EPA must consider and use ''voluntary consensus standards'' (VCS) if available and applicable when developing programs and policies unless doing so would be inconsistent with applicable law or otherwise impractical.

The EPA believes that VCS are inapplicable to this action. Today's action does not require the public to perform activities conducive to the use of VCS.

I. Petitions for Judicial Review

Under section 307(b)(1) of the Clean Air Act, petitions for judicial review of this action must be filed in the United States Court of Appeals for the appropriate circuit by November 29, 1999. Filing a petition for reconsideration by the Administrator of this final rule does not affect the finality of this rule for the purposes of judicial review nor does it extend the time within which a petition for judicial review may be filed, and shall not postpone the effectiveness of such rule or action. This action may not be challenged later in proceedings to enforce its requirements. (See section 307(b)(2).) EPA encourages interested parties to comment in response to the proposed rule rather than petition for judicial review, unless the objection arises after the comment period allowed for in the proposal.

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Hydrocarbons, Ozone.

Dated: September 17, 1999.

John P. DeVillars,

Regional Administrator, Region I.

Part 52 of chapter I, title 40 of the Code of Federal Regulations is amended as follows:

PART 52—[AMENDED]

1. The authority citation for part 52 continues to read as follows:

Authority: 42 U.S.C. 7401 *et seq.*

Subpart EE—New Hampshire

2. Section 52.1520 is amended by adding paragraphs (c)(61) and (62) to read as follows:

§ 52.1520 Identification of plan.

* * * * * *

(c) * * *

(61) Revisions to the State Implementation Plan submitted by the New Hampshire Department of Environmental Services on July 9, 1998.

(i) Additional materials.

(A) ''New Hampshire Stage II Comparability Analysis,'' prepared by the New Hampshire Department of Environmental Services, dated July 1, 1998.

(62) Revisions to the State Implementation Plan submitted by the New Hampshire Department of Environmental Services on June 7, 1994.

(i) Additional materials.

(A) Letter from the New Hampshire Department of Environmental Services dated June 7, 1994 submitting a revision to the New Hampshire State Implementation Plan.

(B) ''Clean Fuel Fleet Equivalency Demonstration,'' prepared by the New Hampshire Department of Environmental Services, dated May, 1994.

[FR Doc. 99–25156 Filed 9–28–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 52

[WI91–01–7322; FRL–6446–7]

Approval and Promulgation of Implementation Plans; Wisconsin; Withdrawal

AGENCY: Environmental Protection Agency (EPA).

ACTION: Withdrawal of direct final rule.

SUMMARY: Because EPA received adverse comment, we are withdrawing the direct final rule for the approval of a site-specific revision to the Wisconsin sulfur dioxide (SO_2) State Implementation Plan (SIP). We published the direct final rule on August 16, 1999 (64 FR 44415), approving alternate SO_2 emission limits for Murphy Oil, located in Superior, Wisconsin. We stated in the direct final rule that if we received adverse comment by September 15, 1999, we would publish a timely notice of withdrawal in the **Federal Register**. We subsequently received adverse comment on the direct final rule. We will address those comments in a subsequent final action based on the parallel proposal also published on August 16, 1999 (64 FR 44451). As stated in the parallel proposal, we will not institute a second comment period on this action.

DATES: As of September 29, 1999, EPA withdraws the direct final rule published at 64 FR 44415, on August 16, 1999.

ADDRESSES: Copies of the SIP revision, public comments on the rulemaking, and other materials relating to this rulemaking are available for inspection at the following address: (It is recommended that you telephone Christos Panos at (312) 353–8328, before visiting the Region 5 Office.) United States Environmental Protection Agency, Region 5, Air and Radiation Division, Air Programs Branch (AR–18J), Regulation Development Section, 77 West Jackson Boulevard, Chicago, Illinois 60604.

FOR FURTHER INFORMATION CONTACT: Christos Panos, Regulation Development Section, Air Programs Branch (AR–18J), Air and Radiation Division, United States Environmental Protection Agency, Region 5, 77 West Jackson Boulevard, Chicago, Illinois 60604, (312) 353–8328.

List of Subjects in 40 CFR Part 52

Environmental protection, Intergovernmental relations, Sulfur dioxide.

Authority: 42 U.S.C. 7401 *et seq.*

Therefore the amendment to 40 CFR part 52 which added § 52.2570(c)(99) is withdrawn.

Dated: September 17, 1999.

Francis X. Lyons,

Regional Administrator, Region 5.

[FR Doc. 99–25311 Filed 9–28–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300929; FRL–6385–6]

RIN 2070–AB78

Pymetrozine; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a permanent tolerance for pymetrozine [1,2,4-triazin-3(2*H*)-one,4,5-dihydro-6-methyl-4-[(3-pyridinylmethylene) amino]] in or on tuberous and corm vegetables (Subgroup 1-C), at 0.02 parts per million (ppm). Novartis Crop Protection, Inc. of Greensboro, North Carolina 27419, requested this tolerance under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996.

DATES: This regulation is effective September 29, 1999. Objections and requests for hearings, identified by docket control number OPP–300929, must be received by EPA on or before November 29, 1999.

ADDRESSES: Written objections and hearing requests may be submitted by mail, in person, or by courier. Please follow the detailed instructions for each

method as provided in Unit VI. of the ''SUPPLEMENTARY INFORMATION'' section. To ensure proper receipt by EPA, your objections and hearing requests must identify docket control number OPP–300929 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Dan Peacock, Registration Division (7504C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–5407; and e-mail address: peacock.dan@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	111 112 311 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 in the ''FOR FURTHER INFORMATION CONTACT'' section.

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 OPP–300929. The official record consists of the documents specifically referenced in this action, 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 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. Persons wishing to review copies of the actual studies summarized in this document need to file a Freedom of Information (FOI) request with Ms. Jeralean Green, Freedom of Information Office (1105), 401 M St., Washington, DC 20460. Specify the MRID number of each study needed. The FOI telephone number is (202) 260–4048.

II. Background and Statutory Findings

In the **Federal Register** of May 20, 1998 (63 FR 27723) (FRL–5773–2), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Public Law 104–170) announcing the filing of a pesticide petition (PP) for tolerance by Novartis Crop Protection, Inc. of Greensboro, NC 27419. This notice included a summary of the petition prepared by Novartis Crop Protection, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR 180.556 be amended by establishing a tolerance for residues of the insecticide pymetrozine [1,2,4-triazin-3(2*H*)-one,4,5-dihydro-6-methyl-4-[(3-pyridinylmethylene) amino]], in or on hops at 5 ppm, fruiting vegetables at 0.05 ppm, and cucurbits and potatoes at 0.02 ppm.

Section 408(b)(2)(A)(i) of the FFDCA allows EPA to establish a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance is ''safe.'' Section 408(b)(2)(A)(ii) defines ''safe'' to mean that ''there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.'' This includes exposure through drinking water and in residential settings, but does not include occupational exposure. Section 408(b)(2)(C) requires EPA to give special consideration to exposure of infants and children to the pesticide chemical residue in establishing a tolerance and to ''ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue....''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. For further discussion of the regulatory requirements of section 408 and a complete description of the risk assessment process, see the final rule on Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997) (FRL–5754–7).

III. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of pymetrozine and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for residues of pymetrozine on tuberous and corm vegetables (Subgroup 1-C), at 0.02 ppm. EPA's assessment of the exposures and risks associated with establishing the tolerance follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children. The nature of the toxic effects caused by pymetrozine are discussed in this unit.

1. *Acute toxicity*. In general, technical pymetrozine has low acute toxicity, being classified as Toxicity Category III for acute dermal and primary eye irritation studies and Toxicity Category IV for acute oral, acute inhalation and primary dermal studies. It is a slight sensitizer.

2. *Subchronic and chronic toxicity*. This section summarizes the results of

the subchronic and chronic toxicity, metabolism, and dermal penetration studies in animals.

i. *Subchronic toxicity*. A subchronic feeding study in rats (MRID No. 44024939, Guideline 82–1a), using 98% pymetrozine, exposed animals for 3 months at dose levels of 0, 50, 500 or 5,000 ppm. These dose levels correspond to 0, 3.42, 32.5 or 360 milligrams/kilograms/day (mg/kg/day) in males and 0, 3.63, 33.9 or 370 mg/kg/day for females. At 5,000 ppm, body weight was decreased. Food and water consumption also decreased. After 14 weeks, the numbers of white blood cells increased (leucocytosis) 42% in males and 73% in females. After the 4–week recovery period, the numbers of white blood cells were still elevated 6% in males and 35% in females. The lowest observable adverse effect level (LOAEL) is 5,000 ppm (~360 mg/kg/day) based primarily on body weight and liver effects. The no observable adverse effect level (NOAEL) is 500 ppm (~32.5 mg/kg/day).

