interest groups are frequently related to contemporary issues, such as identifying methyl bromide replacement chemicals and alternatives to certain organophosphate pesticides.

This work plan represents RD's attempts at balancing these numerous priorities. In no way, however will RD neglect the many other action (e.g., label amendments, me-toos and emergency exemption) requests that are currently pending or will be submitted to the Agency during FY2000. The Agency has constructed this work plan to include some flexibility to respond to emerging public health or environmental issues. While forecasting such issues can be difficult, the Agency is committed to working with all affected parties to address their needs on an expeditious basis. Any submission which creates a modification to the schedule will, of course, require the appropriate justification and scientific data which will allow the Agency to make a sound, health-based decision. Such adjustments, however, may result in the need to modify this work plan during the fiscal year.

With the publication of each year's work plan the Registration Division intends to continue to improve the transparent y of the registration process of new active ingredients and/or new tolerance petitions.

List of Subjects

Environmental protection, Pests and pesticides.

Dated: October 29, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

[FR Doc. 99–32873 Filed 12–21–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–899; FRL–6391–1]

E.I. du Pont de Nemours and Company; Notice of Filing a Pesticide Petition to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by docket control number PF–899, must be received on or before January 21, 2000.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–899 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: James A. Tompkins (PM 25), Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–5697; and e-mail address: tompkins.james@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

Categories	NAICS	Examples of potentially affected entities
Industry	111	Crop production
	112	Animal production
	311	Food manufacturing
	32532	Pesticide manufacturing

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 the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have 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 Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**--Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person.* The Agency has established an official record for this action under docket control number PF–904. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2 (CM #2), 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–904 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by e-mail to: ''*opp-docket@epa.gov*,'' or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–899. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as 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 version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified 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 control 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 certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); 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.

List of Subjects

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

Dated: December 7, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petition

The petitioner summary of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summary of the petitions was prepared by the petitioner and represents the view of the petitioner. EPA is publishing the petition summary verbatim without editing it in any way. 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.

1. PP 3F4268

EPA has received a pesticide petition (PP 3F4268) from E.I. de Nemours and Company (DuPont), 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 permanent tolerances for the combined residues of quizalofop-p-ester (ethyl (*R*)-(2–(4–((6-chloroquinoxalin-2-yl)oxy)phenoxy)propanoate) and its acid metabolite quizalofop-p [R-(2-(4-((6-chloroquinoxalin-2-yl)oxy)phenoxy)propanoic acid), and the S enantiomers of both the ester and the acid, all expressed as quizalofop-p-ethyl ester in or on the raw agricultural commodities foliage of legumes vegetables (except soybeans) at 3.0 parts per million (ppm); legume vegetables (succulent or dried) group at 0.25 ppm; beet, sugar, molasses at 0.2 ppm; beet, sugar, root at 0.1 ppm, and beet, sugar, top at 0.5 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The registrant has provided plant metabolism studies for soybeans, cotton, tomatoes, potatoes, and sugar beets. These studies have been previously reviewed in PP 3F4268.

In summary, quizalofop-p ethyl ester is metabolized by cleavage at three sites as follows:

i. Primary pathway is hydrolysis of the ethyl ester linkage to form the quizalofop-p acid, then;

ii. Cleavage of the enol ether linkage in the acid, between the phenyl and quinoxalinyl rings, to form phenols, and

iii. Cleavage of the ether between the isopropanic group and the phenyl ring to form a phenol.

The plant metabolism data show that quizalofop-p ethyl ester does not translocate, but is rapidly hydrolyzed to the corresponding acid; then the phenols conjugate with the plant sugars. Metabolism studies in soybeans using the racemic mixture quizalofop ethyl ester and the resolved D+ isomer show nearly identical pathways.

The nature of the quizalofop-p ethyl ester residue in cottonseed, potatoes, tomatoes, soybeans, and sugar beets is adequately understood. The residues of concern are quizalofop-p ethyl ester and its acid metabolite, quizalofop-p, and the S enantiomers of both the ester and the acid, all expressed as quizalofop-p ethyl ester.

2. *Analytical method*. An adequate analytical methodology (high-pressure liquid chromatography using either ultraviolet or fluorescence detection) is available for enforcement purposes in Vol. II of the Food and Drug Administration Pesticide Analytical Method (PAM II, Method I). There are currently no actions pending against the registration of this chemical. Any secondary residues expected to occur in eggs, milk, meat, fat, and meat byproducts of cattle, goats, hogs, horses, sheep, and poultry from this use will be covered by existing tolerances.

Adequately validated residue analytical method, DuPont 2829 (Xenos Method XAM-38A, Determination of Quizalofop-P-Ethyl and its Metabolites in Canola, Flax, Lentils, Peas, Dry and Succulent Beans and Sugar Beet Tops and Roots, by Liquid Chromatography). This method determines residues of quizalofop-p-ethyl and its metabolites in oilseed and other crops. It measures levels of quizalofop-p-ethyl, quizalofop-p acid and conjugates as total residues in the form of 2-methoxy-6-chloroquinoxaline (MeCHQ). Quantitation was carried out using normal phase high pressure liquid chromatography with fluorescence detection. The residues were expressed as equivalents of quizalofop-p-ethyl.

A successful tolerance method validation (TMV) on DuPont 2829 (Xenos Method XAM-38A) is not a prerequisite for a tolerance on beans (succulent and dried) as well as sugar beets and sugar beet molasses as there is already an enforcement method in PAM II.

3. *Magnitude of residues* —i. *Magnitude of the residue in plants*. The studies submitted include field trials in three regions for succulent beans, six additional sites for dry beans in four regions and five additional sites in three regions for sugar beets.

In conjunction with previously submitted data, an adequate amount of geographically representative crop field trial residue data were presented which show that the proposed tolerances should not be exceeded when quizalofop ethyl is formulated into Assure® and used as directed.

ii. *Magnitude of the residue in animals.* A ruminant feeding study has been submitted and reviewed by EPA. In summary, three groups of three lactating dairy cows (plus a control group) were fed 0.1, 0.5, and 5.0 ppm quizalofop ethyl ester (encapsulated) for 28 consecutive days. Milk was collected daily and a sub-sample was divided into skim milk and cream. Two cows were sacrificed after 28 days with samples of fat, skeletal muscle, liver, and kidney being collected and analyzed. The remaining cow in each test group was fed a regular diet without encapsulated quizalofop ethyl ester for 7 additional days before sacrifice. Whole milk, skim milk, and cream from the control, and the 0.1 and 0.5 ppm dose groups showed no quizalofop to < 0.02 ppm (0.05 ppm in cream). From the 5 ppm dose, quizalofop residues ranged from 0.01 to 0.02 ppm in whole, and when these samples were separated into cream and skim milk, the quizalofop partitioned into the cream with residues plateauing at 0.26 to 0.31 ppm. No quizalofop to < 0.02 ppm was detected in skeletal muscle, and to < 0.05 ppm was detected in any liver or fat sample from any of the three doses. Quizalofop was detected in one kidney sample as 0.05 ppm from the 5 ppm dose.

From the feed items in this petition, all of the feed items in cattle diets can be treated with quizalofop ethyl ester. A theoretical beef cattle diet consisting of canola meal, bean and pea forage, pea hay, and sugar beet tops which none-the-less maximizes the potential quizalofop exposure of 2.1 ppm. A theoretical dairy cattle diet consisting of pea and bean forage would none-the-less maximize the potential quizalofop exposure at 2.4 ppm. Substitutions of other feed items and varying their percentages in the diets would give a lower dietary quizalofop burden.

The results of the quizalofop ethyl ester bovine feeding study show that finite residues will actually occur in milk and tissues from the feeding of quizalofop ethyl ester treated RACS or their processed feed items when Assure® II is used as directed. The established quizalofop and quizalofop ethyl ester tolerance in milk, and in fat, meat, and meat by-products of cattle, goats, hogs, horse, and sheep are adequate and need not be increased from these additional uses.

A poultry feeding study has been submitted and reviewed. In summary, 3 groups of 20 hens (plus 1 control group) were dosed with encapsulated quizalofop ethyl ester at 0.1, 0.5, and 5 ppm daily for 28 consecutive days. Eggs were collected daily, and after 28 days 3/4 of the hens in each test group were sacrificed, and samples of fat, liver, kidney, breast and thigh muscles were collected and analyzed. Tissues from each test group were pooled prior to analysis. The remaining five hens were fed a regular poultry diet without quizalofop ethyl ester for an additional 7 days before sacrifice. No quizalofop residues were detected in the liver to < 0.05 ppm, and in breast and thigh muscles to < 0.02 ppm for any dose administered. From the 5 ppm dose, one kidney sample showed 0.09 ppm quizalofop, two fat samples were 0.05 and 0.06 ppm quizalofop, and one egg sample was 0.02 ppm quizalofop.

The results of the quizalofop ethyl ester poultry feeding study show that while it is not possible to establish with certainty whether finite residues will actually occur in eggs and tissues from the feeding of quizalofop ethyl ester treated RACS or their processed feed items when Assure® II is used as directed, there is a reasonable expectation for such residues to occur. The established tolerance of quizalofop and quizalofop ethyl ester in eggs, and in fat, meat, and meat by-products of poultry are adequate and need not be changed from these additional uses.

B. Toxicological Profile

1. *Acute toxicity*. Several acute toxicology studies were conducted and the overall results placed technical grade quizalofop ethyl in toxicity Category III. These include the following studies in Category III: acute oral toxicity (LD_{50}s 1,480 and 1,670 for female and male rats, respectively) and eye irritation (mild effects; reversible within 4 days). Dermal toxicity (LD_{50}s > 5,000 mg/kg; rabbit), inhalation toxicity (LC_{50} > 5.8 mg/L; rat) and dermal irritation were classified within Category IV. Technical quizalofop ethyl was not a dermal sensitizer.

2. *Genotoxicty*. Technical quizalofop ethyl was negative in the following genotoxicity tests: bacterial gene mutation assays with *E. coli* and *S. typhimurium*; gene mutation assays in Chinese hamster ovary (CHO) cells; *in vitro* DNA damage assays with *B. subtillis* and in rat hepatocytes; and an *in vitro* chromosomal aberration test in CHO cells.

