Commodity	Parts per million
Horses, meat	0.5
Milk, fat (reflecting 0.1 ppm in whole milk)	1.0
Poultry, fat	0.05
Poultry, mbyp	0.05
Poultry, meat	0.05
Sheep, fat	1.0
Sheep, mbyp	0.1
Sheep, meat	0.5
Strawberries	3.0

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300587; FRL–5757–4]

RIN 2070–AB78

Fipronil; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for combined residues of fipronil (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1*R,S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites MB 46136 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]4-[(trifloumethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile) and MB 45950 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile) in or on field corn grain, stover, and forage; milk fat, (reflecting residues in whole milk); eggs; poultry fat, meat, and meat byproducts; hog fat, meat, meat byproducts, and liver; and liver, fat, meat, and meat byproducts of cattle, goat, horse, and sheep. In petition number 5F4426 Rhone Poulenc AG, Inc. requested this tolerance under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1966 (Pub. L. 104–170).

DATES: This regulation is effective November 26, 1997. Objections and requests for hearings must be received by EPA on or before January 26, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300587], 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–300587], 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. 1132, CM #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@epamail.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–300587]. 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: Marion Johnson, 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: johnson.marion@epamail.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 5F4426) by Rhone Poulenc AG Company, 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, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR part 180 be amended by establishing a tolerance for combined residues of the insecticide fipronil (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1*R,S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites MB 46136 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile) and MB 45950 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile) in or on the following items: corn, field, grain — 0.02 ppm; corn, field, stover — 0.30 ppm; corn, field, forage — 0.15 ppm; Milk, fat (reflecting 0.05 ppm in whole milk) — 1.50 ppm; Liver of cattle, goat, horse and sheep — 0.10 ppm; eggs — 0.03 ppm; Fat of cattle, goat, horse and sheep — 0.40 ppm; poultry fat — 0.05 ppm; meat of cattle, goat, horse and sheep — 0.04 ppm; poultry meat — 0.02 ppm; meat byproducts (except liver) of cattle, goat, horse and sheep — 0.04 ppm; poultry meat byproducts — 0.02 ppm; hog fat — 0.04 ppm; hog liver — 0.02 ppm; hog meat byproducts (except liver) — 0.01 ppm; hog meat — 0.01 ppm.

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) 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 percent 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 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 hundredfold MOE is based on the same rationale as the hundredfold 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, FFDCA section 408 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.

Percent of crop treated estimates are derived from Federal and private market survey data. Typically, a range of estimates are supplied and the upper end of this range is assumed for the exposure assessment. By using this upper end estimate of percent of crop

treated, the Agency is reasonably certain that exposure is not understated for any significant subpopulation group. 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, to pesticide residues. For this pesticide, the most highly exposed population subgroup (non-nursing infants <1 year old) was not regionally based.

II. 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 fipronil and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for combined residues of fipronil (5-amino-1-[2,6- dichloro-4-(trifluoromethyl)phenyl]-4-[(1*R,S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites MB 46136 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile) and MB 45950 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile) in or on the following items at the following levels:

Commodity	Tolerance (in parts per million)
Corn, field, grain	0.02
Corn, field, stover	0.30
Corn, field, forage	0.15
Eggs	0.03
Fat of cattle, goat, horse and sheep.	0.40
Hog fat	0.04
Hog liver	0.02
Hog meat byproducts (except liver).	0.01
Hog meat	0.01
Liver of cattle, goat, horse and sheep.	0.10
Milk, fat (reflecting 0.05 ppm in whole milk).	1.50
Meat of cattle, goat, horse and sheep.	0.04
Meat byproducts (except liver) of cattle, goat, horse and sheep.	0.04
Poultry fat	0.05
Poultry meat	0.02
Poultry meat byproducts	0.02

EPA's assessment of the dietary exposures and risks associated with establishing the tolerance follows.

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 are discussed below.

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

ii. An acceptable acute neurotoxicity study in the rat using technical fipronil concluded the following: The no observed effect level (NOEL) was 0.5 mg/kg for males and females. The low observed effect level (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.

2. *Subchronic toxicity testing.* i. An acceptable subchronic toxicity study in the dog using technical fipronil concluded the following: The LOEL was 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 was 2.0 mg/kg/day for males and 0.5 mg/kg/day for females.

ii. A supplemental subchronic toxicity study in the rat using technical fipronil concluded the following: The LOEL was 30 ppm for males (1.93 mg/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).

iii. An acceptable 21–day dermal toxicity study in the rabbit using technical grade fipronil concluded the following: The Systemic LOEL was 10 mg/kg/day based on decreased body weight gain and food consumption; Dermal irritation LOEL > 10.0 mg/kg/day. The systemic NOEL was 5.0 mg/kg/day; Dermal irritation NOEL was greater than or equal to 10.0 mg/kg/day.

3. *Chronic toxicity studies.* i. An acceptable chronic toxicity study in the dog using technical fipronil concluded the following: The LOEL was 2.0 mg/kg/day based on clinical signs of neurotoxicity and abnormal neurological examinations. The NOEL was 0.2 mg/kg/day.

ii. An acceptable carcinogenicity study in the mouse using technical fipronil concluded the following: The LOEL was 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 was 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.

iii. An acceptable combined chronic toxicity/carcinogenicity study in the rat using technical fipronil concluded the following: The LOEL was 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 was 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).

4. *Developmental and reproduction toxicity studies.* i. An acceptable developmental toxicity study in the rat using technical fipronil concluded the following: 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. The developmental toxicity NOEL was 20 mg/kg/day or higher.

ii. An acceptable developmental toxicity study in the rabbit using technical fipronil concluded the following: The maternal toxicity LOEL was less than or equal to 0.1 mg/kg/day based on reduced body weight gain, reduced food consumption and efficiency. The 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 greater than or equal to 1.0 mg/kg/day.

iii. An acceptable multigeneration reproduction study in the rat using technical fipronil concluded the following: 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 F_2 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).

iv. An acceptable developmental neurotoxicity study using technical fipronil concluded as follows: 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 LOEL was 10 ppm (0.9 mg/kg/day), based on statistically significant decrease in group mean pup weights during lactation and significant increase in time of preputial separation in males. The developmental neurotoxicity LOEL was 10 ppm (0.9 mg/kg/day) based on a significant increase in mean motor activity counts in females on Postnatal Day 17. The NOEL for developmental and developmental neurotoxicity is 0.5 ppm (0.05 mg/kg/day). It is noted that developmental neurotoxicity occurred in the absence of maternal toxicity in this study.

5. *Mutagenicity studies*— i. *Studies conducted with fipronil.* a. An acceptable *Salmonella*/mammalian activation gene mutation assaying technical fipronil concluded as follows: fipronil was not mutagenic in 4 strains of S. typhimurium at concentrations up to 500 µg/plate in the presence or absence of S9 activation.

b. An acceptable *in vitro* gene mutation assay in mammalian cells/Chinese hamster V79 cells using technical fipronil concluded as follows: Fipronil was negative for inducing forward gene mutations at the HGPRT locus in cultured Chinese hamster V79 cells at concentrations up to 385.65 µg/ml both with and without S9 activation.

c. An acceptable *in vitro* micronucleus assay in the mouse using technical fipronil concluded as follows: fipronil was not cytotoxic to the target cell. There was, however, no evidence of a clastogenic or aneugenic effect at any dose or at any harvest time.

d. An acceptable cytogenic assay in human lymphocytes using technical fipronil concluded as follows: there was no evidence of a clastogenic effect when human lymphocytes were exposed *in vitro* to fipronil at doses of 75, 150 or 300 µg/ml with and without S9 activation.

ii. *Studies conducted with fipronil metabolite MB 46136.* a. An acceptable *Salmonella*/mammalian activation gene mutation assay using 98.7% pure metabolite showed that the fipronil metabolite was not mutagenic in 4 strains of *S. typhimurium* at concentrations of up to 200 µg/plate without S9 activation and up to 500 µg/plate in the presence of S9 activation.

b. An acceptable cytogenic assay with human lymphocytes using 98.7% pure metabolite showed that there was no evidence of a clastogenic effect when human lymphocytes were exposed *in vitro* to MB 46136 at doses of 75, 150 or 300 µg/ml with and without S9 activation.