A subchronic feeding study in beagle dogs (MRID No. 44572201, Guideline 82–1), using 98% pymetrozine, exposed animals for 13 weeks (4/sex/dose) at dose levels of 0, 100, 500 or 2,500 ppm. These dose levels corresponded to 0, 3.12, 14, or 54 mg/kg/day for either sex. Mean relative liver weights were increased at all dose levels. At 500 ppm, both absolute (17% males and 18% females) and relative (19% males and 17% female) liver weights were increased. In addition, skeletal muscle myopathy (disease) in 1/4 males and 2/4 females, liver pathology (bile duct proliferation in both sexes and hepatocyte necrosis in females), and lymphohistocytic infiltration (several organs) increased. At 2,500 ppm, there was one death attributable to anemia. Decreases in red blood cell (RBC) parameters and increases in bilirubin were observed at this dose level as well, which are also indicative of anemia. Body weight was decreased in males (24%) and females (30%). Additional pathology was found in the thymus (atrophy and decrease in weight), heart (inflammation and decrease in weight), testis (decrease in spermatogenesis and weight) and uterus (atrophy). The LOAEL is 500 ppm (~14 mg/kg/day) based on liver effects, skeletal muscle atrophy, liver pathology and lymphohistocytic infiltration. The NOAEL is 100 ppm (~3.12 mg/kg/day). Slight liver weight changes at 100 ppm were not considered in the LOAEL.

A subchronic feeding study in the mouse (MRID No. 44024938, Guideline 82–1c), using 98% pymetrozine and designed to determine the dose levels for the definitive carcinogenicity study, exposed mice for 3 months at 0, 1,000, 3,000 or 7,000 ppm. Mean relative liver weights were increased in the low (10.5%), mid (26%) and high (57%) dose males and in the low (12%, not significant), mid (33%) and high (54%) dose females. The liver also showed increases in centrilobular hypertrophy of hepatocytes (swelling of liver cells) with a dose response of 0, 3, 7 and 10 in males and 0, 2, 5, and 10 in females for four dose levels. The liver also was indicated as having ''slight centrilobular perivascular-like aggregates of lymphocytes'' in all dose groups except the control and demonstrated a marked dose response with treatment. Necrosis of the liver was also increased in a dose related manner. Relative spleen weight was also increased at 3,000 ppm (21% in males and 19% in females) and 7,000 ppm (53% in males and 16% in females) and was accompanied by splenic extramedullary hematopoiesis above background. Thus, the liver and blood forming system were indicated as target organs for pymetrozine. Body weight at termination was decreased (17%) in males in the high dose group but was actually slightly increased (7%, not significant) in females.

A 28–day dermal toxicity study in the rat (MRID No. 44024942, Guideline No. 82–2), using 98% pymetrozine, exposed animals at 0, 10, 100 or 1,000 mg/kg/day for 6 hours/day, 5 days/week for 4 weeks. The agent was suspended in distilled water and was applied directly to clipped skin using an occlusive dressing. No treatment-related clinical signs or signs of local irritation were observed. Hematology and clinical biochemistry performed on the test animals revealed no treatment-related effects. Macroscopic and microscopic examination of internal organs and the application site revealed no treatment-related findings. The NOAEL for both systemic effects and dermal irritation is 1,000 mg/kg/day, the highest dose tested (HDT). The LOAEL is greater than 1,000 mg/kg/day.

ii. *Chronic toxicity*. A chronic feeding study in beagle dogs (MRID No. 44024943, Guideline 83–1), using 98% pymetrozine, exposed animals for 12 months at 0, 20, 200 and 1,000 ppm (corresponding to approximately 0, 0.57, 5.33 or 27.8 mg/kg/day in both sexes). At 200 ppm, there were increases in mean absolute (11%) and relative (17%) liver weights in males. At 1,000 ppm, mean absolute (6%) and relative (11%) liver weights were higher in both males and females (absolute (18%) and relative (6%)). In addition, in males, there was also increased inflammatory cell infiltration in the liver (4/6 vs 2/6 in the control group); and myopathy (2/6 vs 0/6 in the control group) in the small and large intestine. Anemia was apparent in two females. The LOAEL is 1,000 ppm (27.8 mg/kg/day), based primarily on myopathy (muscle disease) and presence of anemia (reduction in red blood cells). The NOAEL is 200 ppm (5.33 mg/kg/day). Similar findings in the dog subchronic study (MRID No. 44572201) regarding anemia and liver pathology support the conclusions of this study.

An 18–month definitive carcinogenicity study in mice (MRID No. 44024944, OPPTS No. 870.4200 or Guideline No. 83–2), using 98% pymetrozine, exposed animals (50/sex/dose group) for 18 months at 0, 10, 100, 2,000 or 5,000 ppm. These dose levels correspond to approximately 0, 1.2, 12, 250 and 675 mg/kg/day pymetrozine in either sex. At 2,000 ppm, relative liver weight increased in males (36%) and females (17%), with hepatocyte hypertrophy occurring in most affected animals. Hemosiderosis (increase in storage of insoluble form of iron) and extramedullary hematopoiesis (red blood cell formation) were also increased. Relative liver weight was increased by 78% in males and by 62% in females. The systemic LOAEL was 2,000 ppm (~250 mg/kg/day) based on increases in liver weight as well as hepatocyte hypertrophy and hemosiderosis. The NOAEL is 100 ppm (~12 mg/kg/day). Liver tumors were associated with the higher doses (2,000 and 5,000 ppm) of pymetrozine exposure with 5, 5, 5, 9 and 23 (males) and 0, 0, 0, 0 and 4 (females) hepatocellular carcinomas and 4, 5, 5, 1 and 14 hepatocellular ''benign adenoma'' in females for the control, 10, 100, 2,000 and 5,000 ppm dose groups, respectively. Males did not show increases in adenomas. The increases in liver weight and presence of hypertrophy and hematopoieses may imply that the high dose was excessive for meaningful carcinogenicity evaluation.

A combined chronic feeding/carcinogenicity study in the rat (MRID No. 44024951, Guideline No. 83–5, using 98% pymetrozine, exposed animals for 12 and 24 months. Five groups of 80/sex were dosed at 0, 10, 100, 1,000 or 3,000 ppm in the diet, corresponding to 0, 0.377, 3.76, 38.52 or 123.4 mg/kg/day for males and 0, 0.454, 4.48, 46.26 or 148.3 mg/kg/day for females. Ten/sex/group were sacrificed at 12 months. Fifty/sex/group were reserved for carcinogenicity assessment after dosing for a scheduled 24 months. For the control, 10, 100, 1,000 and 3,000 ppm dietary groups (based on 60/sex),

hepatocellular hypertrophy was present with the following total incidence: for males, 0, 1, 5, 22 and 37 and for females, 2, 1, 0, 12 and 40. At the 1 year interim sacrifice, the incidence in males was 0, 0, 4, 10 and 10 (out of 10/group). Thus indicating in males that the 100 ppm dose is an effect level for induction of hepatocellular hypertrophy. At 1,000 ppm, body weight and gain were reduced (i.e., at 4 weeks males 6% and females 12%, $p < 0.05$ less gain) and relative liver (26%, $p < 0.05$), spleen (24%, $p < 0.05$) and kidney (14%, not significant) weights were increased in males at week 53. At 3,000 ppm, the magnitude of the effects at 1,000 ppm was increased and, in addition, female liver, spleen, kidney, brain and ovary as well as male brain and testis relative weights increased. The uterus showed increased dilation. The systemic LOAEL is 100 ppm (3.76 mg/kg/day) based on hepatocellular hypertrophy in males. The NOAEL is 10 ppm (0.377 mg/kg/day). In females, the systemic LOAEL is 1,000 ppm (46.26 mg/kg/day) and the NOAEL is 100 ppm (4.48 mg/kg/day) based on hepatocellular hypertrophy and reduced body weight and body weight gain. This study was considered positive for induction of liver tumors (benign hepatoma) at 1,000 and 3,000 ppm in females. The presence of hepatocellular hypertrophy at 1,000 and 3,000 ppm and decreased body weight at 3,000 ppm may provide a basis for determining that the dose levels associated with liver tumors were excessive.

3. *Neurotoxicity*. An acute neurotoxicity study in the rat (MRID No. 44411317, Guideline 81–8) exposed animals in groups of 10/sex at dose levels of 0, 125, 500 or 2,000 mg/kg/day. The LOAEL is 125 mg/kg based on decreases in body temperature, function observation battery (FOB) changes, and decreased motor activity (in males) related to decreased activity. The NOAEL is < 125 mg/kg/day.

A 13–week subchronic neurotoxicity study in the rat (MRID No. 44411318, Guideline No. 82–7) exposed groups of 10 animals/sex at dose levels of 0, 500, 1,000 or 3,000 ppm. Systemic effects of treatment were evident at 3,000 ppm only and were limited to decreased body weight gain (10–18% in males and 7–10% in females). At this dose, indications of neurotoxicity were limited to stereotypy (repetition of senseless movements) in males (3/10 affected at week 4 and 1/10 affected at weeks 8 and 13). There were also indications of tiptoe gait or walking on toes in females at all intervals but only statistically significant at week 13. The LOAEL is 3,000 ppm (equivalent to a mean of 201 mg/kg/day for males and 224 mg/kg/day in females) based on decreased weight and stereotypy in males as well as tiptoe gait in females. The NOAEL is 1,000 ppm (equivalent to a mean of 68 mg/kg/day in males and 81 mg/kg/day for females).

