or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950), and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

IV. Submission to Congress and the General Accounting Office

Under 5 U.S.C. 801(a)(1)(A), as added by the Small Business Regulatory Enforcement Fairness Act of 1996, EPA submitted 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 General Accounting Office prior to publication of this rule in today's **Federal Register**. This 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: June 30, 1998.

Peter Caulkins,

Acting Director, Registration Division, 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. 346a and 371.

§ 180.443 [Amended]

2. In § 180.443, by amending paragraph (b) in the table, for the commodities ''Peppermint'' and ''Spearmint'' by changing the date ''July 1, 1998'' to read ''1/31/00''.

[FR Doc. 98–18988 Filed 7–16–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300612; FRL–5768–3]

RIN 2070–AB78

Fipronil; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes new tolerances for combined residues of fipronil, its metabolites MB46136 and MB45950, and its photodegradate MB46513, in or on rice grain and rice straw. In pesticide petition (PP) 7F4832, Rhone Poulenc AG, Inc. requested these tolerances under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1966 (FQPA).

DATES: This regulation is effective July 17, 1998. Objections and requests for hearings must be received by EPA on or before September 15, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, OPP–300612, must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing requests shall be labeled ''Tolerance Petition Fees'' and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees), P.O. Box 360277M, Pittsburgh, PA 15251. A copy of any objections and hearing requests filed with the Hearing Clerk identified by the docket control number, OPP–300612, must also be submitted 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, bring a copy of objections and hearing requests to Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 file format or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number OPP–300612. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: Ann Sibold, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–6788, e-mail: sibold.ann@epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of June 20, 1997 (62 FR 33641) (FRL–5723–7), EPA issued a notice pursuant to section 408 of the FFDCA, 21 U.S.C. 346a(e), announcing the filing of a pesticide petition for a tolerance (PP 7F4832) by Rhone Poulenc AG, Inc., P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709. This notice included a summary of the petition prepared by Rhone Poulenc AG, Inc., the registrant. There were 11 comments received in response to the notice of filing and all supported establishing the tolerance.

The petition proposed to use a 56% flowable solid (FS) formulation (Product name: ICON 6.2 FS Insecticide) to treat rice seed to control the pests rice water weevil and chinch bugs.

The petition further requested that 40 CFR 180.517 be amended by establishing new tolerances for combined residues of the insecticide fipronil, its metabolites MB46136 and MB45950, and its photodegradate MB46513 in or on rice grain at 0.04 parts per million (ppm) and rice straw at 0.10 ppm. Tolerances for residues of fipronil (expressed as fipronil and its metabolites MB45950 and MB46136) in or on animal commodities have recently been established (40 CFR 180.517(a)).

Fipronil is registered in the United States for use on field corn, on golf course and commercial turf, on pets, and in roach and ant bait stations.

I. Risk Assessment and Statutory Findings

New 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) of the FFDCA 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. First, EPA determines the toxicity of pesticides based primarily on toxicological studies using laboratory animals. These studies address many adverse health effects, including (but not limited to) reproductive effects, developmental toxicity, toxicity to the nervous system, and carcinogenicity. Second, EPA examines exposure to the pesticide through the diet (e.g., food and drinking water) and through exposures that occur as a result of pesticide use in residential settings.

A. Toxicity

1. *Threshold and non-threshold effects.* For many animal studies, a dose response relationship can be determined, which provides a dose that causes adverse effects (threshold effects) and doses causing no-observed effects (the ''no-observed effect level'' or ''NOEL'').

Once a study has been evaluated and the observed effects have been determined to be threshold effects, EPA generally divides the NOEL from the study with the lowest NOEL by an uncertainty factor (usually 100 or more) to determine the Reference Dose (RfD). The RfD is a level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. An uncertainty factor (sometimes called a ''safety factor'') of 100 is commonly used since it is assumed that people may be up to 10 times more sensitive to pesticides than the test animals, and that one person or subgroup of the population (such as infants and children) could be up to 10 times more sensitive to a pesticide than another. In addition, EPA assesses the potential risks to infants and children based on the weight of the evidence of the toxicology studies and determines whether an additional uncertainty factor is warranted. Thus, an aggregate daily exposure to a pesticide residue at or below the RfD (expressed as 100% or less of the RfD) is generally considered acceptable by EPA. EPA generally uses the RfD to evaluate the chronic risks posed by pesticide exposure. For shorter-term risks, EPA uses a RfD approach or calculates a margin of exposure (MOE) by dividing the estimated human exposure into the NOEL from the appropriate animal study. Commonly, EPA finds MOEs lower than 100 to be unacceptable. This 100-fold MOE is based on the same rationale as the 100-fold uncertainty factor.

Lifetime feeding studies in two species of laboratory animals are conducted to screen pesticides for cancer effects. When evidence of increased cancer is noted in these studies, the Agency conducts a weight of the evidence review of all relevant toxicological data including short-term and mutagenicity studies and structure activity relationship. Once a pesticide has been classified as a potential-human carcinogen, different types of risk assessments (e.g., linear low-dose extrapolations or MOE calculation based on the appropriate NOEL) will be carried out based on the nature of the carcinogenic response and the Agency's knowledge of its mode of action.

2. *Differences in toxic effect due to exposure duration.* The toxicological effects of a pesticide can vary with different exposure durations. EPA considers the entire toxicity data base, and based on the effects seen for different durations and routes of exposure, determines which risk assessments should be done to assure that the public is adequately protected from any pesticide-exposure scenario. Both short and long durations of exposure are always considered. Typically, risk assessments include ''acute,'' ''short-term,'' ''intermediate-term,'' and ''chronic'' risks. These assessments are defined by the Agency as follows.

Acute risk, by the Agency's definition, results from 1-day consumption of food and water, and reflects toxicity which could be expressed following a single-oral exposure to the pesticide residues. High-end exposure to food and water residues are typically assumed.

Short-term risk results from exposure to the pesticide for a period of 1-7 days, and therefore overlaps with the acute risk assessment. Historically, this risk assessment was intended to address primarily dermal and inhalation exposure which could result, for example, from residential pesticide applications. However, since enaction of FQPA, this assessment has been expanded to include both dietary and non-dietary sources of exposure, and will typically consider exposure from food, water, and residential uses when reliable data are available. In this assessment, risks from average food and water exposure, and high-end residential exposure, are aggregated. High-end exposures from all three sources are not typically added because of the very-low probability of this occurring in most cases, and because the other conservative assumptions built into the assessment assure adequate protection of public health. However, for cases in which high-end exposure can reasonably be expected from multiple sources (e.g. frequent and widespread homeowner use in a specific geographical area), multiple high-end risks will be aggregated and presented as part of the comprehensive risk assessment/characterization. Since the toxicological endpoint considered in this assessment reflects exposure over a period of at least 7 days, an additional degree of conservatism is built into the assessment; i.e., the risk assessment nominally covers 1-7 days exposure, and the toxicological endpoint/NOEL is selected to be adequate for at least 7 days of exposure. (Toxicity results at lower levels when the dosing duration is increased.)

Intermediate-term risk results from exposure for 7 days to several months. This assessment is handled in a manner similar to the short-term risk assessment.

Chronic risk assessment describes risk which could result from several months to a lifetime of exposure. For this assessment, risks are aggregated considering average exposure from all sources for representative population subgroups including infants and children.

B. Aggregate Exposure.

In examining aggregate exposure, section 408 of the FFDCA requires that EPA take into account available and reliable information concerning exposure from the pesticide residue in the food in question, residues in other foods for which there are tolerances, residues in groundwater or surface water that is consumed as drinking water, and other non-occupational exposures through pesticide use in gardens, lawns, or buildings (residential and other indoor uses). Dietary exposure to residues of a pesticide in a food commodity are estimated by multiplying the average daily consumption of the food forms of that commodity by the tolerance level or the anticipated pesticide residue level. The Theoretical Maximum Residue Contribution (TMRC) is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. In evaluating food exposures, EPA takes into account varying consumption patterns of major identifiable subgroups of consumers, including infants and children. The TMRC is a ''worst case'' estimate since it is based on the assumptions that food contains pesticide residues at the tolerance level and that 100% of the crop is treated by pesticides that have established tolerances. If the TMRC exceeds the RfD or poses a lifetime cancer risk that is

greater than approximately one in a million, EPA attempts to derive a more accurate exposure estimate for the pesticide by evaluating additional types of information (anticipated residue data and/or percent of crop treated data) which show, generally, that pesticide residues in most foods when they are eaten are well below established tolerances.