3. *Reproductive and developmental toxicity*. Studies supporting the registration include: A developmental toxicity study in rats administered dosage levels of 0, 30, 100, and 300 mg/kg/day highest dose tested (HDT). The maternal toxicity no observed adverse effect level (NOAEL) was 30 mg/kg/day and a developmental toxicity NOAEL was greater than 300 mg/kg/day (HDT). The maternal NOAEL was based on reduced food consumption and increased liver weights.

A developmental toxicity study in rabbits administered dosage levels of 0, 7, 20, and 60 mg/kg/day with no developmental effects noted at 60 mg/kg/day (HDT). The maternal toxicity NOAEL was 20 mg/kg/day based on decreases in food consumption and body weight gain at 60/mg/kg/day (HDT).

A 2–generation reproduction study in rats fed diets containing 0, 25, 100, or 400 ppm (or approximately 1, 1.25, 5, and 20 mg/kg/day, respectively) with a developmental (systemic effects) NOAEL of 1.25 mg/kg/day for F_{2B} weanlings based on increased liver weights and increased incidence of eosinophilic changes in the livers at 5.0 mg/kg/day. These liver changes were considered to be physiological or adaptive changes to compound exposure among weanlings. When access to the mother's feed is available, it is a common observation that young rats will begin consuming chow prior to

complete weaning at 21 days of age. Consumption could not be quantified; therefore, the maternal consumption was assumed as the NOAEL (if normalized on a body weight basis, exposures to the weanling rats were likely higher). The parental NOAEL of 5.0 mg/kg/day was based on decreased body weight and premating weight gain in males at 20 mg/kg/day (HDT).

4. *Subchronic toxicity.* A 90–day study was conducted in rats fed diets containing 0, 40, 128, 1,280 ppm (or approximately 0, 2, 6.4, and 64 mg/kg/day, respectively). The NOAEL was 2 mg/kg/day. This was based on increased liver weights at 6.4 mg/kg.

A 90–day feeding study in mice was conducted with diets that contained 0, 100, 316, or 1,000 ppm (or approximately 0, 15, 47.4, and 150 mg/kg/day, respectively). The NOAEL was < 15 mg/kg/day lowest dose tested (LDT) based on increased liver weights and reversible histopathological effects in the liver at the LDT.

A 6–month feeding study in dogs was conducted with diets that contained 0, 25, 100, or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOAEL was 2.5 mg/kg/day based on increased blood urea nitrogen at 10 mg/kg/day.

A 21–day dermal study was conducted in rabbits at doses of 0, 125, 500, or 2,000 mg/kg/day. The NOAEL was 2,000 mg/kg/day (HDT).

5. *Chronic toxicity.* An 18–month carcinogenicity study was conducted in CD-1 mice fed diets containing 0, 2, 10, 80, or 320 ppm (or approximately 0, 0.3, 1.5, 12, and 48 mg/kg/day, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 12 mg/kg/day. A marginal increase in the incidence of hepatocellular tumors was observed at 48 mg/kg/day, the HDT, which exceeded the maximum tolerated dose (MTD). (See the discussion by the EPA HED Carcinogenicity Peer Review Committee below.)

A 2–year chronic toxicity/carcinogenicity study was conducted in rats fed diets containing 0, 25, 100 or 400 ppm (or 0, 0.9, 3.7, and 15.5 mg/kg/day for males and 0, 1.1, 4.6, and 18.6 mg/kg/day for females, respectively). There were no carcinogenic effects observed under the conditions of the study at levels up to and including 18.6 g/kg/day (HDT). The systemic NOAEL was 0.9 mg/kg/day based on altered red cell parameters and slight/minimal centrilobuler enlargement of the liver at 3.7 mg/kg/day.

A 1–year feeding study was conducted in dogs fed diets containing 0, 25, 100, or 400 ppm (or approximately 0, 0.625, 2.5, and 10 mg/kg/day, respectively). The NOAEL was 10 mg/kg/day (HDT).

The Carcinogenicity Peer Review Committee (CPRC) of EPA HED has evaluated the rat and mouse cancer studies on quizalofop along with other relevant short-term toxicity studies, mutagenicity studies, and structure activity relationships. The CPRC concluded, after three meetings and an evaluation by the OPP Science Advisory Panel (SAP), that the classification should be a Category D (not classifiable as to human cancer potential). No new cancer studies were required.

The first CPRC review tentatively concluded that quizalofop should be classified as a Category B2 (probable human carcinogen). That classification was based on liver tumors in female rats, ovarian tumors in female mice, and liver tumors in male mice. This classification was downgraded to a Category C (possible human carcinogen) at a second CPRC review. The change in classification was due to a reexamination of the liver tumors in female rats and ovarian tumors in female mice. The first peer review had found a statistically significant positive trend for liver carcinomas in female rats. Subsequent to this conclusion the tumor data was reevaluated, and the revaluation showed a reduced number of carcinomas. Although there remained a statistically significant positive trend for carcinomas in the study, the CPRC concluded that the carcinomas were not biologically significant given the few carcinomas identified (one at the mid-dose and two at the high dose). Noting that this level of carcinomas was within historical levels, the CPRC concluded that administration of quizalofop did not appear to be associated with the liver carcinomas.

As to the ovarian tumors in female mice, the CPRC had first attached importance to the fact that these tumors were statistically significant at the high dose as compared to historical control values although statistically significant when compared to concurrent controls. However, review of further historical control data showed that the level of ovarian tumors in the quizalofop study was similar to the background rate in several other studies. Given this information and that the quizalofop study showed no hyperplasia of the ovary, no signs of endocrine activity related to ovarian function, and no dose response relationship, the CPRC concluded that the ovarian tumors were probably not compound-related.

The findings of the second CPRC review were presented to EPA's SAP. The SAP concurred with the CPRC conclusion that the liver tumors in female rats and the ovary tumors in female mice showed no evidence of carcinogenicity. However, the SAP disagreed with CPRC's classification of quizalofop as a Category C based on the liver tumors in male mice. The SAP concluded that the mouse liver tumors did not support such a classification because the tumors occurred at a dose above the MTD and because they were not statistically significant if a ''p'' value of less than 0.05. The SAP believed that such greater statistical rigor was appropriate for variable tumor endpoints such as male mouse liver tumors.

Following the SAP review, the CPRC changed the classification for quizalofop to Category D. The Category D classification is based on an approximate doubling in the incidence of male mice liver tumors between controls an the high dose. This finding was not considered strong enough to warrant the finding of a Category C (possible human carcinogen) since the increase was of marginal statistical significance, occurred at a high dose which exceeded the predicted MTD, and occurred in a study in which the concurrent control for liver tumors was somewhat low as compared to the historical controls; while the high dose control group was at the upper end of previous historical control-groups.

EPA has found the evidence on the carcinogenicity of quizalofop-p ethyl ester in animals to be equivocal and therefore concludes that quizalofop-p ethyl ester does not induce cancer in animals within the meaning of the Delaney clause. Important to this conclusion was the following evidence:

i. The only statistically significant tumor response that appears compound-related was seen at a single dose in a single sex in a single species.

ii. The response was only marginally statistically significant.

iii. The response was only significant when benign and malignant tumors were combined.

iv. The tumors were in the male mouse liver.

v. The tumors were within historical controls.

vi. The mutagenicity studies were negative.

Although in some circumstances a finding of animal carcinogenicity would be made despite any one, or even several, of the six factors noted, the combination of all of these factors here cast sufficient doubt on the reproducibility of the response in the high dose male mouse that EPA concludes the evidence on carcinogenicity is equivocal.

6. *Animal metabolism*. The metabolism of quizalofop ethyl in animals (rat, goat and poultry) is well understood. ^{14}C-phenyl and ^{14}C-quinoxaline quizalofop ethyl ester metabolism studies have been conducted in each species. There are similarities among these species with respect to metabolism. Quizalofop ethyl is rapidly and extensively metabolized and rapidly excreted by rats. The principal metabolites were the quizalofop-p acid and two dechlorinated hydroxylated forms of the acid. Tissue residues were minimal and there was no evidence of accumulation of quizalofop ethyl or its metabolites in the rat.

The primary pathway in ruminants is hydrolysis of the ethyl ester to form the quizalofop-p methyl ester. In poultry, the primary metabolic pathway is also the hydrolysis of the ethyl ester to form the quizalofop-p acid, then the methyl esterification to form the quizalofop methyl ester becomes a minor pathway.

The nature of the quizalofop ethyl ester residue in livestock is adequately understood. The residues of concern are quizalofop ethyl, quizalofop methyl, and quizalofop, all expressed as quizalofop ethyl.

7. *Metabolite toxicology*. There is no evidence that the metabolites of quizalofop ethyl as identified as 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 quizalofop p-ethyl 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 quizalofop p-ethyl has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. An analysis of chronic dietary risk was conducted to determine the total exposure from current and proposed final tolerances for quizalofop-p-ethyl. A Reference Dose (RfD) of 0.009 mg/kg/day was used in the analyses.

i. *Food*. The first step in the analysis was to run the TAS (Tolerance Assessment System) program using current tolerances with an RfD of 0.009 mg/kg/day. The Theoretical Maximum Residue Concentration (TMRC), based on the current tolerances, was 0.000318 mg/kg/day for the U.S. population (48 contiguous States) and 0.000814 mg/kg/day for the population subgroup with the highest estimated exposure (children 1–6 yrs. old). For the U.S. population subgroup this represents approximately 3.5% of the RfD while for the most exposed population this represents approximately 9.0% of the RfD. Based on the risk estimates arrived at in this analysis, chronic dietary risk from the current uses of Assure® is minimal.

ii. *Drinking water*. Another potential source of dietary exposure to pesticides is residues in drinking water. There is no established Maximum Concentration Level (MCL) for quizalofop ethyl in water. Based on the low use rate of quiza lofop ethyl, and a use pattern that is not widespread (since the current and proposed uses are on minor crops), DuPont does not anticipate residues of quizalofop in drinking water and exposure from this route is unlikely.

2. *Non-dietary exposure*. Quizalofop ethyl is not registered for any use that could result in non-occupational, non-dietary exposure to the general population.

D. Cumulative Effects

There is no evidence to indicate or suggest that quizalofop p-ethyl has any toxic effects on mammals that would be cumulative with those of any other chemicals.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the most sensitive species chronic NOAEL of 0.9 mg/kg and a RfD of 0.009 mg/kg/day, the existing tolerances and proposed uses of quizalofop ethyl are expected to utilize 3.5% of the RfD for the general U.S. population. Generally, exposures below 100% of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to quizalofop ethyl resulting from current and proposed agricultural uses.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of quizalofop ethyl, data were considered from developmental toxicity studies in the rat and rabbit, and a multi-generation reproduction study in rats. There were no developmental effects observed in the absence of maternal toxicity in the rat and rabbit developmental studies. Minimal adaptive or physiological effects were observed in livers of weanlings in the 2–generation rat reproduction study described earlier. However, this effect was only observed at a dose that far exceeds any expected human exposure. Further, the NOAEL of 0.9 mg/kg/day from the 2–year rat study with quizalofop ethyl which was used to calculate the RfD (discussed above), is already lower than any of the NOAELs defined in the developmental and reproductive toxicity studies with quizalofop ethyl.

As indicated above, infants and children have a low potential for quizalofop ethyl exposure. The toxicology profile of quizalofop ethyl demonstrates low mammalian toxicity. Because there was no evidence that offspring were uniquely susceptible to the toxic effects of quizalofop ethyl, an additional 10-fold uncertainty factor should not be required to protect infants and children. Therefore, the RfD of 0.009 mg/kg/day, which utilizes a 100–fold safety factor, is appropriate to assure a reasonable certainty of no harm to infants and children from aggregate exposure to quizalofop ethyl.

F. International Tolerances

Since there are no Mexican or Codex MRLs/tolerances, compatibility is not a problem at this time. Compatibility cannot be achieved with the Canadian negligible residue type limit at 0.1 ppm at the USA use pattern, which had findings of real residues above 0.1 ppm.

2. PP 4F4278

EPA has received a pesticide petition (PP 4F4278) from E.I. DuPont de Nemours and Company, DuPont Agricultural Products, Barley Mill Plaza, Wilmington, DE 19880–0038 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of triflusulfuron methyl: Methyl 2 [[[[[4-(dimethylamino)-6-(2,2,2-trifluoroethoxy)-1,3,5-triazin-2-yl]amino]carbonyl]amino]sulfonyl]-3-methylbenzoate in or on the raw agricultural commodity [beet, sugar, root and beet, sugar, top at 0.05 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. Metabolism of triflusulfuron methyl in sugar beets was

studied using triflusulfuron methyl labeled separately with carbon-14 in the triazine ring and in the ester carbonyl group. Triflusulfuron methyl was extensively metabolized by sugar beets treated at the 4-8-leaf growth stage with 100 grams active ingredient per half acre (g ai/ha). Triflusulfuron methyl levels dropped rapidly from 3 ppm in the sample taken on the day of the treatment to < 0.01 ppm 14 days after treatment. The initial step in the metabolic breakdown of triflusulfuron methyl involves cleavage of the sulfonylurea bridge, which is followed by further metabolism of the initial degradates. The levels of principal radiolabeled metabolites found in plant samples (*N*-desmethyl triazine amine, *N,N*-bis-desmethyl triazine amine, acid sulfonamide, and its glucose conjugate) dropped to < 0.01 ppm at maturity. No significant (> 0.01 ppm) residues of triflusulfuron methyl or its radiolabeled metabolites were detected in mature roots or foliage.

2. *Analytical method.* A method for quantitation of triflusulfuron methyl in sugar beets uses a high performance liquid chromatograph (HPLC) with eluent and column-switching and ultra-violet (UV) detection at 232 nm for the determination of triflusulfuron methyl residues in sugar beet foliage and roots. Sample clean-up is achieved through reversed phase chromatography using eluent-switching. Column-switching provides the resolution required for quantitation of triflusulfuron methyl. The method allows for quantitation of triflusulfuron methyl in sugar beet foliage and roots at levels as low as 0.02 ppm based on a 10–gram sample. Triflusulfuron methyl is detected at levels as low as 0.005 ppm. Triflusulfuron methyl recoveries averaged 98% for forage and 101% for roots.

3. *Magnitude of residues.* Triflusulfuron methyl degraded rapidly in sugar beets to produce the triazine amine which undergoes consecutive demethylations to yield *N*-desmethyl triazine amine and *N,N*-bis-desmethyl triazine amine. Residues of triflusulfuron methyl at harvest were below the detection limits in sugar beet roots and foliage at all application levels. There is no reasonable expectation of residues of triflusulfuron methyl occurring in sugar beet roots or foliage at harvest. The data supports a preharvest interval of 30 days.

Residues of the metabolite triazine amine and *N*-desmethyl triazine amine were at or below the detection limit of 0.02 ppm in sugar beet roots and foliage at all application levels at all test sites. Residues of *N,N*-bis-desmethyl triazine amine were below the detection limit of 0.02 ppm in roots at all application levels at all locations; however, residues in foliage were detected in 7 out of 41 samples at up to 0.05 ppm in samples that were treated at exaggerated rates (70 g ai/ha/application). At the expected maximum seasonal use rate of 60 g ai/ha, residues of *N,N*-bis-desmethyl triazine amine are not expected above the 0.02 ppm detection limit.

The potential of triflusulfuron methyl residues occurring during processing of sugar beet roots treated with triflusulfuron methyl was also determined. Samples of sugar beet roots, harvested at maturity from plots treated with triflusulfuron methyl at a rate of 420 g ai/ha, were processed. Triflusulfuron methyl was below the 0.01 ppm detection limit in sugar beet root and all the processed fractions (sugar, molasses, and dried pulp). The lack of concentration of triflusulfuron methyl even at the exaggerated dose used in this study confirms that at the proposed use rate of triflusulfuron methyl, there is no reasonable expectation of residues in sugar beet roots or processed fractions.

B. Toxicological Profile

1. *Acute toxicity.* Based on EPA criteria, technical triflusulfuron methyl is in acute toxicity Category IV for oral and inhalation routes of exposure, and for dermal irritation. Triflusulfuron methyl is in acute toxicity Category III for dermal toxicity and for eye irritation.

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	LC_{50} > 5.1 mg/L
Primary eye irritation in rabbits	Non-irritant
Primary dermal irritation in rabbits	Non-irritant
Dermal sensitization in guinea pigs	Non-sensitizer
Acute Neurotoxicity	(NOAEL) = 2,000 mg/kg/day highest dose tested (HDT)

2. *Genotoxicty.* Mutagenicity data technical triflusulfuron methyl include a reverse mutation assay (Ames Test) which was negative at concentrations up to 1,000 µg/plate, the highest level tested; a *Salmonella typhimurium* plate incorporation assay which was negative at concentrations up to 3,000 µg/plate, the highest level tested; a Chinese hamster ovary/hypoxanthine-guanine (CHO/HPRT) assay which was negative at concentrations up to 2,000 mg/kg/day, the highest level tested. A chromosomal aberration/human lymphocyte assay was positive in the presence of metabolic activation at concentrations greater than or equal to 1,500 µg/mL. A second chromosomal aberration/human lymphocyte assay was positive in the presence of metabolic activation at concentrations of 2,000 µg/mL. Results in the absence of metabolic activation were inconclusive for both chromosomal aberration studies. The mouse bone marrow micronucleus test was negative at doses up to 5,000 mg/kg, the highest dose level tested. In three *Salmonella typhimurium* plate incorporation assays, metabolites of triflusulfuron methyl were negative up to 5,000 µg/plate, the highest level tested.

3. *Reproductive and developmental toxicity.* In a 2–generation rat reproduction study rats were fed dosages of 0, 0.588, 5.81, 44.0 and 89.5 mg/kg/day (males) and 0, 0.764, 7.75, 58.0, and 115 mg/kg/days (females) with a reproductive toxicity NOAEL equal to or greater than 89.5 and 115 mg/k/day for males and females, respectively, based on the absence of reproductive effects in rats at the highest dose level. The NOAEL for systemic toxicity was 5.81 and 7.75 for males and females, respectively based on decreased body weight/body weight gain and food efficiency in males and females, and decreased weights of offspring from the F^0 generation on days 14 and 21 post-partum at 44.0 and 58.0 mg/kg/day in males and females, respectively.

Technical triflusulfuron methyl was evaluated for developmental toxicity potential in rats and rabbits. Rats were fed dosages of 0, 30, 120, 350, and 1,000 mg/kg/day with a developmental NOAEL equal to or greater than 1,000 mg/kg/day (HDT) and a maternal toxicity NOAEL of 120 mg/kg/day with a lowest observed adverse effect level (LOAEL) of 350 mg/kg/day based on reduced body weight gain in the 350 and 1,000 mg/kg/day animals, reduced food consumption in the 1,000 mg/kg/day animals and lower food efficiency in the 350 and 1,000 mg/kg/day.

Rabbits were fed dosages of 0, 15, 90, 270, and 800 mg/kg/day with a NOAEL for developmental toxicity of 90 mg/kg/day with a LOAEL of 270 mg/kg/day based on the increase in abortions and a decrease in mean fetal body weight. The NOAEL for maternal toxicity is 90 mg/kg/day with a LOAEL of 270 mg/kg/day based on the maternal death and abortions, and increase in clinical signs noted in the mid-high and high dose groups, decreased food efficiency and

increased post mortem finding describing gastrointestinal effects.

4. *Subchronic toxicity.* The subchronic toxicity of technical triflusulfuron methyl was evaluated in rabbits, rats, and dogs. In a 21–day dermal toxicity study with rabbits fed dosages of 50, 300, or 1,000 mg/kg/day, the systemic toxicity NOAEL was equal to or greater than 1,000 mg/kg/day for males and females. The dermal toxicity NOAEL was equal to or greater than 1,000 mg/kg/day for males and females.

Two 90–day studies were conducted in the rat. In one study, rats were fed dosages of 6.2, 127, 646, or 965 mg/kg/day (males) or 7.54, 150, 774, or 1,070 mg/kg/day (females). Triflusulfuron methyl exhibited subchronic toxicity at dietary concentrations of 2,000 ppm (127 and 150 mg/kg/day for males and females) or greater in the form of decreased body weights, decreased body weight gains, decreased food efficiency, increased mean relative liver weights, and regenerative anemia. The NOAEL was 6.2 mg/kg/day (males) and 7.54 mg/kg/day(females). In another study, rats were fed dosages of 6.56, 133, 658, or 1,036 mg/kg/day (males) or 7.71, 153, 783, or 1,124 mg/kg/day (females). Triflusulfuron methyl showed subchronic toxicity at dietary concentrations of 2,000 ppm (133 and 153 mg/kg/day for males and females) or greater in the form of decreased body weight, decreased body weight gain, decreased food efficiency, and increased mean liver weights. The NOAEL was 6.56 mg/kg/day (males) and 7.71 mg/kg/day (females).