4. *Developmental toxicity*. A developmental study in the rat (MRID No. 44024948, OPPTS No. 870.3700 or Guideline No. 83–3a), using 98% pymetrozine, exposed groups of 24 animals in a 0.5% w/w aqueous solution of sodium carboxymethylcellulose at either 0, 30, 100 or 300 mg/kg/day by oral gavage from gestation days 6 through 15, inclusive. Maternal systemic toxicity was seen as reduced body weights gains in the 100 and 300 mg/kg/day dose groups during the dosing period (gestation days 6–16), the dosing period plus post-dosing period (gestation days 6–21 for 300 mg/kg/day) and the corrected body weight gain for the dosing period plus post-dosing period (statistically significant for both 100 and 300 mg/kg/day). There was reduced food consumption in the same groups during the dosing period. The maternal toxicity NOAEL was 30 mg/kg/day and the maternal toxicity LOAEL was 100 mg/kg/day based on reduced body weight gains and food consumption. Developmental toxicity was observed as an increase in skeletal observations at 300 mg/kg/day including dumbbell-shaped thoracic vertebral centers, absent ossification of metatarsal #1, shortened rib #13, absent ossification of the proximal phalanx of anterior digit #5, absent ossification of the proximal phalanx of posterior digit #2, #3 and #4, and absent and poor ossification of the proximal phalanx of posterior digit #5. The developmental toxicity NOAEL was 100 mg/kg/day and the developmental toxicity LOAEL was 300 mg/kg/day based on increased incidence of skeletal anomalies.

A developmental study in the rabbit (MRID No. 44024949, OPPTS No. 870.3700 or Guideline No. 83–3b), using 98% pymetrozine, exposed groups of 20 animals in a 0.5% w/w aqueous solution of sodium carboxymethylcellulose at either 0, 10, 75 or 125 mg/kg/day by oral gavage from gestation days 7 through 19, inclusive. Maternal systemic toxicity was seen as reduced body weight gains in the 75 and 125 mg/kg/day dose groups. There was also reduced food consumption in the mid and high dose groups. There was reduced food efficiency noted in the mid and high dose groups during all periods except for predosing (gestation days 0–7). At 125 mg/kg/day, two dams died and one aborted the entire litter during the dosing period. (Note: these observations were also noted in the rangefinding study). The maternal toxicity NOAEL was 10 mg/kg/day and the maternal toxicity LOAEL was 75 mg/kg/day based on reduced body weight gains and food consumption/efficiency. Developmental toxicity was observed as an increase in additional 13th ribs in the 75 and 125 mg/kg/day dose groups and an increase in skeletal observations at 125 mg/kg/day seen as fused sternebrae #2 & 3, #3 & 4 and #4 & 5, additional caudal vertebral centers, poor ossification of metacarpal #1, poor ossification of the talus of the hind limb, and poor ossification of the anterior digit #5 medial phalanx. Also, there was reduced litter size, increased resorptions and increased post-implantation loss in the 125 mg/kg/day dose group. The developmental toxicity NOAEL was 10 mg/kg/day and the developmental toxicity LOAEL was 75 mg/kg/day based on increased incidence of skeletal anomalies.

5. *Reproductive toxicity*. A multigeneration reproduction study in the rat (MRID No. 44024950, OPPTS No. 870.3800 or Guideline No. 83–4), using 98% pymetrozine, exposed groups of 30 animals at 0, 20, 200 or 2,000 ppm in the diet for two successive generations. Parental systemic toxicity included minimal hepatocellular hypertrophy in 5/30 200 ppm F0 males, 27/30 2,000 ppm F0 males and 2/30 2,000 ppm F0 females, in addition to minimal to moderate hyperplasia of lymphatic follicles of splenic white pulp in 25/30 2,000 ppm F0 females. The F1 animals had minimal hepatocellular hypertrophy in 2/30 200 ppm males, 26/30 2,000 ppm males and 10/30 2,000 ppm females, in addition to minimal to moderate hypertrophy of the basophilic cells in the adenohypophysis in 17/30 2,000 ppm males, compared to 7/30, 8/30, 7/30 for the control, 20 ppm and 200 ppm groups, respectively. Further, there were increased absolute and relative spleen and liver weights in the F0 and F1 2000 ppm animals plus decreased absolute and relative thymus weights in the 2,000 ppm F1 animals. The investigators concluded that the liver was the target organ in both sexes in both generations; in addition, the spleen was the target organ in F0 females, whereas the pituitary gland was affected in F1 males. Systemic toxicity to the paternal animals included reduced body weights, reduced body weight gains, and reduced food consumption. Systemic toxicity to F1 groups, included reduced body weights, reduced body weight gains, and reduced food consumption. The parental (paternal/

maternal) systemic toxicity NOAEL was 20 ppm (1.4–1.7 mg/kg/day for males and 1.6–1.8 mg/kg/day for females) and the parental (paternal/maternal) systemic toxicity LOAEL = 200 ppm (13.9–17.0 mg/kg/day for males and 16.0–18.1 mg/kg/day for females) based on liver effects in the F0 and F1 males. The reproductive toxicity NOAEL is equal to or greater than 2,000 ppm (136.9–179.0 mg/kg/day for males and 151.6–186.5 mg/kg/day for females) and the reproductive toxicity LOAEL is greater than 2,000 ppm (136.9–179.0 mg/kg/day for males and 151.6–186.5 mg/kg/day for females), since no reproductive effects were noted at the highest dose tested. The offspring systemic/developmental toxicity NOAEL was 200 ppm (13.9–17.0 mg/kg/day for males and 16.0–18.1 mg/kg/day for females) and the offspring systemic/developmental toxicity LOAEL was 2,000 ppm (136.9–179.0 mg/kg/day for males and 151.6–186.5 mg/kg/day for females) based on decreased pup weight and delay in eye opening in both F1 and F2 litters.

6. *Mutagenicity*. A reverse gene mutation assay in bacteria (MRID No. 44024952, Guideline No. 84–2), using 98% pymetrozine, exposed cultures of *Salmonella typhimurium* histidine-deficient (*his*-) mutant strains TA98, TA100, TA1535 and TA1537, and the *Escherichia coli* tryptophan-deficient (*try*-) strain WP2 *u*vrA in triplicate to five concentrations ranging from 312.5 to 5,000 µg/plate, in the presence or absence of a mammalian metabolic activation system (S9 plus cofactors) derived from the microsomal fraction (S9) of livers from adult male RAI rats pretreated with Aroclor 1254. In neither the initial nor confirmatory trial were any increased incidences of *his*+ or *try*+ colonies found, compared to solvent control values, in contrast to the strongly positive responses in all mutagen-treated cultures. Therefore, in this *in vitro* test, pymetrozine is considered negative for reverse gene mutation in these strains of bacteria.

A mammalian cell forward gene mutation assay in cultures of Chinese hamster lung (V79) cells (MRID No. 44024954, Guideline No. 84–2), using 98% pymetrozine, exposed cultures in duplicate at four concentrations ranging from 5.21 to 333.3 µg/mL, for 21 hours in the absence of a mammalian metabolic activation system or for 5 hours followed by 16 hours in test article-free tissue culture medium in the presence of activation provided by the microsomal fraction (S9) of livers from adult male RAI rats pretreated with Aroclor 1254. Cultures were negative for the induction of forward gene mutation at the HGPRT locus in this test system.

A mammalian cell cytogenetics (chromosome aberrations) assay in Chinese hamster ovary (CHO) cells (MRID No. 44024953, Guideline No. 84–2), using 98% pymetrozine, exposed cultures at eight concentrations ranging from 2.58 to 330 µg/mL for 18 hours in the absence of mammalian metabolic activation or for 3 hours in the presence of S9 activation (S9 microsomal fraction of livers from adult male rats pretreated with Aroclor 1254, plus co-factors) followed by recovery in treatment-free medium for 15 hours. Cultures were not clastogenic; at none of the concentrations nor harvest times was the incidence of structural chromosome aberrations reported to exceed either the concurrent or historical control values.

A micronucleus test in mice (MRID No. 44024955, Guideline 84–2), using 98% pymetrozine, exposed groups of 8 animals/sex orally by gavage in two series of trials: (1) Three groups at a single maximum tolerated dose (MTD) of 4,000 mg/kg and (2) three groups at single doses of 1,000, 2,000 and 4,000. No statistically significant increases over controls were found in MPCE in any group at any sacrifice time. In addition, no effects of treatment were calculated in PCE/NCE ratios at any time or dose point. CPA-treated positive control animals responded with highly significant increased MPCE.

An unscheduled DNA synthesis assay in primary rat hepatocyte cells (MRID No. 44024956, Guideline No. 84–2), using 98% pymetrozine, exposed cultures in two trials in dimethylsulfoxide (DMSO) at six concentrations ranging from 2.78 to 300 µg/mL for 16–18 hours in the presence of tritiated thymidine. In this genotoxicity mutagenicity test, there was no evidence that the treatment induced unscheduled DNA synthesis, as determined by radioactive tracer procedures (nuclear silver grain counts).

