minutes per speaker or organization. The request should identify the name of the individual making the presentation, the organization (if any) they will represent, any requirements for audio visual equipment (*e.g.,* overhead projector, 35mm projector, chalkboard, etc), and at least 35 copies of an outline of the issues to be addressed or of the presentation itself.

Additional information concerning the Science Advisory Board, its structure, function, and composition, may be found on the SAB Website *(http://www.epa.gov/sab)* and in the Annual Report of the Staff Director which is available from the SAB Publications Staff at (202) 564–4533 or via fax at (202) 501–0256.

Dated: January 13, 2000.

Donald G. Barnes, PhD,

Staff Director, Science Advisory Board.

[FR Doc. 00–1560 Filed 1–21–00; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[OPP–00639; FRL–6488–2]

State FIFRA Issues Research and Evaluation Group (SFIREG) Water Quality and Pesticide Disposal Working Committee; Notice of Public Meeting

ACTION: Notice of public meeting.

SUMMARY: The SFIREG Water Quality and Pesticide Disposal Working Committee will hold a 2-day meeting, beginning on February 7, 2000 and ending on February 8, 2000. This notice announces the location and times for the meeting and sets forth the tentative agenda topics.

DATES: The State FIFRA Issues Research and Evaluation Group (SFIREG) will meet on Monday, February 7, 2000 from 8:30 a.m. to 4:00 p.m. and on Tuesday, February 8, 2000 from 8:30 a.m. to 12:00 noon. There will be a CLOSED SESSION (Open Only to EPA and State Lead Agencies) on Monday, February 7, 2000 from 4:00 p.m.–5:00 p.m.

ADDRESSES: The meeting will be held at The Doubletree Hotel, 300 Army Navy Drive, Arlington-Crystal City, VA 22202.

FOR FURTHER INFORMATION CONTACT: Philip H. Gray, SFIREG Executive Secretary, P. O. Box 1249, Hardwick, VT 05843–1249; (802) 472–6956; fax: (802) 472–6957; e-mail address: aapco@plainfield.bypass.com or Elaine Y. Lyon, Field and External Affairs Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Building, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 305–5306; fax number: (703) 308–1850; e-mail address: lyon.elaine@epa.gov.

SUPPLEMENTARY INFORMATION:

I. Does this Action Apply to Me?

This action is directed to the public in general, but all parties interested in SFIREG's information exchange relationship with EPA regarding important issues related to human health, environmental exposure to pesticides, and insight into the EPA's decision-making process are invited and encouraged to attend the meetings and participate as appropriate.

II. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of the minutes, 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/. You may also obtain electronic copies of the minutes, and certain other related documents that might be available electronically, from the Association of American Pesticide Control Officials (AAPCO) Internet Home Page at http://aapco.ceris.purdue.edu/doc/index.html. To access this document, on the Home Page select ''SFIREG'' and then look up the entry for this document under the ''SFIREG Meetings.''

2. *In person.* The Agency has established an administrative record for this meeting under docket control number OPP–00639. The administrative record consists of the documents specifically referenced in this notice, any public comments received during an applicable comment period, and other information related to the State FIFRA Issues Research and Evaluation Group (SFIREG) Water Quality and Pesticide Disposal Working Committee, including any information claimed as Confidential Business Information (CBI). This administrative 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 administrative record, which includes printed, paper versions of any electronic comments that may be submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., 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.

III. Purpose of Meeting

Tentative Agenda:

1. Working committee issues and updates.
2. Update on pesticide field data plan.
3. Update on Total Maximum Daily Load & National Pollution Discharge Elimination System Draft Straw Proposal.
4. Office of Research and Developments grant to study ''The Impact of Lawn Care Practices on Aquatic Ecosystems in Suburban Watersheds.''
5. Update on Pesticides in Ground Water and Surface Water Data bases.
6. Working committee discussion on survey on aquatic pesticides and National Pollution Discharge Elimination System permitting.
7. Florida State University grant to develop Indicators.
8. Updates from the Office of Pesticide Programs and the Office of Enforcement and Compliance Assurance.
9. Other topics as appropriate.

List of Subjects

Environmental protection,

Dated: January 14, 2000.

Jay Ellenberger,

Director, Field and External Affairs Division, Office of Pesticide Programs.

[FR Doc. 00–1547 Filed 1–21–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–909; FRL–6399–6]

Notice of Filing Pesticide Petitions 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–909, must be received on or before February 23, 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–909 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number/e-mail address	Address	Petition number(s)
Shaja R. Brothers	Rm. 284, CM 2, 703–308–3194, e-mail: brothers.shaja®epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA	PP 9E6025
James A. Tompkins (PM 25).	Rm. 239, CM 2, 703–305–5697, e-mail: tompkins.james®epamail.epa.gov.	Do.	PP 5F4505; PP 6F4791

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 codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing 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–909. 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, 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–909 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, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., 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, Crystal Mall 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–909. 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 pesticide petitions 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 these petitions contain 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: January 7, 2000,

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them 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.