II. Aggregate Risk Assessment and Determination of Safety

The toxicology data base for fipronil has previously been evaluated and was considered adequate to support registration for use on corn (62 FR 62970) (FRL–5757–4). Since that time, MB46513 has been identified. It appears to have greater toxicity than the parent, fipronil. MB46513 is not an animal or plant metabolite. Rather, it forms when the parent compound fipronil is exposed to sunlight. It is not present on corn, but is potentially present on rice due to the foliar application (to germinated rice seed).

Consistent with section 408(b)(2)(D) of the FFDCA, 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 fipronil and to make a determination on aggregate exposure, consistent with section 408(b)(2) of the FFDCA, for tolerances for combined residues of fipronil, its metabolites MB46136 and MB45950, and its photodegradate MB46513 in or on rice grain at 0.04 ppm and rice straw at 0.10 ppm.

A. Toxicology Data Base

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 fipronil and its photodegradate MB46513 are discussed in this unit.

1. *Acute studies*—i. *Technical fipronil.* A battery of acceptable acute toxicity studies place technical fipronil in toxicity Categories II and III. It is classified as a non-sensitizer.

ii. *Icon 6.2 FS (56% fipronil).* A battery of acute toxicity studies submitted for Icon 6.2 FS places it in toxicity categories II and III. This formulation is classified as a sensitizer.

iii. *MB46513.* Based on acute oral and acute dermal studies, MB46513 is classified in toxicity category I. No studies were submitted for acute inhalation, primary eye, primary dermal, and dermal sensitization.

2. *Subchronic toxicity testing.* The data base for subchronic toxicity is considered complete. No additional studies are required at this time.

i. *Fipronil.* a. An acceptable subchronic oral toxicity feeding study in the rat established the lowest observed-effect level (LOEL) to be 30 ppm for males (1.93 milligram (mg)/kilogram (kg)/day) and females (2.28 mg/kg/day) based on alterations in serum-protein values and increased weight of the liver and thyroid. The NOEL was 5 ppm for males (0.33 mg/kg/day) and females (0.37 mg/kg/day).

b. An acceptable subchronic oral toxicity feeding study in the mouse established the LOEL at 25 ppm (3.2 and 4.53 mg/kg/day, for males and females, respectively) based on a possible decreased body-weight gain. The no-observed adverse-effect level (NOAEL) was 10 ppm (1.27 and 1.72 mg/kg/day, for males and females, respectively). The NOEL is less than or equal to 1 ppm (0.13 and 0.17 mg/kg/day for males and females, respectively) based on hepatic hypertrophy at all doses.

c. An acceptable subchronic oral toxicity [capsule] study in the dog established that the LOEL is 10.0 mg/kg/day for males (based on clinical signs of toxicity) and 2.0 mg/kg/day for females (based on clinical signs of toxicity and decreased body-weight gain). The NOEL is 2.0 mg/kg/day for males and 0.5 mg/kg/day for females.

d. An acceptable repeated dose dermal study using the rat found that the systemic LOEL was 10 mg/kg/day based on decreased body-weight gain and food consumption; the dermal irritation LOEL is greater than 10.0 mg/kg/day. The systemic NOEL was 5.0 mg/kg/day; the dermal irritation NOEL was greater than or equal to 10.0 mg/kg/day.

ii. *MB46513.* a. An acceptable subchronic oral toxicity feeding study using the rat found that the LOEL was 3 ppm (0.177 and 0.210 mg/kg/day for males and females, respectively) based on the occurrence of aggressivity, irritability to touch and increased motor activity in one male (these signs are also observed in the mouse). The NOEL was 0.5 ppm (0.029 and 0.035 mg/kg/day for males and females, respectively). The study demonstrates that the metabolite is more toxic than the parent chemical fipronil when administered to rats for 90 days.

b. An acceptable subchronic oral toxicity feeding study using the mouse found that the LOEL is 2 ppm (0.32 mg/kg/day), based on the aggressive and irritable behavior with increased motor activity in males. The NOEL is 0.5 ppm (0.08 mg/kg/day).

c. An acceptable subchronic oral toxicity feeding study using the dog established that the LOEL is 35 ppm (1.05 mg/kg/day), based on behavioral changes in 2 out of 5 females. The NOEL is 9.5 ppm (0.29 mg/kg/day).

3. *Chronic toxicity studies.* The data base for chronic toxicity is considered complete. No additional studies are required at this time.

i. An acceptable chronic feeding study in the rat using fipronil found that the LOEL is 1.5 ppm for males (0.059 mg/kg/day) and females (0.078 mg/kg/day) based on an increased incidence of clinical signs and alterations in clinical chemistry and thyroid parameters. The NOEL is 0.5 ppm for males (0.019 mg/kg/day) and females (0.025 mg/kg/day). The study demonstrated that fipronil is carcinogenic to rats at doses of 300 ppm in males (12.68 mg/kg/day) and females (16.75 mg/kg/day).

ii. An acceptable chronic oral toxicity [capsule] study in the dog using fipronil established a LOEL at 2.0 mg/kg/day based on clinical signs of neurotoxicity and abnormal neurological examinations. The NOEL is 0.2 mg/kg/day.

4. *Carcinogenicity studies.* The data base for carcinogenicity is considered complete. No additional studies are required at this time.

i. The results of a carcinogenicity study in the rat using fipronil is described in Unit II.A.3.i of this preamble.

ii. A acceptable carcinogenicity [feeding] study in the mouse using fipronil found that the LOEL is 10 ppm (1.181 mg/kg/day for males and 1.230 mg/kg/day for females) based on decreased body-weight gain, decreased food conversion efficiency (males), increased liver weights and increased incidence of hepatic histopathological changes. The NOEL is 0.5 ppm (0.055 mg/kg/day for males and 0.063 mg/kg/day for females). The study demonstrated that fipronil is not carcinogenic to CD–1 mice when administered at doses of 30 ppm.

5. *Developmental toxicity studies.* The data base for developmental toxicity is considered complete. No additional studies are required at this time.

i. *Fipronil.* a. An acceptable prenatal developmental study in the rat found that the maternal toxicity LOEL was 20 mg/kg/day based on reduced body-weight gain, increased water consumption, reduced food consumption, and reduced food efficiency. The maternal toxicity NOEL was 4 mg/kg/day. The developmental toxicity LOEL was greater than 20 mg/

kg/day. Developmental toxicity NOEL was 20 mg/kg/day or higher.

b. An acceptable prenatal developmental study in the rabbit found that the maternal toxicity LOEL was 0.1 mg/kg/day or lower, based on reduced body-weight gain, reduced food consumption and efficiency. Maternal toxicity NOEL was less than 0.1 mg/kg/day. The developmental toxicity LOEL was greater than 1.0 mg/kg/day. The developmental toxicity NOEL was 1.0 mg/kg/day or higher.

ii. *MB46513.* An acceptable prenatal developmental study using the rat found that the maternal toxicity LOEL was 2.5 mg/kg/day and the NOEL was 1.0 mg/kg/day based an increase in clinical signs of toxicity (reduced body-weight gain, food consumption and food efficiency). The Developmental Toxicity LOEL was 2.5 mg/kg/day and the NOEL was 1.0 mg/kg/day based on the slight increase in fetal and litter incidence of reduced ossification of several bones.

6. *Reproduction toxicity studies.* The data base for reproductive toxicity is considered complete. No additional studies are required at this time.

An acceptable two-generation reproduction study in the rat using fipronil concluded that the LOEL for parental (systemic) toxicity was 30 ppm (2.54 mg/kg/day for males and 2.74 mg/kg/day for females) based on increased weight of the thyroid glands and liver in males and females; decreased weight of the pituitary gland in females; and an increased incidence of follicular epithelial hypertrophy in the females. The NOEL for parental (systemic) toxicity was 3 ppm (0.25 mg/kg/day for males and 0.27 mg/kg/day for females).