A subchronic neurotoxicity study with rats fed dosages of 0, 6.1, 46.1, 92.7, or 186.2 mg/kg/day (males) or 7.1, 51.6, 104.1, or 205.2 mg/kg/day (females), resulted in a NOAEL of 92.7 (males) and 7.1 mg/kg/day (females). This was based on decreased body weight/body weight gain at the lowest observed effect level of 186.2 mg/kg/day (males) and 51.6 mg/kg/day (females).

In another 90–day subchronic study, dogs were fed dosages of 3.87, 146.1, or 267.6 mg/kg/day (males) or 3.72, 159.9, or 250.7 mg/kg/day (females). Triflusulfuron methyl was found to be hepatotoxic at 4,000 ppm (146.1 mg/kg/day males and 159.9 mg/kg/day (females), and greater (elevated hepatic enzyme levels and postmortem evidence, including elevation in liver weights and microscopic evidence of bile stasis). Other microscopic findings considered to be treatment related were testicular atrophy and decreased testicular weights and hypercellularity of the sternal and femoral bone marrow, with a corresponding increase in reticulocyte and leukocyte counts seen in the high-dose males and females. Based on the microscopic findings in the liver and testes of the 4,000 ppm and greater treated animals, the NOAEL was 3.87 mg/kg/day (males) and 3.72 mg/kg/day (females).

5. *Chronic toxicity.* The chronic toxicity of technical triflusulfuron methyl was evaluated in dogs, mice, and rats. In a 1–year oral toxicity study with dogs fed dosages of 1.0, 26.9, 111.6 mg/kg/day (males) and 1.2, 27.7, and 95.5 mg/kg/day (females), the NOAEL for males was 26.9 mg/kg/day; this was based on increases in alkaline phosphatase, liver weight, and incidence of minimal centrilobular hypertrophy at the LOAEL of 111.6. For females, the NOAEL was 27.7 mg/kg/day; this was based on increased liver weight and increased incidence of minimal centrilobular hepatocellular hypertrophy at the LOAEL of 95.5 mg/kg/day.

In an 18–month carcinogenicity study, mice were fed dosages of 1.37, 20.9, 349, and 1,024 mg/kg/day (males) and 1.86, 27.7, 488, and 1,360 mg/kg/day (females). Male mice had statistically significant positive trends for hepatocellular adenomas and for combined adenoma/carcinoma (driven entirely by adenomas) at 349 and 1,024 mg/kg/day. These increases were not significant in pair-wise comparisons with control groups and were determined not to be carcinogenic effects by the Carcinogenicity Peer Review Committee (CPRC). The NOAEL was based on body and organ weight effects and was 20.9 mg/kg/day (males) and 27.7 mg/kg/day (females).

In the combined chronic toxicity/carcinogenicity study rats were fed dosages of 0, 0.406, 4.06, 30.6 and 64.5 mg/kg/day (males) and 0, 0.546, 5.47, 41.5, and 87.7 mg/kg/day (females). Male rats have a significant increasing trend and significant differences in pair-wise comparisons of the 30.6 and 64.5 mg/kg/day dose groups with controls for interstitial cell adenomas. This effect was determined to be a carcinogenic effect by the CPRC. No carcinogenic effects were noted in females up to and including 87.7 mg/kg/day (highest dose tested). The LOAEL for chronic toxicity is 30.6 mg/kg/day (males) and 41.5 (females) based on decreased body weight and body weight gain, alternations in the hematology parameters (males predominately) and an increased incidence of interstitial cell hyperplasia in males. The NOAEL for chronic toxicity is 4.06 mg/kg/day (males) and 5.47 mg/kg/day (females). This value is adjusted to the lowest concentration level of the chemical at this dosage (60%), resulting in NOAELs of 2.44 mg/kg/day (males) and 3.28 mg/kg/day (females).

6. *Animal metabolism.* For triflusulfuron methyl, in both the rat and the goat, a majority of the administrated dose was excreted in feces and urine. The biotransformation pathway for triflusulfuron methyl in the rat and the goat was similar. The major pathway was demethylation of the dimethylamino substituent on the triazine ring. The intermediate hydroxylated metabolite was also present. The secondary biotransformation pathway was clevage of the sulfonylurea bridge to form methyl saccharin, *N*-desmethyl triazine amine and *N,N*-bis-desmethyl triazine amine. In the lactating goat, triflusulfuron methyl was not excreted to any appreciable level in the milk. Levels of the ester carbonyl-derived residues were generally below the limit of reliable measurement (< 0.0006 µg equivalent triflusulfuron methyl/mL) and triazine-derived residues reached a daily level of about 0.001 ppm.

Therefore, the metabolic pathways in rats and lactating goats were very similar. There were no significant plant metabolites of triflusulfuron methyl that were not found in the rat or goat metabolism studies. In the unlikely event that triflusulfuron methyl were to enter the livestock diet, triflusulfuron methyl and its metabolites would be rapidly excreted and would not accumulate in meat, meat by-products, or milk.

7. *Metabolite toxicology.* The approximate lethal dose (ALD) of the degradation product, *N,N*-bis-desmethyl triazine amine, in male rats was 450 mg/kg/day. Rats were fed dose rates of 200, 300, 450, 670, 1,000, and 2,300 mg/kg of triflusulfuron methyl. Deaths occurred up to test day 7 in rats dosed at 450 mg/kg body weight and above. Clinical signs of toxicity were observed in lethally and nonlethally dosed rats. In an *in vitro* gene mutation study, *N,N*,-bis-desmethyl triazine amine was not mutagenic in *Salmonella typhimurium* up to a dose of 5,000 µg/plate.

For the degradation product, triazine amine, the ALD in male rats was 670 mg/kg/day. The test substance dose was 200, 300, 450, 670, 1,000, or 2,300 mg/kg. Deaths occurred up to test day 4 in rats dosed at 670 mg/kg and above. Clinical signs of toxicity were observed in lethally and nonlethally dosed animals. In an *in vitro* gene mutation study, triazine amine was not mutagenic in *Salmonella typhimurium* up to a dose of 5,000 µg/plate.

8. *Endocrine disruption.* No special studies investigating potential estrogenic or other endocrine effects of

triflusulfuron methyl have been conducted. However, the standard battery of required toxicology studies have 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 triflusulfuron methyl has an adverse effect on the endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. The acute dietary exposure was estimated for triflusulfuron methyl using the Dietary Exposure Evaluation Model (version 6.73) for a number of subpopulation groups. An acute Tier I dietary analysis was based upon the residues for sugar beet (root) at 0.05 ppm and sugar beet (top) at 0.05 ppm. The acute reference dose (aRfD) is 0.9 mg/kg bw/day (based upon a NOAEL of 90 mg/kg bw/day and a 100-fold safety factor). For triflusulfuron methyl, the predicated exposure for the U.S. population was 0.00460 mg/kg bw/day (0.05 % of the aRfD) at the 95th percentile. The subpopulation with the highest predicted exposure was the non-nursing infants subgroup with an exposure of 0.00166 mg/kg bw/day (0.19% of the aRfD) at the 95th percentile. 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 triflusulfuron methyl.

The chronic dietary exposure was estimated for triflusulfuron methyl using the Dietary Exposure Evaluation Model (version 6.74) for a number of subpopulation groups. A chronic Tier I dietary analysis was based upon residues for sugar beet (root) at 0.05 ppm and sugar beet (top) at 0.05 ppm. The chronic RfD is 0.024 mg/kg bw/day (based upon a NOAEL of 2.44 mg/kg bw/day and a safety factor of 100). The estimated exposure for the U.S. population was 0.000146 mg/kg bw/day (0.6% of the RfD). For the subpopulation with the highest level of exposure (non-nursing infants), the exposure was 0.000433 mg/kg bw/day (>1.8% of the chronic reference dose (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 triflusulfuron methyl.

Even though very conservative assumptions were made in predicting acute and chronic exposures to triflusulfuron methyl, the predicted exposures expressed as percentages of the cRfD and aRfD values were found to be well within the acceptable range.

ii. *Drinking water*. Another potential source of dietary exposure is residues in drinking water. Based on the available environmental studies conducted with triflusulfruon methyl, DuPont concludes that there is no anticipated exposure to residues of triflusulfuron methyl in drinking water. In addition, there is no established maximum concentration level (MCL) for residues of triflusulfuron methyl in drinking water.

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

D. Cumulative Effects

Triflusulfuron 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 acetolacate 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 triflusulfuron methyl has any toxic effects on mammals that would be cumulative with those of any other chemical.

E. Safety Determination

1. *U.S. population*. Based on the completeness and reliability of the toxicology data base and using the conservative assumptions presented earlier, EPA has established a chronic RfD of 0.024 mg/kg/day. This was based on the NOAEL for the 2–year chronic rat study (2.44 mg/kg/day) and a 100–fold safety factor. It has been concluded that the aggregate exposure was 0.6% of the cRfD. Generally, exposures below 100% of the cRfD are of no concern because it represents the level at or below which daily aggregrate 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 triflusulfuron methyl residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of triflusulfuron methyl, data from the previously discussed developmental and multi-generation 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 the prenatal and postnatal exposures to the pesticide. The studies with triflusulfuron 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 triflusulfuron 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 triflusulfuron methyl relative to prenatal and postnatal effects for children is complete.

In addition, the NOAEL of 2.44 mg/kg/day in the chronic rat study (and upon which the cRfD is based) is much lower than the NOAELs defined in the reproduction and developmental toxicology studies. The sub-population with the highest level of exposure was non-nursing infants, where exposure was < 1.8% of the cRfD. Based on these conservative analyses, there is reasonable certainty that no harm will result to infants and children from aggregate exposures to triflusulfuron methyl.

F. International Tolerances

There are no Codex Maximum Residue Levels established for triflusulfuron methyl.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–903; FRL–6396–2]

Notice of Filing a Pesticide Petition to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain