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_CCO_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]

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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.