The LOEL for reproductive toxicity was 300 ppm (26.03 mg/kg/day for males and 28.40 mg/kg/day for females) based on clinical signs of toxicity in the F_1 and F_2 offspring; decreased litter size in the F_1 and F_2 litters; decreased body weights in the F_1 and F_2 litters; decrease in the percentage of F_1 parental animals mating; reduction in fertility index in F_1 parental animals; reduced post-implantation survival and offspring postnatal survivability in the F2 litters; and delay in physical development in the F_1 and F_2 offspring. The NOEL for reproductive toxicity was 30 ppm (2.54 mg/kg/day for males and 2.74 mg/kg/day for females).

7. *Neurotoxicity.* The data base for neurotoxicity is considered complete. No additional studies are required at this time.

i. *Fipronil.* a.An acceptable acute neurotoxicity study in the rat concluded the following: The NOEL was 0.5 mg/kg for males and females. The LOEL was 5.0 mg/kg for males and females based on decreased hind-leg splay at the 7 hour post-treatment evaluation in males and females.

b. An acceptable acute neurotoxicity study in the rat concluded that the NOEL was 2.5 mg/kg. The LOEL is 7.5 mg/kg, based on decreased body-weight gains, food consumption and feed efficiency in females, decreased hindlimb splay in males (at 7-hours post test) and decreased grooming in females (14-days post test).

c. An acceptable subchronic neurotoxicity screening battery in the rat concluded the LOEL was 150 ppm (8.89 mg/kg/day, males; 10.8 mg/kg/day, females) based on the results of the functional observational battery (FOB); the NOEL was 5.0 ppm (0.301 mg/kg/day, males; 0.351 mg/kg/day, females).

d. In a developmental neurotoxicity study, fipronil was administered to 30 female rats/group in the diet at dose levels of 0, 0.5, 10, or 200 ppm (0.05, 0.90, or 15 mg/kg/day, respectively) from gestation day 6 to lactation day 10. This study found that the maternal LOEL was 200 ppm (15 mg/kg/day), based on decreased body weight, body-weight gain, and food consumption. The maternal NOEL was 10 ppm (0.90 mg/kg/day). The developmental toxicity LOEL is 10 ppm (0.9 mg/kg/day), based on a marginal but statistically significant decrease in group mean pup weights during lactation and significant increase in time of preputial separation in males. The NOEL for developmental toxicity is 0.5 ppm (0.05 mg/kg/day). The developmental neurotoxicity LOEL is 200 ppm (15 mg/kg/day) based on: Decreased auditory startle response; reduced swimming direction scores, group mean angle measurements, and water ''Y'' maze times trails; and decreased absolute-brain weights. The NOEL for developmental neurotoxicity is 10 ppm (0.90 mg/kg/day).

It is noted that developmental toxicity occurred at a dose lower than the maternal-toxicity NOEL in this study. However, EPA did not consider this to indicate increased susceptibility to infants and children. See Unit II.F.1.ii.d of this preamble for a detailed discussion of this point.

ii. *MB46513.* An acceptable acute neurotoxicity study in the rat concluded that the neurobehavioral LOEL for rats is 12 mg/kg based on decreases in body-weight gains and food consumption for males and females during the week following treatment, significant decreases in locomotor activity 6-hours post dosing for both males and females, decreases in hind-limb splay and rectal temperature at 6-hours post dose in males and females, decreases in the proportion of high-dose males with an immediate righting reflex on days 7 and 14. Decreased forelimb grip strength in males on day 7 and increased forelimb grip strength in high-dose females at 6-hours post dosing was possibly related to the treatment, because there were also slight increases in forelimb grip strength in high-dose males at 6 hours and slight decreases in forelimb grip strength in high dose females at 7 days and in high-dose males and females at 14 days.. The NOEL is 2 mg/kg.

8. *Mutagenicity.* The available studies indicate that fipronil and MB46513 are not mutagenic in bacteria and are not clastogenic *in vitro* or *in vivo* up to doses that showed clear test material interaction with the target cells. Based on these considerations, EPA concluded that there is no concern for mutagenicity. The submitted test battery for both compounds satisfy the new mutagenicity initial testing battery guidelines. No further studies are required at this time.

i. *Fipronil.* a. An acceptable gene mutation/bacteria test using salmonella typhimurium concluded that fipronil was not mutagenic.

b. An acceptable *in vitro* gene mutation assay in mammalian cells/Chinese hamster V79 cells concluded as follows: Fipronil was negative for inducing forward gene mutations at the HGPRT locus in cultured Chinese hamster V79 cells.

c. An acceptable cytogenetic *in vivo* micronucleus assay in the mouse concluded as follows: There was no evidence of a clastogenic or aneugenic effect at any dose or at any harvest time.

d. An acceptable cytogenetic assay in human lymphocytes concluded as follows: There was no evidence of a clastogenic effect when human lymphocytes were exposed *in vitro* to fipronil.

ii. *MB46513.* a. An acceptable gene mutation/bacteria test using salmonella typhimurium showed that there was no evidence of a mutagenic response at any dose.

b. An acceptable gene mutation/*in vitro* assay in mammalian cells considering the HPRT locus in Chinese Hamster Ovary (CHO) cells showed that MB46513 did not induce forward mutations at the HPRT locus in CHO cells at any dose level tested.

c. An acceptable cytogenetics/*in vivo* mouse bone marrow micronucleus assay showed that there was no significant increase in the frequency of MPCEs in bone marrow after any MB46513 treatment time; therefore, the test article is considered negative in this micronucleus assay.

9. *Metabolism study.* The data base for metabolism is considered to be

complete. No additional studies are required at this time.

i. *Fipronil.* An acceptable metabolism study in the rat using 14–C labeled and unlabeled fipronil showed the following: With oral dosing, the rate and extent of absorption appeared similar among all dose groups, but may have been decreased at the high dose. There were no significant sex-related differences in excretion. Feces appeared to be the major route of excretion for fipronil derived radioactivity, where 45–75% of an administered dose was excreted. Excretion in urine was between 5–25%. Major metabolites in urine included two ring-opened products of the metabolite MB45897, two oxidation products (MB46136 and RPA200766), and the parent chemical. In feces, the parent was detected as a significant fraction of the sample radioactivity as well as the oxidation product MB46136 and MB45950. Since MB46513 is not an animal metabolite but a photodegradate, it was not found in this study.

ii. *MB46513.* In a acceptable rat metabolism study, ^{14}C labeled MB46513 was administered to rats by gavage as a single dose or as a single dose following a 14–day pretreatment with unlabeled MB46513. Unchanged MB46513 in urine accounted for less than 0.1% of the dose. Fecal excretion of unchanged MB46513 is the principal pathway for elimination of MB46513 from rats. The high levels of radioactivity in fat compared to blood and the prolonged elimination half-life indicate that there is a potential for bioaccumulation of MB46513 in fatty tissues.

10. *Dermal absorption*—i. *Fipronil.* An acceptable study using the rat found that the quantity of fipronil absorbed was less than 1% at all doses. The system was saturated at 3.88 mg/cm^2. The dermal absorption rat was calculated to be less than 1% at 24 hours.

ii. *MB46513.* An acceptable study in the rat using [^{14}C] labeled MB46513 found that after 24 hours of exposure, dermal absorption of MB46513 was minimal. For all dose groups, the majority of the dose was not absorbed (90.2–102.3%), and only trace amounts (equal to or less than 0.1%) of radioactivity were excreted in the urine and feces. There was 2.35% adhered to the skin and absorbed at the 10 hour time point with the lowest dose applied (0.006 mg/cm^2).

11. *Special studies*—i. *Fipronil.* a. A supplemental thyroid function study in the rat showed the following: The treatment with fipronil or Noxyflex appeared to result in stimulation of the thyroid glands as evidenced by increased accumulation of ^{125}I in the thyroid glands and by increases in the ratios of radioactive distribution between the blood and thyroid. These changes were accompanied by increases in thyroid weight. Treatment with propylthiouracil (PTU) produced decreases in the amount of ^{125}I incorporated in the thyroid and in the blood: Thyroid ratios along with elevated levels of ^{125}I in the blood. However, the weights of the thyroids from these animals were increased by over 2.5 fold compared to the controls and therefore, the ratio of ^{125}I in the blood to thyroid weight was reduced. The administration of perchlorate produced further reductions in the ^{125}I content in the thyroids and in the blood: Thyroid ^{125}I radioactivity ratio. There was no evidence of an inhibition of iodide incorporation by either fipronil or noxyflex.

b. A supplemental thyroxine clearance study in the rat using technical fipronil showed the following: Fipronil had no effect on mortality or other ante mortem parameters. Phenobarbital-treated animals were observed to have collapsed posture, lethargy and shallow breathing on the first day of treatment. There was no effect of fipronil on clearance after 1 day of treatment. However, after 14 days, there was a decrease in terminal half life (52% of control level) and increases in clearance and volume of distribution (261% and 137% of control level, respectively). The effects seen with phenobarbital treatment were similar, although quantitatively not as severe and were evident on day one of treatment.

c. An acceptable 28–day dietary study in the rat concluded that the LOEL is 25 ppm or lower (3.4 mg/kg/day in males; 3.5 mg/kg/day in females), based on clinical laboratory changes, increased absolute liver weights in females and histopathological alterations in the thyroid glands. The NOEL is less than 25 ppm.

ii. *MB46513.* An acceptable 28–day dietary range-finding study in the rat measured thyroid hormone levels as well as standard study parameters. It found that the LOEL is 30 ppm (2.20 and 2.32 mg/kg/day for males and females, respectively), based on clinical signs including piloerection, curling up and thin appearance; and decreased body weights in both sexes. The NOEL is 3 ppm (0.23 and 0.24 mg/kg/day for males and females, respectively).

B. Toxicology Endpoints

The toxicology endpoints for fipronil and MB46513 are presented in this unit.

1. *Fipronil*—i. *RfD.* The RfD for fipronil is 0.0002 mg/kg/day using a NOEL of 0.019 mg/kg/day (0.5 ppm) established from a combined chronic toxicity/carcinogenicity study in rats and an uncertainty factor of 100. The LOEL=1.5 ppm (male (M): 0.059 mg/kg/day; female (F): 0.078 mg/kg/day), based on an increased incidence of clinical signs (seizures and death) and alterations in clinical chemistry (protein) and thyroid parameters.

ii. *Carcinogenic classification and risk quantification.* EPA has classified this chemical as a Group C—Possible Human Carcinogen, based on increases in thyroid follicular-cell tumors in both sexes of the rat, which were statistically significant by both pair-wise and trend analyses. EPA has used the RfD methodology to estimate human risk because the thyroid tumors are due to a disruption in the thyroid-pituitary status. There was no apparent concern for mutagenicity.

iii. *Dermal absorption.* The percent absorbed was less than 1% at 24 hours based on a dermal absorption study.

iv. *Other toxicological endpoints*—a. *Acute dietary (1 day).* In an acute neurotoxicity study in rats the NOEL was 2.5 mg/kg/day based on decreased body-weight gains, food consumption and feed efficiency in females, and decreased hind-limb splay in males at 7-hours post dosing at 7.5 mg/kg/day LOEL. Although a developmental neurotoxicity study with the parent compound fipronil had a lower NOEL, EPA determined that the effects from that study are not attributable to a single exposure (dose) and therefore are not appropriate for acute dietary-risk assessments.

b. *Short- and intermediate-term residential (dermal).* In a 21-day dermal study the NOEL was 5 mg/kg/day based on decreased body-weight gain and food consumption in male and female rabbits observed at the LOEL of 10 mg/kg/day. The dermal NOEL is supported by the oral NOEL of 0.05 mg/kg/day established in a developmental neurotoxicity study when used in conjunction with a dermal absorption factor of 1%. This yields an equivalent-dermal dose of 5 mg/kg/day.

c. *Chronic residential (non-cancer).* In a combined chronic toxicity/carcinogenicity study in the rat, the NOEL is 0.5 ppm (M: 0.019 mg/kg/day; F: 0.025 mg/kg/day), based on an increased incidence of clinical signs (seizures and death) and alterations in clinical chemistry (protein) and thyroid parameters (increased TSH, decreased T4) at 1.5 ppm (M: 0.059 mg/kg/day; F: 0.078 mg/kg/day). Since the NOEL identified is from an oral study, a

dermal absorption factor of less than 1% was used in risk calculations. (This study/dose was also used to establish the chronic RfD).

2. *MB46513*—i. *RfD.* There is no long-term (chronic or carcinogenicity) studies are available for MB46513. However, the toxicity profile of MB46513 indicate this material to be approximately 10 times more potent than the parent compound when the NOELs/LOELs are compared (with the exception of the acute toxicity tests). See table 1 in this preamble.

TABLE 1.—A COMPARISON OF TOXICITIES OF PHOTODEGRADATE MB46513 AND FIPRONIL

Study	Photodegradate MB46513	Fipronil
Acute Oral.	LD_{50}= 16 mg/kg	LD_{50}= 92 mg/kg
Acute Neurotoxicity.	NOEL/LOEL= 2/12 mg/kg	NOEL/LOEL= 2.5/7.5 mg/kg NOEL/LOEL= 0.5/5.0 mg/kg
28–Day Oral—Rat.	NOEL/LOEL= 0.23/2.2 mg/kg/day	NOEL/LOEL= 3.4 mg/kg/day lowest dose tested (LDT)
90–Day Oral—Mouse.	NOEL/LOEL= 0.08/0.32 mg/kg/day	NOEL/LOEL= 1.7/3.2 mg/kg/day
90–Day Oral—Rat.	NOEL/LOEL= 0.029/0.18 mg/kg/day	NOEL= 0.33/1.9 mg/kg/day
Developmental—Rat.	Maternal NOEL/LOEL= 1/2.5 mg/kg/day Developmental NOEL/LOEL= 1/2.5 mg/kg/day	Maternal NOEL/LOEL= 4/20 mg/kg/day Developmental NOEL/LOEL= 20 mg/kg/day highest dose tested (HDT)

As shown in table 1 of this preamble, the 28-day and 90-day subchronic oral studies and oral developmental studies consistently demonstrated an approximately 10-fold greater potency of MB46513 as compared to the parent compound, fipronil. In the acute oral tests, the difference between the LD_{50} values for MB46513 and fipronil is not considered significant due to the insensitivities inherent in this test.

EPA concluded that there is sufficient experimental evidence to warrant the application of a 10-fold Potency Adjustment Factor (PAF) to the chronic NOEL for the parent compound to calculate a chronic NOEL for MB46513 in the absence of test data on the chemical. An adjusted NOEL was established at 0.0019 mg/kg/day for MB46513.

An Uncertainty Factor (UF) of 100 was applied to account for inter (10 x)- and intra-(10x) species variation.

ii. *Carcinogenic classification and risk quantification*. No carcinogenicity studies are available with MB46513. Fipronil, the parent compound, was classified as a Group C Carcinogen (Possible Human Carcinogen). This classification is based on increased incidence of thyroid follicular-cell tumors in rats. EPA used the RfD methodology for the quantification of human risk because the thyroid tumors are related to a disruption in the thyroid-pituitary status and there was no apparent concern for mutagenicity or available information from structurally related analogs. EPA has no reason to believe MB46513 is more carcinogenic than the parent. EPA determined that it was appropriate to use the RfD methodology to quantify chronic risk for MB46513. The NOEL used for the chronic RfD has been adjusted by the PAF to account for the fact that MB46513 is about 10 times more toxic than the parent (except for acute toxicity).

iii. *Dermal absorption*. The percent absorbed is estimated at approximately 2% at 10 hours based on a dermal absorption study with MB46513.

iv. *Other toxicological endpoints*—a. *Acute dietary*. The NOEL is 2 mg/kg in an acute neurotoxicity study in rats (with MB46513) based on significant decreases in locomotor activity in both sexes during the first 30 minutes as well as decreases in hind-limb splay and rectal temperature in both sexes at 6-hours post dosing at 12 mg/kg/day LOEL. Effects were seen on the day of treatment after a single-oral exposure (dose) and thus is appropriate for this risk assessment. For reasons noted in Unit II.B.1.iv of this preamble, EPA did not use a developmental neurotoxicity study with the parent compound fipronil for this risk assessment.

b. *Short- and intermediate-term dermal exposure (1 to 7 days) (1 week to several months).* The adjusted dose of 0.5 mg/kg/day was derived by dividing the study NOEL of 5 mg/kg/day by the PAF of 10 (5/10= 0.5 mg/kg/day). The LOEL was based on decreases in body-weight gain and food consumption. The dose and endpoint from the 21-day dermal study with the parent compound was used for the following reasons:

(1) A 21–dermal toxicity study with MB46513 is not available.

(2) There is low potential for risk from dermal exposure due to minimal dermal absorption as indicated for both the parent (< 1%) and the MB46513 (2%) materials.

(3) The developmental/developmental neurotoxicity NOEL of 0.05 mg/kg/day for fipronil (established in the developmental neurotoxicity study), adjusted for 1% dermal absorption (DA), results in a comparable dermal dose of 5 mg/kg/day (i.e., 0.05 mg/kg/day ′ 1% DA= 5 mg/kg/day) which essentially is the same as the NOEL for fipronil in the 21–day dermal toxicity study.

Residential exposure to MB46513 is not expected while spraying or handling a recently treated pet as these are brief periods usually occurring indoors, and MB46513 forms upon exposure to sunlight. Post-application exposure to the degradate is also not expected due to the products reportedly strong affinity to the sebum and epidermis of pets.

c. *Chronic dermal exposure (several months to lifetime).* Based on the current use pattern for MB46513 (i.e., 1 application/year at planting), long-term exposure via the dermal route is not expected. Residential exposures are not chronic in nature as label uses for pets indicate treatment every 1 to 3 months.

d. *Recommendation for aggregate exposure risk assessments.* An aggregate systemic (oral) and dermal exposure-risk assessment is not appropriate due to differences in the toxicity endpoints observed between the oral (neurotoxicity and alterations in clinical chemistry and thyroid parameters) and dermal (decreases in body-weight gain and food consumption) routes. An aggregate oral and inhalation risk assessment is not required due to the lack of exposure potential via the inhalation route based on the current use pattern.

C. Exposures and Risks

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.517) for the combined residues of fipronil in or on on corn, eggs, meat, milk, and poultry. Risk assessments were conducted by EPA to assess dietary exposures and risks from fipronil and MB46513 as follows:

i. *Acute dietary risk*. An acute dietary risk assessment is required for fipronil and its metabolites and degradate. The NOEL of 2.5 mg/kg was selected as the endpoint to be used for fipronil, MB46136, MB45950, and MB46513. Since MB46513 does not appear to be significantly more acutely toxic than the parent, it was incorporated into the acute dietary risk evaluation system (DRES) run for rice. If further refinements in the acute dietary risk assessment are required in the future, a separate DRES run for MB46513 only will be performed.

TABLE 2.—ACUTE RISK FOR FIPRONIL, ITS METABOLITES, AND DEGRADATE

Subgroup	RfD (mg/kg/day)	Level of concern	Exposure (mg/kg/day)	Percent of RfD
General U.S. Population.	0.025	100% RfD	0.0018	7
Infants (< 1 year).	0.025	100% RfD	0.003	12
Children (1–6 years).	0.025	100% RfD	0.003	12
Females (13+ years).	0.025	100% RfD	0.0012	5

TABLE 2.—ACUTE RISK FOR FIPRONIL, ITS METABOLITES, AND DEGRADATE—Continued

Subgroup	RfD (mg/kg/day)	Level of concern	Exposure (mg/kg/day)	Percent of RfD
Males (13+ years).	0.025	100% RfD	0.0014	6

EPA does not consider the acute dietary risks to exceed the level of concern.

ii. *Chronic dietary risk*. A chronic dietary risk assessment is required for fipronil, MB46136, and MB45950. The RfD used for the chronic dietary analysis for parent fipronil and 2 metabolites is 0.0002 mg/kg/day. The RfD used for MB46513 is 0.00002 mg/kg/day. The analysis evaluates individual food consumption as reported by respondents in the United States Department of Agriculture (USDA) 1977–78 Nationwide Food Consumption Survey (NFCS) and accumulates exposure to the chemical for each commodity.

Chronic DRES for fipronil, MB46136, MB45950, and MB46513 are summarized in Table 3 of this preamble. The DRES analysis utilized the anticipated residues calculated from field-trial data for all animal, corn, and rice commodities. The proposed fipronil uses result in an Anticipated Residue Contribution (ARC) that is equivalent to the following percent of the RfD:

TABLE 3.—CHRONIC DIETARY RISK

Subgroups	Fipronil, MB46136, and MB45950	Photodegradate MB46513	Total
U.S. Population (48 states)	4.8%	1.7%	6.5%
Hispanics	6.2%	2.9%	8.1%
Non-Hispanic Others	5.8%	3.9%	9.7%
Nursing Infants (< 1 year old)	2.8%	2.3%	5.1%
Non-Nursing Infants (< 1 year old)	11.2%	5.5%	16.7%
Females (13+ years, pregnant)	3.3%	1.2%	4.5%
Females (13+ years, nursing)	4.2%	1.6%	5.8%
Children (1–6 years old)	11.4%	3.8%	15.2%
Children (7–12 years old)	7.6%	2.3%	9.9%
Females (20+ years, not pregnant, not nursing).	3.0%	1.2%	4.2%

EPA does not consider the chronic dietary risk to exceed the level of concern.

Anticipated residues. Section 408(b)(2)(E) of the FFDCA authorizes EPA to consider 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.

Percent crop treated. Section 408(b)(2)(F) of the FFDCA states that the Agency may use data on the actual percent of food treated for assessing chronic dietary risk onl if the Agency can make the following findings:

(1) 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.

(2) That the exposure estimate does not underestimate exposue for any significant subpopulation group.

(3) If data are available on pesticide use and food consumption in a particular area, the exposure estimate does not understate exposue 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 percent crop treated as required by the section 408 (b)(2)(F) of the FFDCA, EPA may require registrants to submit data on percent crop treated.

Anticipated residues, based on average field trial values, and percent crop treated information were used to estimate dietary risk for the chronic dietary risk assessment. For the acute dietary risk assessment, anticipated residues in blended commodities (such as corn and rice processed commodities) were used, without the adjustment for percent crop treated. However, tolerance level residues were used for fat; meat by-products; meat of cattle, goats, hogs, horses, sheep, and poultry; and eggs. Since milk is a blended commodity, an anticipated residue value was used. As required by the FQPA, EPA will issue a data call-in under section 408(f) of the FFDCA to all fipronil registrants for data on anticipated residues, to be submitted no later than 5 years from the date of issuance of these tolerances.

The percent of crop treated estimates for fipronil and MB46513 were based on an estimate of percent crop treated by existing products used to control rice water weevil and chinch bugs. In addition, as set forth in 62 FR 62970, market share estimates were used for corn. They were based on an estimate of percent crop treated by other insecticides to control corn rootworm, wireworm, and corn borer. EPA considers these data reliable. A range of estimates are supplied by this data and the upper end of this range was used for the exposure assessment. By using this upper end estimate of percent crop treated, the Agency is reasonably certain that exposure is not underestimated for any significant subpopulation. Further, regional consumption information is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Review of this regional

data allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency.

To provide for the periodic evaluation of these estimates of percent crop treated and to meet the requirement for data on anticipated residues, EPA may require fipronil registrants to submit data on percent crop treated.

2. *Dietary exposure (drinking water source).* EPA does not have monitoring data available to perform a quantitative drinking water risk assessment for fipronil at this time. Using environmental fate data, EPA developed ground and surface water exposure estimates for use on corn and rice.

i. *Ground water (tiered assessment).* The environmental fate data for fipronil indicate a moderate to high persistence and relatively low mobility in terrestrial environments. Based on the SCI-GRO model, acute drinking water concentrations in shallow ground water on highly vulnerable sites are not likely to exceed the values set forth in tables 4–7 of this preamble:

TABLE 4.—ESTIMATED GROUND WATER RESIDUES OF FIPRONIL AND ITS METABOLITES

	Corn parts per billion (ppb)	Rice (ppb)
Fipronil	0.055	0.00804
MB46136	0.001	0.00038
MB45950	0.00036	0.000685
Total:	0.05636	0.009105

TABLE 5.—ESTIMATED GROUND WATER RESIDUES OF PHOTODEGRADATE MB46513

	Corn (ppb)	Rice (ppb)
Photodegradate MB46513.	0.00026	0.004138

Chronic concentrations are not expected to be higher than acute values. Highly vulnerable sites are those with low-organic matter, coarse textured soils (e.g., sands and loamy sands) and shallow-ground water. The fate data for fipronil and its degradates indicate a higher potential mobility on coarse-textured soils (sand or loamy sands).

ii. *Surface water (tiered assessment).* Based on the environmental fate assessment, fipronil, MB46513, MB46136, and MB45950 can potentially move into surface waters. Since fipronil is used as an in-furrow application on field corn, the runoff potential of fipronil residues is expected to be lower than for unincorporated surface application techniques. Since photodegradation is a major route of degradation for fipronil, its dissipation is expected to be dependent on physical components of the water (i.e. sediment loading) which affect sunlight penetration. The maximum fipronil concentration for acute (peak concentration) and chronic (56–day average) based on the Tier 1 GENEEC surface water modeling is shown in the table 6 of this preamble:

TABLE 6.—SURFACE WATER CONCENTRATIONS FOR FIPRONIL AND ITS METABOLITES BASED ON GENEEC MODELING

	Corn		Rice	
	Acute Peak Estimated Environmental Concentration (EEC)	Chronic 56–day EEC	Acute Peak EEC (ppb)	Chronic 56–day EEC (ppb)
Fipronil	2.05	0.78	1.45	0.40
MB46136	0.168	0.062	0.061	0.004
MB45950	0.039	0.019	0.1296	0.013
Total	2.257	0.861	1.6406	0.417

TABLE 7.—SURFACE WATER CONCENTRATIONS FOR PHOTODEGRADATE MB46513 BASED ON GENEEC MODELING

	Corn		Rice	
	Acute Peak EEC	Chronic 56–day EEC	Acute Peak EEC (ppb)	Chronic 56–day EEC (ppb)
Photodegradate MB46513	0.014	0.009	0.359	0.066

iii. *Drinking water risk (acute and chronic).* To calculate the Drinking Water Level of Concern (DWLOC) for acute exposure relative to an acute toxicity endpoint, the acute dietary food exposure (from the DRES analysis) was subtracted from acute RfD to obtain the acute exposure to fipronil (plus MB45950 and MB46136) in drinking water. To calculate the DWLOC for chronic (non-cancer, cancer) exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure (from DRES) was subtracted from the chronic RfD to obtain the acceptable chronic (non-cancer) exposure to fipronil, MB45950, and MB46136 in drinking water. DWLOCs were then calculated using default body weights and drinking water consumption figures.

a. *Acute risk.* EPA has calculated DWLOCs for acute exposure to fipronil, MB45950, MB46136, and MB46513 in surface and ground water for the U.S. population and children (1–6 yrs). They are 810 and 220 ppb, respectively.

b. *Chronic risk.* For chronic (non-cancer) exposure to fipronil (plus MB45950 and MB46136) in surface and ground water, the drinking water levels of concern are 6.67 and 1.77 ppb for U.S. population and children (1–6 years old), respectively.

c. *Maximum and Average concentrations.* Estimated maximum concentrations of fipronil, MB45950, MB46136, and MB46513 in surface and ground water are 2.271 and 0.05662 ppb (with 0.00026 ppb from MB46513 included), respectively. The estimated average concentration of fipronil, MB45950, and MB46136 in surface water is 0.861 ppb. Chronic concentrations in ground water are not expected to be higher than the acute concentrations. For the purposes of the screening-level assessment, the maximum and average concentrations in

ground water are not believed to vary significantly.

The maximum estimated concentrations of fipronil, MB45950, and MB46136 in surface and ground water are less than EPA's levels of concern for fipronil, MB45950, and MB46136 in drinking water as a contribution to acute aggregate exposure.

The estimated average concentrations of fipronil, MB45950, and MB46136 in surface and ground water are less than EPA's levels of concern for fipronil, MB45950, and MB46136 in drinking water as a contribution to chronic aggregate exposure. Therefore, taking into account the present uses and uses proposed in this action, EPA concludes with reasonable certainty that residues of fipronil, MB45950, and MB46136 in drinking water (when considered along with other sources of exposure for which EPA has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

d. *MB46513 (chronic only).* For chronic (non-cancer) exposure to MB46513 in surface and ground water, the drinking water levels of concern are 0.69 and 0.19 ppb for U.S. population, children (non-nursing infants, < 1 year old), respectively. To calculate the DWLOC for chronic (non-cancer, cancer) exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure (from DRES) was subtracted from the RfD to obtain the acceptable chronic (non-cancer) exposure to MB46513 in drinking water. DWLOCs were then calculated using default body weights and drinking water consumption figures.

Estimated maximum concentrations of MB46513 in ground water is 0.00026 ppb. The estimated average concentration of MB46513 in surface water is 0.009 ppb. Chronic concentrations in ground water are not expected to be higher than the acute concentrations. For the purposes of the screening-level assessment, the maximum and average concentrations in ground water are not believed to vary significantly. The estimated average concentrations of MB46513 in surface and ground water are less than EPA's levels of concern for MB46513 in drinking water as a contribution to chronic aggregate exposure. Therefore, taking into account the present uses and uses proposed in this action, EPA concludes with reasonable certainty that residues of MB46513 in drinking water (when considered along with other sources of exposure for which EPA has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

3. *From non-dietary exposure.* The residential uses of fipronil include the use of ant and cockroach bait traps ranging from 0.01 to 0.05 percent active ingredient. In addition, three fipronil products are registered to control fleas and ticks on dogs and cats. These products are applied to the fur of the animal as a ready-to-use pump spray or as a ready-to-use, pour-on, spot treatment made along the back of the animal between the shoulder blades.

i. *Ant and roach baits.* Exposure from the use of fipronil in self contained bait stations is expected to result in low exposures since there is no contact with the pesticide.

ii. *Pet care.* For purposes of setting a tolerance, an aggregate short-term and intermediate-term systemic (oral) and dermal exposure risk assessment which includes the pet care products is not appropriate due to differences in the toxicity endpoints observed between the oral (neurotoxicity and alterations in clinical chemistry and thyroid parameters) and dermal (decreases in body-weight gain and food consumption) routes. Further, though fipronil is currently registered for residential uses, no chronic residential exposure is anticipated.

4. *Cumulative exposure to substances with common mechanism of toxicity.* Fipronil is structurally similar to other members of the pyrazole class of pesticides (i.e., tebufenpyrad, pyrazolynate, benzofenap, etc.). Further, other pesticides may have common toxicity endpoints with fipronil.

Section 408(b)(2)(D)(v) of the FFDCA 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.'' The Agency believes that ''available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides as to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

EPA does not have, at this time, available data to determine whether fipronil has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, fipronil 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 fipronil has a common mechanism of toxicity with other substances.

5. *Endocrine disruption.* EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect...''. The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry, and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3 years from the passage of FQPA (August 3, 1999) to implement this program. At that time, EPA may require further testing of this active

ingredient and end use products for endocrine disrupter effects.

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

1. *Acute aggregate exposure and risk*. Using refined exposure assumptions (anticipated residues for blended commodities), a high-end exposure estimate (food only) was calculated for these subgroups: females 13+ years, for the general U.S. population, infants (< 1 year), children (1–6 years), and males 13+. These risk estimates are the same as those displayed in table 2 of this preamble.

The maximum estimated concentrations of fipronil in surface and ground water are less than EPA's levels of concern for fipronil in drinking water as a contribution to acute aggregate exposure.

2. *Short- and intermediate-term aggregate exposure and risk*. An aggregate systemic (oral) and dermal exposure risk assessment is not appropriate due to differences in the toxicity endpoints observed between the oral (neurotoxicity and alterations in clinical chemistry and thyroid parameters) and dermal (decreases in body-weight gain and food consumption) routes.

3. *Chronic aggregate exposure and risk*. Chronic dietary exposure estimates for fipronil, MB46136, MB45950, and MB46513 utilized anticipated residues and a projected market share and are thus highly refined. For the U.S. population, 6.5% of the RfD is occupied by dietary (food) exposure. Though fipronil is currently registered for residential uses, no chronic residential exposure is anticipated. The estimated average concentrations of fipronil in surface and ground water are less than EPA's levels of concern for fipronil in drinking water as a contribution to chronic aggregate exposure.

4. *Aggregate cancer risk for U.S. population*. For fipronil plus MB46136 and MB45950, EPA finds that the dietary risk concerns due to long-term consumption of fipronil residues are adequately addressed by the DRES chronic exposure analysis using the RfD. For MB46513, EPA finds that the dietary risk concerns due to long-term consumption of MB46513 residues are adequately addressed by the DRES chronic exposure analysis using the RfD.

5. *Safety finding*. Based on Unit II.C. of this preamble, EPA concludes that there is a reasonable certainty of no harm from aggregate exposure to fipronil.

F. 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 fipronil, EPA considered data from developmental toxicity studies in the rat and rabbit, a two-generation reproduction study in the rat, and a developmental neurotoxicity study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. Growth, survival and general toxicity are evaluated for two generations of offspring. Developmental Neurotoxicity studies are designed to evaluate adverse effects on the nervous system of the developing organism resulting from pesticide exposure of the pregnant and nursing mother during several critical stages of prenatal and postnatal development.

Section 408 of the FFDCA provides that EPA shall apply an additional 10-fold margin of safety for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the database 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 MOE and uncertainty factor (usually 100 for combined inter- and intra-species variability)) and not the additional tenfold MOE/uncertainty factor 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 the standard MOE/safety factor.

ii. *Data on Susceptibility*—a. *Neurotoxicity*. Fipronil has demonstrated neurotoxicity in the acute and subchronic rat neurotoxicity studies as well as in the rat chronic/oncogenicity and chronic dog studies.

b. *Developmental toxicity*. There are acceptable rat and rabbit developmental toxicity studies with fipronil. There is no evidence of developmental toxicity in either study. EPA also considered a developmental study conducted for MB46513. In that study, pregnant rats received oral administration of MB46513 (99.2%). For maternal toxicity, the NOEL was 1.0 mg/kg/day and the LOEL was 2.5 mg/kg/day based on an increase in clinical signs of toxicity (hair loss) and on reduced body-weight gain, food consumption, and food efficiency. For developmental toxicity, the NOEL was 1.0 mg/kg/day and the LOEL was 2.5 mg/kg/day based on a slight increase in fetal and litter incidence of reduced ossification of several bones (hyoid, 5th/6th sternebrae, 1st thoracic vertebral body, pubic bone, and one or two metatarsi). Most of the reduced ossification is weak evidence of a developmental effect. Although the minor decrement in fetal weight at 2.5 mg/kg/day has questionable biological relevance, the decrement is supported by the delayed ossification.

c. *Reproductive toxicity*. There is an acceptable two-generation reproduction study in the rat with fipronil. Toxicity to the offspring (clinical signs of toxicity, decreased litter size, decreased body weights, decreased pre- and postnatal survival, and delays in physical development.) occurred only at levels where there was maternal toxicity (including maternal mortality).

d. *Developmental neurotoxicity*. In an acceptable study with fipronil, developmental neurotoxicity (behavioral changes and decreased absolute brain weights) was seen only at levels where there was maternal toxicity (decreased body weight, body-weight gain and food consumption). However, developmental toxicity (including marginal but statistically significant decrease in group mean pup weights during lactation, and significant increase in time of preputial separation in males) was seen at levels below levels of maternal toxicity.

e. *Adequacy of data*. An acceptable two-generation reproduction study in rats and acceptable prenatal developmental toxicity studies in rats and rabbits have been submitted to the Agency, meeting basic data requirements, as defined for a food-use chemical. In addition, an acceptable developmental neurotoxicity study was conducted with fipronil and reviewed by the Agency. Further, EPA has a developmental toxicity study for MB46513. Where specific data on MB46513 are not available, the toxicity of the photodegradate can be reliably estimated by comparing the fipronil and MB46513 data bases and taking into consideration the PAF. Therefore, additional data on MB46513 are not required at this time. There are no data

gaps for the assessment of the effects of fipronil on developing animals following in utero and/or early postnatal exposure.

f. *Determination of susceptibility*. Although there is no evidence of enhanced pre or post natal susceptibility in infants and children in the developmental and reproduction studies for fipronil and MB46513, the developmental neurotoxicity study for fipronil identified a developmental NOEL (0.05 mg/kg/day) which is less than the maternal NOEL of 0.9 mg/kg/day indicating an apparent susceptibility issue. However, EPA determined that the evidence regarding susceptibility was not convincing due to the equivocal nature of the findings. Of principal importance were the following conclusions:

(1) The effects observed in the offspring at the LOEL of 0.9 mg/kg/day, although statistically significant, were marginal and appeared to define a threshold response level for this study.

(2) The body weight findings of this study are not supported by results of the two-generation reproduction study in rats at similar treatment levels.

EPA concluded that the apparent increased susceptibility in the developmental neurotoxicity study was not supported by the overall weight-of-the-evidence (including no evidence for increased susceptibility in the developmental and reproduction studies) from the fipronil data base.

iii. *Determination of the FQPA safety factor*. There is a complete toxicity data base for fipronil and exposure data is complete or is estimated based on data that reasonably accounts for potential exposures. Further, as discussed in Unit II.F.1.f of this preamble, EPA has concluded that the studies do not show that there is an increased susceptibility for developmental effects. Accordingly, EPA believes reliable data are available to remove the additional 10-fold safety factor for the protection of infants and children.

2. *Acute risk*. The total dietary (food only) percents of the acute RfD for these population subgroups females 13+ years, for the general U.S. population, infants (< 1 year), children (1–6 years), and males 13+ ranged from 6–12%. This calculation was based on an acute neurotoxicity study NOEL in rats of 2.5 mg/kg/day for fipronil and 2.0 mg/kg/day for MB46513. Despite the potential for exposure to fipronil in drinking water, EPA does not expect the acute aggregate exposure to exceed EPA's level of concern. The small percent of the acute dietary RfD calculated for females 13+ years old provides assurance that there is a reasonable certainty of no harm for both females 13+ years and the pre-natal development of infants.

3. *Chronic risk*. EPA has concluded that the percentage of the RfD that will be utilized by chronic dietary (food only) exposure to residues of fipronil ranges from 5.1% for nursing infants less than 1 year old, up to 16.7% for non-nursing infants less than 1 year old. Despite the potential for exposure to fipronil in drinking water, EPA does not expect the chronic aggregate exposure to exceed 100% of the RfD. There are uses of fipronil that result in residential exposure, but is not expected to result in chronic exposure. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from acute, short- and intermediate-term, or chronic aggregate exposure to fipronil residues. That data call-in [will] require such data to be submitted every 5 years as long as the tolerances remain in force.

III. Other Considerations

A. Metabolism In Plants and Animals

1. Rhone Poulenc AG, Inc. has submitted data from a study investigating the metabolism of fipronil in rice. The qualitative nature of the residue in rice is adequately understood based on this metabolism study. Fipronil was detected in all rice commodities. MB46513 was also detected in all commodities. MB45950 and MB46136, among other metabolites, were also identified. EPA determined that the fipronil residues of concern for the tolerance expression and dietary risk assessment in plants animals are the parent and its metabolites MB46136 and MB45950 and photodegradate MB46513. The Agency, therefore, has determined that the residues of concern for the proposed tolerances are fipronil, MB45950, MB46136, and MB46513.

2. Residues in eggs, meat, milk, and poultry. Rice bran, grain, hulls, and straws are animal feed items.

i. *Fipronil*. The maximum theoretical dietary burden of fipronil to beef and dairy cattle, based on the required tolerances of 0.04 ppm for rice and 0.10 ppm for rice straw, is 0.04 ppm. The maximum theoretical dietary burden of fipronil to poultry, based on the proposed tolerances of 0.04 ppm for rice and 0.10 ppm for rice straw, is 0.04 ppm. Acceptable cow and poultry feeding studies were submitted and reviewed in conjunction with the pesticide petition for corn. Based on these studies, the Agency has already established appropriate tolerance levels for fipronil residues in/on animal commodities.

ii. *MB46513*. Based on low potential for residues in eggs, meat, and milk, EPA will not require animal feeding studies to be conducted with MB46513.