6. *Metabolism study*. An acceptable metabolism study in the rat using ^{14}-C 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. Distribution data showed significant amounts of residual radioactivity in carcass, G.I. tract, liver, adrenals, and abdominal fat at 168 hours post-dose for all rats in all dose groups. Repeated low oral dosing or a single high oral dose resulted in an overall decrease in the amount of residual radioactivity found, but an increase in the amount in abdominal fat, carcass, and adrenals. 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%. Increases in the percentages excreted in urine and feces were observed with repeated low oral dosing or a single high dose, while the percentage found in all tissues combined decreased. There were no significant sex-related differences in excretion. Major metabolites in urine included two ring-opened products of the metabolite MB 45897, two oxidation products (MB 46136 and RPA 200766), and parent chemical (MB 46030). In feces, parent MB 46030 was detected as a significant fraction of the sample radioactivity as well as the oxidation products MB 46136 and MB 45950.

7. *Special studies.* i. A supplemental thyroid function study in the rat using technical fipronil showed the following: Four groups of 27 male rats per group were administered either methylcellulose (vehicle control), 10 mg/kg/day fipronil, 200 mg/kg/day propylthiouracil (PTU) or 50 mg/kg/day Noxyflex for 14 days. On Day 15, each animal received Na^{125}I at a dose level of 1 µCi ^{125}I. Six hours later, 9 males per group received either 10 or 25 mg/kg potassium perchlorate or 0.9% saline solution. 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 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.

ii. A supplemental thyroxine clearance study in the rat using technical fipronil showed the following: Six groups of six male rats per group were administered either fipronil (10 mg/kg/day by gavage), phenobarbital (80 mg/kg/day intraperitoneally) or 0.5% methylcellulose (vehicle control at 5 ml/kg by gavage) for a duration of either 1 day or 14 days. Four hours after the final dose of either test substance, each rat received [^{125}I] thyroxine at a dosage of 10 µCi/kg. 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 1 of treatment.

iii. An acceptable 28–day study in the rat by dietary administration using 96.2% pure fipronil metabolite RPA 200766 showed the following: The NOEL was 50 ppm (3.80 mg/kg/day for males and 4.44 mg/kg/day for females). The LOEL was 500 ppm (38.16 mg/kg/day for males and 43.97 mg/kg/day for females) based on decreased

hemoglobin values, increased cholesterol values and increased liver weights in both sexes.

iv. An acceptable 28–Day Study in the rat using technical fipronil showed that: the LOEL is ≤ 25 ppm (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 < 25 ppm.

B. Toxicology Profile

The toxicology endpoints and dose levels of concern have been identified for use in this fipronil exposure and risk assessment as set forth below:

1. *Residential exposure—i. Short - and intermediate - term exposure (1 to 7 days).* a. A dermal absorption factor is set at less than 1% at 24 hours based on a dermal absorption study.

b. For short- and intermediate-term residential exposure for females age 13+ years, the NOEL is 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 in the 21–day dermal study.

In the supporting study of developmental toxicity and developmental neurotoxicity, the developmental NOEL was 0.5 ppm (0.05 mg/kg/day) based on decreased mean pup weights during lactation and a significant increase in time to preputial separation in male rats observed at the developmental LOEL of 10 ppm (0.9 mg/kg/day). The developmental neurotoxicity LOEL was 10 ppm (0.9 mg/kg/day) based on an increase in mean motor activity counts for females on Postnatal Day 17.