7. *Absorption, distribution and metabolism*. A metabolism study in rats (MRID No. 44024957, Guideline 85–1), using radiolabeled pymetrozine, exposed animals orally or intravenously in groups of 5 animals/sex to evaluate absorption and excretion. Within the first 24 hours post-dosing, the urine from all orally-dosed groups contained from 52.0% to 73.5% of the administered radioactivity. The intravenous treated rats also had comparable 24-hour urine levels which were 63.6% and 68.3% of the administered dose in males and females, respectively. At study termination (7 days post-dosing), the recovered radioactivity in urine (56.3–80.3%), expired air (0.2–1.4%), tissues (0.3–3.8%), feces (15.4–38.9%), and cage washes (0.2–0.7%) accounted for a total recovery of 91–100.7% of the administered dose in all groups. The relatively high urinary level of unchanged test material suggests metabolic saturation at the high dose of 100 mg/kg.

A metabolism study in female rats (MRID No. 44517720, OPPTS No. 870.7485, Guideline No. 85–1), using radiolabled pymetrozine, exposed animals orally to a single low dose (0.5 mg/kg) or a high dose (100 mg/kg). Irrespective of the label site, the time to maximum blood concentrations (tmax) were attained at 1 hour (0.1 ppm for both labels) and at 8 hours (41 ppm for triazine and 52 ppm for pyridine) following low and high oral dosing, respectively. While the peak blood levels were dependent on the dose but independent of the labeling site, the pyridine label was more persistent than the triazine label. At all time points and irrespective of the dose or labeling site, tissue residue levels (ppm) were highest in the kidneys and liver. For the low/high doses, the peak kidney levels were 0.6/75 ppm (triazine) and 0.6/101 ppm (pyridine), while the peak liver levels were 0.4/59 ppm (triazine) and 0.5/176 ppm (pyridine). Of all tissues (with the exception of the GI tract), the skeletal muscle had the highest percent of the administered dose (both labels) accounting for 7 to 8% of the low dose at 1 hour and for 19 to 21% of the high dose at 8 hours. The calculated half life times ($t_{1/2}$) for the triazine residue depletion from all the tissues ranged from 2.9 to 4.8 hours (low dose) and from 1.9 to 3.5 hours (high dose) and for the pyridine radiolabel depletion, from 31.7 to 110.3 hours (low dose) and from 2.5 to 13.9 hours (high dose).

Absorption was lower at the high dose representing nearly 82% of the administered dose for both radiolabels. Irrespective of the labeling site, the biliary excretion was higher at the low dose than at the high dose. The total 48–hour excretion, including cage wash, was higher at both dose levels for the triazine label (low dose/ high dose: 103%/95%) than the pyridine label (low dose/high dose: 85%/81%). These results confirm other findings (above) that of the two moieties, pyridine is more persistent than triazine.

8. *Dermal absorption*. A dermal absorption study in male rats (MRID No. 44024958, Guideline No. 85–3), using 98.1–99.5% radiolabeled pymetrozine, exposed 24 male animals in 0.5% carboxy-methyl cellulose aqueous suspension at dose levels of 0.084, 0.503, or 4.69 mg/rat (0.0067, 0.0402, or

0.375 mg/cm^2). After blood collection, four rats/dose were killed for assessment of dermal absorption after 0.5, 1, 2, 4, 10, and 24 hours of exposure. Urine and feces were also collected at the time of killing. After 24 hours of exposure, dermal absorption of CGA–215944 was minimal (0.05%, 0.01%, and <0.005% for the low, mid, and high dose groups, respectively). For all dose groups, the majority of the dose (81.4–100.0%) was not absorbed and was recovered in the skin wash. For all dose groups, adsorption to skin from the test site (0.18–8.84%) accounted for the next largest proportion of the dose and only trace amounts (≤0.05%) of radioactivity were excreted in the urine and feces. Within each dose group, radioactivity remaining in/on the skin after washing did not seem to increase with the duration of exposure; likewise, absorption (measured as amount excreted plus amount retained in the body) did not seem to increase over time.

9. *Special studies.* A cell proliferation study in young adult male mice (MRID No. 44024923), using 97.4% pymetrozine, exposed 15 groups of animals in a basal diet as follows: (i) Two groups at dietary concentrations of 0 and 5,000 ppm for 4 days (corresponding to intakes of 0 and 891.6 mg/kg/day); (ii) six groups at concentrations of 0, 10, 100, 500, 2,000 and 5,000 ppm for 14 days (intakes of 0, 1.6, 15.6, 83.9, 323.4 and 876.7 mg/kg/day); (iii) six groups at concentrations of 0, 10, 100, 500, 2,000 and 5,000 ppm for 42 days (intakes of 0, 1.6, 13.3, 70.7, 299.9 and 767.1 mg/kg/day); and (iv) a single group at a concentration of 5,000 ppm for 14 days (intake of 1,006 mg/kg/day), followed by a recovery period of 28 days, in order to test for reversibility of any treatment-related changes. No clinical signs of toxicity were observed in any group throughout the treatment and/or recovery periods. Absolute and relative liver weights were slightly increased at 4–days treatment with 5,000 ppm, but significantly so after 14 and 42 days at this high concentration as well as 2,000 ppm, indicating hypertrophy. Absolute and relative liver weights returned to control levels in the 14–day treatment/28 day recovery animals. Significant decreases in the mean number of total nuclei were recorded at 2,000 ppm (≈16%) and at 5,000 ppm (≈17–18%) after 14 and 42 days. These findings, in conjunction with evidence that the enlarged hepatocytes at 5,000 ppm (14 and 42 days) often contained vacuoles, slight focal single cell necrosis and PCNA+ inflammatory cell infiltration that occurred at a higher frequency in the livers of mice at 5,000 ppm (14 and 42 days) than in the vehicle control liver samples, indicate that the test material induced a cytotoxic effect on the target organ. Immunohistochemical staining of liver sections revealed significant increases in PCNA values in both 2,000 and 5,000 ppm groups at all time points. Cell proliferation effects were reversible in animals treated at 5,000 ppm for 14 days followed by a 28–day recovery. Thus, these results show that the observed hepatomegaly in mouse liver at the 2,000 and 5,000 ppm treatment levels was the combined result of hypertrophy and hyperplasia. Accordingly, the LOAEL is 2,000 ppm, based on increased liver weight, reduced total hepatocytes, microscopic evidence of necrosis and significant increases in the LI for cell proliferation; the NOAEL is 500 ppm level. Overall, the findings of this study offer support for the hypothesis that the increased incidence of hepatocellular carcinomas in a previous 18–month carcinogenicity study in mice was due to (reversible) replicative DNA synthesis, with a threshold effect at a NOAEL = 500 ppm.

A special study in male rats (MRID No. 44517723), using 97.8% pymetrozine and conducted to evaluate possible mechanisms for liver tumor formulation, exposed 6 groups of 16 animals in diets containing 0, 25, 50, 100 or 1,000 ppm for 18 weeks. Assessments were limited to cage side observations for clinical signs, body weight and food plus water consumption. Pathology was limited to organ assessment of the liver and thyroid for weight and macroscopic and histopathological lesions but also included a special assessment for the immunohistological evaluation of the glutathione S-transferase placental from positive hepatocyte (GST-P) foci, a foci induced by the presence of the initiators. Pymetrozine produced its expected increase in liver and thyroid weight but did not increase the GST-P foci thus was not considered positive for a promotional effect of proliferative lesions in the liver. Pymetrozine was associated with an increase ($p < 0.05$) in follicular cell adenomas only in the 100 ppm dose group but there was no associated increase in thyroid hyperplasia or similar effect at 1,000 ppm. Overall, it could not be concluded that pymetrozine resulted in promotion of proliferative lesions in either the rat liver or thyroid at dose levels up to and including 1,000 ppm.

B. Toxicological Endpoints

1. *Acute dietary toxicity* — i. *Females 13 years and older.* The Agency selected a NOAEL of 10 mg/kg/day from the rabbit developmental study (MRID No. 44024949) for the acute dietary endpoint, based on reduced body weight gains and reduced food consumption and efficiency in mothers and an increased incidence of skeletal anomalies in pups at the LOAEL of 75 mg/kg/day. The selection of the rabbit developmental toxicity study is comparable to the rat developmental toxicity study, which had a maternal NOAEL and LOAEL of 30 and 100 mg/kg/day, respectively.

ii. *Acute dietary toxicity (General Population and Infants and Children).* The Agency selected the LOAEL of 125 mg/kg (lowest dose tested) from the acute rat neurotoxicity study (MRID No. 44411317) for the acute dietary endpoint for the general population, including infants and children, based on decreased body temperature, decreased motor activity, and FOB parameters associated with decreased activity.

2. *Short- and intermediate-term toxicity.* For dermal exposure, the Agency selected a NOAEL of 1,000 mg/kg/day from a 28–day dermal toxicity in the rat (MRID No. 44024942) because there were no effects at the highest dose tested. Based on these results, the Agency did not perform a short- or intermediate-term dermal risk assessments.

For short-term (1–7 days) inhalation exposure, the Agency selected (in the absence of an inhalation study) an oral NOAEL of 10 mg/kg/day from a developmental study in the rabbit (MRID No. 44024949), based on reduced body weight gains and food consumption and efficiency in mothers and an increased incidence of skeletal anomalies in pups at the LOAEL of 75 mg/kg/day.

For intermediate (7 days to several months) inhalation exposure, the Agency selected (in the absence of an inhalation study) an oral NOAEL of 10 ppm (0.377 mg/kg/day) from a chronic feeding study in the rat (MRID No. 44024951), based on hepatocellular (liver) hypertrophy in males at an LOAEL of 100 ppm (3.76 mg/kg/day).

3. *Chronic toxicity.* For chronic dietary exposure, EPA has selected an oral NOAEL of 10 ppm (0.377 mg/kg/day) from a chronic feeding study in the rat (MRID No. 44024951), based on hepatocellular (liver) hypertrophy in males at an LOAEL of 100 ppm (3.76 mg/kg/day).

4. *Carcinogenicity.* EPA has classified pymetrozine as a''likely human carcinogen'' and recommended that quantification of risk be estimated for combined (benign hepatomas and/or carcinomas) liver tumors in male and

female mice and female rats. EPA selected a unit risk, Q1*, of 2.05 x 10–1 (mg/kg/day)–1 for quantification of the cancer risk and has determined the cancer dose to be 0.0000049 mg/kg/day. The Agency reviewed ''mechanism of action'' studies, but these were insufficient to affect the classification of carcinogenicity.

5. *Dermal penetration*. The dermal penetration study (MRID No. 44024958) in rats indicated that the amount of pymetrozine capable of penetrating the skin is very small (no more than 0.28%). However, because the EPA concluded that the study may have underestimated the actual amount of dermal penetration, the Agency has used a dermal penetration value of 1% in risk assessments.

6. *Long-term (several months to lifetime) dermal and inhalation endpoints*. The current use pattern does not indicate a concern for long-term dermal or inhalation exposure potential.

7. *Safety (uncertainty) factors, including FQPA safety factor*. The Agency will use the above NOAELs and LOAELs levels to assess the risks of using pymetrozine to the general population and certain subgroups of the general population. However, the Agency first modifies these values numerically, downward, by dividing the NOAEL dose by one or more safety factors. These safety factors may represent the uncertainty of the individual variation among animals for all studies (10 fold safety or uncertainty factor), of using animal studies to assess human risk for all studies (10 fold safety factor); and of using a LOAEL in place of a NOAEL to estimate the risk (3 fold safety factor).

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children. As noted, EPA has added an additional three-fold factor to the acute dietary risk assessment for infants and children due to the lack of a NOAEL in the critical study. An additional 3-fold factor is also needed due to the uncertainty resulting from the data gap for the developmental neurotoxicity study in rats. This latter safety factor is applicable to the following subgroup populations: Females 13–50; infants, children (1–6 years), and children (7–12 years) for all risk assessment scenarios for acute and chronic dietary and residential scenarios. No greater additional factor is needed because:

- There was no evidence of developmental effects being produced in fetuses at lower doses as compared to maternal animals nor was there evidence of an increase in severity of effects at or below maternally toxic doses following *in utero* exposure in the prenatal developmental toxicity studies in rats and rabbits.
- In the prenatal/postnatal 2–generation reproduction study in rats, there was no evidence of enhanced susceptibility in pups when compared to parental animals (i.e., effects noted in offspring occurred at maternally toxic doses or higher).
- There was no evidence of abnormalities in the development of the fetal nervous system in the prenatal/postnatal studies submitted to the Agency.
- Adequate actual data, surrogate data, and/or modeling outputs are available to satisfactorily assess food exposure and to provide a screening level drinking water exposure assessment.

i. *Acute dietary toxicity (females 13 years and older)*. The Agency divided the NOAEL dose of 10 mg/kg/day from the rabbit developmental study (MRID No. 44024949) by 300 (10 for individual variation x 10 for species variation x 3 for lack of a developmental neurotoxicity study) to calculate an acute population-adjusted dose (aPAD) of 0.033 mg/kg for females 13 years or older.

ii. *Acute dietary toxicity* (general population and infants and children). The Agency divided the LOAEL dose of 125 mg/kg from the acute neurotoxicity study (MRID No. 44411317) by 300 (3 for lack of a NOAEL x 10 for individual variation x 10 for species variation) to calculate an aPAD of 0.42 mg/kg for the general population (300-fold FQPA safety factor) and by dividing by an additional 3-fold FQPA safety factor for lack of a developmental neurotoxicity study to calculate an aPAD of 0.14 mg/kg for infants and children (900-fold safety factor).

iii. *Chronic toxicity*. EPA divided the NOAEL dose of 0.377 mg/kg/day from a chronic feeding study in the rat (MRID No. 44024951) by 100 (10 for individual variation x 10 for species variation) to calculate a chronic population-adjusted dose (cPAD) of 0.0038 mg/kg/day for the general population by dividing by a additional 3-fold FQPA safety factor to calculate a cPAD of 0.0013 mg/kg/day for females 13 years and older and for infants and children.

C. Exposures and Risks

1. *Proposed uses*. Pymetrozine is a new insecticide of the pyridine azomethine type. Pymetrozine is proposed for the control of aphids and suppression of whiteflies in a variety of crops. The mode of action of pymetrozine has not been precisely determined biochemically; physiologically, it appears to act by preventing these insects from inserting their stylus into the plant tissue.

Pymetrozine is proposed for use on tuberous and corm vegetables (Subgroup 1-C) and tobacco under Fulfill™ and ornamental plants under Relay™. Currently, there are no requested homeowner applications for pymetrozine. However; post-application (residential) exposure could occur due to contact with treated ornamental plants. As both Fulfill™ and Relay™, pymetrozine is formulated as a water-dispersible granule containing 50% active ingredient.

Fulfill™ may be applied by either ground or aerial broadcast equipment, in a minimum of 10 gallons of water per acre; chemigation is not permitted. Pymetrozine is applied to the foliage of affected plants where it is quickly absorbed. Potato and tobacco crops may be treated up to twice, each at a maximum rate of 0.09 pound (lb) active ingredient per acre (ai/acre). The maximum seasonal use rate is 0.17 lb ai/acre. The retreatment and pre-harvest intervals are 7 and 14 days, respectively. The label for Fulfill™ specifies a restricted-entry interval of 12 hours.

Relay™ is to be broadcast-applied to ornamentals at a rate not to exceed 10 oz./acre/application. Multiple applications may be made on a 7– to 14–day interval. For indoor use, the yearly application rate is not to exceed 100 oz./acre/year; for outdoor use, the maximum rate is 48 oz./acre/year.

The above uses result in food and feed, drinking water, and non-dietary (residential) exposures as outlined below (2–4).

2. *From food and feed uses*. This rule establishes the first tolerance for pymetrozine.

Section 408(b)(2)(E) authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide chemicals that have been measured in food. If EPA relies on such information, EPA must require that data be provided 5 years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the levels anticipated. Following the initial data submission, EPA is authorized to require similar data on a time frame it deems appropriate. As required by section 408(b)(2)(E), EPA will issue a data call-in for information relating to anticipated residues to be submitted no

later than 5 years from the date of issuance of this tolerance.

Section 408(b)(2)(F) states that the Agency may use data on the actual percent of crop treated (PCT) for assessing chronic dietary risk only if the Agency can make the following findings: That the data used are reliable and provide a valid basis to show what percentage of the food derived from such crop is likely to contain such pesticide residue; that the exposure estimate does not underestimate exposure for any significant subpopulation group; and if data are available on pesticide use and food consumption in a particular area, the exposure estimate does not understate exposure for the population in such area. In addition, the Agency must provide for periodic evaluation of any estimates used. To provide for the periodic evaluation of the estimate of PCT as required by section 408(b)(2)(F), EPA may require registrants to submit data on PCT.

Most of the dietary risk assessments performed on pymetrozine used a Tier 1 approach for fruiting vegetables, cucurbits, and potatoes, crops originally requested in the petition. That is, the Agency assumed 100% crop treated and tolerance level residues. For carcinogenicity risk assessment, the Agency used a Tier 3 chronic dietary exposure analysis for only tuberous and corm vegetables. This was based on 20% of the crop treated and an anticipated residue of 0.0046 ppm to refine the cancer risk. Novartis supplied this estimate of PCT to the Agency. Based on the number of existing alternatives, the PCT could be much lower. However, the market is looking for rotational alternatives to prevent the buildup of resistance and to replace organophosphate (OP) insecticides threatened by FQPA. The Agency reviewed Novartis' estimate and found it reasonable.

The Agency believes that the three conditions, discussed in section 408 (b)(2)(F) in this unit concerning the Agency's responsibilities in assessing chronic dietary risk findings, have been met. EPA finds that the PCT information is reliable and has a valid basis. Before the petitioner can increase production of product for treatment of greater than 340,000 acres (20% of 1,700,000 total acres for the tuberous and corm subgroup), permission from the Agency must be obtained. The regional consumption information and consumption information for significant subpopulations is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Use of this consumption information in EPA's risk assessment process ensures that EPA's exposure estimate does not understate exposure for any significant subpopulation group and allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency. Other than the data available through national food consumption surveys, EPA does not have available information on the consumption of food in a particular area.

i. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1–day or single exposure.