I. Acetochlor Registration Partnership

PP 5F4505 and 6F4791

EPA has received pesticide petitions (PP 5F4505 and 6F4791) from Acetochlor Registration Partnership, c/o Zeneca Ag Products, 1800 Concord Pike, Wilmington DE 19850 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 acetochlor (2-chloro-2′-methyl-6′-ethyl-*N*-ethoxymethylacetanilide and it metabolites containing the ethyl methyl aniline (EMA) and the hydroxy methyl aniline (HEMA) moiety, to be expressed as acetochlor, EMA and HEMA and expressed as acetochlor equivalents in or on the raw agricultural commodity field, corn, forage at 3.0 part per million (ppm) (5F4505); corn, sweet, grain (K+CHWHR) at 0.05 ppm; corn, sweet, fodder at 1.0 ppm; and corn, sweet, forage at 1.4 ppm. (6F4791). PP 5F4505 also proposes to divide 40 CFR 180.470 into two sections: (a) Specific tolerances (containing the tolerances for field corn and sweet corn) and (b) Indirect or inadvertent tolerances (containing the tolerances for the rotational crops sorghum, soybean, wheat, and nonanimal grass feeds). PP 6F4791 also proposes that tolerances be established for the indirect or inadvertent residues of acetochlor in or on the the raw agricultural commodities when present therein as a result of the application of acetochlor to growing crops and other nonfood crops as follows: nongrass animal feeds, forage at 0.6 ppm and nongrass animal feeds, hay at 1.0 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 metabolism of acetochlor has been studied in corn and soybeans. The major metabolic pathways are: (i) Uptake of soil metabolites and subsequent metabolism, (ii) uptake of acetochlor followed by oxidative metabolism and conjugation, and (iii) uptake of acetochlor, conjugation with glutathione and subsequent catabolism. Acetochlor is completely metabolized in plants to produce a number of polar metabolites. EPA has determined that the residues of concern are those which contain the EMA and HEMA.

2. *Analytical method*. An adequate enforcement method for residues of acetochlor in crops has been approved. Acetochlor and its metabolites are hydrolyzed to either EMA or to HEMA which are determined by GC-MSD and expressed as acetochlor.

3. *Magnitude of residues*. Field residue trials in field corn with acetochlor were conducted in 32 plots in 8 states. The maximum combined residues (acetochlor and metabolites) were 2.52 ppm in corn forage, 0.217 ppm in corn fodder and <0.04 ppm in corn grain.

Fourteen field residue trials in sweet corn with acetochlor were conducted in 12 states. The maximum combined residues (acetochlor and metabolites) were 1.35 ppm in corn forage, 0.97 ppm in corn fodder and <0.05 ppm in grain.

Seventeen rotational crop residue trials were conducted in 17 states representing the top corn, alfalfa and clover producing regions in the U.S. The maximum combined residue (acetochlor and metabolites) in alfalfa forage was 0.540 ppm and the maximum alfalfa hay residue was 1.870 ppm. The maximum clover forage residue was 0.567 ppm, the maximum clover residue was 1.244 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicology data place technical acetochlor in toxicity category III for eye irritation, toxicity category III for acute oral, acute dermal, and acute inhalation. Technical acetochlor is in category IV for primary skin irritation and it is a skin sensitizer.

2. *Genotoxicty*. In mutagenicity testing, submitted by Monsanto, acetochlor was weakly positive in the Chinese hamster ovary/hypoxanthine-guanine phosphoribosyltransferase (CHO/HGPRT) gene mutation assay with and without activation in the mouse lymphoma assay. Acetochlor was negative in a DNA damage repair assay in rat hepatocytes, a *Salmonella* assay, and two (2) *in vivo* chromosomal aberration studies.

In mutagenicity tests conducted by ZENECA, acetochlor induced a reproducible, positive, mutagenic response in strain TA 1538 of *Salmonella typhimurium* with metabolic activation at 100 milligrams /plate (mg/p) (however, this was less than the 2X background mutation, but was significant at p less than 0.05). Significant increases in number of revertant colonies were not induced in strains TA 1535, TA 1537, TA98, and TA100. The effect in strain TA1538 although reproducible in the first study was not observed in a more extensive follow up study. Acetochlor was not

clastogenic in a mouse micronucleus test at doses tested (898 and 1,436 milligrams/kilograms (mg/kg) in males; 1,075 and 1,719 mg/kg in females). Acetochlor was clastogenic in cultured human lymphocytes both in the presence and absence of S9 mix at 100 mg/milliliters (ml), and in the absence of S9 mix at 50 mg/ml. It has subsequently been shown that the chloroacetyl substituent on acetochlor is the clastogenic moeity, however two structurally related chemicals containing this moiety have been shown to be non-carcinogens as defined by the US NTP.

Acetochlor induced a weak DNA repair (measured by UDS) in rat hepatocytes derived from animals exposed *in vivo* at 2,000 mg/kg. At this dose there is significant hepatotoxicity (depletion of glutathione, severe liver necrosis and substantial release of hepatic enzymes). Acetochlor was negative in the unscheduled DNA synthesis (UDS) assay at a maximum tolerated dose (MTD) of 1,000 mg/kg. In a structural chromosome aberration study, acetochlor at doses 1,000 and 2,000 mg/kg resulted in reduced pregnancy incidence, decreased implants per pregnancy incidence, increased preimplantion loss, and decreased time implant per pregnancy at weeks 2, 3 and 4 of this study. Early and late intrauterine deaths were not affected in this study. The Agency concluded there was positive evidence of mutagenicity at the mid- and high-dose levels in this study. The Acetochlor Registration Partnership has submitted new data which show that there were no mutagenic effects in this study. Acetochlor was negative in a DNA damage (comet) assay conducted using nasal tissue derived from rats treated with a supra-MTD of 1,750 ppm of acetochlor in the diet for either 7 days or 18 weeks.

3. *Reproductive and developmental toxicity*. In a developmental study submitted by Monsanto, with rats fed dosages of 0, 50, 200, and 400 mg/kg/day, acetochlor did not induce developmental toxicity in rats up to 400 mg/kg/day, the highest dose tested (HDT). The maternal no observed adverse effect level (NOAEL) was 200 mg/kg/day based on matting and/or staining of the anogenital region, a decrease in mean maternal weight gain during the treatment period, and in adjusted mean weight gain on gestation day 20 at 400 mg/kg/day (HDT).