It should be noted that the NOEL established after dermal administration in the 21–day dermal toxicity study is 5 mg/kg/day. When the co-critical study NOEL based on oral administration in the developmental neurotoxicity study, 0.05 mg/kg/day is corrected for the less than 1% dermal absorption, exposure is essentially the same as the critical study (5 mg/kg/day).

c. For short- and intermediate-term residential exposure for the general population, including infants and kids, the NOEL is 5.0 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 in the 21–day dermal toxicity study.

ii. *Chronic or residential exposure (several months to lifetime).* The NOEL is 0.5 ppm, 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 the LOEL of 1.5 ppm in a combined chronic toxicity/carcinogenicity study in the rat. Since the NOEL identified is from an oral study, a dermal absorption factor of < 1% should be used in risk calculations.

2. *Dietary exposure*—i. *Acute risk* . The NOEL is 0.5 mg/kg, based on decreased hind leg splay in male and female rats observed at LOEL = 5 mg/kg in the acute neurotoxicity study in rats.

ii. *Chronic risk.* The RfD (reference dose) for fipronil is 0.0002 mg/kg/day. This RfD is based on a NOEL of 0.019 mg/kg/day and an uncertainty factor of 100; the NOEL was established from the combined chronic toxicity/carcinogenicity study in rats where the LOEL was 1.5 ppm, based on an increased incidence of clinical signs (seizures and death) and alterations in clinical chemistry (protein) and thyroid parameters (increased TSH, decreased T4).

iii. *Cancer risk*. Fipronil has been classified 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. The RfD methodology should be used to estimate human risk because the thyroid tumors appear to be related to a disruption in the thyroid-pituitary status. There was no apparent concern for mutagenicity (no mutagenic activity).

B. Exposures and Risks

1. *From food and feed uses*. In today's action, tolerances will be established (40 CFR 180.517) in or on a variety of raw agricultural commodities as follows:

Commodity	Tolerance (in parts per million)
Corn, field, grain	0.02
Corn, field, stover	0.30
Corn, field, forage	0.15
Eggs	0.03
Fat of cattle, goat, horse and sheep.	0.40
Hog Fat	0.04
Hog Liver	0.02
Hog Meat Byproducts (except liver).	0.01
Hog Meat	0.01
Liver of cattle, goat, horse and sheep.	0.10
Milk, fat (reflecting 0.05 ppm in whole milk).	1.50
Meat of cattle, goat, horse and sheep.	0.04
Poultry Fat	0.05
Poultry Meat	0.02
Meat Byproducts (except liver) of cattle, goat, horse and sheep.	0.04
Poultry Meat Byproducts	0.02

Risk assessments were conducted by EPA to assess dietary exposures and risks from fipronil as follows:

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 acute dietary exposure endpoint of concern for fipronil is neurotoxicological. As this endpoint is not developmental, all population subgroups are of potential concern. EPA calculated MOE values of 277 for the U.S. population, 167 for non-nursing infants (< 1 year old) and 167 for children (1–6 years years old). Anticipated residues were used for milk and corn commodities in this assessment.

ii. *Chronic exposure and risk.* Chronic dietary residues exposure estimates (DRES) for fipronil were calculated using anticipated residues derived from field-trial data for all commodities. In addition, an anticipated market share of 7% was used for corn grain, forage, and stover. The proposed fipronil tolerances result in an Anticipated Residue Contribution (ARC) that is equivalent to the following percents of the RfD:

U.S. Population (48 States)	4.6%
Hispanics	5.9%
Non-Hispanic Others	5.2%
Non-Nursing Infants (< 1 year old).	10.1%
Females (13+ years, pregnant)	3.2%
Females (20+ years, not pregnant, not nursing).	3.0%
Females (13+ years, nursing)	4.1%
Children (1–6 years old)	11.1%
Children (7–12 years old)	7.4%

The subgroups listed above are: (1) the U.S. population (48 states); (2) infants and children; and, (3) the other subgroups for which the percentage of the RfD occupied is equal to, or greater than, that occupied by the subgroup U.S. population (48 states).

iii. *Percent crop treated and anticipated residues.* Section 408(b)(2)(E) 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 timeframe it deems appropriate. Section 408(b)(2)(F) allows the Agency to use data on the actual

percent of crop treated when establishing a tolerance only where the Agency can make the following findings:

a. That the data used are reliable and provide a valid a basis for showing the percentage of food derived from a crop that is likely to contain residues.

b. That the exposure estimate does not underestimate the exposure for any significant subpopulation.

c. Where data on regional pesticide use and food consumption are available, that the exposure estimate does not understate exposure for any regional population. In addition the Agency must provide for periodic evaluation of any estimates used.