The Tier 1 Dietary Exposure Evaluation Model (DEEM™) analysis indicates that acute dietary (food only) exposure to pymetrozine from all in the original petition (tuberous and corm, fruiting, and curcubits) will be below EPA's level of concern (100% of the aPAD) and will not occupy more than 7% (of the aPAD for any population subgroup, including those of infants and children. For the maximum-exposed subgroup, the 95th percentile of exposure (children ages 1–6 years) is predicted to be 3.3% of the aPAD. Due to pymetrozine's lower acute endpoint for females 13–50 years (0.033 mg/kg) versus that of other population subgroups (0.14 mg/kg), the percentage of the aPAD occupied for females 13–50 years (6.5%) is slightly higher than that estimated for children 1–6 years. For a Tier 1 analysis, EPA considers exposure at the 95th percentile of exposure. Even at the 99.9th percentile of exposure, the acute risk is well below EPA's level of concern.

ii. *Chronic exposure and risk*. The Tier 1 DEEM™ chronic analysis indicates that exposure to pymetrozine from tuberous and corm vegetables (Subgroup 1-C), cucurbits and fruiting vegetables will occupy less than 74% of the cPAD for children ages 1–6 (the most highly exposed population subgroup). Chronic dietary risk to all other subgroups is less than that of children ages 1–6. See Table 1 below.

Table 1. Chronic Dietary (Food Only) Tier 1 Exposure and Risk Estimates for Pymetrozine Use

Population Subgroup	cPAD, mg/kg/day[b]	Exposure, mg/kg/day	% cPAD[c]
U.S. Population (total)[a]	0.0038	0.000455	12
Hispanics	0.0038	0.000496	13
Children 1–6 yrs	0.0013	0.000958	74
Females 13–19 (not pregnant or nursing)	0.0013	000.480	37
Males 13–19 yrs	0.0038	0.000500	13

[a]Population subgroups shown include the U.S. general population and the maximally exposed subpopulation of adults, infants and children, and women of child-bearing age.
[b]cPAD values incorporate the different FQPA Safety Factors for the various population subgroups.
[c]% cPAD = Exposure (mg/kg/day) ÷ cPAD (mg/kg/day) 100.

iii. Cancer exposure and risk. The Agency used a Tier 3 DEEM™ analysis for cancer risk estimates to the U.S. population. Based on use of pymetrozine on tuberous and corm vegetables only, the food only cancer risk is 1.7 10^{-7}, which is below the Agency's level of concern.

3. *From drinking water*. Pymetrozine is not persistent, breaking down in the environment through a number of mechanisms and degradation pathways including hydrolysis and aqueous and soil photolysis. Laboratory studies indicate that pymetrozine is a ''low mobility'' to ''no mobility'' chemical with respect to leaching. The environmental fate profile and application rates suggest that there should not be any notable concerns in the areas of soil mobility and persistence for pymetrozine resulting from its agriculture use to control aphids and whiteflies. Based on the low application rate, the field dissipation data, and the minimal concentrations relative to the parent (<10%, total), pymetrozine degradates should not enter ground and surface water to any appreciable extent.

EPA used the Screening Concentration In GROund Water (SCI-GROW) model to predict the

Environmental Estimated Concentrations (EECs) for pymetrozine in ground water. SCI-GROW is a regression model based on actual ground water monitoring data. SCI-GROW appears to provide realistic estimates of pesticide concentrations in shallow, highly vulnerable ground water sites. Using the highest application rate of 0.187 lb ai/acre (hops), SCI-GROW estimates the concentration of pymetrozine in ground water to be 0.015 µg/L. As there is relatively little temporal variation in ground water, this estimate can be used for both acute and chronic exposure scenarios.

In addition, EPA used the Tier 2 GENeric Estimated Environmental Concentration (GENEEC) and Pesticide Root Zone Model-EXAMS (PRZM-EXAMS) model to obtain Estimated Environmental Concentrations (EECs) in surface water. The standard PRZM-EXAMS runoff modeling scenario is based on a 10 ha field draining into a 1 ha by 2 meter deep small water body. This scenario represents a watershed drainage area:water volume ratio of 5 m^2/m^3. Each PRZM modeling scenario represents a unique combination of climatic conditions (e.g., rainfall), crop specific management practices, soil specific properties, site specific hydrology, and pesticide specific application and dissipation processes. Each PRZM simulation is conducted for multiple years to provide a probabilistic exposure characterization for a single site. Based on 2 applications of pymetrozine on sweet potato, each at 0.176 lb ai/acre, PRZM- EXAMS estimates acute (peak) EEC of pymetrozine in surface water to be 1.85 µg/L and estimates the chronic (36–year mean) EEC of pymetrozine in surface water to be 0.222 µg/L.

The EEC's for surface water (1.85 µg/L and 0.222 µg/L) are higher than those for ground water (0.015 µg/L). Therefore, surface water EEC's will be used to: (1) Estimate actual concentrations of pymetrozine in water and (2) to compare those conentrations with the Drinking Water Levels of Comparison (DWLOCs) in µg/L. DWLOCs are acceptable concentrations of pymetrozine in drinking water as theoretical upper limits in light of total aggregate exposure to that pesticide from food, water, and residential uses. EPA calculates each DWLOC by subtracting the food and residential exposures (if appropriate) from the PAD or Cancer Dose and by converting this resulting dose, called the Maximum Water Exposure (in mg/kg/day), into a concentration of pymetrozine in water expressed in µg/L. Only pymetrozine was included in the drinking water assessment on the basis that the metabolites would not be found in drinking water.

Table 2 shows the DWLOC's for acute and chronic exposure.

Table 2. Drinking Water Levels of Comparison for Aggregated Exposures

Scenario/Population Subgroup[a]	Population-Adjusted Dose, mg/kg/day	Exposure mg/kg/day[b]	Maximum Water Exposure mg/kg/day	DWLOC µg/L[c]
ACUTE EXPOSURE				[EEC=1.9]
U.S. Population	0.42	0.001980	0.418020	15000
Hispanic	0.42	0.002285	0.417715	15000
Children (1–6 yrs)	0.14	0.004556	0.135444	1400
Females (13–19, not pregnant or nursing)	0.033	0.0021 39	0.030861	930
Males (13–19 yrs)	0.42	0.002052	0.417948	15000
SHORT-TERM[d] EXPOSURE				
Toddlers	0.033	0.00097	0.032030	320
CHRONIC EXPOSURE				[EEC=0.22]
U.S. Population	0.0038	0.000455	0.003345	120
Hispanic	0.00380	0.000496	0.003304	120
Children (1–6 yrs)	0.0013	0.000958	0.000342	3.4
Females (13–19, not pregnant or nursing)	0.0013	0.000480	0.000820	25
Males (13–19)	0.0038	0.000500	0.003300	120

[a]Population subgroups shown include the U.S. general population and the maximally exposed subpopulation of adults, infants and children, and women of child-bearing age for each exposure scenario.

[b]Exposure is the sum of dietary and non-dietary exposure. For the case of pymetrozine, only the short-term and cancer DWLOC have a non-dietary component. See Section 5.4 for clarification.

[c]DWLOC = Maximum Water Exposure (mg/kg/day) 1,000 µg/mg body weight (70 kg general population/males 13+, 60 kg females 13+, 10 kg infants and children) ÷ Water Consumption (2 L/day adults, 1 L/day infants and children). The acute EEC is 1.9 µg/L, the chronic and cancer EEC is 0.22 µg/L.

[d]For short-term exposure, the short-term oral NOAEL was converted to a PAD by applying the 100x and 3x safety factors. Chronic food exposure for children ages 1–6 was used to estimate background food exposure.

i. *Acute exposure and risk.* For acute aggregate exposure scenarios, the DWLOC values (930–15,000 µg/L) are all in excess of the modeled acute EEC values (1.9 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure.

ii. *Chronic exposure and risk.* For chronic (non-cancer) aggregate exposure scenarios, the DWLOC values (3.4–120 µg/L) are all in excess of the modeled EEC values (0.22 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure.

iii. *Cancer exposure and risk.* Preliminary analysis suggested that drinking water may be a significant contributor towards cancer risk. Therefore, the Agency did an aggregate quantitative risk assessment which is discussed in section D3 of this unit.

4. *From non-dietary exposure.* As currently proposed, pymetrozine could be used on the following residential non-food sites: ornamentals (landscape, ground-covers, interiorscapes); home nurseries, non-bearing orchards, and greenhouses. The end-use product, Relay™, may not be applied by homeowners, but post-application exposure could occur. There are no intermediate-term exposure scenarios for which a risk assessment is required. Short-term exposures are not applicable for adults but are applicable for toddlers.

Since there was no chemical-specific data to determine dislodgeable residues, EPA used its Standard Operating Procedures (SOPs) for Residential

Exposure Assessment (Draft, December 18, 1997) to estimate post-application exposure. This SOP does not include a scenario for ornamentals, landscapes and groundcover. Therefore, this assessment used the garden plants scenarios to determine post-application exposures.

The post-application scenarios and associated Margins of Exposure (MOEs) included: (1) Incidental non-dietary hand-to-mouth transfer of pesticide residues (770,000) (2) incidental non-dietary ingestion of pesticide-treated plants (not significant), and (3) incidental non-dietary ingestion of soil from pesticide-treated areas (660,000). The following assumptions were used for estimating post-application for the three post-application scenarios.

Hand-to-mouth transfer (incidental non-dietary ingestion)

-Maximum application rate of 0.3125 lbs ai/A as specified on the label

-20% of the application rate are available on the foliage as dislodgeable residue

-Exposure is assessed on the same day the pesticide is applied

-Medium surface area of both hands is 350 cm^2 for a toddler (age 3 yrs)

-Mean rate of hand-to-mouth activity is 1.56 events/hr

-Duration of exposure was assumed to be 0.18 hrs/day (10 mins) for toddlers

-A body weight of 15 kg was assumed for toddlers

-Short term NOAEL = 10 mg/kg/day (acute dietary)

-Hand-to-mouth exposure is not considered an intermediate-term exposure scenario

Accidental Ingestion of Plant Material

-According to the HED SOP for Residential Exposure, exposure via this route is considered negligible

Accidential Ingestion of Soil

-Maximum application rate of 0.3125 lbs ai per acre as specified on the label

-20% of the application rate are available on the foliage as dislodgeable residue

-Exposure is assessed on the same day the pesticide is applied

-The fraction of ai available in uppermost cm of soil is 1cm

-The assumed soil ingestion rate for children (ages 1–6 yrs) is 100 mg/day

-A body weight of 15 kg was assumed for toddlers

-Short term NOAEL = 10 mg/kg/day (acute dietary);

-Exposure from soil ingestion is not considered an intermediate-term exposure scenario

These exposure estimates are based on upper-percentile (i.e., maximum application rate, available residues and duration of exposure) and some central tendency (i.e., transfer coefficient, surface area, hand-to-mouth activity, and body weight) assumptions and are considered to be representative of high-end exposures. The uncertainties associated with this assessment stem from the use of an assumed amount of pesticide available from gardens, and assumptions regarding dissipation, transfer of chemical residues, and hand-to-mouth activity. The estimated exposures are believed to be reasonable high-end estimates based on observations from chemical-specific field studies and professional judgement.

EPA determined that the FQPA Safety Factor to protect infants and children should be reduced to 3x and that the factor should apply to female (13–50 years), infant, and children population subgroups for all risk assessments. Thus, the levels of concern for these post-application exposure scenarios are MOEs that are less than 100 for adult populations and less than 300 for female (13–50), infant, and children populations.

i. *Chronic exposure and risk*. Based on the proposed uses of pymetrozine, EPA does not believe there will be chronic non-occupational exposure to this insecticide.

ii. *Cancer exposure and risk*. EPA has estimated the lifetime average daily dose for non-occupational exposure resulting from prining and planting treated ornamental plants is 0.0000012 mg/kg/day.

A quantitative cancer risk assessment was performed for post-application non-occupational exposure to treated ornamentals (e.g., a home garden). Exposures were estimated using EPA's default activity scenarios, transfer coefficients and input parameters as follows:

- The fraction of active ingredient retained on foliage is assumed to be 20% (0.2) on day zero (= percent dislodgeable foliar residue, DFR, after initial treatment). This fraction is assumed to further dissipate at the rate of 10% (0.1) per day on following days. These are EPA's default values for exposure.
- An application rate of 0.3125 lbs ai/acre (electrostatic spray, pulsfog and low volume systems) was used to represent the worst case scenario.
- Transfer coefficient of 4,500 was used to represent heaviest day of activity (planting, transplanting, and pruning) for contact with treated ornamental plants.
- Assumed homeowner worked 0.67 hours per day (Residential SOP for Gardening).
- Assumed homeowner worked a total of 2 days per year performing heaviest activities (planting, pruning) at time points shortly after pymetrozine application.
- Assumed homeowner would be exposed for 50 years of their life.
- Dermal absorption = 1%.
- Body weight = 70 kg.
- Life expectance = 70 years.
- Cancer Q* (mg/kg/day) = 2.05×10^{-1}.

The cancer risk estimate for this post-application exposure is 2.4×10^{-7} and does not exceed EPA's level of concern (in the range of 1×10^{-6}) for the general population.

iii. *Short- and intermediate-term exposure and risk*. EPA did not calculate MOEs for adults since there are no short-term dermal exposure scenarios. However, short-term oral exposures and risks were calculated for toddlers. For toddlers, the MOEs for short-term post-application exposure scenarios are 770,000 and 660,000 for hand-to-mouth and soil ingestion scenarios. These values are all greater than either of the threshold values; thus, short-term risks are below the Agency's level of concern.

4. *Cumulative exposure to substances with a common mechanism of toxicity*. Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.''

According to our information, there are no other pesticides that have a common mechanism of toxicity with pymetrozine. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, pymetrozine does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that pymetrozine has a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the final rule for Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997).

D. Aggregate Risks and Determination of Safety for U.S. Population

1. *Acute risk*. The risk from aggregate acute exposure from food and drinking water from pymetrozine is below EPA's level of concern for the following reasons. As indicated in Table 2, the Tier 1 DEEM™ analysis indicates that acute dietary (food only) exposure to pymetrozine from fruiting vegetables, cucurbits, and tuberous and corm

vegetables (Subgroup 1-C) will occupy less than 1/2% (0.001980/0.42) of the aPAD for the U.S. Population, which is below EPA's level of concern of 100% of the aPAD. In addition, for drinking water, the DWLOC value (15000 µg/L) for the U.S. Population is greatly in excess of the modeled acute EEC value (1.9 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure.

2. *Chronic risk*. As indicated in Table 1, the Tier 1 DEEM™ analysis indicates that chronic dietary (food only) exposure to pymetrozine will utilize less than 12% (0.000455/0.0038) of the cPAD for the U.S. population. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. In addition, for drinking water, the DWLOC value (120 µg/L) for the U.S. Population is greatly in excess of the modeled EEC values (0.222 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure. Despite the potential for exposure in the diet, drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate chronic exposure to exceed 100% of the cPAD.

3. *Aggregate cancer risk for U.S. population*. For tuberous and corm vegetables (Subgroup 1-C), EPA based its cancer risk assessment on a Tier 3 estimate of dietary exposure, which incorporates anticipated residues for pymetrozine and an estimate that 20% of the crops will be treated. At this level of refinement, EPA's estimate of food exposure and cancer risk were 0.0000008 mg/kg/day and 1.7 $x10^{-7}$. EPA also calculated a lifetime average daily dose of 0.0000012 mg/kg/day for non-occupational exposure resulting from pruning and planting treated ornamental plants.

EPA does not generally use surface water modeling values for quantitative risk assessment. However, due to the statistical uncertainties regarding the significance of cancer risks, which are near 1 x 10^{-6}, EPA has calculated the cancer risk resulting from 0.22 µg/L in drinking water to be 1.3 x 10^{-6}. The aggregate cancer risk is thus 1.7 x 10^{-6} (1.7 x 10^{-7} for food, 1.3 x 10^{-6}, for water, and 2.4 x 10^{-7} for post-application residential exposure).

4. *Determination of safety*. EPA believes that the total risk estimate for pymetrozine from food, drinking water, and residential exposures of 1.7 x 10^{-6} generally represents a negligible risk, as EPA has traditionally applied that concept. EPA has commonly referred to a negligible risk as one that is in the range of 1 in 1 million (1 x 10^{-6}). Quantitative cancer risk assessment is not a precise science. There are a significant number of uncertainties in both the toxicology used to derive the cancer potency of a substance and in the data used to measure and calculate exposure. The Agency does not attach great significance to numerical estimates for carcinogenic risk that differ by less than a factor of 2. However, as a condition of product registration, the Agency will require the registrant to submit monitoring data. These data are expected to confirm that the actual concentration of pymetrozine in drinking water is less than the level of concern for all sub-populations and endpoints.

E. Aggregate Risks and Determination of Safety for Infants and Children

1. *Safety factor for infants and children* —i. *In general*. In assessing the potential for additional sensitivity of infants and children to residues of pymetrozine, EPA considered data from developmental toxicity studies in rabbit, an acute neurotoxicity study in the rat, and a chronic feeding study in the rat. See the Toxicological Profile (section A. of this unit) for a discussion of these tests.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined interspecies and intraspecies variability) and the additional 3-fold MOE/uncertainty factors, as described above, when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of these safety factors.

ii. *Conclusion*. EPA considered the available data and determined that the 10-fold FQPA factor could be reduced to 3. A discussion of these considerations may be found in B7 of this unit.

2. *Acute risk*. The risk from aggregate acute exposure from food and drinking water from pymetrozine is below EPA level of concern for the following reasons. The Tier 1 DEEM™ analysis indicates that acute dietary (food only) exposure to pymetrozine from tuberous and corm vegetables (Subgroup 1-C), fruiting vegetables and curcubits will occupy less than 4% (0.004556/0.14) of the aPAD for children (1 to 6 years old), which is below EPA's level of concern of 100% of the aPAD. In addition, for drinking water, the DWLOC value (1,400 µg/L) for children (1 to 6 years old) is greatly in excess of the modeled acute EEC values (1.9 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure.

3. *Chronic risk*. Using the residue concentration exposure assumptions described in this unit, the risk from aggregate chronic exposure from food and drinking water from pymetrozine is below EPA's level of concern for the following reasons. As indicated in the previous table, the Tier 1 DEEM™ analysis indicates that chronic dietary (food only) exposure to pymetrozine will utilize less than 74% (0.000958/0.0013) of the cPAD for children (1 to 6 years old). EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. In addition, for drinking water, the DWLOC value (3.4 µg/L) for children (1 to 6 years old) exceeds the modeled chronic EEC values (0.222 µg/L); thus, drinking water is not expected to be a significant contributor towards this type of exposure. Despite the potential for exposure in the diet, drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate chronic exposure to exceed 100% of the cPAD.

4. *Short-term risk*. In aggregating short-term risk, EPA considered background average dietary exposure and short-term, non-dietary oral exposure. Non-dietary oral exposure may occur as hand-to-mouth transfer of residues from ornamental plants or incidental ingestion of surrounding soil. The lowest short-term MOE value is for toddlers. Combining this MOE (660,000) with that from dietary exposure (Short-term oral NOAEL/chronic dietary exposure = 10/0.00096 ≈ 10,000) results in an aggregate MOE of ≈ 10,000. As this value is greater than 300, the short-term aggregate risk is below the Agency's level of concern. Aggregated short-term exposure results in a DWLOC of 320 µg/L. This value is in excess of the peak EEC for pymetrozine (1.9 µg/L; see Table 2).

5. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty of no harm to infants and children from aggregate exposure to pymetrozine residues.

IV. Other Considerations

A. Metabolism in Plants and Animals

Data concerning the metabolism of pymetrozine in plants and animals have been previously submitted. The nature of residues in plants and animals is adequately understood. The tolerance expression is for pymetrozine *per se*. The residues of concern for risk assessment are pymetrozine; the plant metabolites GS–23199 [6-methyl-1,2,4-triazin-3,5 (2*H*,4*H*)-dione], CGA-215525 [4-amino-4,5-dihydro-6-methyl-1,2,4-triazin-3(2*H*)-one], CGA-249257 [4,5-dihydro-6-methyl-1,2,4-triazin-3(2*H*)-one], CGA-294849 [4-amino-6-methyl-1,2,4-triazin- 3,5(2*H*,4*H*)-dione]; and the ruminant metabolite CGA-313124 [4,5-dihydro-6-hydroxymethyl-4-[(3-pyndynyl methylene)amino]-1,2,4-triazin-3(2*H*)-one] (free acid conjugated).

B. Analytical Enforcement Methodology

Adequate enforcement methodology for pymetrozine (Novartis Analytical Method AG–643) is currently being validated. Following validation, it will be available to enforce the tolerance expression. At that time the method may be requested from: Calvin Furlow, PIRIB, IRSD (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 305–5229; e-mail address: furlow.calvin@epa.gov..

C. Magnitude of Residues

The crop field trial data support the proposed tolerances for residues of ''pymetrozine, *per se*.''

D. International Residue Limits

The are no established European (CODEX), Canadian, or Mexican Maximum Residue Limits (MRLs) for pymetrozine. There are provisional MRLs in Germany for hops (10 ppm) and potatoes (0.02 ppm). The European Union is currently evaluating a proposed tolerance of 5 ppm on hops. At this time, international harmonization of residue levels is not an issue.

E. Rotational Crop Restrictions

The label has been revised to include only the following sites: Tuberous and corm vegetables (Subgroup 1-C) and tobacco. The label also includes a plant back restriction of not less than 120 days for all leafy and root crops, and not less than 365 days for all other crops.

F. Pre-harvest Intervals

The pre-harvest interval for pymetrozine on the tuberous and corm vegetables (Subgroup 1-C) is 14 days.

V. Conclusion

Therefore, the tolerance is established for residues of pymetrozine *per se* in tuberous and corm vegetables (Subgroup 1-C), at 0.02 ppm.

VI. Objections and Hearing Requests

Under section 408(g) of the FFDCA, as amended by the FQPA, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. EPA procedural regulations which govern the submission of objections and requests for hearings appear in 40 CFR part 178. Although the procedures in those regulations require some modification to reflect the amendments made to the FFDCA by the FQPA of 1996, EPA will continue to use those procedures, with appropriate adjustments, until the necessary modifications can be made. The new section 408(g) provides essentially the same process for persons to ''object'' to a regulation for an exemption from the requirement of a tolerance issued by EPA under new section 408(d), as was provided in the old FFDCA sections 408 and 409. However, the period for filing objections is now 60 days, rather than 30 days.

A. What Do I Need to Do to File an Objection or Request a Hearing?

You must file your objection or request a hearing on this regulation in accordance with the instructions provided in this unit and in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket control number OPP–300929 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk on or before November 29, 1999.

1. *Filing the request*. Your objection must specify the specific provisions in the regulation that you object to, and the grounds for the objections (40 CFR 178.25). If a hearing is requested, the objections must include a statement of the factual issues(s) on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the objector (40 CFR 178.27). Information submitted in connection with an objection or hearing request may be claimed confidential 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. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

Mail your written request to: Office of the Hearing Clerk (1900), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. You may also deliver your request to the Office of the Hearing Clerk in Rm. M3708, Waterside Mall, 401 M St., SW., Washington, DC 20460. The Office of the Hearing Clerk is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Office of the Hearing Clerk is (202) 260–4865.

2. *Tolerance fee payment*. If you file an objection or request a hearing, you must also pay the fee prescribed by 40 CFR 180.33(i) or request a waiver of that fee pursuant to 40 CFR 180.33(m). You must mail the fee to: EPA Headquarters Accounting Operations Branch, Office of Pesticide Programs, P.O. Box 360277M, Pittsburgh, PA 15251. Please identify the fee submission by labeling it ''Tolerance Petition Fees.''

EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding the waiver of these fees, you may contact James Tompkins by phone at (703) 305–5697, by e-mail at tompkins.jim@epa.gov, or by mailing a request for information to Mr. Tompkins at Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

If you would like to request a waiver of the tolerance objection fees, you must mail your request for such a waiver to: James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

3. *Copies for the Docket*. In addition to filing an objection or hearing request with the Hearing Clerk as described in Unit VI.A. of this preamble, you should also send a copy of your request to the PIRIB for its inclusion in the official record that is described in Unit I.B.2. of this preamble. Mail your copies, identified by docket number OPP–300929, to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In

person or by courier, bring a copy to the location of the PIRIB described in Unit I.B.2. of this preamble. You may also send an electronic copy of your request via e-mail to: *opp-docket@epa.gov*. Please use an ASCII file format and avoid the use of special characters and any form of encryption. Copies of electronic objections and hearing requests will also be accepted on disks in WordPerfect 6.1/8.0 file format or ASCII file format. Do not include any CBI in your electronic copy. You may also submit an electronic copy of your request at many Federal Depository Libraries.

B. When Will the Agency Grant a Request for a Hearing?

A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is a genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues(s) in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32).

VII. Regulatory Assessment Requirements

This final rule establishes a tolerance under section 408(d) of the FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4). Nor does it require prior consultation with State, local, and tribal government officials as specified by Executive Order 12875, entitled *Enhancing the Intergovernmental Partnership* (58 FR 58093, October 28, 1993) and Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19,1998), or special consideration of environmental justice related issues under Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994) or require OMB review in accordance with Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). The Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 12612, entitled *Federalism* (52 FR 41685, October 30, 1987). This action directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(b)(4). This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). In addition, since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply.

VIII. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this rule in the **Federal Register**. This rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: September 23, 1999.

Susan B. Hazen,

Acting Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180—[AMENDED]

1. The authority citation for part 180 continues to read as follows:

Authority: 21 U.S.C. 321(q), (346a) and 371.

2. Section 180.556 is added to read as follows:

§ 180.556 Pymetrozine; tolerances for residues.

(a) *General.* Tolerances are established for residues of the insecticide pymetrozine [1,2,4-triazin-3(2H)-one,4,5-dihydro-6-methyl-4-[(3-pyridinylmethylene) amino]] in or on the following raw agricultural commodities. The tolerance level for each commodity is expressed in terms of the parent insecticide only, which serves as an indicator or the use of pymetrozine on these raw agricultural commodities.

Commodity	Parts per million	Expiration/Revocation Date
Corm and Tuberous Vegetables Subgroup 1-C.	0.02	None

(b) *Section 18 emergency exemptions.* [Reserved]

(c) *Tolerances with regional registrations.* [Reserved]

(d) *Indirect or inadvertent residues.* [Reserved]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300921; FRL–6382–1]

RIN 2070–AB78

Diflubenzuron; Pesticide Tolerances for Emergency Exemptions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a time-limited tolerance for residues of diflubenzuron (*N*-[[4-