B. Analytical Enforcement Methodology

1. *Plants*. In conjunction with the cotton petition, gas chromatography/electron capture detector (GC/ECD) method EC–95–303 has been proposed for enforcement of tolerances for residues of fipronil and its metabolites MB45950, MB46136, and photodegradate MB46513, and RPA200766 in/on plant commodities. The GC methods used for the analyses of samples collected from the rice crop field trials and processing study analyze for each compound separately and are adequate for collection of residue data. Adequate method validation and concurrent method recovery have been submitted for these methods. These methods are similar to the GC method proposed for cottonseed which has undergone a successful pesticide method validation (PMV). The registrant has been notified that all directions pertaining to RPA200766 should also be removed as this metabolite has been determined to not be of regulatory concern.

2. *Animals*. A method for the determination of residues of fipronil, MB45950, and MB46136 in animal commodities was previously reviewed in conjunction with a petition for corn and animal raw agricultural commodities (RACs), and has undergone a successful PMV.

3. *Multiresidue methods*. A report on multiresidue testing of fipronil, MB45950, and MB46136 has been received and forwarded to the Food and Drug Administration (FDA). Acceptable recoveries of fipronil, MB45950, and MB46136 were obtained in corn grain. A report on multiresidue testing of MB46513 has been received and forwarded to FDA. Acceptable recoveries of MB46513 were obtained in corn forage and cottonseed.

C. Magnitude of Residues

1. *Plants*. The submitted data indicate that the combined residues of fipronil, MB45950, MB46136, and MB46513 will not exceed the proposed tolerance for rice straw (0.10 ppm), or the proposed tolerance for rice grain (0.04 ppm) in/on samples harvested at maturity following either a preplant incorporated (PPI) broadcast application of the 80% water dispersable granular (WDG) formulation or seed treatment with a 10% liquid formulation at about 0.05 lb active ingredient (ai)/acre (A) (1 x the proposed maximum rate).

Based on the highest residue value obtained from samples harvested following the proposed PPI or seed treatments at the proposed maximum use rate, the proposed tolerance level of 0.10 ppm for rice straw is appropriate. No residues of fipronil or MB46136, MB45950, or MB46513 were detected in rice grain, so the proposed tolerance level for rice grain at the combined limits of quantitation for fipronil, MB46136, MB45950, and MB46513 (0.04 ppm) is appropriate.

2. *Processed food/feed*. Rhone Poulenc AG, Inc. submitted data depicting the potential for concentration of fipronil residues in the processed commodities of rice. The submitted rice processing data are adequate. The data indicate that total residues of fipronil, MB45950, MB46136, and MB46513, and RPA200766 are less than the limit of quantitation (LOQ) (0.01 ppm) in/on rice grain harvested at maturity following PPI broadcast application of the 80% Because treatment at 5–6 x the label application rate did not result in quantifiable levels of fipronil residues of concern in rice grain, all further requirements for the processing study are waived, and no tolerances are required for the processed commodities of rice. As a result of this use, residues of fipronil are not expected to exceed the proposed or existing tolerances.

D. International Residue Limits

There are no CODEX, Canadian, or Mexican MRLs established for fipronil in/on rice RACs. Therefore, no compatibility problems exist.

E. Rotational Crop Restrictions

An acceptable confined rotational crop study with grain, grain sorghum, lettuce, radishes, and wheat was submitted and reviewed in conjunction with the corn petition.

The rotational crop restrictions specified on the labels (1 month for leafy vegetables, 5 months for root crops, and 12 months for small grains and all other crops) are supported by the results of the confined rotational crop study.

IV. Conclusion

Therefore, the tolerances established at 40 CFR 180.517 are amended to include combined residues of the insecticide fipronil, MB46136, MB45950, and MB46513 in or on rice grain at 0.04 ppm and rice straw at 0.10 ppm.

V. Objections and Hearing Requests.

The new FFDCA section 408(g) provides essentially the same process for persons to ''object'' to a tolerance regulation issued by EPA under new section 408(e) and (l)(6) as was provided in the old section 408 and in section 409. However, the period for filing objections is 60 days, rather than 30 days. EPA currently has procedural regulations which govern the submission of objections and hearing requests. These regulations will require some modification to reflect the new law. However, until those modifications can be made, EPA will continue to use those procedural regulations with appropriate adjustments to reflect the new law.

Any person may, by September 15, 1998, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33. If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is 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 in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). 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.

VI. Public Record

EPA has established a record for this rulemaking under docket control number OPP–300612 (including any comments and data submitted electronically). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in room 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall ı2, 1921 Jefferson Davis Hwy., Arlington, VA.

Electronic comments may be sent directly to EPA at:

opp-docket@epamail.epa.gov.

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption.

The official record for this rulemaking, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer any copies of objections and hearing requests received electronically into printed, paper form as they are received and will place the paper copies in the official rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the Virginia address in **ADDRESSES** at the beginning of this document.

VII. Regulatory Assessment Requirements

This final rule establishes tolerances under FFDCA section 408(d) 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) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by 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).

In addition, since these tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances 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. Nevertheless, the Agency has previously assessed whether establishing tolerances, exemptions from tolerances, raising tolerance levels or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950) and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

VIII. Submission to Congress and the General Accounting Office

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 the 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: July 2, 1998.

Peter Caulkins,

Acting Director, Registration Division, 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. 346a and 371.

2. In § 180.517 by revising the introductory text of paragraph (a) and adding the following entries to the table in paragraph (a) to read as follows:

§ 180.517 Fipronil; tolerances for residues.

(a) *General.* Therefore, tolerances are established for combined residues of the insecticide fipronil (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1R,S)-(trifluoromethyl)sulfinyl]-1H-pyrazole-3-carbonitrile) and its metabolites 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1H-pyrazole-3-carbonitrile and 5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1H-pyrazole-3-carbonitrile and its photodegradate 5-amino-1-(2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1R,S)-(trifluoromethyl)]-1H-pyrazole-3-carbonitrile in or on the following items at the levels specified:

Commodity	Parts per million (ppm)
* * * *	* *
Rice grain	0.04
Rice straw	0.10

* * * * *

[FR Doc. 98–18987 Filed 7–16–98; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300681; FRL–6016–7]

RIN 2070–AB78

Pseudomonas Fluorescens Strain PRA-25; Temporary Exemption From the Requirement of a Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This rule establishes a temporary exemption from the requirement of a tolerance for residues of the microbial pest control agent *pseudomonas fluorescens* strain PRA-25 on peas, snap beans, sweet corn, supersweet corn when applied/used on vegetable seeds in the planter box immediately before planting to reduce seed rot and damping-off disease cause by *Pythium spp.* and root rot caused by *Aphanomyces euteiches.* Good Bugs, Inc. submitted a petition to EPA under the Federal Food, Drug, and Cosmetic Act (FFDCA) as amended by the Food Quality Protection Act of 1996 (FQPA) (Pub. L. 104-170) requesting the temporary/time-limited tolerance. This regulation eliminates the need to establish a maximum permissible level for residues of *pseudomonas fluorescens* strain PRA-25. The tolerance will expire on July 31, 2001.

DATES: This regulation is effective July 17, 1998. Objections and requests for hearings must be received by EPA on or before September 15, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number [OPP–300681], must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing requests shall be labeled ''Tolerance Petition Fees'' and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees) and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees), P.O. Box 360277M, Pittsburgh, PA 15251. A copy of any objections and hearing requests filed with the Hearing Clerk identified by the docket control number, [OPP-300681], must also be submitted 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, bring a copy of objections and hearing requests to Rm. 119, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Copies of electronic objections and hearing requests must be submitted as an ASCII file avoiding 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 5.1/6.1 file format or ASCII file format. All copies of electronic objections and hearing requests must be identified by the docket number [OPP-300681]. No Confidential Business Information (CBI) should be submitted through e-mail. Copies of electronic objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: Linda A. Hollis, c/o Product Manager (PM) 90, Biopesticides and Pollution Prevention Division (7511C), Environmental Protection Agency, 401