to review the toxicity studies on flufenpyr-ethyl and has not established toxic endpoints. For chronic oral exposure, Valent has chosen the NOAEL from the second rat reproduction study of 100 ppm (5 mg/kg/day nominal) as the toxic endpoint. There is no study with flufenpyr-ethyl that showed toxicity that could be associated with a single, or acute, oral exposure. Therefore no acute endpoint could be identified, and no acute oral risk analyses are performed.

1. *Acute toxicity*. The acute toxicity of technical grade flufenpyr-ethyl is low by all routes. Flufenpyr-ethyl produces minimal toxicity following acute oral, dermal or inhalation exposures. The technical material is essentially non-irritating to the eye, is not irritating to the skin and does not cause dermal sensitization in guinea pigs. Flufenpyr-ethyl technical will be classified as Toxicity Class IV.

2. *Genotoxicity*. Flufenpyr-ethyl does not present a genetic hazard. Flufenpyr-ethyl technical was negative in the following tests for mutagenicity: Ames assay with and without S9, *in vitro* mammalian cell gene mutation assay using L5178Y/TK$^{+/-}$ mouse lymphoma cells, and the *in vivo* mouse bone marrow micronucleus test.

3. *Reproductive and developmental toxicity*. Developmental toxicity studies have been performed in rats and rabbits, and multigenerational effects on reproduction were tested in rats.

i. *Rats*. In the developmental toxicity study conducted with rats, technical flufenpyr-ethyl was administered by gavage at levels of 0, 100, 300, and 1,000 mg/kg/day during gestation days 6 through 19. There were no adverse maternal or fetal effects observed. The NOAEL for both maternal and developmental toxicity was found to be 1,000 mg/kg/day, the highest dose tested.

ii. *Rabbits*. Flufenpyr-ethyl technical was tested in a developmental toxicity study in rabbits at doses of 0, 100, 300 and 1,000 mg/kg/day during gestation days 6 through 28. Maternal mortality occurred at the two highest doses tested but the deaths at 300 ppm were not considered to be the result of systemic toxicity. In surviving animals and their fetuses, there were no adverse effects. Based on these results, the maternal toxicity NOAEL was 300 mg/kg/day and the developmental toxicity NOAEL was 1,000 mg/kg/day.

A second developmental toxicity study was conducted to confirm the maternal NOAEL at dose levels of 0, 100, 200, 300 or 1,000 mg/kg/day during gestation. Again, maternal mortality occurred, but at all dose levels. Detailed examination of these animals showed in the majority of cases the cause of death to be test material aspiration into the lungs. The cause of death for several animals at the high dose could not be determined. Their deaths were therefore attributed to systemic toxicity. There were no other adverse effects in the surviving dams or fetuses. The NOAEL for this study (and overall for both rabbit developmental toxicity studies) were found to be 300 mg/kg/day (maternal) and 1,000 mg/kg/day (developmental).

iii. *Reproduction*. In the rat reproduction study, flufenpyr-ethyl technical was administered in the diet at levels of 0, 200, 2,000, and 20,000 ppm for 2–generations. Parental toxicity was observed at all dose levels, although the effects at the low dose were minimal. Parental toxicity was exhibited by dose-related microscopic changes in the kidney in high dose F_0 animals, in all treated F_1 males, and in high dose F_1 females. There were also 2 high dose F_1 males that died possibly as a result of treatment. Midzonal cytoplasmic vacuolation of the hepatocytes was also observed in the liver of all groups of treated animals in both generations. Based on the results of this study, the NOAEL for parental toxicity was considered to be less than 200 ppm. The NOAEL for reproductive and neonatal toxicity was considered to be 20,000 ppm.

A second 1–generation reproduction study was performed to establish a clear NOAEL for adult kidney lesions using the dose levels of 20, 50 and 100 ppm. The results of the study indicate that the NOAEL for histological changes in the kidneys of F_1 male rats was 100 ppm. No other treatment related findings were noted at any dose level indicating 100 ppm as the NOAEL for treatment and reproductive effects evaluated in the study.

A mechanistic study was also conducted to investigate the reproducibility and reversibility of the kidney lesions observed in the initial 2-generation reproduction study. In the first study, the effects observed at 200 ppm in the F_1 males, basophilic tubules and interstitial inflamation, were minimal but slightly increased in incidence and severity and a slight increase in interstitial fibrosis of the cortex was also observed. In this mechanistic study, using dose levels of 0 and 2,000 ppm, the NOAEL for histological changes in the kidneys of F_0 and F_1 male rats and reproductive effects was 2,000 ppm. The histological changes seen in the kidneys in the original study was not reproducible.

4. *Subchronic toxicity*. Subchronic oral toxicity studies conducted with flufenpyr-ethyl in the rat, mouse and dog indicate a low level of toxicity.

i. *Rats*. Pure (99.4%) flufenpyr-ethyl was tested in rats at dose levels of 0, 600, 2,000, 6,000, and 20,000 ppm in the diet for 13 weeks. Effects observed included urinary incontinence, increased food and water consumption, slight hematological and blood biochemistry changes, decreased spleen weights, an increase in the incidence and severity of basophilic tubules of the kidneys and slight to mild diffusely distributed vacuolation in the liver. Based on these results, the NOAEL was 2,000 ppm (134.2 mg/kg/day) for the males and 20,000 ppm (1,509.6 mg/kg/day) for the females.

In an additional study, flufenpyr-ethyl technical was tested in rats at dose levels of 0, 1,000, 10,000, and 20,000 ppm in the diet for 13 weeks. Effects observed included urinary incontinence, increased food and water consumption, and mild urinalysis, hematological and blood biochemistry changes. Thymus weights were slightly increased. Diffusely distributed hepatic vacuolation was seen in the high dose males. Based on these findings, the NOAEL was 10,000 ppm (595.2 mg/kg/day) in the males and 20,000 ppm (1,377.5 mg/kg/day) in the females.

ii. *Mice*. In a 4–week study, CD-1 mice were fed pure flufenpyr-ethyl at dose levels of 0, 300, 1,000, 3,000, and 7,000 ppm. Effects were slight anemia, changes in blood biochemistry, increased liver and thymus weights, and enlarged liver. Centrilobular hepatocellular hypertrophy and vacuolation and increases in the severity and incidence of hepatic focal and single cell necrosis were observed. Based on these findings, the NOAEL was 300 ppm (44.9 mg/kg/day) for males and 1,000 (210.5 mg/kg/day) for females. In a 13-week study, flufenpyr-ethyl technical was administered to mice at dose levels of 0, 300, 1,000, 3,000, and 7,000 ppm. Slight anemia and blood biochemistry changes were noted. Liver weights were increased and ovary weights were decreased. Histopathological findings included: Hepatocellular fatty vacuolation. The NOAEL for this study in both sexes was

1,000 ppm (128.4 mg/kg/day for males and 155.7 mg/kg/day for females).

iii. *Dogs*. Flufenpyr-ethyl technical was administered for 13 weeks via capsule to Beagle dogs at levels of 0, 100, 300 or 1,000 mg/kg/day. The effects were very minimal. Only small nonsignificant decreases in body weight and slightly elevated alkaline phosphatase values were noted. In the absence of other effects, the NOAEL in both sexes was determined to be 1,000 mg/kg/day.

iv. *Dermal*. A 21–day dermal toxicity study in rats with flufenpyr-ethyl technical did not produce any signs of dermal or systemic toxicity at 1,000 mg/kg/day, the highest dose tested.

5. *Chronic toxicity*. Flufenpyr-ethyl technical has been tested in chronic studies with dogs, rats and mice.

i. *Rats*. In a 104–week combined chronic/oncogenicity study in rats, flufenpyr-ethyl technical was administered at dose levels of 0, 100, 1,000, 10,000, or 20,000 ppm in the diet for 24 months. Urinary incontinence, increased food and water consumption, changes in urinalysis, hematological and blood biochemistry changes were observed but the effects were not toxicologically significant. No neoplastic lesions were observed. The NOAEL was found to be 20,000 ppm (777.5 mg/kg/day for males and 1024 mg/kg/day for females).

ii. *Mice*. In a 78–week oncogenicity study with mice, flufenpyr-ethyl technical was administered at dose levels of 0, 350, 3,500, and 7,000 ppm. Male animals exhibited slight anemia. Females had increased liver and kidney weights (week 53 only). Slight to moderate hepatocellular fatty vacuolation and necrosis were observed. There were no increases in incidence of pre-neoplastic or neoplastic lesions. Based on these results, the NOAEL was 350 ppm for both sexes (39.9 mg/kg/day for males and 43.7 mg/kg/day for females).

iii. *Dogs*. In a 52–week chronic study, flufenpyr-ethyl technical was administered by capsule to Beagle dogs at dose levels of 0, 50, 200, and 1,000 mg/kg/day. There were very few observations related to treatment. Slightly elevated alkaline phosphatase values were again observed, but they were not accompanied with any other findings and were thus considered not to be an adverse effect. The NOAEL was determined to be 1,000 mg/kg/day, the highest dose tested.

iv. *Carcinogenicity*. Flufenpyr-ethyl is not a carcinogen. Studies with flufenpyr-ethyl technical have shown that repeated high dose exposures produced minimal signs of toxicity, including slight hematologic, liver and kidney effects, but did not produce cancer in test animals. No oncogenic response was observed in a rat 2–year chronic feeding/oncogenicity study or in a 78-week study on mice. Valent anticipates that the oncogenicity classification of flufenpyr-ethyl will be ''E'' (no evidence of carcinogenicity for humans).

6. *Animal metabolism*. Following oral administration of ^{14}C-phenyl-labeled flufenpyr-ethyl to rats at 50 mg/kg, the majority of the radiocarbon is eliminated from the body within 2 days. Approximately half is excreted in the urine and the balance is excreted in the feces. Tissue residues are very low 7 days after administration (<0.09% of the administered dose). The major metabolite was identified as [2-chloro-4-fluoro-5-(5-methyl-6-oxo-4-trfluoro-methyl-1,6-dihydropyridazin-1-yl)phenoxy]acetic acid (*S*-3153-acid) which accounted for 93.2% of the dose. Two other minor metabolites each accounted for less than 5% of the administered radiocarbon. Flufenpyr-ethyl was detected only in feces (0.5%). The major reaction was cleavage of the ester linkage; minor reactions were hydroxylation of the 5-methyl of pyridazine ring and cleavage of the ether linkage between the phenyl group and the carboxymethyl group.

7. *Metabolite toxicity*. Metabolism studies of flufenpyr-ethyl in rats, goats and hens, as well as the fish bioaccumulation study demonstrate that the parent is very rapidly metabolized and eliminated. Because parent and metabolites are not retained in the body, the potential for acute toxicity from *in situ* formed metabolites is low. The potential for chronic toxicity is adequately tested by chronic exposure to the parent at the maximum tolerated dose (MTD) and consequent chronic exposure to the internally formed metabolites. One plant metabolite, *S*-3153-acid-4-*OH* was not detected as an animal metabolite. This compound was tested for acute oral toxicity in rats, and for mutagenicity Ames testing with and without mixed function oxidation (S9 mix). The metabolite caused no mortality in rats at 5,000 mg/kg the highest dose tested, and was not mutagenic at up to 5,000 micrograms per plate.

8. *Potential endocrine effects*. No special studies to investigate the potential for estrogenic or other endocrine effects of flufenpyr-ethyl 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 flufenpyr-ethyl 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 flufenpyr-ethyl. EPA has not had the opportunity to review the toxicity studies on flufenpyr-ethyl and has not established toxic endpoints of concern for use in risk analyses. For chronic oral exposure, Valent has chosen the NOAEL from the second rat reproduction study of 100 ppm (5 mg/kg/day nominal) as the toxic endpoint. There is no study with flufenpyr-ethyl that showed toxicity that could be associated with a single, or acute, oral exposure. Therefore no acute endpoint could be identified, and no acute oral risk analyses are performed. The chronic RfD using the standard 100-fold uncertainty factor is 0.05 mg/kg/day, and because there is no evidence of enhances susceptibility of infants and children, the FQPA extra 10-fold uncertainty factor is removed. Thus, the Population Adjusted Dose for chronic oral exposure (cPAD) used in these risk assessments is 0.05 mg/kg/day.

i. *Food—a. Acute dietary exposure*. There is no acute oral toxic endpoint identified, and so no acute exposure and risk analysis was performed.

b. Chronic dietary exposures to flufenpyr-ethyl residues were calculated for the U.S. population and 25 population subgroups. This Tier I analysis includes residue contribution from the field corn, soybean and sugarcane uses and assumes tolerance-level residues and 100% of the crops treated. The results from several representative subgroups are listed below. For all population subgroups, chronic dietary exposure was below 0.2% of the cPAD. Generally, the Agency has no cause for concern if total chronic exposure to residues contributed by published and proposed tolerances is less than 100% of the cPAD.

TIER I—CALCULATED CHRONIC DIETARY EXPOSURES TO THE TOTAL U.S. POPULATION AND SELECTED SUB-POPULATIONS TO FLUFENPYR-ETHYL RESIDUES IN FOOD (cPAD = 0.05 MG/KG/DAY)

Population Subgroup	Exposure (mg/kg/day)	Percent of cPAD
Total U.S. population	0.000025	0.05
Males (13 - 19 years)	0.000032	0.06
Females (13 + (nursing))	0.000019	0.04
Females (13 + (pregnant/not nursing))	0.000021	0.04
Children (7 - 12 years)	0.000043	0.09
Children (1 - 6 years)	0.000056	0.11
All infants (< 1 year)	0.000067	0.13
Non-nursing infants	0.000082	0.16
Nursing infants	0.000017	0.03

ii. *Drinking water*. Since flufenpyr-ethyl is applied outdoors to growing agricultural crops and can be applied by aircraft, the potential exists for the parent or its metabolites to reach ground water or surface water that may be used for drinking water. Because of the physical and environmental fate properties of flufenpyr-ethyl, it is unlikely that flufenpyr-ethyl or its metabolites can leach to potable ground water. To quantify potential exposure from drinking water, surface water concentrations for flufenpyr-ethyl were estimated using Generic Expected Enviornmental Concentration (GENEEC 1.2.). The 56–day average GENEEC concentration was 0.027 ppb. Using standard assumptions about body weight and water consumption, the maximum chronic exposure from this drinking water would be 0.000000763 and 0.00000267 mg/kg/day for adults and children, respectively; 0.0053 percent of the cPAD of 0.05 mg/kg/day for children. The contribution of drinking water to chronic dietary exposures is much smaller than that from food, and adds negligible risk.

2. *Non-dietary exposure*. Flufenpyr-ethyl is proposed only for agricultural uses and no homeowner, turf, or industrial 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.

There are other pesticidal compounds that are structurally related to flufenpyr-ethyl and may have similar effects on animals. In consideration of potential cumulative effects of flufenpyr-ethyl 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 flufenpyr-ethyl would be cumulative with those of other chemical compounds. Thus, only the potential risks of flufenpyr-ethyl have been considered in this assessment of aggregate exposure and effects.

Valent will submit information for EPA to consider concerning potential cumulative effects of flufenpyr-ethyl consistent with the schedule established by EPA in the **Federal Register** of August 4, 1997 (62 FR 42020) (FRL–5734–6) and other subsequent EPA publications pursuant to the Food Quality Protection Act of 1996 (FQPA).

E. Safety Determination

The FQPA introduced a new standard of safety, a reasonable certainty of no harm. To make this determination, at this time the Agency should consider only the incremental risk of flufenpyr-ethyl in its exposure assessment. Since the potential chronic and acute exposures to flufenpyr-ethyl are small (<100% of cPAD and aPAD) the provisions of the FQPA will not be violated.

1. *U.S. population*—i. *Acute risk*. There is no acute oral toxic endpoint available, so no risk analysis was performed.

ii. *Chronic risk*. Using the dietary exposure assessment procedures described above for flufenpyr-ethyl, calculated chronic dietary exposure resulting from residue exposure from proposed uses of flufenpyr-ethyl is minimal. The estimated chronic dietary exposure from food for the overall U.S. population and many non-child/infant subgroups is 0.064 to 0.042% of the cPAD. Addition of the small but worse case potential exposure from drinking water (calculated above) increases exposure by only 0.000000763 mg/kg/day (0.0053% of cPAD) and the maximum occupancy of the cPAD from 0.064% to 0.066%. Generally, the Agency has no cause for concern if total residue contribution is less than 100% of the cPAD. It can be concluded that there is a reasonable certainty that no harm will result to the overall U.S. population and many non-child/infant subgroups from aggregate, chronic exposure to flufenpyr-ethyl residues.

2. *Safety factor for infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of flufenpyr-ethyl, FFDCA section 408 provides that EPA shall apply an additional margin of safety, up to 10-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.

In assessing the potential for additional sensitivity of infants and children to residues of flufenpyr-ethyl, EPA considers the completeness of the human health effects data, particularly those studies that evaluate toxicity to reproduction and to fetal and developing young experimental animals. These studies include developmental toxicity studies in the rat and rabbit and a 2–generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to prenatal and postnatal effects from exposure to the pesticide, information on the reproductive capability of both male and female mating animals and data on systemic toxicity.

3. *Developmental toxicity*. Flufenpyr-ethyl is not a developmental toxicant in either rats or rabbits. In the developmental toxicity study conducted with rats, the NOAEL for both maternal and developmental toxicity was found to be 1,000 mg/kg/day, the highest dose tested.

Flufenpyr technical was tested in two developmental toxicity studies in rabbits because of unexpected maternal mortality. In the first study maternal mortality occurred at the two highest doses tested. In surviving animals and their fetuses, there were no adverse effects. Based on these results, the maternal toxicity NOAEL was 100 mg/kg/day and the developmental toxicity NOAEL was 1,000 mg/kg/day. In the second study maternal mortality again occurred, but at all dose levels. Detailed examination of most these animals showed the cause of death to be test material aspiration into the lungs. There were no other adverse effects in the surviving dams or fetuses. The NOAEL for this study and the overall NOAEL for rabbits was found to be 300 mg/kg/day (maternal) and 1,000 mg/kg/day (developmental).

4. *Reproduction*. In the rat reproduction study, flufenpyr-ethyl technical was administered for 2–generations. Parental toxicity (kidney and liver effects) was observed at all dose levels, although the effects at the low dose were minimal. There were no effects at any dose on any reproductive parameter. Based on the results of this study, the NOAEL for parental toxicity was considered to be less than 200 ppm. The NOAEL for reproductive and neonatal toxicity was considered to be 20,000 ppm.

A second 1–generation reproduction study was performed to establish a clear NOAEL for adult kidney lesions using the dose levels of 20, 50 and 100 ppm. The results of the study indicate that the NOAEL for histological changes in the kidneys for F_1 male rats was 100 ppm. No other treatment-related findings were noted at any dose level indicating 100 ppm as the NOAEL for treatment and reproductive effects evaluated in the study.

A mechanistic study was also conducted to investigate the reproducibility and reversibility of the kidney lesions observed in the initial 2–generation reproduction study. In the first study, the effects observed at 200 ppm in the F_1 males, basophilic tubules and interstitial inflamation, were minimal but slightly increased in incidence and severity and a slight increase in interstitial fibrosis of the cortex was also observed. In this mechanistic study, using dose levels of 0 and 2,000 ppm, the NOAEL for histological changes in the kidneys of F_0 and F_1 male rats and reproductive effects was 2,000 ppm. The histological changes seen in the kidneys in the original study was not reproducible.

The toxicological data base for evaluating prenatal and postnatal toxicity for flufenpyr-ethyl is complete with respect to current data requirements. Valent concludes that there is no evidence that fetal, or developing young experimental animals are any more susceptible to the effects of flufenpyr-ethyl than adult animals. Therefore there is no need for an extra FQPA uncertainly factor to be further protective of infants and children.

5. *Acute exposure and risk*. There is no acute oral toxic endpoint available, so no risk analysis was performed.

6. *Chronic exposure and risk*. Using the conservative exposure assumptions described above, the percentage of the cPAD that will be utilized by dietary (food only) exposure to residues of flufenpyr-ethyl ranges from 0.16% for non-nursing infants, to 0.03% for nursing infants. Adding the worse case potential incremental exposure to infants and children from flufenpyr-ethyl in drinking water (0.00000267 mg/kg/day) increases the aggregate, chronic dietary exposure by 0.0053% The addition of the exposure attributable to drinking water increases the occupancy of the cPAD for Non-Nursing Infants from 0.164 to 0.169 percent. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. It can be concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate, chronic exposure to flufenpyr-ethyl residues.

7. *Safety determination summary*. Aggregate chronic dietary exposure to various sub-populations of children and adults demonstrate acceptable risk. Chronic dietary exposures to flufenpyr-ethyl occupy considerably less than 100% of the cPAD. Acute dietary risk to children from flufenpyr-ethyl should not be of concern. Further, flufenpyr-ethyl 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 exposures to flufenpyr-ethyl residues resulting from proposed uses. There is no evidence that acute oral exposures to flufenpyr ethyl causes appreciable toxicity, and no exposure and risk analyses are appropriate.

F. International Tolerances

There are no existing U.S. tolerances or Codex Maximum Residue Limits for flufenpyr-ethyl.

[FR Doc. 03–16033 Filed 6–24–03; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPPT–2003–0031; FRL–7315–1]

Certain New Chemicals; Receipt and Status Information

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: Section 5 of the Toxic Substances Control Act (TSCA) requires any person who intends to manufacture (defined by statute to include import) a new chemical (i.e., a chemical not on the TSCA Inventory) to notify EPA and comply with the statutory provisions pertaining to the manufacture of new chemicals. Under sections 5(d)(2) and 5(d)(3) of TSCA, EPA is required to publish a notice of receipt of a premanufacture notice (PMN) or an application for a test marketing exemption (TME), and to publish periodic status reports on the chemicals under review and the receipt of notices of commencement to manufacture those chemicals. This status report, which covers the period from May 26, 2003 to June 2, 2003, consists of the PMNs and