In a developmental study submitted by ZENECA , with rats fed dosages of 0, 40, 150, and 600 mg/kg/day, the developmental NOAEL was 150 mg/kg/day based on increased resorptions, post-implantation loss, and decrease in mean fetal weight at 600 mg/kg/day (HDT). The maternal toxicity NOAEL for this study was 150 mg/kg/day based on animals sacrificed moribund, clinical observations, and decreased body weight gain at 600 mg/kg/day (HDT).

In a developmental study submitted by Monsanto, with rabbits fed dosages of 0, 15, 50, and 190 mg/kg/day, acetochlor did not induce developmental toxicity in rabbits up to 190 mg/kg/day (HDT). The maternal toxicity NOAEL was 50 mg/kg/day based on loss of body weight during dosing at 190 mg/kg/day (HDT).

In a developmental study submitted by ZENECA, with rabbits fed dosages of 0, 30, 100, and 300 mg/kg/day, acetochlor did not induce either maternal or developmental toxicity up to 300 mg/kg/day (HDT).

In a 2–generation reproduction study submitted by Monsanto, with rats fed dosages of 0, 30.4, 74.1, and 324.5 mg/kg/day (males) or 0, 44.9, 130.1, and 441.5 mg/kg/day (females), the reproductive NOAEL was 30.4 mg/kg/day for males and 44.9 mg/kg/day for females, based on decreased body weight gain of F2b pups at 74.1 mg/kg/day for males and 130.1 mg/kg/day for females. A NOAEL for systemic effects was not established.

In a 2–generation reproduction study submitted by ZENECA, with rats fed dosages of 0, 1.6, 21, and 160 mg/kg/day, the reproductive NOAEL was 21 mg/kg/day based on significant reductions in pup weight at lactational day 21 and total body weight gain during lactation at 160 mg/kg/day (HDT). The parental NOAEL was 21 mg/kg/day based on reductions in body weight, accompanied by slight reductions in food consumption and significant increases in relative organ weights at 160 mg/kg/day (HDT).

Conclusion. Acetochlor is not considered to be a material that causes developmental or reproductive toxicity. The lowest NOAEL for fetotoxicity was 21 mg/kg/day in a 2–generation reproduction study and the lowest NOAEL for fetotoxicity in a developmental study was 150 mg/kg/day.

4. *Subchronic toxicity*. A 3–month feeding study submitted by Monsanto with rats fed dosages of 0, 40, 100, and 300 mg/kg/day resulted in a NOAEL of 40 mg/kg/day based on loss of body weight and decreased food consumption at 100 mg/kg/day.

A 3–week dermal study submitted by Monsanto with rabbits fed dosages of 0, 100, 400, and 1,200 mg/kg/day resulted in a NOAEL for systemic effects of 400 mg/kg/day based on mortality and decreased body weight at 1,200 mg/kg/day, (HDT). The lowest effect level (LEL) for dermal irritation was 100 mg/kg lowest dose tested (LDT). A NOAEL for dermal irritation was not established.

A 3–week dermal study submitted by ZENECA with rats fed dosages of 0.1, 1.0, 10, or 100 mg/kg/day resulted in minimal to mild skin irritation after 21 days. Signs of systemic toxicity were not apparent at any level. Higher doses were not possible because of severe dermal toxicity at higher doses.

5. *Chronic toxicity*. In a 1–year feeding study submitted by Monsanto, with dogs fed dosages of 0, 4, 12, and 40 mg/kg/day, the NOAEL was 12 mg/kg/day based on decreased body weight gains in males, decreased terminal body weight in females, testicular atrophy with accompanying decreases in absolute and relative testicular weight, increase in relative liver weights in male and females, and clinical chemistry changes at 40 mg/kg/day (HDT).

In a 1–year feeding study submitted by ZENECA, with dogs fed dosages of 0, 2, 10, and 50 mg/kg/day, the NOAEL was 2 mg/kg/day based on increased salivation, ornithine carbamyl transferase, and triglyceride values accompanied by decreased blood glucose levels and liver glycogen levels at 10 mg/kg/day. Interstitial nephritis, tubular degeneration of the testes and hypospermia were reported.

In a chronic feeding/carcinogenicity study submitted by Monsanto, in which rats were fed dose levels of 0, 22, 69, and 250 mg/kg/day, a NOAEL for chronic effects was not established.

In a repeat chronic feeding/carcinogenicity study submitted by Monsanto, in which rats were fed dose levels of 0, 2, 10, and 50 mg/kg/day, the NOAEL for chronic effects was 10 mg/kg/day.

In a chronic feeding/carcinogenicity study submitted by ZENECA, in which rats were fed dose levels of 0, 0.8, 7.9, and 79.6 mg/kg/day, the NOAEL for chronic effects was 7.9 mg/kg/day.

Conclusion. The lowest NOAEL for chronic effects in dogs was 2 mg/kg/day and the lowest NOAEL for chronic effects in rats was 7.9 mg/kg/day. EPA has established the Reference Dose (RfD) for acetochlor at 0.02 mg/kg/day based on the 2.0 mg/kg/day NOAEL in the ZENECA dog study and the application of a 100–fold safety factor.

In a chronic feeding/carcinogenicity study submitted by Monsanto with mice fed dosages of 0, 75, 225, and 750 mg/kg/day (high dose determined to be 973 mg/kg/day by the ARP) carcinogenic effects noted included increased incidence of liver carcinomas in high-

dose males, total lung tumors in females at all dose levels, carcinomas of lungs in females fed 75 and 750 (973) mg/kg/day, uterine histiocytic sarcomas in females at all dose levels, and total benign ovarian tumors in mid-dose females. Other dose-related changes included: (1) Increased mortality and decreased mean body weights in both high-dose males and females, (2) decreased red blood cell count, hematocrit, and hemoglobin in high-dose females at terminal sacrifice, (3) increased white blood count in high-dose males at terminal sacrifice, (4) increased platelet count in mid-and high-dose females at terminal sacrifice, (5) increased mean liver weight and liver-to-body-weight ratios at study termination in all dose groups of males and in high-dose females; increased absolute and relative kidney weights in all dose groups of males at termination; increased absolute and relative adrenal weights in all groups of males and in high-dose females at study termination; and (6) increased interstitial nephritis in high-dose males and females.

In a chronic feeding/carcinogenicity study submitted by ZENECA with mice fed dosages 0, 1.1, 11, and 116 mg/kg/day in males and 0, 1.4, 13, and 135 mg/kg/day in females, carcinogenic effects noted included an increase in pulmonary adenoma in both male and females at the high dose. Pulmonary tumors were confirmed as adenomas or carcinomas of the lung parenchyma and were all of the alveolar type. The NOAEL for systemic toxicity in females was 13 mg/kg/day based on a significant increase in anterior polar vacuoles in the lens of the eye at 135 mg/kg/day.

In a chronic feeding/carcinogenicity study submitted by Monsanto, with rats fed dosages of 0, 22, 69, and 250 mg/kg/day (males) or 0, 30, 93, and 343 mg/kg/day (females), carcinogenic effects noted at 250 (highest dose determined to be 297 mg/kg/day) mg/kg/day in males and 343 mg/kg/day in females included hepatocellular carcinoma in both sexes and thyroid follicular cell adenoma in males. Nasal papillary adenomas were noted in male rats at 69 mg/kg/day and above and in females at 93 mg/kg/day. A NOAEL for chronic effects was not established.

In a repeat chronic feeding/carcinogenicity study submitted by Monsanto, in rats fed dosages of 0, 2, 10, and 50 mg/kg/day oncogenic effects noted at 50 mg/kg/day (HDT) included neoplastic nodules of the liver, follicular adenoma/cystadenoma of the thyroids and papillary edema of the mucosa of the nose/turbinates in high dose animals. The NOAEL for chronic effects was 10 mg/kg/day based on decreased body weights and body weight gain in both sexes, high cholesterol levels in males, increased absolute and relative kidney and liver weight in males, and increased testicular weights at 50 mg/kg/day (HDT).

In a 2–year chronic feeding/carcinogenicity study submitted by ZENECA, with rats fed dosages of 0, 0.8, 7.9, and 79.6 mg/kg/day, carcinogenic effects noted at 79.6 mg/kg/day (HDT) included a significant increase in nasal epithelial adenomas and thyroid follicular cell adenomas in both sexes at 79.6 mg/kg/day. Also, at that dose nasal carcinoma was present in two males and one female rat at this dose. Rare tumors in the form of benign chondroma of the femur and basal cell tumor of the stomach were also observed at 79.6 mg/kg/day. The systemic NOEL was 7.9 mg/kg/day based on decreased body weight gain, decreased food efficiency, increased organ to body weight ratios, increased plasma GGT and cholesterol at 79.6 mg/kg/day (HDT).

Conclusions. Three oncogenicity studies have been conducted with acetochlor in rats and two have been conducted in mice. In rats, increased incidences of tumors in nasal, thyroid and liver tissues were found only at dose levels equal to or exceeding the MTD. Liver tumors were found in only one rat study and at the highest dose tested (297mg/kg/day), a dose which greatly exceeded the MTD. The nasal tumors, found only at and above the MTD, are the only biologically relevant and reproducible oncogenic effect in rats.

In mice, increased incidences of tumors in liver, lung, and uterine tissues were observed. The liver tumors were observed only in one study, at the HDT (973mg/kg/day) a dose which greatly exceeded the MTD as evidenced by increased mortality of approximately 90%. The lung tumors and uterine histiocytic sarcomas were observed in all treated female groups in one study, but there was no dose-response relationship which makes the relationship to treatment and relevance equivocal. Lung tumors occurred only in high dose animals in the second mouse study and their incidence rate was within the historical control range for the laboratory. The rat and mouse liver tumors and the mouse lung and histiocytic sarcomas have been subjected to an independent pathology peer review.

Overall, the only clear oncogenic responses in rats or mice are found only at high dose levels at or above the MTD. This suggests that such tumors are not produced by genotoxic mechanisms, but by other threshold-dependent mechanisms. The weight of the evidence of all the genotoxicity studies conducted with acetochlor also supports the conclusion that tumor formation is not driven by genotoxic mechanisms. An overview of the genotoxicity studies with acetochlor has been reported by Ashby, et al. in Human and Experimental Toxicology, 15, 702, 1996 (EPA MRID NO. 44069503).

Mechanistic studies with alachlor, a structural analog of acetochlor which produces the same nasal and thyroid tumors in the rat, provide additional evidence that rodent tumors incident to acetanilide dosing are produced by indirect threshold mechanisms that are unique to the rat and not relevant to humans under realistic exposure levels. The Acetochlor Registration Partnership (ARP) has conducted and submitted a number of studies on the mechanism of tumor formation with acetochlor. The ARP believes these studies establish the basis for the use of a Margin of Exposure (MOE) for the cancer risk assessment for acetochlor.

6. *Animal metabolism*. The metabolism of acetochlor has been studied in goats, laying hens and rats. EPA has concluded that the nature of the residue in ruminants and poultry are adequately understood and the residue of concern is the same as that in corn.

7. *Metabolite toxicology*. EPA has determined that the residues of concern are those which contain the EMA and HEMA.

8. *Endocrine disruption*. Acetochlor is not a member of a class of chemicals associated with direct adverse effects on the endocrine system. The subchronic, chronic, developmental and reproductive studies with acetochlor satisfy the present data requirements, and they have measured many toxic endpoints which are sensitive to endocrine-modulation activity. Acetochlor has not produced effects in these toxicity studies that can be related to direct interference with female or male endocrine systems.

C. Aggregate Exposure

1. *Dietary exposure*. The nature of the residue in plants and animals is understood. Acetochlor metabolizes extensively to yield a number of polar metabolites. Tolerances have been established at 40 CFR 180.470 for raw agricultural commodities of field corn and indirect or inadvertent residues in or on sorghum, soybean and wheat. The tolerances are combined acetochlor, and metabolites that contain the EMA and HEMA moieties expressed as acetochlor. No tolerances have been established for livestock commodities because there is

no reasonable expectation of finite residues based on the results of exaggerated rate feeding studies.

i. *Food —a. Acute*. An acute dietary analysis was performed based on the EPA selected acute NOAEL of 150 mg/kg/day for developmental toxicity. The results of this analysis produced MOEs of greater than 70,000 for all 23 subgroups of the U.S. population. The most highly exposed subgroup, non-nursing infants, has a MOE of 77,000. EPA generally considers MOEs of greater than 100 to provide adequate acute dietary safety. Therefore, this evaluation demonstrates that acetochlor does not represent an acute dietary concern.

b. Chronic. The theoretical maximum residue contribution (TMRC) for the general U.S. population from all established uses combined with the proposed tolerance on corn forage is 1.11×10^{-4} mg/kg/day. For non-nursing infants less than 1 year old, the most highly exposed subgroup, the TMRC is 3.24×10^{-4} mg/kg/day. The TMRC is calculated assuming that all of the corn crop is treated with acetochlor, that all crop commodities bear tolerance-level residues, and that all rotation crops are grown in soil treated with acetochlor and thus all rotation crop commodities have tolerance level residues. A refined dietary exposure estimate, based on 30% of corn acres treated, actual maximum residues found in crop commodities, and reduction of residues in some processed commodities was calculated for the same population groups. The refined and more accurate exposure estimate, called the Anticipated Residue Contribution (ARC), is 1.0×10^{-5} for the U.S. general population and 2.7×10^{-5} for non-nursing infants. The TMRC represents only 0.55% of the RfD for the general population. The ARC represents only 0.05% of the RfD.

ii. *Drinking water*. Acetochlor is not registered for direct application to bodies of water. Seasonal run-off from treated fields can be transported to surface water. Since March 1995, the ARP has been monitoring drinking water from 175 community water systems (CWSs) which take their water supplies from surface water sources. The 175 CWSs take water from watersheds of all sizes in major acetochlor use areas but primarily from small watersheds located in areas of high-intensity corn production. Water samples taken every 2 weeks from mid March through early September from each CWS are analyzed for acetochlor. The results to date show that acetochlor was non-detected in about 80% of all individual samples of drinking water, with peak concentrations occurring mainly in May and June, the peak use season for acetochlor. Only about 10% of the participating CWSs had time-weighted annualized mean concentrations (AMC) above 0.1 parts per billion (ppb). There were no CWSs that had AMCs exceeding 2 ppb, the annual AMC limit set for acetochlor in the EPA-ARP registration agreement.

Although acetochlor is not expected to leach through most agricultural soils, there is a potential for limited ground water contamination in areas of highly permeable soils. To address this possibility, acetochlor products are labeled to prohibit use in fields where the depth to ground water is less than 30 feet and where the soils are ''sands'' with less than 3% organic matter; ''loamy sands'' with less than 2% organic matter; or ''sandy loams'' with less than 1% organic matter. However, shallow ground water contamination can also result from misuse, improper well construction and the movement of surface water into direct conduits to ground water. The ARP has been conducting a ground water monitoring (GWM) program consisting of 175 wells immediately adjacent to acetochlor treated fields since 1995. The wells are located in a variety of soil types to cover the range from light permeable soils to heavy less vulnerable soils, reflecting the soils on which corn is grown in the seven major corn-producing states. The ARP GWM wells are agricultural monitoring wells and do not adequately represent the drinking water wells across the entire country. Therefore, sporadic detections at very low levels cannot be extrapolated to provide accurate estimates of acetochlor in drinking water derived from ground water. A series of eight Prospective Ground Water (PGW) studies are being conducted by the ARP to monitor the movement of acetochlor to ground water under intensively instrumented fields, across a range of soil textures. Two studies initiated during 1995 are nearing completion and neither show any indication of acetochlor movement. Four studies commenced during 1996 and continue to show no acetochlor ground water contamination. Traces of acetochlor were detected at one of these sites at one sampling interval, soon after application. The residues were extremely low (0.06 ppb) and had dissipated by the next sampling interval.

The conditions of the registration of acetochlor include cancellation triggers based on detection scenarios in the Surface Water Monitoring Program, the Ground Water Monitoring Program and the Prospective Ground Water Program that will preclude any significant, widespread contamination of drinking water.

For the purpose of chronic risk assessment, a level of 0.1 ppb seems to represent a reasonable, upper-bound level for acetochlor in drinking water. Based on 0.1 ppb in the water and an assumed water consumption of 2 liters per day for an adult weighing 70 kg, the upper bound exposures would be 2.9×10^{-6} mg/kg/day.

For the purpose of assessing short term risk, a level of 2 ppb, the probable MCL, represents a reasonably conservative, upper bound level for acetochlor in drinking water. Based on 2 ppb in the water and an assumed water consumption of 2 liters per day for an adult weighing 70 kg and 1 liter per day for a child weighing 10 kg, the short-term exposure for the adult would be 5.7×10^{-5} mg/kg/day and for the child, 2.0×10^{-4} mg/kg/day.

2. *Non-dietary exposure*. Acetochlor is not registered for any use which would result in non-occupational, non-dietary exposure for the general population. Acetochlor is registered for use on corn, a commercial crop which is grown in fields remote from public-use areas. Acetochlor products are Restricted Use, for use only by Certified Applicators which means the general public cannot buy or use acetochlor.

D. Cumulative Effects

Toxicological testing of the chloroacetamide herbicide family in animals with high doses has produced a number of observed effects. Certain effects in some tissues are observed in two, three, or four members of the family, but there is no single effect that represents conclusive evidence of a common mechanism of toxicity existing throughout the chloroacetamide family.

EPA has not established procedures for determining when pesticides share a common toxic mechanism, or provided a definition of ''concurrent exposure.'' At this time there is no established procedure for risk assessment of pesticides which may have a common mechanism by may differ in potency and exposure. Following an EPA proposal to the FIFRA Scientific Advisory Panel meeting on March 20, 1997 (Docket No. OPP–00466) that nasal tumors in alachlor, acetochlor, butachlor, and perhaps metolachlor may be formed by a common toxic mechanism, Monsanto Company has derived an equation to calculate a MOE for the combined, concurrent exposure to multiple chloroacetamide herbicides that may share a common mechanism for nasal tumors. The mechanism is thought to be metabolic production of

an electrophilic 3,5-dialkyl-benzoquinone-4-imine (DABQI) or similar compounds at sufficient levels to cause cytotoxicity, proliferation of nasal cells and neoplasms in nasal tissues. The equation, as presented in ''Summary Information and Assessment as Required for the Reregistration of Alachlor by the Food Quality Protection Act of 1996'' [MRID 44252200] is:

$$MOE=1\div([ala]\div[ala]_{10}) + ([Chlor1]\div[Chlor1]) + ([Chlor2]\div[Chlor2]_{10}) + ([Chlor3]\div[Chlor3]_{10}) + Etc$$

In which, [ChlorX] represents the Aggregate Exposure to each individual chloroacetamide herbicide which shares the common mechanism with alachlor (ala), and $[ChlorX]_{10}\leq$ represents the toxicological dose of that same herbicide which produced a measurable (10%) increase in tumors in tested animals (i.e., the ED_{10}). This equation gives the cumulative MOE relative to the ED_{10} and is valid assuming approximately constant relative potencies among the chloroacetamides at exposures below the ED_{10}. Since the ED_{10} will almost always exceed the NOAEL, this MOE will be smaller than the NOAEL-based MOE.

The ARP adopts this equation for the purpose of the cumulative risk assessment for chloroacetamides and to show that acetochlor uses meet the FQPA standard of reasonable certainty of no harm even if a common mechanism of toxicity is presumed to exist for several chloroacetamides.

E. Safety Determination

1. *U.S. population* —i. *U.S. general population -acetochlor alone*. The upper bound Aggregate Exposure estimate for short-term exposures to acetochlor is 6.7 x 10^{-5} mg/kg/day. The Toxicity Endpoint Reference Committee has established 150 mg/kg/day as the acute dietary endpoint for risk assessment. Comparing the aggregate exposure to this endpoint indicates that short-term exposures have a margin of safety of 2,238,805.

The Aggregate Exposure estimate for chronic exposures to acetochlor is 1.29 x 10^{-5} mg/kg/day. This exposure utilizes only 0.065% of the RfD of 0.02 mg/kg/day. EPA generally has no concern about exposures below 100% of the RfD for the U.S population.

For cancer risk assessment, the ARP proposes that acetochlor be assessed by the MOE method that has been approved for cancer risk assessment of alachlor, a close structural analog which produces the same nasal and thyroid tumors in the rat. The appropriate cancer reference endpoint for acetochlor is the lowest NOAEL for tumors which is 26 mg/kg/day, the NOAEL for nasal tumors in the rat. Comparison of the aggregate exposure estimate of 1.29 x 10^{-5} mg/kg/day to the 26 mg/kg/day cancer endpoint gives a MOE (relative to this minimum NOAEL) of 2,015,504. The margins of safety for short-term exposure, chronic exposure and carcinogenicity are all adequate and support the conclusion that there is a reasonable certainty of no harm resulting from the established and proposed uses of acetochlor.

ii. *U.S. general population—acetamides common nasal mechanism*. The Aggregate Exposure (chronic) estimate for acetochlor is given above as 1.29 x 10^{-5} mg/kg/day. Using Aggregate Exposure estimates and ED_{10} derived by Monsanto for alachlor, butachlor and metolachlor, and this refined Aggregate Exposure estimate for acetochlor, the Common Mechanism MOE for all four pesticides was calculated.

Because some of these active ingredients have more than one chronic rat study, MOE ED_{10} was calculated using the lowest or worst case ED_{10}'s. (The lowest ED_{10}'s were 8.5 mg/kg/day for alachlor, 40.7 mg/kg/day for acetochlor and 85.1 mg/kg/day for butachlor. For metolachlor there were insufficient data to estimate an ED_{10} and a worst-case value of 150 mg/kg/day was used. The aggregate exposure estimates used for alachlor, butachlor, and metolachlor were 1.7 x 10^{-5}, 5.2 x 10^{-7}, and 2.1 x 10^{-4} mg/kg/day, respectively.) The Combined Mechanism MOE relative to the ED_{10} was 268596. This MOE is sufficiently large to demonstrate that there is a reasonable certainty of no harm from cumulative exposure to these chloroacetamides even if they are considered to share a common toxic mechanism.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of acetochlor, EPA considers data from developmental studies in the rat and the rabbit and a 2–generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure to female test animals. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals data on systemic toxicity and the survival, growth and development of the offspring.

Based on the current toxicological data requirements, the acetochlor data base is complete and sufficient for assessing prenatal and postnatal effects on children. There are two developmental studies with acetochlor in both the rat and the rabbit and there are two reproduction studies in the rat. In the four developmental studies and two reproduction studies with acetochlor, the fetal NOAEL's were either equal to or higher than the maternal (systemic) NOAEL's, indicating that there is no increased sensitivity for offspring. The NOAEL of 2 mg/kg/day in the dog study which was used to establish the RfD is lower than the lowest developmental NOAEL by a factor of 75, and lower than the lowest reproductive NOAEL by a factor of 10, suggesting that the RfD is appropriate for assessing aggregate risk to infants and children. The results of the acetochlor testing establishes that there is reasonable certainty of no harm to infants and children from the proposed uses of acetochlor.

The upper bound Aggregate Exposure for infants or children is 2.27 x 10^{-4} mg/kg/day, representing the combination of dietary exposure for non-nursing infants less than 1 year old (the most highly exposed subgroup) with potential short-term exposure to drinking water containing 2.0 ppb acetochlor. This potential short-term exposure provides a margin of safety of 660,793 when compared to the toxicological reference point of 150 mg/kg/day for acute dietary exposures. Chronic exposure at this level would utilize only 1.1% of the RfD. EPA generally has no concern about chronic exposures that utilize less than 100% of the RfD. Cancer risk assessment for children is considered to be included in the adult assessment because of the long induction period for carcinogenic effects. The cumulative risk assessment for chloroacetamides is based on the proposed common mechanism for induction of nasal tumors, a process requiring a long dosing period. Therefore, the data presented support the conclusion that there is a reasonable certainty of no harm to infants or children will result from the established and proposed uses for acetochlor.

F. International Tolerances

There are no Codex Alimentarius Commission (CODEX) Maximum Residue Levels established for residues of acetochlor on agricultural commodities.

II. Interregional Research Project Number 4

9E6025

EPA has received a pesticide petition (9E6025) from the Interregional Project Number 4 (IR-4), New Jersey

Agricultural Experiment Station, Rutgers University, New Brunswick, New Jersey 08903 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of pyridate, 0-(6-chloro-3-phenyl-4-pyridazinyl)-S-octyl carbonothioate and its metabolite 6-chloro-3-phenyl-pyridazine-4-ol (known as SAN 1367), and conjugates of SAN 1367 in or on the raw agricultural commodities peppermint tops and spearmint tops at 0.20 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 support granting of the petition. Additional data may be needed before EPA rules on the petition. This summary was prepared by Novartis Crop Protection, Inc., Greensboro, NC, 27419.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of pyridate in plants is well understood based on studies with broccoli, corn, and peanut. Pyridate is rapidly broken down by hydrolysis to its major degraded, SAN 1367. The SAN 1367 metabolite is further conjugated to glucoside and degraded.

2. *Analytical method*. The proposed analytical method is ''Method of Analysis of Determination of Residues of Pyridate and its Metabolites CL 9673 and Conjugated CL 9673 in Plant Materials.''

B. Toxicological Profile

1. *Acute toxicity*. Results of a rat acute oral study showed a lethal dose $(LD)_{50}$ of 4,690 mg/body weight (bwt)/day (5,993 mg/kg in males and 3,544 mg/kg in females).

In a rat acute dermal study, the LD_{50} was shown to be > 2,000 mg/kg. A rat acute inhalation study yielded a LD_{50} > 4.37 mg/milliliter (ml).

Results of a primary eye irritation study in the rabbit indicated that pyridate is a mild irritant.

A primary dermal irritation study showed pyridate to be a moderate skin irritant, whereas, a dermal sensitization study indicated it is a sensitizer.

2. *Genotoxicity*. Pyridate was tested in the Ames test, mouse micronucleus assay, chromosome aberration assay with Chinese hamster ovary cells (CHO), the REC assay, and rat hepatocyte unscheduled DNA synthesis assay. Results were negative for mutagenicity and chromosome aberrations.

3. *Reproductive and developmental toxicity*. A developmental toxicity study in the rat dosed at 0, 55, 165, 400, or 495 mg/kg/day showed a maternal no observed adverse effect level (NOAEL) of 165 mg/kg/day, and a developmental NOAEL > 495 mg/kg/day.

A developmental toxicity study in the rabbit with doses of 0, 150, 300, or 600 mg/kg/day showed a maternal NOAEL of 300 mg/kg/day and a developmental NOAEL > 600 mg/kg/day.

Results of a multi-generational reproduction study with rats dosed at 0, 2.2, 10.8, or 67.5 mg/kg/day showed a NOAEL of 10.8 mg/kg/day for maternal and developmental toxicity.

4. *Subchronic toxicity*. Results of a 21–day dermal study showed a NOAEL > 1,000 mg/kg. A 90–day feeding study in rats dosed at 0, 62.5, 177, and 500 mg/kg/day showed a NOAEL of 62.5 mg/kg/day. No neuropathological effects were found.

A 90–day feeding study in dogs with doses of 0, 20, 60, or 200 mg/kg/day showed a NOAEL of 20 mg/kg/day. Slight degenerative myelopathy in the peripheral nerves was observed at the highest dose level, which is much higher than the NOAEL and the expected exposure from field use.

5. *Chronic toxicity*. A 1–year feeding study in dogs was conducted with doses of 0, 5, 20 or 60 mg/kg/day for 34 weeks. After week 34, the doses were increased to 30, 100, or 150 mg/kg/day because no toxic effects were evident at the lower doses. The final results showed a systemic NOAEL of 20 mg/kg/day.

A lifespan (121 week) chronic/carcinogenicity study in rats treated with analytical levels of 0, 2.2, 10.8, or 67.5 mg/kg/day (equivalent to 0, 48, 240, or 1,500 ppm) showed a systemic NOAEL of 10.8 mg/kg/day (240 ppm) based on body weight depression. No carcinogenic potential was observed.

