625.3 Transportation to GPL Facility by the USPS

For mailers who do not transport the mail as a drop-shipment, the Postal Service will transport the packages by truck to the GPL processing facility according to a schedule agreed to by the Postal Service and the customer. For mailers whose plants are located within 500 miles from a GPL facility, there will be no charge for the transportation. If the plant from which the GPL mailing originates is located more than 500 miles of a GPL processing facility the mailing is subject to a transportation charge (see 623.443).

Stanley F. Mires,

Chief Counsel, Legislative.

[FR Doc. 97–28524 Filed 10–28–97; 8:45 am]

BILLING CODE 7710–12–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 156

[OPP–250124; FRL–5753–2]

Flammability Labeling Requirements for Total Release Fogger Pesticides; Notification to the Secretary of Agriculture

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notification to the Secretary of Agriculture.

SUMMARY: Notice is given that the Administrator of EPA has forwarded to the Secretary of Agriculture a final regulation under section 25(a) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). The rule requires specific precautionary labeling relating to the flammability of total release fogger pesticides. This action is required by FIFRA section 25(a)(2).

FOR FURTHER INFORMATION CONTACT: By mail: Jim Downing, Labeling Team, Field and External Affairs Division (7506C), 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, 703–308–9071, e-mail: downing.jim@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: Section 25(a)(2) of FIFRA provides that the Administrator shall provide the Secretary of Agriculture with a copy of any final regulation at least 30 days before signing it for publication in the **Federal Register**. If the Secretary comments in writing regarding the final regulation within 15 days after receiving it, the Administrator shall issue for publication in the **Federal Register**, with the final regulation, the comments of the Secretary, if requested by the Secretary, and the response of the Administrator concerning the Secretary's comments. If the Secretary does not comment in writing within 15 days after receiving the final regulation, the Administrator may sign the regulation for publication in the **Federal Register** anytime thereafter. As required by FIFRA section 25(a)(3), a copy of the final regulation has been forwarded to the Committee on Agriculture of the House of Representatives and the Committee on Agriculture, Nutrition, and Forestry of the Senate.

List of Subjects in Part 156

Environmental protection, Labeling, Occupational safety and health, Pesticides and pests, Reporting and recordkeeping requirements.

Authority: 7 U.S.C. 136 *et seq.*

Dated: October 20, 1997.

Anne E. Lindsay,

Acting Director, Office of Pesticide Programs.

[FR Doc. 97–28654 Filed 10–28–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300565; FRL–5750–2]

RIN 2070–AB78

4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1H-pyrrole-3-carbonitrile; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in or on potatoes . The Ciba-Geigy Corporation submitted a petition to EPA under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170) requesting this tolerance.

DATES: This regulation is effective October 29, 1997. Objections and requests for hearings must be received by EPA on or before December 29, 1997.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300565], 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–300565], 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 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–300565]. 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: Mary L. Waller, 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) 308-9354, e-mail: waller.mary@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of February 5, 1997 (62 FR 5403) (FRL–5584–1), EPA, issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) announcing the filing of a pesticide petition (PP6F4694) for tolerance by the Ciba-Geigy Corporation, 410 Swing Road, Greensboro, NC 27401. This notice included a summary of the petition prepared by the Ciba-Geigy Corporation, 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 the fungicide, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile, in or on potatoes at 0.02 parts per million (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% 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, which could occur for residential uses of a pesticide, 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 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,'' 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 enactment of FQPA this risk 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 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile on potatoes at 0.02 ppm. EPA's assessment of the dietary exposures and risks associated with establishing the tolerance follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children. The nature of the toxic effects caused by 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile are discussed below.

1. A battery of acute toxicity studies placing technical fludioxonil in Toxicity Category III for eye irritation, Category IV for oral LD50, Category IV for inhalation LC50 and dermal irritation, and Category III for dermal LD50. 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile is a non-sensitizer.

2. A subchronic oral toxicity study in rats dosed orally with technical fludioxonil at levels of 0, 10, 100, 1,000, 7,000, and 20,000 ppm (0, 0.8, 6.6, 64, 428, and 1,283 mg/kg/day in males; 0, 1.0, 7.1, 70, 462, and 1,288 mg/kg/day in females) resulted in the Lowest Effect Level (LEL) of 428 mg/kg/day in males and 462 mg/kg/day in females, based on the increased incidence of microscopic pathology of the kidney and liver, and deceased body weight gain. The Noel is 64 mg/kg/day in males; 70 mg/kg/day in females.

3. A subchronic oral toxicity study in dogs administered doses of 0, 200, 2,000, and 15,000/10,000 ppm (15,000 ppm for 17 days and 10,000 ppm from day 18 until study termination) for 13 weeks with a LEL of 2,000 ppm in males and females, based on the observation of diarrhea at this dose level. These dose levels correspond to nominal doses of 0, 5, 50, or 375/250 mg/kg/day, as actual intake data were not provided. The NOEL is 5 mg/kg/day in males and females.

4. A subchronic oral toxicity study in mice administered doses of 0, 10, 100, 1,000, 3,000, or 7,000 ppm (0, 1.3, 13.9, 144, 445, or 1,052 mg/kg/day in males; 0, 1.9, 16.8, 178, 559, or 1,307 mg/kg/day in females) with a LEL of 1,052 mg/kg/day in males, and 1,307 mg/kg/day in females based on decreased body weight gain in female mice, changes in serum chemistry in male and female mice, observed increase in liver to body weight ratio, and the increased incidence of nephropathy and centrilobular hypertrophy of the liver in both sexes. The NOEL is 445 mg/kg/day in males and 559 mg/kg/day in females.

5. A dermal toxicity test in rats exposed as a repeated dermal dose under occlusive dressing 6 hrs/day, 5 days/week, for 4 weeks at 0, 40, 200, and 1,000 mg/kg/day. For dermal irritation, the LEL and NOEL are both greater than 1,000 mg/kg for males and females. The LEL for systemic toxicity for females is 1,000 mg/kg based on increased AST and adrenal weight, and 1,000 mg/kg for males based on increased creatinine and adrenal weight. The NOEL is 200 mg/kg/day for males and females.

6. A chronic oral toxicity study in dogs dosed for 52 weeks at 0, 100, 1,000, and 8,000 ppm in the diet (0, 3.1, 33.1, and 297.8 mg/kg/day in males; 3.3, 35.5, and 330.7 mg/kg/day in females. The LEL is 297.8 mg/kg/day for male dogs based on decreased body weight, hematology alterations (increase in platelets and fibrin), clinical chemistry alterations (increase in cholesterol and alkaline phosphatase) and increased liver weight. The LEL is 35.5 mg/kg/day for female dogs based on a marked decrease in body weight gain for weeks 1 - 13 and weeks 1 - 52 of the study. The NOEL is 33.1 mg/kg/day for male dogs and 3.3 mg/kg/day in female dogs.

7. A combined chronic toxicity/carcinogenicity study in rats fed 0, 10, 30, 100, 1,000 and 3,000 ppm for either 12 or 24 months (males: 0, 0.37, 1.1, 3.7, 37 and 113 mg/kg/day, respectively; females: 0, 0.44, 1.3, 4.4, 44 and 141 mg/kg/day respectively). The 3,000 ppm dose level is considered adequate for carcinogenicity testing, based on decreased body weight and body weight gain in both sexes, slight anemia in females at 12 months, and an increased incidence and severity of liver histopathology changes in both sexes. Rats from the control and 3,000 ppm groups were fed the test diets for 12 months and then allowed to recover for one month prior to sacrifice. There was no treatment-related effect on food or water consumption. Males dosed at 1,000 and 3,000 ppm, and females dosed at 3,000 ppm exhibited a number of effects including higher incidence of dark stool and urine, staining (mostly blue) around the pelvic region and abdomen, higher frequency of diarrhea (males only), and decrease body weight gain. Females dosed at 3,000 ppm had some evidence of slight anemia at the 12-month evaluation. At necropsy,

males at the 3,000 ppm dose level exhibited an increased incidence of enlarged livers, and kidneys with discolored foci or general discoloration and an increased severity of progressive nephropathy; kidneys with cysts were reported at both the 1,000 and 3,000 ppm dose levels. For females in the 1,000 and 3,000 ppm dose levels there was an increase incidence of general discoloration of the the kidneys. Males and females in the 3,000 ppm group had an increased incidence and more severe grade of histopathological changes in the liver. There was an increase incidence of hepatocellular tumors in both sexes of the 3,000 ppm group, however the increase in males was not statistically significant. The statistically significant finding in females was an increase in combined adenomas and carcinomas (0/70, 1/60, 0/60, 1/60, 2/60 and 5/70 in the 0, 10, 30, 100, 1,000 and 3,000 ppm groups, respectively). Males and females in the 3,000 ppm group had an increased incidence of basophilic foci in the liver; males also had an increase in hepatocellular hypertrophy. The LEL for males and females was 113 and 141 mg/kg/day, respectively (3,000 ppm) based on decreased body weight and weight gain, slight anemia in females at 12 months, and increased incidence and severity of histopathology changes in the liver. The NOEL for males and females was 37 and 44 mg/kg/day, respectively. 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile technical was not carcinogenic in male rats. There was a statistically significant increase in the incidence of combined adenomas and adenocarcinomas of the liver in female rats in the 3,000 ppm dosed group.

8. A carcinogenicity study in mice administered in the diet nominal dose levels at 0, 10, 100, 1,000, and 3,000 ppm (0, 1.1, 11.3, 112, and 360 mg/kg/day for male mice; 0, 1.4, 13.5, 133, and 417 mg/kg/day for female mice). Male mice at the 3,000 ppm level exhibited clinical toxicity in the form of an incidence of male mice which ''convulsed'' when handled. No significant effects on body weight, weight gain, food consumption, hematology, or microscopic non-neoplastic pathology was reported in either sex. Increased liver weight (9%) and spleen weight (34%) were observed in male mice at the 3,000 ppm dose level, which correlated with the macroscopic observations of enlarged spleen and raised foci of their liver. Female mice showed a statistically significant increase in liver weight at the 3,000 ppm dose level, and this is also supported by the macroscopic observation of enlarged liver at the 3,000 ppm dose level in female mice. Other macroscopic changes in female mice were an increased incidence of enlarged thymus, spleen, mediastinal lymph node, and liver, and an increased incidence of lymphoma in these organs. The LEL is 112 mg/kg/day for male mice, based on the increased incidence of clinical toxicity in male mice (specifically, the increased incidence of mice convulsing when handled), and 417 mg/kg/day for female mice, based on the increase in liver weight of female mice, and the increase in incidence of macroscopic pathology. The NOEL is 11.3 mg/kg/day and 133 mg/kg/day in male and female mice, respectively. There was evidence of carcinogenicity in female mice based on an increase incidence of lymphoas, which contributed to death. This effect was due to the early onset and high incidence of lymphoma at the 3,000 ppm dose relative to the control group. Total incidence of lymphoma was reported as 11/59, 10/59, 13/60, 12/60, and 18/60 for the 0, 10, 100, 1,000, and 3,000 ppm dose levels in female mice. This increase in total lymphoma was significant by a trend test, but not by pair wise comparison. Whether an adequate dose level was used in this study to assess the carcinogic potential of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile is complicated by the observation of an increased lymphoma incidence at the 3,000 ppm dose level. This dose level produced some systemic effects, such as an increased incidence of male mice which 'convulsed' when handle and macroscopic pathology in both sexes. But this dose level produced no significant effects on body weight, weight gain, food consumption, hematology, or microscopic non-neoplastic pathology in either sex.

In a second carcinogenicity study in mice, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile technical was administered in the diet at nominal dose levels of 0, 3, 30, 5,000, and 7,000 ppm (0, 0.33, 3.3, 590, and 851 mg/kg/day for male mice; 0, 0.41, 4.1, 715, and 1,008 mg/kg/day for female mice). In male and female mice, the 7,000 ppm dose level produced significant systemic effects in addition to significant nephropathy. The nephropathy in both sexes of mice dosed at 7,000 ppm contributed to death in a majority of the mice. Survival in female mice was below 25%, and exceeded the guideline criteria for survival in a mouse carcinogenicity study. Changes in liver weights were observed in both sexes at the 5,000 and 7,000 ppm dose levels, but could not be related to histological alterations in the liver. Therefore the LEL is 851 mg/kg/day in males, and 1,008 mg/kg/day in females. The NOEL is 590 mg/kg/day in males, and 715 mg/kg/day in females. The 7,000 ppm dose is adequate for testing carcinogenic potential in male mice, based on the significant systemic effects and nephropathy observed at this dose. For female mice, the 7,000 ppm dose level is considered excessive, based on the reduction in survival of the test animals. There was no evidence of increased incidence of tumors in this study for male or female mice.

9. A developmental toxicity study in rats administered doses of 0, 10, 100, and 1,000 mg/kg/day by oral gavage in 0.5% carboxymethylcellulose to pregnant female rats on gestation days 6 - 15 inclusive. Maternal Toxicity was evident at 1,000 mg/kg/day, with a 16% reduction in corrected body weight gain. Developmental Toxicity was evident at the 1,000 mg/kg/day dose level with increased fetal and litter incidence of dilated renal pelvis and dilated ureter. Based on these observations, the Maternal LEL is 1,000 mg/kg/day and the Maternal NOEL is 100 mg/kg/day. The Developmental Toxicity LEL is 1,000 mg/kg/day, and the Developmental toxicity NOEL is 100 mg/kg/day.

10. A developmental toxicity (teratology) study in rabbits dosed at 0, 10, 100, and 300 mg/kg/day in a 0.5% methylcellulose solution in distilled water by oral gavage from gestation days 6 through 18, inclusive. Maternal toxicity as less body weigh was noted in the mid and high dose groups during the dosing period (gestation days 6 through 18), for the overall dosing plus post dosing periods (gestation days 6 through 28), and for the entire gestation period; maternal toxicity as decreased corrected body weight gains was observed for the dosing plus post dosing periods. The high dose group consumed less food than the control group during the dosing period (gestation days 6 - 18), the post dosing period (gestation days 19 -28), the dosing plus post dosing period (gestation days 19 - 28), and for the overall gestation period. However, food efficiency was decreased in the mid and high dosed groups during the dosing plus post dosing periods, and for the entire gestation period. The Maternal Toxicity LEL is 100 mg/kg/day, and the Maternal Toxicity NOEL is 10 mg/kg/day based on decreased body weight gains and decreased food efficiency. No developmental toxicity was noted at the dose levels tested. The Developmental Toxicity LEL is greater than 300 mg/kg/day, and the

Developmental Toxicity NOEL is equal to or greater than 300 mg/kg/day.

11. A reproduction toxicity study in rats receiving 0, 30, 300, and 3,000 ppm (equivalent to 0, 2.19, 22.13, and 221.61 mg/kg/day for males, and 0, 2.45, 24.24, and 249.67 mg/kg/day for females) fludioxonil technical in the diet for 2 generations. The Parental Systemic Toxicity LEL is 221.61 mg/kg/day for males, and 249.67 mg/kg/day for females. The Parental Systemic Toxicity NOEL is 22.13 mg/kg/day for males, and 24.24 mg/kg/day for females based on clinical observations, reduced body weight and body weight gains, and reduced food consumption. Treatment related effects are noted in the high dose groups in both the F1 and F2 pups as reduced mean pup body weights starting at postnatal day 4 through 21; this was considered a developmental toxic effect rather than a true reproductive toxic effect , because the reduced mean pup body weights are an effect on the growth of the pup. The Reproductive/Developmental Toxicity LEL is 221.61 mg/kg/day for males, and 249.67 mg/kg/day for females. The Reproductive/Developmental Toxicity NOEL is 22.13 mg/kg/day for males, and 24.24 mg/kg/day for females based on reduced pup body weights.

12. Studies on gene mutation and other genotoxic effects: an Ames Salmonella Assay which provided evidence of cytotoxicity at 1,250 µg/plate and 5,000 µg/plate concentrations; an Unscheduled DNA Synthesis Assay with apparent cytotoxicity at 313 µg/ml; an In Vitro Chromosome Aberrations assay in Chinese hamster ovary (CHO) cells, with and without S9-activation which provided convincing evidence that technical fludioxonil is a clastogen, and a potent inducer of polyploidy in this cultured mammalian cell assay; an In Vitro Chromosome Aberrations assay in Chinese hamster bone marrow cells with the occurance of hyperploidy in one mid-dose female and trisomy in one high dose male; an In Vivo Micronucleus Assay using rat hepatocytes, no definitive conclusions were made, and this study should be repeated; A Dominant Lethal Assay in mice with no indication the test material induced dominant lethal mutations in male mouse germinal cells over the entire period of spermatogenesis; a Point Mutation Test in CHO cells in vitro, with and without S9-activation, with no increase in the number of thioguanine-resistant colonies, mutation frequency, or mutation factor with or without S9-activation; and a Mouse Micronucleus Assay in a mouse bone morrow micronucleus test which was negative.

B. Toxicological Endpoints

1. *Acute toxicity*. There is no concern for an acute dietary risk. The the available data do not indicate any evidence of significant toxicity from one day or single event exposure by oral exposure.

2. *Chronic toxicity*. EPA has established the RfD for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile at 0.03 mg/kg/day. This RfD is based upon the 1-year toxicity study in dogs with a NOEL of 3.3 mg/kg/day in female dogs, and an uncertainty factor of 100 to account for both interspecies extrapolation and intraspecies variability.

3. *Carcinogenicity*. This chemical has been classified as a Group D - not classifiable as to Human Carcinogenicity. That is, the evidence is inadequate and cannot be interpreted as showing either the presence or absence of a carcinogenic effect. The Group D classification was also based on the increase in liver tumors in female rats that was statistically significant for combined adenoma/carcinoma only, the lack of a tumorigenic response in male rats or in either sex of the mouse, and the need for additional mutagenicity studies.The mutagenicity studies will be required as a Condition of Registration for products containing 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile, and consists of a repeat of the in vivo rat hepatocyte study with a primary interest in determining the mechanism (s) for inducing genetic damage and a repeat of the bone marrow micronucleus assay using lower doses.

C. Exposures and Risks

1. *From food and feed uses.* This is the first tolerance for residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile, in or on a raw agricultural commodity. Risk assessments were conducted by EPA to assess dietary exposures and risks from 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile as follows:

Chronic exposure and risk. The RfD used for the chronic dietary analysis is 0.03 mg/kg/day. A tolerance of 0.02 ppm in/on potatoes was used. Tolerances in animal commodities or in potato granules/flakes are not required for this seed piece use on potatoes. 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile is currently registered for use as a seed treatment on corn and sorghum, and for use in greenhouses on nonfood crops. Since the residues were non-quantifiable, no exposure was assumed to result for the registered use on corn or sorghum, and these uses did not require tolerances. Using the tolerance level residue (0.02 ppm) and assuming that 100% of the crop is treated, the risk assessment resulted in use of less then 1% of the RfD for the general population and all 22 subgroups, including infants under 1 year old and children under 13 years of age.

2. *From drinking water.* Because of the requested and currently registered use patterns, including the treatment of potato seed pieces at a low use rate (approximately 0.06 lbs ai/A), seed treatment of field, sweet and popcorn, and sorghum, and ornamental plants grown in greenhouses or other enclosed structures, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile is not expected to impact ground or surface waters. Thus the likelihood of residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in drinking water is considered negligible from the above mentioned use patterns. Therefore, it is concluded that a drinking water risk assessment is not required at this time, and there is no drinking water risk assessment to aggregate with the chronic dietary (food sources) risk assessment. The aggregate dietary risk is therefore the dietary risk which is less than 1% for the general population and all 22 subgroups.

Acute exposure and risk. There is no concern for an acute dietary exposure to fludioxonil from drinking water as stated above, and because the available data do not indicate any evidence of significant toxicity from a one day or single event exposure by the oral route. Therefore, an acute exposure risk assessment is not required for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile at this time.

3. *From non-occupational non-dietary exposure.* 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile is currently not registered for use on residential non-food sites, therefore no non-occupational non-dietary exposure is expected.

4. *Cumulative exposure to substances with common mechanism of toxicity.* Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' 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 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile 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, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile 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 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile has a common mechanism of toxicity with other substances.

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

Chronic risk. Using the Theoretical Maximum Residue Contribution (TMRC) exposure assumptions described above, EPA has concluded that aggregate exposure to 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile from food will utilize less then 1% of the RfD for the U.S. population and the 22 subgroups, including infants and children. 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 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile residues.

E. Aggregate Cancer Risk for U.S. Population

This chemical has been classified as Group D - not classifiable as to Human Carcinogenicity. The available carcinogenicity studies in the rat and mouse shows some increase in the combined tumors only in the female rat above that in the concurrent controls. However, this statistical increase in liver tumors in female rats was only at the high dose. Some of this significant increase was due to the lack of any liver tumors in the concurrent control whereas the historical control from the same lab indicated a range of 1.4 to 15% for combined liver tumors. Therefore based on available information, a carcinogenic risk analysis is not appropriate. EPA believes that this pesticide does not pose a significant cancer risk.

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

1. *Safety factor for infants and children— In general.* In assessing the potential for additional sensitivity of infants and children to residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction 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.

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. The toxicity database for fludioxonil includes as acceptable two-generation reproduction study in rats and an acceptable prenatal developmental toxicity studies in rats and rabbits. The data did not suggest any additional sensitivity to the embryo or neonate following in utero or early postnatal exposure to fludioxonil. The maternal NOEL, and the developmental (fetal and pup) Toxicity NOEL were both 100 mg/kg/day in the rat developmental study. In the rabbit developmental study, the maternal NOEL was 10 mg/kg/day. No developmental toxicity was noted at any dosing level. The developmental NOEL was set equal to or greater than 300 mg/kg/day, the highest dose tested. Results from the 2-generation reproduction study for rats indicated a developmental/reproduction NOEL of 22.13 mg/kg/day for males and 24.24 mg/kg/day for females. The developmental/reproductive NOEL is at least 600 fold higher then the RfD (0.03 mg/kg/day), and should be protective for infants and children; no additional safety factors are warrented.

2. *Chronic risk.* Using the conservative exposure assumptions described above, EPA has concluded that aggregate exposure to 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile from food will utilize less then 1% of the RfD for infants and children. 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 to infants and children from aggregate exposure to 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile residues.

III. Other Considerations

A. Metabolism In Plants and Animals

The metabolism in plants is adequately understood for this potato seed piece treatment use. The residue of regulatory concern is the parent compound only, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile. Since it has been determined that secondary residues in livestock commodities are not likely to

result from this use, metabolism of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in animals is not relevent to this requested use on potato seed treatment.

B. Analytical Enforcement Methodology

The method accepted by EPA for enforcement of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in plants is Ciba-Geigy's Method AG-597B. A method, Ciba-Geigy's Method AG-616B (MRID#s 4360412 - 4360415), is also available for quantifying residues in meat and milk. These methods are available from the Docket under docket control number [OPP–300565] at the address stated above.

C. Magnitude of Residues

The submitted residue data indicate that residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile were below the level of quantitation (LOQ), <0.01 ppm, in/on immature and mature potato tubers grown from seed pieces treated with 0.5% Dust formulation of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile at 1.75 or 2.5 g ai/100 kg seed pieces (0.7X or 1X the labeled rate, respectively). Residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in/on immature and mature tubers treated at a 2X application rate ranged from less then 0.01 ppm to 0.04 ppm. Harvest times varied from 45 to 143 days after planting treated seed pieces. Residue data was also submitted at 6X and 10X the label application rate, with reported residues ranging <0.01 - 0.06 ppm and <0.01 - 0.09 ppm at the 6X rate for immature and mature tubers, respectively; and <0.01 - 0.48 ppm amd <0.01 - 0.06 ppm at the 10X rate for immature and mature tubers, respectively. Based on the submitted residue data, the requested tolerance of 0.02 ppm is adequate for this potato seed piece use. Potato processing studies were also submitted to determine whether concentration of residues occur in potato chips, granules, and wet peels and trimmings from potatoes grown from treated potato seed pieces. Based on the submitted processing studies, concentration of the pesticide chemical residues of 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in the processed foods is not expected to be greater than the tolerance of 0.02 ppm requested and prescribed in this **Federal Register** document for the pesticide chemical residue in the raw agricultural commodity, potatoes. Therefore, the tolerance of 0.02 ppm prescribed for potatoes will also cover the residues of fludioxonil up to 0.02 ppm resulting in potato processed products from this seed piece use.

D. International Residue Limits

There are currently no CODEX, Canadian, or Mexican listings for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile residues, therefore there are no harmonization issues for this action.

E. Rotational Crop Restrictions

The submitted confined rotation studies provided adequate results to conclude that a 30-day plantback interval is sufficient for all crops.

IV. Conclusion

Therefore, the tolerance is established for 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile in or on potatoes at 0.02 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 December 29, 1997, 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 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 Docket

EPA has established a record for this rulemaking under docket control number [OPP–300565] (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 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 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 for 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: October 10, 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. Section 180.516 is added to read as follows:

§ 180.516 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1H-pyrrole-3-carbonitrile ; tolerances for residues.

(a) *General.* A tolerance is established for residues of the herbicide, 4-(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile, in or on the following food commodity:

Commodity	Parts per million
Potatoes	0.02

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

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

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

[FR Doc. 97–28644 Filed 10–28–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300567; FRL–5750–8]

RIN 2070–AB78

Avermectin; Pesticide Tolerances for Emergency Exemptions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a time-limited tolerance for the combined residues of avermectin in or on basil. This action is in response to an emergency exemption request under section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act permitting use of the pesticide on basil. This regulation establishes a maximum permissible level for residues of avermectin B_1 and its delta-8,9-isomer 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 is revoked on September 30, 1998.

DATES: This regulation is effective October 29, 1997. Objections and requests for hearings must be received by EPA on or before December 29, 1997.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300567], 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–300567], 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–300567]. 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: Daniel Rosenblatt, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone