Users of these products who desire continued use should contact the applicable registrant before January 3, 2005, to discuss withdrawal of the application for amendment. This 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.

TABLE 2.—REGISTRANTS REQUESTING AMENDMENTS TO DELETE USES IN CERTAIN PESTICIDE REGISTRATIONS

EPA Company No.	Company Name and Address
7501	Gustafson, LLC 1400 Preston Road, Suite 400, Plano, TX 75093
62719	Dow Agrosciences, LLC 9330 Zionsville Road, Indianapolis, IN 46268

IV. 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.

V. Procedures for Withdrawal of Request

Registrants who choose to withdraw a request for use deletion must submit the withdrawal in writing to Katie Hall using the instructions listed under **FOR FURTHER INFORMATION CONTACT**. The Agency will consider written withdrawal requests postmarked no later than January 3, 2005.

VI. Provisions for Disposition of Existing Stocks

Existing stocks are those stocks of registered pesticide products which are currently labeled in the United States and which have been packaged, labeled, and released for shipment prior to the effective date of the cancellation action.

The Agency intends to authorize the registrants to sell or distribute product under the previously approved labeling through December 31, 2004, after approval of the revision, unless other restrictions have been imposed, as in special review actions. Stocks in the hands of dealers and distributors other than the registrants could be sold or distributed until December 31, 2005. The Agency anticipates that use of the products proposed for cancellation will end December 31, 2006. Any future tolerance modifications would be calculated from the December 31, 2006, date. EPA will issue a **Federal Register** notice with the cancellation order and final existing stock provisions.

List of Subjects

Environmental protection, Pesticides and pests.

Dated: June 18, 2004.

Debra Edwards,

Director, Special Review and Reregistration Division, Office of Pesticide Programs.

[FR Doc. 04–15209 Filed 7–6–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0180; FRL–7364–8]

Tribenuron Methyl; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket identification (ID) number OPP–2004–0180, must be received on or before August 6, 2004.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: James A. Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5697; e-mail address: *tompkins.jim@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are a 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–2004–0180. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1801 South Bell St., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

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

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

facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in EPA's 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.1. 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–2004–0180. 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–2004–0180. 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–2004–0180.

3. *By hand delivery or courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1801 South Bell St., Arlington, VA, Attention: Docket ID Number OPP–2004–0180. 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 pesticide 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 pesticide petition. Additional data may be needed before EPA rules on the pesticide petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: June 22, 2004.

Lois Rossi,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner's summary of the pesticide petition (PP) 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.

E. I. du Pont de Nemours and Company

PP 0F6135

EPA has received a pesticide petition (0F6135) from E. I. du Pont de Nemours and Company, DuPont Agricultural Products, Barley Mill Plaza, Wilmington, DE 19880–0038 proposing, pursuant to FFDCA section 408(d), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of tribenuron methyl (methyl 2-[[[[(4-methoxy-6-methyl-1,3,5- triazin-2-yl)methylamino] carbonyl]amino]sulfonyl]benzoate) in or on the raw agricultural commodity imazethapyr tolerant canola at 0.02 parts per million (ppm), cotton seed at 0.02 ppm, cotton gin trash at 0.02 ppm, and Crop Development Center (CDC) triffid flax at 0.02 ppm. EPA has determined that the pesticide 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 pesticide petition. Additional data may be needed before EPA rules on the pesticide petition.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues of tribenuron methyl is adequately understood. Tribenuron methyl is rapidly metabolized in wheat plants with a half-life of less than 4 days. A major metabolic reaction was *N*-demethylation of tribenuron methyl to form metsulfuron methyl. Metsulfuron methyl was further metabolized, primarily through rapid hydroxylation of the phenyl ring, followed by conjugation with glucose. Hydrolysis of the sulfonylurea bridge of tribenuron methyl to release sulfonamide and triazine amine was also observed. The sulfonamide may be further metabolized to hydroxylated sulfonamide or cyclized to saccharin. The presence of α-hydroxy triazine amine, *N*-demethyl triazine amine, and *O*-demethyl *N*-demethyl triazine amine demonstrated that the released triazine moiety of tribenuron methyl was also extensively degraded in wheat. Metabolism studies were conducted with radioactive ^{14}C-tribenuron methyl on wheat under field conditions. Wheat plants were treated with 72–75 gram (g) active ingredient (a.i.)/health advisory (ha) of ^{14}C-phenyl and ^{14}C-triazine labeled tribenuron methyl at the tillering stage. Samples were harvested 0, 4, 8, 14, 21, 28, and 63 days after treatment. Total ^{14}C-residue levels in the foliage declined rapidly from 5.5 ppm at time of application to 0.55 ppm in the mature straw and 0.05 ppm in the grain (^{14}C-phenyl), and from 4.2 ppm to 0.37 ppm in the mature straw and 0.01 ppm in the grain (^{14}C-triazine). Analysis of the wheat foliage and straw extracts by high performance liquid chromotography (HPLC) and threshold level ceiling (TLC) revealed that tribenuron methyl was rapidly and extensively metabolized. Metabolites were identified based on chromatography with authentic standards. The major metabolites were the glucose conjugate of hydroxylated metsulfuron methyl, hydroxylated saccharin, the glucose conjugate of hydroxylated saccharin, saccharin, triazine amine, *O*-demethyl triazine amine, and *O*-hydroxy triazine amine.

A metabolism study was conducted with ^{14}C-tribenuron methyl on acetolactate synthase (ALS)-tolerant canola. ^{14}C-tribenuron methyl was applied at 25 g/ha as a topical spray treatment at 2 true leaf stage to bolting. Whole canola plants were harvested at 0, 2 days, 35 days, and at maturity, 78 days after treatment. Total reactive residue (TRR) in canola foliage, when expressed as tribenuron methyl equivalents, declined from, on average, 0.26 ppm at day 0 to 0.04 ppm at day 35. TRR in immature 35–day canola seed pods was not higher than 0.04 ppm, and was 0.02 ppm in 78–day seed samples. ^{14}C-tribenuron methyl accounted for greater than 81% of the radioactive residue in the 0 to 2–day foliage samples. Other minor components were polar metabolites or conjugates, each less than 10% of the TRR. No single component in the polar metabolites exceeded 0.01 ppm. In the 35–day foliage samples, ^{14}C-tribenuron methyl accounted for only about 11–25.5% of the TRR which is less than 0.01 ppm. The average half life for ^{14}C-tribenuron methyl was 15 days. Several metabolic processes in the foliage are involved. They include a hydrolytic cleavage of tribenuron methyl as well as *N*-demethylation of tribenuron methyl. Other demethylation and hydroxylation processes continued up to final harvest. The results of the study suggest that the tribenuron methyl metabolic process in canola follows a typical plant metabolism pattern, and no accumulation of tribenuron methyl is anticipated in canola when it is used in accordance with the proposed labels.

A metabolism study was conducted to determine the nature and magnitude of the residues of tribenuron methyl in

cotton plants after treatment with 2-^{14}C-tribenuron methyl. Soil treatments were applied at 0.3 ounce a.i./acre as a direct spray in an aqueous suspension containing inert dry-flowable (DF) formulation ingredients. The application was performed immediately after planting to provide the data for the shortest anticipated time between application and planting. No terminal residues at or above 0.01 ppm were observed in any triazine-label treated fractions of mature cotton after treatment with tribenuron methyl. No detectable residues were found in the undelinted seed and very low residues of 0.028 ppm were observed in the gin trash after treatment. Tribenuron methyl and its known metabolites are not expected to be present in the terminal residues in gin trash or undelinted seed, when applied according to the proposed label.

A confined crop rotation study with ^{14}C-phenyl tribenuron methyl was conducted using cabbage, red beets, sorghum, soybeans, and wheat planted in pots of sandy loam soil 30 and 120 days following a single application of ^{14}C-phenyl-labeled tribenuron methyl. For the 30–day aging period, samples from both treated and control crops were taken at 28, 49, and 67 days after planting with additional samples taken from the sorghum and soybeans plantings at 90 and 115 days. At maturity, all remaining plants were harvested and subdivided into edible and nonedible portions. Harvest dates, in days after planting were: 90 days (cabbage), 115 days (beets and wheat), and 168 days (sorghum and soybeans). Samples from all crops from the 120–day aging study were taken at 28, 48, 69, and 90 days (maturity for beets, cabbage, and wheat,) and 120 days and 169 days (maturity for sorghum and soybeans). Tribenuron methyl dissipated rapidly in the soil with none of the intact material detected after the 30–day aging period. The major radiolabeled residue extracted from the soil was saccharin which remained in the soil at very low levels throughout the study. Some accumulation of total radioactive residues was apparent in the mature sorghum foliage, soybean, and wheat due to the dehydrated nature of samples harvested. The major residue in the plants was identified as saccharin.

A confined crop rotation study with ^{14}C-triazine tribenuron methyl was conducted using cabbage, red beets, and sorghum. Sandy loam soil was treated at 32 g a.i./ha ^{14}C-phenyl tribenuron methyl in the greenhouse. Rotational crops were sown 30 and 120 days post-treatment. Tribenuron methyl degraded rapidly in the soil with no detectable intact material present 30 days post-treatment. The major radiolabeled metabolite was the triazine amine. No significant accumulation (less than 0.01 ppm) of radiolabeled materials from the soil were observed in the mature crops of cabbage foliage. Some accumulation of the radioactivity was observed in the mature beet foliage in the 30–day study (0.029 ppm) and the 120–day study (0.011 ppm). Major metabolites were *N*-demethyl triazine amine and *O*-hydroxy triazine amine. Accumulation of radioactivity was observed in the mature sorghum straw due to the dehydrated nature of this plant tissue at harvest. Levels of radiolabeled materials detected were 0.108 and 0.057 ppm in the 30–day and 120–day studies. The major metabolites were highly polar materials. Tribenuron methyl rapidly decomposes in soil to the triazine amine, which is then degraded, not accumulated, in plants.

Based on the absence of detectable residue in food commodities (barley and oat grain) and on the expected low residue levels of individual substances in feed items (straw) under normal conditions, and the Residue Chemistry Test Guidelines (OPPTS 860.1300(c)(2)(D)(ii) which states that; one metabolism study will be required for each of the crop groups defined in 40 CFR 180.34(f) except for herbs and spices, a plant metabolism study in barley and oat was not required. Additionally, based on the results of three metabolism studies on dissimilar crops having similar metabolic routes (canola, cotton, and wheat), an additional metabolism study for flax is not required.

2. *Analytical method.* There is an analytical method for determination of residues of tribenuron methyl in barley, wheat grain, straw, and wheat grain forage samples. The method is based on extraction of tribenuron methyl from crops with acetonitrile, and cleanup on a silica cartridge. Final determination is by normal phase liquid chromatography using a photoconductivity detector. Recoveries for grain, straw, and green forage samples fortified between 0.01 and 0.10 ppm averaged 88% with a standard deviation of 14%. The lower level of quantitation (LOQ) for grain and green forage is 0.01 ppm and for straw it is 0.02 ppm.

Another analytical method for determination of tribenuron methyl in wheat grain and straw uses 2 HPLC with ultra-violet (UV) detection at 254 nanometer (nm). The method provides a means to quantitate tribenuron methyl in these matrices at levels as low as 0.05 ppm based on a 5–gram sample.

An analytical method to detect tribenuron methyl at a level of 0.02 ppm or above in grass seed, straw, and seed screenings consists of using gel permeation chromatography and solid-phase extraction. Purified column eluent is taken to dryness, dissolved in ethyl acetate, and analyzed by capillary gas chromatography using a mass spectral detector. In fortification recovery trials, an average recovery of 87.6% with a standard deviation of 21% was obtained for 18 grass seed samples over a fortification range of 0.02 to 0.06 ppm. Tribenuron methyl residues in canola and flax samples were determined by an analytical method based on the use of liquid chromatography with eluent and column switching with photometric detection at 258 nm at levels as low as 0.02 ppm LOQ using a 5–gram sample.

Residues in cotton seed and gin trash were determined based on the use of column-switching liquid chromatography with detection via positive ion electrospray mass spectroscopy. The LOQ was determined to be 20 nanograms (ng)/g and the LOD was estimated to be 6 ng/g, based on a 5–gram sample.

3. *Magnitude of residues*—i. *Wheat, barley, grain, and straw.* A study was conducted to determine the extent of residues of tribenuron methyl in wheat when applied at the maximum use rate (0.25 ounce a.i./acre) 40 days before maturity. Samples of mature wheat, grain, and straw were taken from treated and control plots at pre-harvest intervals (PHI) ranging from 25 to 40 days after the test substance was applied. A 2–step HPLC method was used to determine tribenuron methyl at levels as low as 0.0075 ppm in wheat grain based on a 20–gram sample, and 0.014 ppm in wheat straw based on a 10–gram sample. No grain or straw samples showed quantifiable or detectable residues of tribenuron methyl.

A study was conducted to determine the extent of residues of tribenuron methyl in barley when applied at the maximum use rate (0.25 ounce a.i./acre) 40 days before maturity. Samples of mature barley grain and straw were taken from each plot at PHI ranging from 24 to 43 days after the test substance was applied. A 2–step HPLC method was used to determine tribenuron methyl at levels as low as 0.0066 ppm in barley grain based on a 20–gram sample, and 0.013 ppm in barley straw based on a 10–gram sample. One grain sample showed a detectable residue (0.0064 ppm) of tribenuron methyl, which is below the established grain tolerance of 0.05 ppm. A straw sample from one of the sites

contained tribenuron methyl at 0.034 ppm, which is below the established straw tolerance of 0.10 ppm. The remaining grain and straw samples showed no detectable or quantifiable residues of tribenuron methyl.

The results of the analyses of grain and straw from wheat and barley show that no residues were found in either grain or straw from plants treated at or below the maximum recommended application rate (0.25 ounce a.i./acre). The PHI ranged from 42–140 days (0.020 ppm–0.050 ppm LOQ). A small percentage of plants treated at higher rates showed some residues in straw.

ii. *Forage, grass, and hay*. Established plots of bluegrass, tall fescue, and perennial ryegrass grown for production of grass seed were each treated with 0.25 ounce a. i./acre and 0.50 ounce a.i./acre of ''express'' herbicide (formulated as a 75 DF water-dispersible granule). A total of 4 test sites were included in the study—2 for bluegrass and 1 each for tall fescue and perennial ryegrass. Sampling PHI ranged from 56 to 85 days. Reliable detected residues of tribenuron methyl (0.016 ppm or above) were not found in any crop fraction from any test site, with one exception of a residue level of 0.004 ppm for the 0.25 ounce a.i./acre treatment, and 0.006 ppm for the 0.50 ounce a.i./acre treatment. An attempt to reconfirm this result by reextracting a second screening waste sample failed to confirm the presence of these tribenuron methyl residues.

iii. *Grain, oat, and straw*. A study was conducted to determine the extent of residues of tribenuron methyl in oats when applied at 1 to 2 times the maximum use rate approximately 40 days before harvest. Samples of mature oat grain and straw were taken from both treated and control plots at PHI ranging from 39 to 57 days after the application of the test substance. A 2–step HPLC method was used to detect tribenuron methyl residues in oat grain at levels as low as 0.0055 ppm based on a 20–gram sample and in oat straw at levels as low as 0.018 ppm based on a 10 gram sample. Residues of tribenuron methyl in oat grain from oats treated at 1x and 2x were below the quantitation level of 0.013 ppm and 0.01 ppm, respectively. The residues of tribenuron methyl in oat straw were below the quantitation level of 0.018 ppm and 0.04 ppm respectively and also below reported detection level of 0.009 ppm and 0.018 ppm, respectively, in oat straw from oats treated at 1x and 2x rates.

iv. *Canola and flax*. Magnitude of residue studies were conducted on seed fractions of canola varieties containing the Smart™ trait and CDC triffid flax. The post-emergent broadcast application of Refine Extra® herbicide at a use rate of 15 to 30 g a.i./ha (representing 5 to 10 g a.i./ha of tribenuron methyl) which represents 1 to 2 times the proposed use rate for Refine Extra® herbicide on these canola and flax varieties. The study included treatment of 15 sites for canola containing the Smart™ trait and 11 sites for CDC triffid flax. No tribenuron methyl residues were found above the LOQ of 0.02 ppm in any seed samples treated with the test substance at a use rate of 15 to 30 g a.i./ha Refine Extra® herbicide.

v. *Cotton seed and gin trash*. Magnitude of residue studies were also conducted to determine residues of tribenuron methyl in cotton seed and cotton gin trash at 9 test sites. The study consisted of 3 treatments:

- One broadcast application at 0.45 ounce a.i./acre, applied approximately 14 days prior to planting.
- One broadcast application at 0.45 ounce a.i./acre, applied pre-plant, on the day of planting.
- One broadcast application at 2.25 ounce a.i./acre, applied pre-plant, the day of planting.

The anticipated target PHI was approximately 120 days after the last application of the test substance; actual PHIs ranged from 123 to 196 days. The experimentally determined LOQ was 20 parts per billion (ppb) for both analytes. The LOD was estimated to be 6 ppb. No tribenuron methyl residues were found above the LOQ of 0.02 ppm in any cotton seed and cotton gin trash samples treated with the test substance.

B. Toxicological Profile

1. *Acute toxicity*. Based on EPA criteria, technical tribenuron methyl is in acute toxicity category IV for oral and inhalation routes of exposure, and for skin irritation. Tribenuron methyl is in acute toxicity category III for the dermal route of exposure, and for eye irritation. It is not a skin sensitizer.

Acute oral toxicity in rats	Lethal dose $(LD)_{50}$ >5,000 milligrams/kilogram (mg/kg)
Acute dermal toxicity in rabbits	LD_{50} >2,000 mg/kg
Acute inhalation toxicity in rats	Lethal concentration $(LC)_{50}$ >5.0 mg/Liter (L)
Primary eye irritation in rabbits	Moderate effects reversed within 3 days
Primary dermal irritation in rabbits	Slight skin irritant
Dermal sensitization	Non-sensitizer

2. *Genotoxicity*. Technical tribenuron methyl has shown no genotoxic or mutagenic activity in the following *in vitro* and *in vivo* tests:

In vitro mutagenicity Ames Assay	Negative
In vitro mutagenicity chinese hampster ovary/hypoxanthine guanine phophoribosyl transferase (CHO/HGPRT) Assay	Negative
In vitro unscheduled deoxyribonucleic acid (DNA) synthesis	Negative
In vivo Cytogenetic	Negative
In vivo micronuclei induction (mouse)	Negative

Tribenuron methyl was negative for mutagenicity in an *in vitro* bacterial gene mutation assay using *Salmonella typhimurium* and in an *in vitro* mammalian cell gene mutation assay using chinese hampster ovary (CHO) cells. In cultured primary rat hepatocytes *in vitro*, thifensulfuron methyl was negative for the induction of unscheduled DNA synthesis.

In a test measuring clastogenic damage *in vivo*, tribenuron methyl was negative for the induction of chromosome aberrations in male and female rat bone marrow cells. A study measuring chromosome damage *in vivo* was conducted. The study included the evaluation of micronuclei in bone marrow polychromatic erythrocytes of male and female mice. The result was negative when exposures were conducted at 5,000 mg/kg body weight.

3. *Reproductive and developmental toxicity*. On long-term dietary administration, tribenuron methyl did not affect the reproduction or lactation performance of rats. Developmental studies in the rat and rabbit by gavage administration indicated that tribenuron methyl did not present a unique toxic risk to the fetus. Embryo-fetal and maternal NOAELs were equivalent in all cases.

There were no effects in reproduction or lactation in rats in a 1–generation reproduction study with rats fed for 90 days with diets that contained 0; 100; 1,750; or 5,000 ppm a.i. The no observed effect level (NOEL) was 100 ppm (7 mg/kg/day for males and 8 mg/

kg/day for females) based on lower mean dam and pup body weights for the intermediate and high dose groups.

There were no effects on fertility observed in a 2–generation reproduction study, in rats fed for at least 90 days with diets that contained 0, 25, 250, or 1,000 ppm a.i. The NOEL was 25 ppm based on lower body weights for the dams and offspring at 250 and 1,000 ppm. There were no differences attributed to administration of tribenuron methyl in the number of litters produced or other indices of reproductive performance. No compound-related effects on male fertility were noted. No effect on the number of pups born or pup survival were observed in any tribenuron methyl treated group.

In a study to evaluate developmental toxicity potential in rats, tribenuron methyl did not produce birth defects after administering via oral intubation to pregnant rats dosage levels of 0, 20, 125, and 500 mg/kg/day. The NOEL for this study was 20 mg/kg/day for both maternal and developmental toxicity. This was based on maternal effects at the 125 and 500 mg/kg/day. The effects included decreased body weight gain and food consumption and an increased incidence of excess salivation. Fetal effects included decreased body weights and increased number of resorptions (only at (hightest dose tested HDT)). In the rabbit developmental toxicity study, rabbits were fed dosage levels of 0, 5, 20, and 80 mg/kg/day. The NOEL for maternal and developmental toxicity was 20 mg/kg/day. This was based on maternal effects which included decreased feed consumption and an increased incidence of abortions (at the HDT). Fetal effects included slightly reduced body weights at 80 mg/kg/day.

4. *Subchronic toxicity*. The most sensitive species to subchronic exposure of tribenuron methyl was the rat. In the rat study, rats were fed dosage levels of 0; 100; 1,750; or 5,000 ppm tribenuron methyl for 90 days. The findings show that the NOEL for tribenuron methyl was 100 ppm for both male and female rats (90–day dietary). This concentration is equivalent to 7 and 9 mg/kg/day in male and female rats, respectively. The NOEL was based on the decreased body weight and decreased feed consumption noted in the 1,750 and 5,000 ppm groups. The NOEL for the 90–day mouse feeding study was 500 ppm (70 mg/kg/day for males and 90 mg/kg/day for females) based on liver and spleen effects at 1,250; 2,500; and 5,000 ppm at 4 weeks. An increase in liver weights at 2,500 ppm was noted with no histologic effects at any level. The NOEL for subchronic (90–day dietary) exposure in dogs was 500 ppm (15.1 mg/kg/day for male and 14.9 mg/kg/day for female dogs). This was based on lower mean body weights of male dogs fed the 2,500 ppm diet. A specific target organ was not identified in any of the species studied.

5. *Chronic toxicity*. The NOEL for chronic (18–month dietary) exposure in mice was 200 ppm (equivalent to 25 and 31 mg/kg/day in male and female mice, respectively). This was based on lower body weights for mice in the high-dose group (1,500 ppm). There were no neoplastic or other histopathological effects associated with this compound and no target organ was identified. Additionally, no evidence of tribenuron methyl induced oncogenicity was observed in the mouse.

The NOEL for chronic (2–year dietary) exposure in rats was 25 ppm (0.95 and 1.2 mg/kg/day for male and female rats, respectively). Lower body weights, which paralleled lower food consumption and organ weight effects, were observed in the 250 and 1,250 ppm groups. There were no clinical or histopathological effects associated with these organ weight effects. The incidence of mammary adenocarcinomas was greater than controls for female rats in the 1,250 ppm group. This effect was only observed in this high-dose group and under conditions of significant physiological stress (body weights for female rats were 42% lower than the controls).

In a 1–year feeding study in dogs, the NOEL was determined by DuPont to be 250 ppm (8.16 and 8.18 mg/kg/day for male and female dogs, respectively). This was based on slightly lower body weights and increased serum creatinine concentrations for dogs in the high-dose group (1,500 ppm). Upon review by EPA, the NOEL was set at 25 ppm (0.79 mg/kg/day). There were no neoplastic or other histopathological effects associated with compound administration.

6. *Animal metabolism*. Metabolism of tribenuron methyl was evaluated in rats using both phenyl and triazine labeling. Tribenuron methyl was extensively and rapidly converted to polar metabolites and primarily excreted in the urine and feces. Urinary excretion accounted for 2 to 4 times the amount of radiolabel excreted via feces in all groups. Essentially all of the tribenuron methyl and its metabolites were excreted in the urine and feces of the rat within 96 hours after dosing. Levels of radiolabeled residues in tissues were correspondingly higher in those groups with slower elimination kinetics, but no evidence of bioconcentration was seen. None of the dosed label was expired as carbon dioxide or volatile metabolites.

The average excretion half-life values for male and female rats in the low-dose group (20 mg/kg) were approximately the same (26–33 hours) and independent of dietary preconditioning. The average excretion half-lives for male and female rates in the high-dose groups (1,700; 1,800; and 2,000 mg/kg) were approximately 51–54 hours (males) and 68–96 hours (females). These results indicate that the metabolism of tribenuron methyl in male and female rats is qualitatively similar; however, female rats metabolize and excrete this product much slower than male rats at the high doses. The low residual radioactivity in the rat indicated that tribenuron methyl does not covalently bind to tissue macromolecules. Based on these data, the body burden of this compound is not expected to increase significantly upon repeated, long-term administration.

The major metabolites of tribenuron methyl are those expected from the enzymatic hydroxylation and dealkylation activities of the hepatic microsomal mixed function oxidase system. The major urinary metabolites were identified as metsulfuron methyl and saccharin (phenyl labeled groups) and metsulfuron methyl and *O*-demethyl triazine amine (triazine labeled groups); no evidence of glucuronide or sulfate conjugation was seen.

Results from a metabolism study with 2 radioactive forms of tribenuron methyl (^{14}C-triazine and ^{14}C-phenyl) in lactating goats show that most of the dosed radioactivity was recovered in the urine (61–71%) and feces (15–20%). In the urine, intact tribenuron methyl and metsulfuron methyl accounted for 17–23% and 20–22% of the administered dose, respectively. The third major component in phenyl-dosed goat urine was saccharin (23.5% of the dose); the third major metabolite in the triazine-dosed goat urine was *O*-demethyl *N*-demethyl triazine amine (10.9%). The highest levels of residues observed in the milk were 0.09 ppm (tribenuron methyl equivalents) from the triazine-dosed goat, and 0.006 ppm from the phenyl-dosed goat. Recoveries of the administered dose were 82.2% for the goat given the triazine label, and 86.8% for the goat dosed with the phenyl label. Throughout the dosing phase, the goats did not display any signs of toxicity, and there was no effect on milk production.

There were no significant levels of unique plant metabolites of thifensulfuron methyl found in food or feed products at crop maturity. Hence,

toxicity testing of other degradation products of thifensulfuron methyl is not needed.

7. *Metabolite toxicology*. There is no evidence that the metabolites of tribenuron methyl as identified in either the plant or animal metabolism studies are of any toxicological significance.

8. *Endocrine disruption*. In a previous 2–year feeding study, female rats fed, 1,250 ppm tribenuron methyl had an approximately 3–fold increase in mammary adenocarcinoma incidence when compared to control. This concentration of tribenuron methyl exceeded the maximum tolerated dose, producing a 43% decrease in body weight. In contrast, an 18–month feeding study demonstrated that tribenuron methyl was not oncogenic in mice. Because tribenuron methyl is also negative in five short-term tests for genotoxicity, a non-genotoxic mechanism was investigated. A study was designed to investigate whether tribenuron methyl can alter the hormonal system of female rats, which would support a non-genotoxic mechanism for the tribenuron methyl-induced mammary adenocarcinoma. The integrity of the endocrine system was assessed by monitoring the estrous cycle, measuring serum hormone levels, characterizing the estrogen and progesterone receptors from the uterus and mammary gland, and weighing reproductive organs.

The data from this study indicate that the endocrine system may have been affected at a relatively high dose, 5,000 ppm. These data further suggest that the hormonal effects served to enhance the growth of preinitiated mammary cells in this susceptible rat strain. Such hormone-mediated effects are considered to have a threshold below which growth of mammary tissue will not be affected. Adequate margins of safety protect humans from these threshold effects.

C. Aggregate Exposure

1. *Dietary exposure*. The chronic reference dose (RfD) of 0.008 mg/kg/day is based on the NOEL of 0.79 mg/kg/day from a 1–year dog feeding study and a 100X safety factor (SF). The acute RfD of 0.20 mg/kg/day is based the NOEL of 20 mg/kg/day from the rabbit and rat developmental studies and a 100X safety factor.

i. *Food*. Chronic dietary exposure assessment. Chronic dietary exposure, resulting from the proposed use of tribenuron methyl on barley, canola, cotton, flax, grass, oats, and wheat, is well within the acceptable limits for all sectors of the population, as predicted by the chronic module of the Dietary Exposure Evaluation Model ((DEEM), Novigen Sciences, Inc., 1999 Version 6.74). The percentage or proportion of a crop that is treated can have a significant effect on the exposure profile. In this case, it was assumed for the crop that 100% was treated with tribenuron methyl. Based on a comparison with the use profile for most other herbicides, this is an extremely conservative estimate. The predicted chronic exposure for the U.S. population subgroup was 0.000094 mg/kg body weight/day (bwt/day). The population subgroup with the highest predicted level of chronic exposure was the children 1 to 6 years subgroup with an exposure of 0.000213 mg/kg bwt/day. Based on a chronic NOEL of 0.79 mg/kg bwt/day and a 100–fold (SF), the chronic RfD would be 0.008 mg/kg bwt/day. For the U.S. population, the predicted exposure is equivalent to 1.2% of the chronic RfD. For the population subgroup with the highest level of exposure (children 1 to 6 years), the exposure would be equivalent to 2.7% of the chronic RfD. Because the predicted exposures, expressed as percentages of the chronic RfD, are well below 100%, there is reasonable certainty that no chronic effects would result from dietary exposure to tribenuron methyl.

ii. *Acute dietary exposure*. The predicted acute exposure for the U.S. population subgroup was 0.000262 mg/kg bwt/day (95^{th} percentile). The population subgroup with the highest predicted level of acute exposure was the children 1 to 6 years subgroup with an exposure of 0.000475 mg/kg bwt/day (95^{th} percentile). Based on an acute NOEL of 20 mg/kg bwt/day and a 100–fold SF, the acute RfD would be 0.20 mg/kg bwt/day. For the U.S. population the predicted exposure (at the 95^{th} percentile) is equivalent to 0.13% of the acute RfD. For the population subgroup with the highest level of exposure (children 1 to 6 years), the exposure (at the 95^{th} percentile) would be 0.24% of the acute RfD. Because the predicted exposures, expressed as percentages of the acute RfD, are well below 100%, there is reasonable certainty that no acute effects would result from dietary exposure to tribenuron methyl.

iii. *Drinking water*. Surface water exposure was estimated using the Generic Expected Environmental Concentration (GENEEC) model. Ground water exposures were estimated using screening concentration in ground water (SCI-GROW).

EPA uses drinking water levels of comparisons (DWLOCs) as a surrogate measure to capture risk associated with exposure to pesticides in drinking water. A DWLOC is the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses. A DWLOC will vary depending on the residue level in foods, the toxicity endpoint and with drinking water consumption patterns and body weights for specific subpopulations.

The acute DWLOCs are 7 ppm for the U.S. population and 2 ppm for the subpopulation with the highest exposure (children 1 to 6 years). The estimated maximum concentration of tribenuron methyl in surface water 0.7 ppb are derived from GENEEC is much lower than the acute DWLOCs. Therefore, one can conclude with reasonable certainty that residues of tribenuron methyl in drinking water do not contribute significantly to the aggregate acute human health risk.

The chronic DWLOCs are 0.3 ppm for the U.S. population and 0.01 ppm for the subpopulation with the highest exposure (children 1 to 6 years). These DWLOC values are substantially higher than the GENEEC 56–day estimated environmental concentration of 0.3 ppb for tribenuron methyl in surface water. Therefore, one can conclude with reasonable certainty that residues of tribenuron methyl in drinking water do not contribute significantly to the aggregate chronic human health risk.

2. *Non-dietary exposure*. Tribenuron methyl is not registered for any use which could result in non-occupational or non-dietary exposure to the general population.

D. Cumulative Effects

Tribenuron methyl belongs to the sulfonylurea class of crop protection chemicals. Other structurally similar compounds in this class are registered herbicides. However, the herbicidal activity of sulfonylureas is due to the inhibition of ALS, an enzyme found only in plants. This enzyme is part of the biosynthesis pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the relatively low toxicity of sulfonylurea herbicides in animals. There is no reliable information that would indicate or suggest that thifensulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population*. Tribenuron methyl is the active ingredient in two DuPont herbicides with new proposed uses on the following commercial crops:

Imazethapyr tolerant canola, cotton, and CDC triffid flax. There are no residential uses for any tribenuron methyl containing herbicides.

Based on data and information submitted by DuPont, EPA previously determined that the establishment of tolerances of tribenuron methyl on the following raw agricultural commodities would protect the public health, including the health of infants and children:

Wheat	Barley	Grass	Oats
Grain	Grain	Forage	Grain
Straw	Straw	Hay	Straw

Establishment of new tolerances for tribenuron methyl on imazethapyr tolerant canola seed at 0.02 ppm, cotton seed at 0.02 ppm, cotton gin trash at 0.02 ppm, and CDC triffid flax at 0.02 ppm, will not adversely impact public health.

Using the conservative exposure assumptions described in this unit, and based on the most sensitive chronic NOEL of 0.79 mg/kg/day and an RfD of 0.008 mg/kg/day, the aggregate dietary exposure will utilize 2.7% of the RfD for the U.S. population. Generally, exposure below 100% of the RfD are of no concern because the RfD represents the level at or below which daily dietary exposure over a lifetime will not pose risk to human health. We therefore conclude that there is reasonable certainty that no harm will result from aggregate exposure to tribenuron methyl residues.

2. *Infants and children.* Chronic dietary exposure of the most highly exposed subgroup in the population, children 1 to 6, is 0.000213 mg/kg/day or 2.7% of the chronic RfD. The acute dietary exposure of the most exposed subgroup, children 1 to 6, is 0.24% of the acute RfD (95th percentile). For non-nursing infants (<1–year), the acute dietary exposure is 0.15% acute RfD (95th percentile).

There are no residential uses of tribenuron methyl and contamination of drinking water is extremely unlikely. Based on the completeness and reliability of the toxicity data, the lack of toxicological endpoints of special concern, the lack of any indication of greater sensitivity of children, and the conservative exposure assessment, there is a reasonable certainty that no harm will result to infants and children from the aggregate exposure to residues of tribenuron methyl from all anticipated sources of dietary and non-occupational exposure. Accordingly, there is no need to apply an additional safety factor for infants and children.

F. International Tolerances

The maximum residue level (MRL) in Canada for tribenuron methyl on canola is 0.1 ppm. No Mexican or Codex MRLs exist for tribenuron methyl on canola. There are no Canadian, Mexican or Codex MRLs for tribenuron methyl on cotton and flax.

[FR Doc. 04–15208 Filed 7–6–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0132; FRL–7362–5]

Flonicamid; 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–2004–0132, must be received on or before August 6, 2004.

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: Ann Sibold, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6502; e-mail address: *sibold.ann@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you [grow brassica crops or mustard greens or consume them] Potentially affected entities may include, but are not limited to:

- Crop production (NAICS code 111)
- Other vegetable (except potato) Farming (NAICS 111219)
- Farming (NAICS code 112)
- Food manufacturing (NAICS 311)
- Fruit and vegetable preserving and specialty food manufacturing (NAICS code 3114)
- Pesticide manufacturing (NAICS code 32532)
- Entomological; services, agrecultural; insect control for crops (NAICS code 115112)
- Agricultural production or harvesting crews (NAICS code 115115)

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–2004–0132. 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–2004–0132. 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–2004–0132. 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–2004–0132.

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–2004–0132. 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 21, 2004.

Lois Rossi,

Director, Registration Division, Office of PesticidePrograms.

Summary of Petition

The petitioner's summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by ISK Biosciences 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.

ISK Biosciences Corporation

PP 4F6832P

EPA has received a pesticide petition PP 4F6832 from ISK Biosciences Corporation, 7470 Auburn Road, Suite A, Concord, Ohio, 44077, 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 tolerances for the combined residues of the insecticide flonicamid (N-(cyanomethyl)-4-trifluoromethyl)-3-pridinecarboxamide (CA) or N-cyanomethyl-4-trifluoromethylnicotinamide (IUPAC) and its metabolites, TFNA [4-trifluoromethylnicotinic acid, TFNA-AM (4-trifluoromethylnicotinamide) and TFNG N-(4-trifluoromethylnicotinoyl)-glycine) in or on the raw agricultural commodities: Brassica, head and stem, subgroup 5-A, at 1.5 parts per million (ppm), and mustard greens at 11 ppm.

EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* Wheat, potato and peach metabolism studies were conducted using 14_{C}-pyridyl-flonicamid. The metabolic profile was similar for all three matrices. The major metabolites for the various crops were: TFNA in peach, TFNA and TFNG in potato and TFNG in wheat. The metabolism of flonicamid in plants shows the main pathway of metabolism involves hydrolysis of -CN and $CONH_2$ functional groups in the molecule. The metabolism of flonicamid in plants is well understood.

2. *Analytical method.* Analytical methodology has been developed to determine the residues of flonicamid and its three major plant metabolites, TFNA, TFNG, and TFNA-AM in various crops. The residue analytical method for the majority of crops includes an initial extraction with acetonitrile (ACN)/deionized (DI) water, followed by a liquid-liquid partition with ethyl acetate. The residue method for wheat straw is similar, except that a C_{18} solid phase extraction (SPE) is added prior to the liquid-liquid partition. The final sample solution is quantitated using a liquid chromatograph (LC) equipped with a reverse phase column and a triple quadruple mass spectrometer (MS/MS).

3. *Magnitude of residues.* Residue data were collected on mustard greens and the Brassica leafy vegetables, head and stem subgroup during field trials. Maximum total residues for head and stem Brassica (total of 12 field trials) ranged from 0.590 ppm (broccoli) to 1.281 ppm (cabbage). Maximum total residues for mustard greens (total of 6 field trials) ranged from 2.115 ppm to 10.113 ppm.

B. Toxicological Profile

1. *Acute toxicity.* A battery of acute toxicity studies was conducted which placed flonicamid technical in Toxicity Category III for oral LD_{50}, Category IV for dermal LD_{50}, inhalation LC_{50}, dermal irritation, and eye irritation. Flonicamid technical is not a dermal sensitizer. In an acute neurotoxicity study, the no observed adverse effect levels (NOAELs) for neurotoxicity were 600 milligrams/kilogram (mg/kg) in males and 1,000 mg/kg in female highest doses tested (HDT). The systemic NOAELs were 600 mg/kg in males and 300 mg/kg in females.

2. *Genotoxicty.* Flonicamid technical did not cause mutations in the bacterial reverse mutation or mouse lymphoma tests with or without metabolic activation, chromosome damage in the mouse micronucleus or cytogenetics tests with and without metabolic activation, an increase in DNA damage in the comet assay or in an *in vivo* rat unscheduled DNA synthesis (UDS) study. Based on the weight of evidence, it is concluded, that flonicamid technical is not genotoxic.

3. *Reproductive and developmental toxicity* A developmental toxicity study in rats resulted in the maternal and developmental no observed effect levels (NOELs) of 100 mg/kg/day. The maternal lowest observed effect level (LOEL) was 500 mg/kg/day based on the treatment-related effects observed on the liver and kidney of the dams in the highest dose group. The developmental LOEL was 500 mg/kg/day based on the increases in placental weights and incidences of fetal skeletal variations seen only at maternally toxic doses of 500 mg/kg/day.

In the rabbit developmental toxicity study, the maternal and developmental NOELs were 7.5 mg/kg/day and 25 mg/kg/day HDT, respectively. The maternal LOEL was 25 mg/kg/day based on

decreased body weights and food consumption. No adverse effects on the fetuses were observed at the highest dose.

In the multigeneration rat reproduction study, the NOAEL was 300 ppm for both parental animals (13.5-32.8 and 16.3-67.0 mg/kg/day, respectively, for males and females) and their offspring. The effects at the highest dose of 1,800 ppm included the following: Increased kidney weights and gross and histopathological alterations in the kidney. Findings noted in the top dose females included delayed vaginal opening and increased liver, kidney and spleen weights in the F1 generation and reduced ovary and adrenal weights in the parental generation and decreased uterine weights in the F1 female weanlings. There was an increase in the follicle stimulating hormone (FSH) and luteinizing hormone (LH) levels in F1 females tested for these endpoints. These findings did not affect the reproductive performance or survival of offspring in the study.

4. *Subchronic toxicity.* The no observed adverse effect level (NOAEL) for flonicamid technical in the rat 28–day dermal toxicity study was 1,000 mg/kg/day, which was the highest dose tested.

In a 90–day rat feeding study the NOAEL was established at 200 ppm (12.11 mg/kg/day) for males and 1,000 ppm (72.3 mg/kg/day) for females. The NOAELs were based on effects on hematology, triglycerides, and pathology in the liver and kidney.

In a 13–week mouse study, the NOAEL was 100 ppm (15.25 mg/kg/day in males and 20.1 mg/kg/day in females). The LOAEL is 1,000 ppm (153.9 mg/kg/day in males and 191.5 mg/kg/day in females) based on hematology effects and changes in glucose, creatinine, bilirubin, sodium, chloride and potassium levels, increased liver and spleen weights and histopathology findings in the bone marrow, spleen and kidney.

In a subchronic toxicity study in dogs with capsule administration, the NOAEL was 20 mg/kg/day based on findings of severe toxicity at a dose exceeding the maximum tolerated dose; symptoms included collapse, prostration and convulsions leading to early sacrifice at the LOAEL of 50 mg/kg/day.

In a subchronic neurotoxicity study in rats, the NOAEL for dietary administration was 1,000 ppm (67 mg/kg/day in males and 81 mg/kg/ day in females) for systemic toxicity based on body weight and food consumption effects. The NOAEL for neurotoxicity was 10,000 ppm (625 and 722 mg/kg/day in males and females, respectively highest dose tested.

5. *Chronic toxicity*. In the chronic dog study with administration via using capsules, the NOEL was 8 mg/kg/day. The LOAEL was 20 mg/kg/day based on reduced body weights in females and effects on the circulating red blood cells.

In a rat 24–month combined chronic and oncogenicity study, flonicamid technical was not carcinogenic in rats. The NOAEL was 200 ppm (7.32 mg/kg/day) for males and 1,000 ppm (44.1 mg/kg/day) for females. The LOAEL was 1,000 ppm for males and 5,000 ppm for females based on histopathology in the kidney, hematology effects, hepatic effects including changes in biochemical parameters, increased organ weights, and histopathological changes. Atrophy of striated muscle fibers, cataract and retinal atrophy observed in the high dose females were considered to be due to acceleration of spontaneous age-related lesions.

In the 18–month mouse study, effects were observed in the lung, liver, spleen and bone marrow at 250 ppm or higher. Findings included centrilobular hepatocellular hypertrophy, extramedullary hematopoiesis and pigment deposition in the spleen and decreased cellularity (hypocellularity) in the bone marrow. There were statistically significant increases in the incidence of alveolar/bronchiolar adenomas in both sexes of treated groups with hyperplasia/hypertrophy of epithelial cells in terminal bronchioles. There was a statistically significant increase in the incidence of alveolar/bronchiolar carcinomas in males at 750 ppm and 2,250 ppm and in females at 2,250 ppm only. These effects in the lungs of mice were not life threatening as most of effects were observed at the terminal sacrifice and there was no effect of treatment on mortality in the study. A no observed adverse effect level (NOAEL) could not be determined from the dose levels administered. Mechanism-of-action studies have indicated that the lung effects are unique to the mouse and are not likely to translate to other species including the rat. A second 18–month mouse study was conducted in CD-1 mice at dose levels ranging from 10 to 250 ppm to establish a NOAEL for hyperplasia/hypertrophy of epithelial cells in terminal bronchioles and for the incidence of alveolar/bronchiolar adenomas and carcinomas in both sexes. There was a statistically significant increase in the incidence of alveolar/bronchiolar adenomas in males at 250 ppm. In females, there was no statistically significant increase in the incidence of pulmonary neoplastic lesions at any dose level. The incidence of hyperplasia/hypertrophy of epithelial cells lining the terminal bronchioles of the lungs was statistically increased at 250 ppm in both sexes. There were no treatment-related increases in neoplastic or non-neoplastic lesions at dose levels of 80 ppm or lower in either sex. The NOAEL was 80 ppm, equivalent to 10.0 and 11.8 mg/kg body weight/day for males and females, respectively. This study confirmed a threshold for these effects at 80 ppm, which had been indicated in studies on the mechanism. Mechanism-of-action studies have indicated that the lung effects are unique to the mouse and are not likely to translate to other species including the rat. Flonicamid technical was not carcinogenic in the rat.

6. *Animal metabolism.* Rat, goat and poultry metabolism studies were conducted using ^{14}C-pyridyl-flonicamid. The majority of the dose was rapidly excreted. Flonicamid was a major component of rat urine 48 hours after dosing. TFNA-AM was the major metabolite found in rats (urine), goats (milk and tissues) and in laying hens (tissues and eggs). TFNG was found between 8%–24% of the total radioactive residue (TRR) in the livers of rats sacrificed at intervals between 0.5–6 hours after dosing. The liver samples at these time intervals had ^{14}C-residues of 2.3%–4.6% of the dose. TFNA was not a major component in animal tissues. The metabolism of flonicamid in animals shows the main pathway of metabolism involves hydrolysis of -CN and -$CONH_2$ functional groups in the molecule, identical to plant metabolism. The main metabolic reactions were hydrolysis of cyano to the amide function and ring hydroxylation. In rats flonicamid was further metabolized by several routes, including nitrile hydrolysis, amide hydrolysis, N-oxidation, and hydroxylation of the pyridine ring, leading to multiple metabolites. The metabolism of flonicamid in animals is well understood.

7. *Metabolite toxicology.* The main metabolites of flonicamid were examined in acute oral toxicity studies in rats and bacterial reverse mutation tests. All the metabolites were less toxic than flonicamid and not mutagenic.

8. *Endocrine disruption.* No special studies investigating potential estrogenic or other endocrine effects of flonicamid have been conducted. Some suggestions of possible endocrine effects were reported at the highest dose tested (1,800 ppm) in the multi-generation reproduction study which showed increased FSH and LH levels, a delay in the time to vaginal opening in the F1

generation, and reduced ovary and adrenal weights in the parental generation. However, there were no effects on reproductive performance or survival of the offspring in the study. At levels that are expected to be found in the environment, flonicamid will not cause any endocrine-related effects.

C. Aggregate Exposure

1. *Dietary exposure.* Potential dietary exposures from food were estimated using the proposed tolerances for all crops using the Dietary Exposure Evaluation Model (DEEM-FCID™) and percent crop treated of 100%. The following raw agricultural commodities were included: Head and stem Brassica, mustard greens, leaf lettuce, head lettuce, celery, spinach, cotton, potatoes, fruiting vegetables, cucurbits, stone fruits, pome fruits and resulting secondary residues in meat, milk, poultry and eggs.

a. *Food.* Acute dietary exposure was compared to the acute population adjusted dose (aPAD) of 3.0 mg/kg/day based on the NOEL of 300 mg/kg from the acute neurotoxicity study in rats and a 100–fold uncertainty factor. The U.S. population exposure is 0.31% of the aPAD and the most highly exposed subpopulation is children 1–2 years of age with 0.93% of the aPAD 95th percentile.

Based on the available data, an appropriate chronic population adjusted dose (cPAD) is 0.073 mg/kg/day based on the NOEL of 7.32 mg/kg/day from the chronic toxicity study in rats and a 100–fold uncertainty factor. The U.S. population exposure is 3.6% of the cPAD and the most highly exposed subpopulation exposure is children 1–2 years of age with 12.2% of the cPAD.

b. *Drinking water.* A drinking water level of comparison (DWLOC) was calculated by subtracting the chronic/acute food exposures calculated using DEEM™ from the cPAD/aPAD to obtain the acceptable chronic/acute exposure to flonicamid in drinking water. The estimated average and maximum concentration of flonicamid in surface water is 1.07 parts per billion (ppb) and 7.33 ppb, respectively. These are both well below the lowest chronic (641 ppb) and acute (29,720 ppb) DWLOC values for flonicamid. Therefore, taking into account all proposed uses, it can be concluded, with reasonable certainty that residues of flonicamid in food and drinking water will not result in unacceptable levels of human health risk.

2. *Non-dietary exposure.* There are currently no residential uses of flonicamid registered or pending action that need to be added to the total risk from exposure.

D. Cumulative Effects.

In consideration of potential cumulative effects of flonicamid and other substances that may have a common mechanism of toxicity, to our knowledge there are currently no available data or other reliable information indicating that any toxic effects produced by flonicamid would be cumulative with those of other chemical compounds; thus, only the potential risks of flonicamid have been considered in this assessment of its aggregate exposure. If ISK Biosciences Corporation learns of any other compound with the same mechanism of toxicity they will submit information for the EPA to consider concerning potential cumulative effects of flonicamid consistent with the schedule established by EPA in the **Federal Register** of August 4, 1997 (62 FR 42020), and other EPA publications pursuant to the Food Quality Protection Act.

E. Safety Determination

1. *U.S. population.* Using conservative exposure assessment analyses, the acute dietary exposure estimates are well below the aPAD of 3 mg/kg bwt/day for all population subgroups. In addition, the chronic dietary exposure estimates for the various population groups are well below the cPAD of 0.073 mg/kg bwt/day. Based on this information, ISK Biosciences Corporation concludes, that there is reasonable certainty that no harm will result from acute or chronic exposure to flonicamid.

2. *Infants and children.* Based on the available developmental and reproductive data on flonicamid, ISK Biosciences Corporation concludes, that reliable data support use of the standard 100-fold uncertainty factor, and that an additional uncertainty factor is not needed to protect the safety of infants and children under the Food Quality Protection Act (FQPA). Although, the reproduction study indicated signs of toxicity to some reproductive organs/systems at the high dose of 1,800 ppm in the diet, other signs of toxicity such as effects on the kidney accompanied these; there were no effects observed at a dose level of 300 ppm. There were no effects on reproduction or survival at any dose level. Since acute and chronic aggregate exposure assessments are well below the aPAD and cPAD respectively, there is reasonable certainty that no harm will result to infants and children from aggregate exposure to flonicamid residues.

F. International Tolerances

There are no Canadian or Mexican residue limits or Codex MRLs for the insecticide flonicamid and its metabolites TFNA, TFNA-AM and TFNG.

[FR Doc. 04–15206 Filed 7–6–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2004–0181; FRL–7364–7]

Thifensulfuron Methyl; 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–2004–0181, must be received on or before August 6, 2004.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: James A. Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5697; e-mail address: *tompkins.jim@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Crop production (NAICS code 111)
- Animal production (NAICS code 112)
- Food manufacturing (NAICS code 311)
- Pesticide manufacturing (NAICS code 32532)

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be

affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. 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–2004–0181. 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–2004–0181. 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–2004–0181. 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–2004–0181.

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–2004–0181. 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 22, 2004.

Lois Rossi,

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 E. I. du Pont de Nemours and Company, 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.

E. I. du Pont de Nemours amd Company

PP 0F6152

EPA has received a pesticide petition PP 0F6152 from E. I. du Pont de Nemours and Company, DuPont Agricultural Products, Barley Mill Plaza, Wilmington, DE 19880–0038 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 thifensulfuron methyl: Methyl-3-[[[[(4-methoxy-6-methyl-1,3,5-triazin-2-yl)amino]carbonyl]amino sulfonyl]-2-thiophenecarboxylate in or on the raw agricultural commodity imazethapyr tolerant canola seed at 0.02 parts per million (ppm), cotton seed at 0.02 ppm, cotton gin trash at 0.02 ppm and CDC triffid flax at 0.02 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.* The qualitative nature of the residues of thifensulfuron methyl is adequately understood. Plant metabolism studies on wheat, corn, and soybeans were conducted. No significant difference in metabolic profile was observed. The plant metabolism studies in wheat and in corn were conducted with 14_C-labeled thiophene and triazine rings to follow the degradation pathway from the two most stable portions of thifensulfuron methyl. The metabolism in those plants shows similar patterns and involves cleavage of the urea bridge and metabolism of the methoxy group on the triazine ring and hydrolysis of the methyl ester group on the thiophene ring. The thiophene portion of thifensulfuron methyl in wheat degraded to 2-acid-3-sulfonamide and 14_C-polar compounds that further broke down to 14_CO_2. The triazine ring of thifensulfuron methyl metabolized to triazine urea and triazine amine. In corn, the thiophene portion of thifensulfuron methyl degraded to 2-acid-3-sulfonamide as well, and the triazine ring metabolized primarily to triazine urea and triazine amine. The primary thifensulfuron methyl metabolic pathways in soybean and wheat are the same. Minor differences in the formation and decline of the short-lived intermediate precursors to 2-acid-3 sulfonamide and O-demethyl triazine amine were found. These differences were not environmentally significant because of the very low levels of these intermediate metabolites in crops.

Metabolism studies conducted with radioactive 14_C-thifensulfuron methyl

on wheat under field conditions showed no significant residues of thifensulfuron methyl or its degradation products (>0.01 ppm) in field wheat grain at maturity. Mature forage and straw total residues were 0.80 to 0.45 ppm for the thiophene and triazine-labeled tests respectively. No single metabolite was greater than 0.06 ppm in the mature wheat. Major metabolites in wheat straw were thifensulfuron methyl, thifensulfuron methyl acid, 2-acid-3-sulfonamide, O-demethyl thifensulfuron methyl, triazine urea, and triazine amine.

There were no detectable residues of thifensulfuron methyl or its transformation products in corn grain (<0.01 ppm) or foliage (<0.02 ppm) at maturity. Analysis of earlier foliar samples showed extensive metabolism of thifensulfuron methyl. Among the residues detected were thifensulfuron methyl, 2-acid-3-sulfonamide, triazine urea, triazine amine, O-demethyl triazine urea, and O- demethyl triazine amine, however no thifensulfuron methyl acid was detected.

Metabolism studies were conducted with soybeans under greenhouse conditions. There were no detectable residues (<0.01 ppm) in the bean or pods at either rate or label at final harvest. Analysis of earlier foliar samples showed extensive metabolism of thifensulfuron methyl. Among the residues detected were thifensulfuron methyl, thifensulfuron methyl acid, 2-ester-3-sulfonamide, 2-acid-3-sulfonamide, triazine amine, O-demethyl triazine amine.

Two different crop rotation scenarios were investigated, one involving a bare ground application, the other one with a cover crop. No significant difference in metabolic profile was observed.

A confined greenhouse crop rotation study (following application to bare soil) was conducted planting beets, peas, and sunflowers at either a 30–day or 120–day treatment-to-planting interval. The application rate used was 34.8–38 grams/active ingredient/acre (g a.i./acre). There were no substantial residues (0.001 to 0.005 ppm) in food items (beet root, peas, sunflower seeds) in crops planted 30 or 120 days following soil treatment. There were minor detectable residues (0.02 to 0.05 ppm) in animal feed items (beet foliage and sunflower foliage). Thifensulfuron methyl was the only component identified (0.002 ppm) in sunflower foliage 73 days after treating the soil. Thifensulfuron was the only major radiolabeled component observed in the treated soil at the 30–day crop planting interval.

A confined greenhouse crop rotation study following treated wheat was conducted using beet root, peas, pea pods, and sunflower as following crops. The study used an application rate of 14.6 g a.i./acre, and a 45 or 75 day treatment-to-planting interval. There were no substantial residues (less than 0.01 ppm) in food items (beet root, peas, pea pods, sunflower (seeds and heads)) in crops planted 45 or 75 days following treated wheat incorporation into the soil. There were minor detectable residues in animal feed items. Pea and sunflower foliage contained 0.053–0.040 ppm and 0.015–0.008 ppm for the 45 and 75 day planting, respectively. Small amounts of triazine amine (<0.032 ppm), triazine urea, and O-demethyl triazine amine were identified in these fractions. Triazine urea was the major soil degradate at the 45 and 75 days planting interval.

Given the uniform lability of thifensulfuron methyl in plants, and that no residues above the limit of quantitation were found in treated canola plants with the ''Smart'' trait, it is unlikely that there would be any significant accumulation of metabolites in the harvested portions of treated canola and CDC triffid flax. No significant difference in metabolite distribution is anticipated for cotton use either. This is due to the significant soil interception that occurs during either a preemergence or postemergence application when thifensulfuron methyl is applied to small weeds for effective weed control.

2. *Analytical method.* For wheat, barley, and soybeans, the analytical methods use liquid chromatography and a photoconductivity detector for thifensulfuron methyl. Coupled with extraction, cleanup and isolation procedures, these methods provide a means of determining thifensulfuron methyl in soybeans and in wheat and barley straw with a detection limit of 50 parts per billion (ppb) nanogram/gram (50 ng/g), based on a 5–gram sample (soybeans) or a 10–gram sample (wheat and barley).

For corn forage and whole ears, an analytical method uses liquid chromatography and a photoconductivity detector for thifensulfuron methyl. Coupled with extraction, cleanup and isolation procedures, this method provides a means of determining thifensulfuron methyl in kernels with a detection limit of 20 ppb (20 ng/g), based on a 25-gram sample, and 50 ppm (50 ng/g) based on a 10 gram sample for green forage and whole ears. For determination of thifensulfuron methyl residues in corn processed fractions (processed corn oil and processed corn meal), the method uses HPLC with UV detection at 254 nm. This method provides a means to determine thifensulfuron methyl at levels as low as 0.02 ppm, based on a 10 gram sample.

Thifensulfuron methyl residues in canola and flax samples were determined by an analytical method based on the use of liquid chromatography with eluent and column switching with photometric detection at 254 nm at levels as low as 0.02 ppm (limit of quantitation) using a 5 gram sample.

Residues in cotton seed and gin trash were determined based on the use of column-switching liquid chromatography with detection via positive ion electrospray mass spectroscopy. The limit of quantitation was determined to be 20 ng/g and the limit of detection was estimated to be 6 ng/g, based on a 5 gram sample.

3. *Magnitude of residues*—a. *Wheat and barley grain and straw.* Field tests were conducted on wheat and on barley at 20 representative sites in the United States. Residues of thifensulfuron methyl were determined in wheat and barley grain and straw after single postemergence applications of thifensulfuron methyl at rates of 0–0.28 kg a.i./hectare (a.i./ha) in wheat and 0-0.14 kg a.i./ha in barley. The pre-harvest interval (PHI) was 41–140 days for the wheat grain and straw samples, 49–116 days for barley grain, and 60–89 days for barley straw. No quantifiable residues (<0.02 ppm for grain, <0.05 ppm for straw) were found in any samples.

In separate studies, wheat was treated with thifensulfuron methyl at a rate of 0.50 oz. a.i./acre or higher, and harvested at PHIs ranging from 25–42 days. No thifensulfuron methyl residues were detected in wheat grain (<0.02 ppm) or straw (<0.05 ppm) in any of the trials. Barley was treated with thifensulfuron methyl at a rate of 0.50 oz a.i./acre. Samples of mature barley grain and straw were taken from the test plots at a PHI of approximately 40 days after the test substance was applied. All results were below the established tolerance of 0.05 ppm for grain, and 0.1 ppm for straw.

b. *Corn grain, forage and fodder.* Field tests were conducted in the U.S. at 15 sites representative of the major U.S. corn growing regions. Tests included two decline studies. Residues of thifensulfuron methyl were determined in corn grain, forage, and fodder after a single postemergent application of thifensulfuron methyl at rates from 0 to 0.070 kg a.i./ha. PHIs were 80–154 days for the grain sample, 0–97 days for forage, and 82–154 days for fodder. No

residues above the quantitation limit (<0.02 milligrams/kilogram (mg/kg) for grain, <0.05 mg/kg for forage/fodder) were found in any grain or fodder samples. Residues in forage declined very rapidly with time. Even with treatment, at several times the typical use rate, residues were below the limit of quantitation within 14 days after treatment. In another study, plots were treated with thifensulfuron methyl at rates of 0.5, 1.0, and 2.0 oz a.i./acre. No thifensulfuron methyl was detected (quantitation limit of 0.02 ppm) in grain from the 2.0 oz. sample. No residues of thifensulfuron methyl were detected in the processed fractions (corn oil and corn meal).

c. *Soybeans.* A study was conducted to evaluate the magnitude of residues of thifensulfuron methyl in soybeans at either 0.125 oz a.i./acre or 0.25 oz a.i./acre. All applications were made approximately 60 days before harvest and were postemergence foliar broadcast. All thifensulfuron methyl residues in treated soybeans were below the limit of quantitation of 0.050 ppm; the current tolerance for thifensulfuron methyl in soybeans is 0.1 ppm.

d. *Oat grain and straw.* In a study using either 0.45 oz. a.i./acre or 0.90 oz. a.i./acre thifensulfuron methyl on oats, samples of mature oat grain and straw were taken from plots at preharvest intervals ranging from 39–57 days after the application of the test substance. Results show that all residues for thifensulfuron methyl were below the limit of quantitation (0.0055 ppm for oat grain, and 0.018 ppm for oat straw).

e. *Canola and flax.* Magnitude of residue studies were conducted on a variety of canola containing the ''Smart'' trait at 15 test sites, and on CDC triffid flax at 11 test locations. All treatment plots received an application at a rate of 15 or 30 g a.i./ha as a broadcast foliar application. The canola variety containing the ''Smart'' trait ranged from cotyledon up to the 8 leaf stage at application. CDC triffid flax staging at application ranged from 5 to 20 cm in height. No thifensulfuron methyl residues were found above the limit of quantitation of 0.02 ppm in any seed samples treated with the test substance.

f. *Cotton seed and gin trash.* Magnitude of residue studies were also conducted to determine residues of thifensulfuron methyl in cotton seed and cotton gin trash at nine test sites. The study consisted of three treatments. Treatment 1: One broadcast application at 0.45 oz a.i./acre, applied approximately 14–days prior to planting. Treatment 2: One broadcast application at 0.45 oz a.i./acre, applied pre-plant, on the day of planting. Treatment 3: One broadcast application at 2.25 oz. a.i./acre, applied pre-plant, the day of planting. The anticipated target PHI was approximately 120–days after the last application of the test substance; actual PHIs ranged from 123–196 days. The experimentally determined limit of quantitation was 20 ppb for both analytes. The limit of detection was estimated to be 6 ppb. No thifensulfuron methyl residues were found above the limit of quantitation of 0.02 ppm in any cotton seed and cotton gin trash samples treated with the test substance.

B. Toxicological Profile

1. *Acute toxicity.* Based on EPA criteria, technical thifensulfuron methyl is in acute toxicity Category IV for oral and inhalation routes of exposure, and for eye irritation. Thifensulfuron methyl is in acute toxicity Category III for the dermal route of exposure and for dermal irritation. It is not a skin sensitizer.

Acute oral toxicity in rats	LD_{50} >5,000 mg/kg
Acute dermal toxicity in rabbits	LD_{50} >2,000 mg/kg
Acute inhalation toxicity in rats	LD_{50} >7.9 milligrams/Liter (mg/L)
Primary eye irritation in rabbits	Minimal effects reversed within 24 hours
Primary dermal irritation in rabbits	Effects reversed within 48 hours
Dermal sensitization in guinea pigs	Non-senitizer

2. *Genotoxcity.* Technical thifensulfuron methyl has shown no genotoxic or mutagenic activity in the following *in vitro* and *in vivo* tests:

- *In vitro* Mutagenicity Ames Assay Negative
- *In vitro* mutagenicity Chinese hampster ovary/hypoxanthine guanine phophoribosyl transferase (CHO/HPRT) Assay Negative
- *In vitro* unscheduled DNA synthesis negative
- *In vivo* micronuclei induction (Rat) negative

Thifensulfuron methyl was not mutagenic with or without metabolic activation in an *in vitro* bacterial gene mutation assay using *Salmonella typhimurium.* Thifensulfuron methyl also was not mutagenic in the *in vitro* CHO/HPRT assay at concentrations up to 2,712 mg/L (in Chinese hamster ovary cells). In cultured primary rat hepatocytes, thifensulfuron methyl was negative for the induction of unscheduled DNA synthesis up to 2,712 mg/L.

An *in vivo* chromosome aberration study was conducted on rats. This included the assessment of chromosome aberrations by metaphase analysis in bone marrow of male and female rats. Thifensulfuron methyl did not induce cytogenic damage in bone marrow cells at a dose of 5,000 mg/kg.

3. *Reproductive and developmental toxicity.* The results of a series of studies indicated that there were no reproductive, developmental or teratogenic hazards associated with the use of thifensulfuron methyl. In a 1–generation reproduction study in rats, the suggested no observed effect level (NOEL) was 7,500 ppm (559 mg/kg/day males, 697 mg/kg/day females). In a rat multigeneration reproduction study, the NOEL for reproductive effects of thifensulfuron methyl in adult rats and their offspring was 2,500 ppm, the highest dietary level tested. This level was based on the absence of significant compound related effects observed in this study and is equivalent to 175–180 mg/kg/day in adult male rats and 212–244 mg/kg/day in adult female rats. There were no effects on fertility, lactation, litter size, or pup survival. Thifensulfuron methyl is not considered a reproductive toxin.

In studies conducted to evaluate developmental toxicity potential, thifensulfuron methyl was neither teratogenic nor uniquely toxic to the conceptus (i.e., not considered a developmental toxin). In the rat study, there was evidence of maternal toxicity (small decrease in body weight gain) and developmental toxicity (increase in sum of fetuses with developmental variations and variations due to retarded development) at a dose level of 800 mg/kg/day. No significant indications of maternal or fetal toxicity were evident at the other dose levels (0, 30, and 200 mg/kg/day). Therefore, the maternal and developmental no observed adverse effect level (NOAEL) for rats was considered to be 200 mg/kg/day. Upon review by the EPA, the NOEL was set at 159 mg/kg/day. In the rabbit developmental toxicity study, there was slight maternal toxicity (decreased body weight gain) at a dose of 650 mg/kg/day. No significant indications of maternal toxicity were evident at the lowest dose level (30 mg/kg/day). No compound-related effects on fetal weights or the incidences of malformations or variations were seen at any dose. The maternal NOEL was 200 mg/kg/day and the developmental NOEL was 650 mg/kg/day for rabbits dosed with thifensulfuron methyl by gavage on gestation days 7–19. Upon review by the

EPA, the maternal NOEL was set at 158 mg/kg/day and the developmental NOEL 511 mg/kg/day.

4. *Subchronic toxicity.* The most sensitive species to subchronic exposure of thifensulfuron methyl was the rat. The findings show that the NOEL for thifensulfuron methyl were 100 ppm for male and female rats (90–day dietary). These levels were based on the decreased body weight and food efficiency noted in the 2,500 and 7,500 parts per million (ppm) groups. This concentration is equivalent to 7 and 9 mg/kg/day in male and female rats, respectively. For mice, in both the 4–week range-finding and the 90–day studies, the NOEL for both male and female mice under the conditions of this study was 7,500 ppm; this was based on the lack of compound-related effects at the highest concentration. 7,500 ppm is equivalent to 1,427 mg/kg/day in male mice and 2,287 mg/kg/day in female mice. The NOEL for subchronic (90–day dietary) exposure in dogs was 1,500 and 7,500 ppm in male and female dogs, respectively. The NOELs were equivalent to 40.4 mg/kg/day in male dogs and 159.7 mg/kg/day in female dogs. These levels were based on lower body weight in males and a lack of adverse effects in females at 7,500 ppm, the highest concentration tested. In females, a compound-related decrease in body weight was observed at 7,500 ppm but was not considered adverse, based on the small magnitude of effect. Therefore, the NOEL in males and females was 1,500 ppm (26.1 mg/kg/day female, 40.4 mg/kg/day male). No compound-related pathologic lesions were observed and no target organ was identified in all of the above tests.

5. *Chronic toxicity.* The NOEL for chronic (18–month dietary) exposure in mice was 7,500 ppm (equivalent to 979 and 1,312 mg/kg/day in male and female mice, respectively). No biologically significant compound-related effects were seen in male or female mice at 7,500 ppm, the highest concentration tested. Thifensulfuron methyl was not an oncogen in mice.

The NOEL for chronic (2–year dietary) exposure in rats was 500 ppm (20 and 26 mg/kg/day in male and female rats, respectively). The NOEL was based on body weight effects in male and female rats at 2,500 ppm. The NOEL in female rats was 25 ppm (1.3 mg/kg/day) based on a non-adverse reduction in serum sodium concentration at 500 ppm. Thifensulfuron methyl was not an oncogen in rats.

In a 1–year feeding study in dogs, the NOEL of thifensulfuron methyl was 750 ppm in male and female beagle dogs (equivalent to 19.7 mg/kg/day males and 22.5 mg/kg/day females), based on decreased body weights, body weight gains, and food efficiency in females and increased liver with gall bladder weights in males, all at 7,500 ppm. The liver weight effects in males are not considered to be adverse effects; therefore, the lowest observed effect level (LOEL) was considered to be 7,500 ppm (195.3 mg/kg/day) in male dogs and 750 ppm (22.5 mg/kg/day) in female dogs.

6. *Animal metabolism.* The proposed major metabolic pathway for thifensulfuron methyl involved hydrolysis to 2-ester-3-sulfonamide (which may chemically condense to yield thiophene sulfonimide) or non-specific esterase activity to yield thifensulfuron methyl acid. The tissue data did not indicate potential retention or accumulation of thifensulfuron methyl or its metabolites.

Rats were dosed with two radioactive forms of thifensulfuron methyl (14_C-thiophene and 14_C-triazine). In the thiophene study, the thifensulfuron methyl was primarily excreted unchanged by rats following low dose (20 mg/kg), low dose following 21–days dietary preconditioning 100 ppm, and high dose (2,000 mg/kg) routines. From 70% to 85% of the excreted radioactivity was thifensulfuron methyl. The urine was the primary excretion route and contained from 71% to 92% of the original dose from the low and low-dose preconditioned groups. Combined urinary and fecal elimination was rapid, with over 90% of excretion completed by 48 hours after dosing for both low-dose groups. The high-dose group peak elimination was delayed by approximately 24 hours compared to the other dose levels. Tissue radioactivity levels were low at sacrifice (96 hours after dosing) for all dosing groups with no enhanced retention of radioactivity by any organ or tissue. Mass spectral analysis confirmed thifensulfuron methyl as the primary radiolabeled excretion product. Structural confirmation was also obtained for the 2-ester-3-sulfonamide metabolite. In the triazine study, thifensulfuron methyl was excreted primarily unchanged in urine and feces by male and female rats after administration of approximately 2,000 mg/kg by oral gavage. Urine was the primary route of excretion, averaging 58.7% of the dose in males and 75.5% in females. Fecal excretion of the dose averaged 21.2% for the male rats and 15.8% for the females. Greater than 50% of the dose was excreted by 48 hours post-dosing. Essentially no elimination of the dose as radiolabeled CO_2 or volatile compounds occurred. These results are similar to those reported on the thiophene-labeled thifensulfuron methyl. Intact thifensulfuron methyl was identified by mass spectrometry as the principal radioactive compound in urine (>94%) and feces (>77%). Three minor metabolites, each less than 3% of the dose, were identified in urine and feces by chromatographic retention comparison; they were thifensulfuron methyl acid, O-Demethyl thifensulfuron methyl, and triazine amine.

Results from a metabolism study with two radioactive forms of thifensulfuron methyl (14_C-triazine and 14_C-thiophene) in lactating goats show that most of the dosed radioactivity was rapidly excreted (primarily in the urine) and recovered as intact thifensulfuron methyl. Radioactivity in the milk (0.1-0.2 ppm) was comprised of mostly intact thifensulfuron methyl and a small amount of triazine amine and several very minor metabolites. Radioactivity did not accumulate in the tissues. After its absorption, the major metabolic pathway involved cleavage of the carboxyl ester linkage, resulting in the formation of thifensulfuron methyl acid. Oxidative O-demethylation occurred to a limited extent.

There were no significant levels of unique plant metabolites of thifensulfuron methyl found in food or feed products at crop maturity. Hence, toxicity testing of other degradation products of thifensulfuron methyl is not needed.

7. *Metabolite toxicology.* There is no evidence that the metabolites of thifensulfuron methyl as identified in either the plant or animal metabolism studies are of any toxicological significance.

8. *Endocrine disruption.* No special studies investigating potential estrogenic or other endocrine effects of thifensulfuron methyl have been conducted. However, the standard battery of required toxicology studies has been completed. These include an evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following repeated or long-term exposure to doses that far exceed likely human exposures. Based on these studies there is no evidence to suggest that thifensulfuron methyl has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure.* The chronic reference dose (RfD) of 0.013 mg/kg/day is based on the NOEL of 1.25 mg/kg/day from a 2–year rat feeding study and a 100X safety factor. The acute RfD of 1.59 mg/kg/ day is based on the NOEL of 159

mg/kg/day from a rat developmental study and a 100X safety factor.

i. *Food*—a. Chronic dietary exposure assessment dietary exposure, resulting from the proposed use of thifensulfuron methyl on barley, canola, cotton, flax, field corn, oats, soybeans and wheat, is well within the acceptable limits for all sectors of the population, as predicted by both the Chronic and Acute Modules of the Dietary Exposure Evaluation Model (DEEM™, Novigen Sciences, Inc., 1999 Version 6.74). The percentage or proportion of a crop that is treated can have a significant effect on the exposure profile. In this case, it was assumed for the crop that 100% was treated with thifensulfuron methyl. Based on a comparison with the use profile for most other herbicides, this is an extremely conservative estimate.

The predicted chronic exposure for the U.S. population subgroup was 0.000140 milligrams/kilogram body weight/day (mg/kg bwt/day). The population subgroup with the highest predicted level of chronic exposure was the non-nursing infants subgroup with an exposure of 0.000382 mg/kg bwt/day. Based on a chronic NOEL of 1.25 mg/kg bwt/day and a 100–fold safety factor, the chronic reference dose (cRfD) would be 0.013 mg/kg bwt/day. For the U.S. population, the predicted exposure is equivalent to 1.1% of the cRfD. For the population subgroup with the highest level of exposure (non-nursing infants), the exposure would be equivalent to 2.9% of the cRfD. Because the predicted exposures, expressed as percentages of the cRfD, are well below 100%, there is reasonable certainty that no chronic effects would result from dietary exposure to thifensulfuron methyl.

b. *Acute dietary exposure.* The predicted acute exposure for the U.S. population subgroup was 0.000364 mg/kg bwt/day (95th percentile). The population subgroup with the highest predicted level of acute exposure was the non-nursing infants subgroup with an exposure of 0.000846 mg/kg bwt/day (95th percentile). Based on an acute NOEL of 159 mg/kg bwt/day and a 100–fold safety factor, the acute reference dose (aRfD) would be 1.59 mg/kg bwt/day. For the U.S. population the predicted exposure (at the 95th percentile) is equivalent to 0.02% of the aRfD. For the population subgroup with the highest level of exposure (non-nursing infants subgroup), the exposure (at the 95_{th} percentile) would be equivalent to 0.05% of the aRfD. Because the predicted exposures, expressed as percentages of the aRfD, are well below 100%, there is reasonable certainty that no acute effects would result from dietary exposure to thifensulfuron methyl.

ii. *Drinking water.* Surface water exposure was estimated using the Generic Expected Environmental Concentration (GENEEC) model. Ground water exposures were estimated using Screening Concentration in Ground water (SCI-GROW).

EPA uses drinking water levels of comparison (DWLOCs) as a surrogate measure to capture risk associated with exposure to pesticides in drinking water. A DWLOC is the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses. A DWLOC will vary depending on the residue level in foods, the toxicity endpoint and with drinking water consumption patterns and body weights for specific subpopulations.

The acute DWLOCs are 56 ppm (parts per million) for the U.S. population and 16 ppm for the subpopulation with the highest exposure (non-nursing infants). The estimated maximum concentration of thifensulfuron methyl in surface water (1.2 ppb or parts per billion) derived from GENEEC is much lower than the acute DWLOCs. Therefore, one can conclude with reasonable certainty, that residues of thifensulfuron methyl in drinking water do not contribute significantly to the aggregate acute human health risk.

The chronic DWLOCs are 0.45 ppm for the U.S. population and 0.13 ppm for the subpopulation with the highest exposure (non-nursing infants). These DWLOC values are substantially higher than the GENEEC 56–day estimated environmental concentration of 0.65 ppb for thifensulfuron methyl in surface water. Therefore, one can conclude with reasonable certainty, that residues of thifensulfuron methyl in drinking water do not contribute significantly to the aggregate chronic human health risk.

2. *Non-dietary exposure.* Thifensulfuron methyl is not registered for any use which could result in non-occupational or non-dietary exposure to the general population.

D. Cumulative Effects

Thifensulfuron methyl belongs to the sulfonylurea class of crop protection chemicals. Other structurally similar compounds in this class are registered herbicides. However, the herbicidal activity of sulfonylureas is due to the inhibition of acetolactate synthase (ALS), an enzyme found only in plants. This enzyme is part of the biosynthesis pathway leading to the formation of branched chain amino acids. Animals lack ALS and this biosynthetic pathway. This lack of ALS contributes to the relatively low toxicity of sulfonylurea herbicides in animals. There is no reliable information that would indicate or suggest that thifensulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population.* Thifensulfuron methyl is the active ingredient in two DuPont herbicides with new proposed uses on the following commercial crops: Imazethapyr tolerant canola, cotton and CDC triffid flax. There are no residential uses for any thifensulfuron methyl containing herbicides. Based on data and information submitted by DuPont, EPA previously determined that the establishment of tolerances of thifensulfuron methyl on the following raw agricultural commodities would protect the public health, including the health of infants and children:

- Barley: grain, straw
- Oats: grain, straw
- Wheat: grain, straw
- Field corn: grain, fodder
- Soybeans
- Forage

Establishment of new tolerances for thifensulfuron methyl on canola seed at 0.02 ppm, cotton seed at 0.02 ppm, cotton gin trash at 0.02 ppm, and flax at 0.02 ppm will not adversely impact public health.

Based on the completeness and reliability of the toxicology data base and using the conservative assumptions presented earlier, EPA has established an RfD of 0.013 mg/kg/day. This was based on the NOEL for the chronic rat study, females (1.25 mg/kg/day) and a 100–fold safety factor. It has been concluded, that the aggregate exposure was approximately 1.1% of the RfD. Generally, exposures below 100% of the RfD are of no concern because it represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is reasonable certainty that no harm will result from aggregate exposures to thifensulfuron methyl residues.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of thifensulfuron methyl, data from the previously discussed developmental and, multigeneration reproductive toxicity studies were considered.

Developmental studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to

reproductive and other effects on adults and offspring from prenatal and postnatal exposures to the pesticide. The studies with thifensulfuron methyl demonstrated no evidence of developmental toxicity at exposures below those causing maternal toxicity. This indicates that developing animals are not more sensitive to the effects of thifensulfuron methyl administration than adults.

FFDCA section 408 provides that EPA may apply an additional uncertainty factor for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. Based on current toxicological data requirements, the data base for thifensulfuron methyl relative to prenatal and postnatal effects for children is complete. In addition, the NOEL of 1.25 mg/kg/day in the chronic rat study (and upon which the RfD is based) is much lower than the NOELs defined in the reproduction and developmental toxicology studies. The sub-population with the highest level of exposure was non-nursing infants (<1 yr), where exposure was less than 1% of the RfD. Based on these conservative analyses, there is reasonable certainty that no harm will result to infants and children from aggregate exposures to thifensulfuron methyl.

F. International Tolerances

The MRL in Canada for thifensulfuron methyl on canola is 0.1 ppm. No Mexican or Codex MRLs exist for thifensulfuron methyl on canola. There are no Canadian, Mexican or codex MRLs for thifensulfuron methyl on cotton and flax.

[FR Doc. 04–15212 Filed 7–6–04; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPPT–2004–0102; FRL–7368–5]

Approval of Test Marketing Exemption for a Certain New Chemical

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces EPA's approval of an application for test marketing exemption (TME) under section 5(h)(1) of the Toxic Substances Control Act (TSCA) and 40 CFR 720.38. EPA has designated this application as TME–04–5. The test marketing conditions are described in the TME application and in this notice.

DATES: Approval of this TME is effective June 29, 2004.

FOR FURTHER INFORMATION CONTACT: *For general information contact*: Colby Lintner, Regulatory Coordinator, Environmental Assistance Division (7408M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (202) 554–1404; e-mail address: *TSCA-Hotline@epa.gov*.

For technical information contact: Adella Watson, CCD (7405M), Office of Pollution Prevention and Toxics, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (202) 564–9364; e-mail address: *watson.adella@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

This action is directed in particular to the chemical manufacturer and/or importer who submitted the TME to EPA. This action may, however, be of interest to the public in general. Since other entities may also be interested, the Agency has not attempted to describe all the specific entities that may be affected by this action. If you have any questions regarding the applicability of this action to a particular entity, consult the technical 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 OPPT–2004–0102. 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 EPA Docket Center, Rm. B102-Reading Room, EPA West, 1301 Constitution Ave., NW., Washington, DC. The EPA Docket Center is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The EPA Docket Center Reading Room telephone number is (202) 566–1744 and the telephone number for the OPPT Docket, which is located in EPA Docket Center, is (202) 566–0280.

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.

II. What is the Agency's Authority for Taking this Action?

Section 5(h)(1) of TSCA and 40 CFR 720.38 authorizes EPA to exempt persons from premanufacture notification (PMN) requirements and permit them to manufacture or import new chemical substances for test marketing purposes, if the Agency finds that the manufacture, processing, distribution in commerce, use, and disposal of the substances for test marketing purposes will not present an unreasonable risk of injury to health or the environment. EPA may impose restrictions on test marketing activities and may modify or revoke a test marketing exemption upon receipt of new information which casts significant doubt on its finding that the test marketing activity will not present an unreasonable risk of injury.

III. What Action is the Agency Taking?

EPA approves the above-referenced TME. EPA has determined that test marketing the new chemical substance, under the conditions set out in the TME application and in this notice, will not present any unreasonable risk of injury to health or the environment.

IV. What Restrictions Apply to this TME?

The test market time period, production volume, number of customers, and use must not exceed specifications in the application and this notice. All other conditions and restrictions described in the application and in this notice must also be met.

TME–04–05

Date of Receipt: May 14, 2004.

Notice of Receipt: June 14, 2004 (69 FR 33015) (FRL–7365–3).

Applicant: CBI.

Chemical: (G) reaction products of fatty acids and hydroxy acids.