In an 18–month carcinogenicity study, mice were fed doses of 0, 400, 800, 1,600 or 7,000 ppm of pyridate. In males, dose levels were approximately 0, 47.7; 97.1; 169.5, and 882.6 mg/kg bwt/day; in females, dose levels were approximately 0, 54.5, 114.6, 204.3, and 1,044.6 mg/kg bwt/day with a NOAEL at 800 ppm (97.1 mg/kg in males and 114.6 mg/kg in females). Results showed no evidence of carcinogenicity.

Carcinogenicity. Existing data demonstrate that there is no evidence of carcinogenicity in rats at 1,500 ppm (67.5 mg/kg/day) or mice at 7,000 ppm (883 mg/kg bwt/day in males, and 1,044.6 mg/kg bwt/day in females). These data have been obtained at dosing in excess of any dietary exposure.

6. *Animal metabolism*. Pyridate has been tested in rats, dogs, cattle, goats, and hens. In every study, pyridate was hydrolyzed to SAN 1367 and rapidly excreted, primarily through the urine as SAN 1367 or its glucoside or glucuronide conjugates. Pyridate and its metabolites are not persistent and do not accumulate in animal systems.

C. Aggregate Exposure

1. *Dietary exposure*. Pyridate is registered for use in corn, peanut, and cabbage. The pending petition add the use in/on peppermint tops and spearmint tops. The potential dietary exposure of the population to residues of pyridate or its metabolites is calculated based on Theoretical Maximum Residue Contribution (TMRC) for all crops with pyridate use. The TMRC is a worst case estimate of dietary exposure since it assumes that 100% of all crops for which tolerances are established are treated with pyridate, and that pesticide residues are present at the tolerance levels. Novartis maintains that this method of calculation result in an overestimation of the exposure and is considered conservative. Dietary exposure is not expected in meat, milk, poultry, or eggs, based on cow and hen feeding studies, animal metabolism studies, and the fact the residue studies indicate that residues are not present in crops fed to animals above the limit of detection.

i. *Chronic effects*. The chronic population adjusted dose (cPAD) has been established based on the chronic toxicity data base. The cPAD = 0.11 mg/kg bwt/day based on the NOAEL of 10.8 from the lifespan rat carcinogenicity study due to body weight depression in males, and assuming a safety factor of 100.

ii. *Acute effects*. Acute dietary analysis compared the daily dietary exposure to the lowest NOAEL for subchronic studies. EPA's current policy for Tier I analysis uses the conservation assumption that all residues are at a high end estimate or maximum, typically taken as the tolerance value. Acute dietary assessment for pyridate was generated by comparing the ratio of exposure and the NOAEL from the 90–day feeding study in dogs of 20 mg/kg bwt/day to determine a margin of exposure (MOE). The exposure estimate includes all current and pending tolerances from Sandoz Agro, Inc. and IR-4. A MOE of 100 or more is considered acceptable. For all subgroups evaluated, the MOE is greater than 140,000.

2. *Drinking water*. Drinking water is not expected to be a means of exposure to pyridate. Environmental studies indicate that pyridate binds to the soil and is rapidly hydrolyzed into its

metabolites. The metabolites are then photolyzed and further degraded and finally mineralized to CO_2. Leaching studies and lysimeter studies indicate that under typical agricultural conditions, neither pyridate nor its metabolites were detected below 30 centimeters. Ground water monitoring studies conducted in Europe have not confirmed any detection of pyridate or metabolites. Therefore, significant movement of pyridate is not likely and is not a considerable factor in assessing human health risk.

3. *Non-dietary exposure.* There are no registered uses for pyridate on residential or recreational turf. Therefore, non-dietary exposure of pyridate is not likely and not a factor in assessing human health risk.

D. Cumulative Effects

Pyridate belongs to the pyridazine group of herbicidal compounds and has a unique mode of action in plants. Sandoz does not have data to indicate a common mechanism of toxicity to other compounds in humans. Therefore cumulative effects from common mechanisms of action are unlikely.

E. Safety Determination

1. *U.S. population.* The cPAD is calculated to be 0.11 mg/kg bwt/day. The estimates of exposure are based on conservative assumptions that all crops with a tolerance for pyridate are treated and that all residues found are at the maximum or tolerance level. The dietary exposure to the U.S. population for the current uses plus the corn grain, peanut butter, and cabbage uses is estimated at most to be 6.0×10^{-5} mg/kg/bwt/day, which is 0.1% of the cPAD. Therefore, Novartis concludes that there is reasonable certainty of no harm from aggregate exposure of residues of pyridate or its metabolites including all dietary and other non-occupational exposures.

2. *Infants and children.* Pyridate is not a reproductive or developmental toxicant. Therefore no specific effects on infants and children are expected. Based on the weight of evidence of the toxicity studies, an additional safety factor is not warranted.

Using the same assumptions as above, the exposure to infants and children is presented as a percent of cPAD. The dietary exposure for the current uses plus the corn grain, peanut butter, and cabbage uses for non-nursing infants is estimated as 1.25×10^{-4} mg/kg/bwt/day, which is 0.1% of the cPAD. For children age 1–6, the estimated exposure is 1.43×10^{-4} mg/kg/day, 0.1% of the cPAD. Therefore, Sandoz concludes that there is reasonable certainty of no harm from aggregate exposure of residues of pyridate or its metabolites including all dietary and other non-occupational exposures.

F. International Tolerances

No international tolerances have been established for pyridate on peppermint tops and spearmint tops by CODEX Alimentarius Commission.

[FR Doc. 00–1553 Filed 1–21–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–914; FRL–6486–8]

Notice of Filing Pesticide Petitions 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–914, must be received on or before February 23, 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–914 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Mary Waller, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–9354; e-mail address: waller.mary@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 codes	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–914. 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, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m.,