The percent of crop treated estimates for fipronil were derived from Federal and market survey data. 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. Such evaluation will likely be conducted no sooner than 5 years after date of issuance of this tolerance. Further, as required by the FQPA, EPA will issue a Data Call-In under section 408(f) to all fipronil registrants for data on anticipated residues, to be submitted no later than 5 years from the date of issuance of this tolerance.

2. *From drinking water.* EPA does not have monitoring data available to perform a quantitative drinking water risk assessment for fipronil at this time. EPA estimated ground and surface water exposure using the Generic Expected Environmental Concentration (GENEEC) model, a screening level model for determining concentrations of pesticides in surface water. GENEEC uses the soil/water partition coefficient, hydrolysis half life, and maximum label rate to estimate surface water concentration. In addition, the model contains a number of conservative underlying assumptions. Therefore, the drinking water concentrations derived from GENEEC for surface water are likely to be overestimated. As fipronil is relatively immobile in soil, residues in groundwater are expected to be less than those in surface water.

i. *Acute exposure and risk.* The exposure estimate for surface water is 247 ppt (peak concentration). Based on an acute NOEL of 0.5 mg/kg/day and water consumption of 1 L/d for a 10 kg child, the worst-case estimates of residues in drinking water (247 ppt) result in a child exposure of 2.5×10^{-5} mg/kg/day. This exposure value corresponds to a MOE of 20,000 for the most highly exposed subgroup for acute exposure (children 1–6 years old). As this value exceeds 100, fipronil residues in surface drinking water do not pose an acute risk.

ii. *Chronic exposure and risk.* The exposure estimate for surface water is 48.8 ppt (54–day average). Based on a RfD of 0.0002 $(mg/kg/day)^{-1}$ and water consumption of 2 L/d for a 70 kg adult (male) and of 1 L/d for a 10 kg child (1–6 years old), the worst-case estimates of residues in drinking water (48.8 parts per trillion (ppt)) result in the following exposures: Adult exposure is 1.4×10^{-6} mg/kg/day and exposure for children is 4.9×10^{-6} mg/kg/day. These exposure values correspond to 0.7% of the RfD for adult males and 2.4% of the RfD for children (1–6 years old).

3. *From non-dietary exposure.* Fipronil is currently registered for use on the following residential non-food sites: ant and cockroach bait traps ranging from 0.01 to 0.05% active ingredient; and flea and tick control products for dogs and cats, including a pump spray (0.29% RTU (ready to use) and a 9.7% RTU spot treatment in which a premeasured small amount is applied between the pet's shoulder blades. The flea and tick spray use is expected to result in the highest exposure of fipronil products. Based on the high MOE's resulting from these uses (see below), the application of small amounts between the pet's shoulder blades was not addressed. This use is expected to result in much lower exposure based on lower duration and a considerably smaller area being treated. Exposure from the use of fipronil in self contained bait stations is also expected to result in lower exposures since there is no contact with the pesticide.

i. *Acute exposure and risk.* For incidental non-dietary (acute) exposures, the endpoint selected for acute dietary (oral) assessments is used. The NOEL is 0.5 mg/kg/day. The MOE for a child/hand-to-mouth exposure after petting a wet or recently treated pet is 5,000 to 8,000.

ii. *Chronic exposure and risk.* Fipronil is reportedly strongly bound to the skin and does not come off the dog once dry. Therefore, the use of fipronil products in residential situations is not expected to result in chronic exposures. It should be noted that an exposure study assessing exposures resulting from the pet uses will be submitted in the fall of 1997. The risk assessment may be refined at that time.

iii. *Short- and intermediate-term exposure and risk.* Label directions on pet care products state that applications of fipronil are expected to occur several times per year in residential settings, resulting in acute and short- and intermediate-term exposures. The endpoint selected for short and intermediate-term non-occupational exposure assessments is based on the results of a 21–day dermal toxicity study. The systemic toxicity NOEL is 5.0 mg/kg/day. The MOE for applicators of the 0.29% ready-to-use formulation on dogs and cats is 50,000. The MOE for a child/dermal contact with a wet or recently treated pet is 1,000 to 2,000.

iv. *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) 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 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.

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

1. *Acute risk.* For the most highly exposed subgroup (children 1–6 years old), the calculated MOE value is 160 (the reciprocal of the sum of the reciprocal food, residential and water MOEs). (The MOE is 167 for food, 5,000 for residential (oral) and 20,000 for water). This aggregate MOE does not exceed the HED's level of concern for acute dietary exposure.

2. *Chronic risk.* Based on the available data and assumptions for dietary/water/residential exposure and risk estimates, the population group estimated to be most highly exposed is children (1–6 years old) with a risk estimate from combined sources equaling 13.5% of the RfD (11.1% dietary + 2.4% water). As previously noted, no chronic residential exposure is anticipated. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fipronil residues.

3. *Short- and intermediate-term risk.* Short- and intermediate-term aggregate exposure should take into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. However, the short and intermediate term end points for fipronil are based on dermal exposure, and chronic endpoints are based on dietary exposure. The two exposure scenarios use different toxicological end points, and thus are not comparable in toxicological terms. At the present time, EPA does not know how to aggregate dermal and oral exposures for this chemical. For this reason, EPA has not developed a short and intermediate term risk assessment for fipronil. Further, as indicated above, when viewed independently, neither oral nor dermal exposure posed a risk of concern.

E. Aggregate Cancer Risk for U.S. Population

Based on the Cancer Peer Review Committee recommendation that the RfD approach be used to quantify carcinogenicity, a quantitative dietary cancer risk assessment was not performed. Dietary risk concerns due to long-term consumption of fipronil residues are adequately addressed by the chronic exposure analysis using the RfD.

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. 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. The developmental neurotoxicity study provided further information about the acute and chronic neurotoxic effects during prenatal and postnatal development.

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 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.

2. *FQPA considerations.* EPA has evaluated the chemical fipronil for FQPA considerations. The following discussion represents the information EPA considered.

i. *Developmental toxicity studies.* 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 by 40 CFR part 158.

ii. *Reproductive toxicity study.* An acceptable two-generation reproduction study in rats has been submitted to the Agency, meeting basic data requirements, as defined for a food-use chemical by 40 CFR part 158.

iii. Developmental neurotoxicity study. An acceptable developmental neurotoxicity study was conducted with fipronil and reviewed by the Agency.

iv. Pre- and post-natal sensitivity. There are no data gaps for the assessment of the effects of fipronil on developing animals following *in utero* and/or early postnatal exposure.

v. *Conclusion.* The available data contained evidence of increased sensitivity of rats to alterations in functional development following pre- and/or postnatal exposure with fipronil. Specifically, in a developmental neurotoxicity study in rats, the developmental and developmental-neurotoxicity NOEL of 0.5 ppm (0.05 mg/kg/day) was lower than the maternal toxicity NOEL of 10 ppm (0.9 mg/kg/day). In the offspring, decreased pup weights, increased time of preputial separation in males, and increased

motor activity counts in female pups were observed at the developmental LOEL of 10 ppm (0.9 mg/kg/day), while maternal toxicity (decreased body weight, body weight gain, and food consumption) was observed at the maternal LOEL of 200 ppm (15 mg/kg/day).

Previously conducted studies with fipronil did not identify any issues of increased sensitivity in the fetuses or pups following pre- and/or postnatal exposure. In the prenatal developmental toxicity study in rats, there was no evidence of developmental toxicity at the highest doses tested (20 mg/kg/day). Maternal toxicity (decreased body weight gain, food consumption and/or water consumption) was observed at this dose (20 mg/kg/day) with the maternal NOEL established at 4 mg/kg/day. In the prenatal developmental toxicity study in rabbits, there was also no evidence of developmental toxicity at the highest doses tested (1.0 mg/kg/day). Maternal toxicity (decreased body weight gain, food consumption and/or water consumption) was observed at this same dose (1.0 mg/kg/day) and lower, with the maternal NOEL established at < 0.1 mg/kg/day.

Additionally, in the two-generation reproduction study in rats, offspring toxicity was observed only in the presence of parental toxicity. The offspring NOEL was 30 ppm (2.54–2.74 mg/kg/day), based upon clinical signs of toxicity, decreased litter size, decreased body weights, decreased pre- and postnatal survival, and delays in physical development at the LOEL of 300 ppm (26.0–28.4 mg/kg/day). In the parental animals, reproductive toxicity (reductions in mating and fertility) was also observed at the 30 ppm dietary level. The systemic NOEL for the parental animals was 3 ppm (0.25–0.27 mg/kg/day), based upon increased weight of the thyroid gland and liver in both sexes, decreased weight of the pituitary gland in the females, and increased incidence of thyroid follicular epithelial hypertrophy in the females at the LOEL of 30 ppm.

In considering whether additional uncertainty factors were needed to protect children, EPA noted that the developmental neurotoxicity NOEL of 0.05 mg/kg/day, when adjusted for 1% dermal absorption, yields an equivalent NOEL of 5 mg/kg/day, the value established as the systemic NOEL in the 21–day dermal study in rabbits. This value was selected for use in the short term and intermediate risk assessment calculations for fipronil. The NOEL used for the RfD calculation was 0.019 mg/kg/day from the combined chronic toxicity-carcinogenicity study in the rat, a value that is even lower than the NOEL used for short- and intermediate-term exposure. Therefore, it was concluded that the risk assessment calculations as defined, will provide adequate protection for sensitive subpopulations, including infants and children. The Committee determined that the third uncertainty factor in the risk assessment of fipronil, under the provisions of the FQPA mandate to ensure the protection of infants and children, was not warranted for chronic or less than life time exposure and could be removed.

EPA believes that reliable data support using the hundredfold margin/factor, rather than the thousandfold margin/factor, when EPA has a complete data base under existing guidelines, and when the severity of the effect in infants or children, the potency or unusual toxic properties of a compound, or the quality of the exposure data do not raise concerns regarding the adequacy of the tenfold margin/factor.

For the reasons outlined above, EPA has determined there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to residues of fipronil following its use on field corn and other uses registered to date.

III. Other Considerations

A. Endocrine Disrupter Effects

EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inert ingredients) ''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.

B. Metabolism In Plants and Animals

EPA considers the nature of the residue in corn to be understood. Fipronil is metabolized by: (1) hydrolysis to the amide (RPA 200766) with further hydrolysis to the carboxylic acid (RPA 200761) or (2) oxidation to the sulfone MB 46136. The EPA Metabolism Committee has concluded that the residues of concern for the tolerance expression and dietary risk assessment in corn and animal RACs are fipronil, MB 46136, and MB 45950.

C. Analytical Enforcement Methodology

Analytical methodology suitable for the enforcement of the proposed tolerance is available. For corn RACs, the registrant has submitted a proposed analytical enforcement method which measures the parent and its metabolites (MB 45950, and MB 46136) in a single chromatographic separation using GC with ECD. The limit of quantitation (LOQ) for each compound is 0.01 ppm in grain and 0.02 ppm in forage and fodder. This method has undergone a successful Petition Method Validation (PMV).

For animal RACs, the registrant has submitted a proposed analytical enforcement method which measures the parent and its metabolites (MB 45950 and MB 46136) in a single chromatographic separation using GC with ECD. The LOQ of cattle, goat, horse and sheep for each compound is < 0.02 ppm. This method has also undergone a successful PMV.

D. Magnitude of Residues

As a result of this use, residues of fipronil are not expected to exceed the following levels:

corn, field, grain	0.02 ppm
corn, field, stover	0.30 ppm
corn, field, forage	0.15 ppm

Secondary residues in animal commodities from this proposed use on corn are not expected to exceed the following levels:

Eggs	0.03 ppm
Fat of cattle, goat, horse and sheep	0.40 ppm
Hog Fat	0.04 ppm
Hog Liver	0.02 ppm
Hog Meat Byproducts (except liver)	0.01 ppm
Hog Meat	0.01 ppm
Milk, fat (reflecting 0.05 ppm in whole milk)	1.50 ppm
Liver of cattle, goat, horse and sheep	0.10 ppm
Meat Byproducts (except liver) of cattle, goat, horse and sheep	0.04 ppm
Meat of cattle, goat, horse and sheep	0.04 ppm
Poultry Fat	0.05 ppm
Poultry Meat	0.02 ppm
Poultry Meat Byproducts	0.02 ppm

E. International Residue Limits

There are no CODEX, Canadian, or Mexican MRLs established for fipronil

in/on corn and animal RACs. Therefore, no compatibility problems exist.

F. Rotational Crop Restrictions

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

IV. Conclusion

Therefore, the tolerance is established for combined residues of the insecticide fipronil (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1*R,S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites MB 46136 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile) and MB 45950 (5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile) in or on the following items at the levels specified:

Commodity	Tolerances (in parts per million)
Corn, field, grain	0.02
Corn, field, stover	0.30
Corn, field, forage	0.15
Eggs	0.03
Fat of cattle, goat, horse and sheep	0.40
Hog fat	0.04
Hog liver	0.02
Hog meat byproducts (except liver)	0.01
Hog meat	0.01
Liver of cattle, goat, horse and sheep	0.10
Meat byproducts (except liver) of cattle, goat, horse and sheep	0.04
Meat of cattle, goat, horse and sheep	0.04
Milk, fat (reflecting 0.05 ppm in whole milk)	1.50
Poultry fat	0.05
Poultry meat	0.02
Poultry meat byproducts	0.02

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 January 26, 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(i). 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 Confidential Business Information (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 and Electronic Submissions

EPA has established a record for this rulemaking under docket control number [OPP–300587] (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 1132 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, 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 a tolerance 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 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. 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 Advocacy of the Small Business Administration.

VIII. 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, the Agency has 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: November 14, 1997.

Stephen L. Johnson,

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. 346a and 371.

2. By adding a new § 180.517 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-[(1*R,S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile and 5-amino-1-[2,6-dichloro-4-(trifluoromethyl) phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile in or on the following items at the levels specified:

Commodity	Parts per million
Corn, field, grain	0.02
Corn, field, stover	0.30
Corn, field, forage	0.15
Eggs	0.03
Fat of cattle, goat, horse and sheep	0.40
Hog Fat	0.04
Hog Liver	0.02
Hog Meat	0.01
Hog Meat Byproducts (except liver)	0.01
Liver of cattle, goat, horse and sheep.	0.10
Milk, fat (reflecting 0.05 ppm in whole milk).	1.50
Meat Byproducts (except liver) of cattle, goat, horse and sheep.	0.04
Meat of cattle, goat, horse and sheep.	0.04
Poultry Fat	0.05
Poultry Meat	0.02
Poultry Meat Byproducts	0.02

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

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

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

[FR Doc. 97–30949 Filed 11–25–97; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300569; FRL–5751–1]

RIN 2070–AB78

Tebufenozide; Pesticide Tolerances for Emergency Exemptions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a time-limited tolerance for residues of tebufenozide in or on sugarcane. This action is in response to EPA's granting of an emergency exemption under section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act authorizing use of the pesticide on sugarcane. This regulation establishes a maximum permissible level for residues of tebufenozide in this food commodity pursuant to section 408(l)(6) of the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996. The tolerance will expire and be revoked on December 31, 1998.

DATES: This regulation is effective November 26, 1997. Objections and requests for hearings must be received by EPA on or before January 26, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300569], 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–300569], 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. 1132, 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@epamail.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–300569]. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing request on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: David Deegan, 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: CM #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 308–9358, e-mail: deegan.dave@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA, on its own initiative, pursuant to section 408(e) and (l)(6) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) and (l)(6), is establishing