under 5 U.S.C. 605(b) that this regulation will not have a significant economic impact on a substantial number of small entities. The EEOC's debt collection activities do not affect a substantial number of small entities. Moreover, as found by the Department of the Treasury, wage garnishment requirements do not have a significant economic impact on small entities. Employers of delinquent debtors must certify certain information about the debtor, such as the debtor's employment status and earnings. This information is contained in the employer's payroll records. Therefore, it will not take a significant amount of time or result in a significant cost for an employer to complete the certification form. Even if an employer is served withholding orders on several employees over the course of a year, the cost imposed on the employer to complete the certification would not have a significant economic impact on that entity. Employers are not required to vary their normal pay cycles in order to comply with a withholding order issued pursuant to this rule. For these reasons, a regulatory flexibility analysis is not required.

Executive Order 12866

This rule is not a significant regulatory action as defined in Executive Order 12866 and is therefore not subject to review by the Office of Management and Budget.

Paperwork Reduction Act

This regulation contains no information collection requirements subject to review by the Office of Management and Budget under the Paperwork Reduction Act of 1980 (44 U.S.C. chapter 35).

List of Subjects in 29 CFR Part 1650

Administrative practice and procedure, Claims, Debts, Garnishment of wages, Hearing and appeal procedures, Salaries, Wages.

For the reasons stated in the preamble, 29 CFR Part 1650 is amended as set forth below.

PART 1650—DEBT COLLECTION

1. The authority citation for 29 CFR Part 1650 is revised to read as follows:

Authority: 5 U.S.C. 5514; 31 U.S.C. 321, 3701, 3711, 3716, 3720A, 3720D; EO 13019, 61 FR 51763, 3 CFR 1996 Comp., p. 216; 5 CFR 550.1101.

2–3. Section 1650.101 is amended by adding two new sentences at the end of the section to read as follows:

§ 1650.101 Purpose.

* * * The general standards and procedures governing the collection, compromise, termination, and referral to the Department of Justice of claims for money and property that are prescribed in the regulations issued jointly by the General Accounting Office and the Department of Justice pursuant to the Federal Claims Collection Act of 1966 (4 CFR Parts 101–105) apply to the administrative collection activities of the EEOC. The Director of the Financial Management Division shall act on all claims arising out of the activities of the EEOC.

4. Section 1650.201 is amended by adding two new sentences at the end of the section to read as follows:

§ 1650.201 Purpose.

* * * The general standards and procedures governing the collection, compromise, termination, and referral to the Department of Justice of claims for money and property that are prescribed in the regulations issued jointly by the General Accounting Office and the Department of Justice pursuant to the Federal Claims Collection Act of 1966 (4 CFR Parts 101–105) apply to the administrative collection activities of the EEOC. The Director of the Financial Management Division shall act on all claims arising out of the activities of the EEOC.

5. Section 1650.301 is amended by adding two new sentences at the end of the section to read as follows:

§ 1650.301 Purpose.

* * * The general standards and procedures governing the collection, compromise, termination, and referral to the Department of Justice of claims for money and property that are prescribed in the regulations issued jointly by the General Accounting Office and the Department of Justice pursuant to the Federal Claims Collection Act of 1966 (4 CFR Part 101–105) apply to the administrative collection activities of the EEOC. The Director of the Financial Management Division shall act on all claims arising out of the activities of the EEOC.

6. A new Subpart D is added to 29 CFR Part 1650 to read as follows:

Subpart D—Procedures for the Collection of Debts by Administrative Wage Garnishment

§ 1650.401 Purpose and regulatory procedures for the collection of debts by administrative wage garnishment.

The Commission hereby adopts by cross-reference the administrative wage garnishment regulation issued by the Department of the Treasury at 31 CFR 285.11. The general standards and procedures governing the collection, compromise, termination, and referral to the Department of Justice of claims for money and property that are prescribed in the regulations issued jointly by the General Accounting Office and the Department of Justice pursuant to the Federal Claims Collection Act of 1966 (4 CFR Parts 101–105) apply to the administrative collection activities of the EEOC. The Director of the Financial Management Division shall act on all claims arising out of the activities of the EEOC.

Dated: May 20, 1999.

For the Commission.

Ida L. Castro,

Chairwoman.

[FR Doc. 99–13342 Filed 5–27–99; 8:45 am]

BILLING CODE 6570–01–M

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300866; FRL–6082–7]

RIN 2070–AB78

Fenhexamid; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for fenhexamid (*N*-2,3-dichloro-4-hydroxyphenyl)-1-methyl cyclohexanecarboxamide) in or on grapes at 4.0 parts per million (ppm), strawberries at 3.0 ppm, and raisins at 6.0 ppm. The TM–402 Fungicide Task Force comprised of Tomen Agro, Inc. and Bayer Corporation requested these tolerances under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996.

DATES: This regulation is effective May 28, 1999. Objections and requests for hearings must be received by EPA on or before July 27, 1999.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300866], 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–300866], must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, Crystal Mall 2 (CM #2), 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may be submitted electronically by sending electronic mail (e-mail) to: opp-docket@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–300866]. 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, Product Manager 21, 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: Rm. 249, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 308–9354, waller.mary@epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of November 20, 1998 (63 FR 64498) (FRL–6042–1), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Pub. L. 104–170) announcing the filing of a pesticide petition (PP 7F4890) for tolerances by the TM–402 Fungicide Task Force comprised of Tomen Agro, Inc. and Bayer Corporation. The notice included a summary of the petition prepared by the TM–402 Fungicide Task Force. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR part 180 be amended by establishing tolerances for the fungicide, fenhexamid in or on grapes at 4.0 ppm, strawberries at 3.0 ppm, and raisins at 6.0 ppm.

I. Background and Statutory Findings

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

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

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 fenhexamid and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances in or grapes at 4.0 ppm, strawberries at 3.0 ppm, and raisins at 6.0 ppm. EPA's assessment of the dietary exposures and risks associated with establishing the tolerances 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 fenhexamid are discussed in this unit.

1. *Acute toxicity*—i. The acute oral LD_{50} and acute dermal LD_{50} for rats was > 5,000 milligrams/kilograms (mg/kg) for both sexes. The acute LC_{50} for rats was > 5.06 mg/liters (L) for both sexes. Fenhexamid was not an eye or skin irritant and was not a dermal sensitizer.

ii. In an acute neurotoxicity study, rats were gavaged with a single oral dose of fenhexamid at dose levels of 0, 200, 630, or 2,000 mg/kg. The rats were observed for 14 days. Functional Observational Battery and motor activity testing were performed 7 days prior to dosing, approximately 20 minutes to 3 hours post-dosing, and on days 7 and 14. The no observed adverse effect level (NOAEL) in males was 630 mg/kg. The NOAEL in females was 2,000 mg/kg. The lowest observed adverse effect level (LOAEL) in males was 2,000 mg/kg based on a marginally decreased mean body temperature (the only treatment-related effect noted in the study). The LOAEL in females was not established.

2. *Subchronic toxicity*—i. In an inhalation toxicity range-finding study, 10 rats/sex/dose were exposed (head/nose only) to fenhexamid at concentrations of 0, 11.8, 97.7 or 1,092.6 mg/m^3 in air for 6 hours per day for 5 days. One-half of the rats were sacrificed 7 days after the first exposure and the other one-half were sacrificed 21 days after the first exposure. The NOAEL was 0.098 mg/L and the LOAEL was 1.092 mg/L based on the observations of macroscopic grey coloration of the lungs and marginally increased lung weights.

ii. In a 21–day dermal toxicity study, fenhexamid was applied to the shaved skin of 5 male and female rabbits at a dose level of 1,000 mg/kg/day for 17 days over a 3–week period. There were no compound related effects. The NOAEL was 1,000 mg/kg/day and the LOAEL was > 1,000 mg/kg/day for both systemic and local effects on the skin.

iii. In a 28–day oral toxicity range finding study, 10 rats/sex/dose were gavaged at dose levels of 0, 100, 300, or 1,000 mg/kg/day for 28 days. There were no compound-related effects in mortality, clinical signs, body weight, food consumption, hematology, clinical chemistry, organ weights, or gross and histologic pathology. The NOAEL was 1,000 mg/kg/day.

iv. In a 90–day oral toxicity study, 10 rats/sex/dose were fed fenhexamid at dose levels of 0, 2,500, 5,000, 10,000 or 20,000 ppm (0, 202, 415, 904, and 1,904 mg/kg/day for males and 0, 270, 549, 1,132, and 2,824 mg/kg/day for females). No treatment-related changes were seen in clinical signs, mortality, opthalmoscopic examinations, hematology, urinalyses, or gross pathology. The NOAEL was 5,000 ppm in males and 10,000 ppm in females.

The LOAEL in males was 10,000 ppm based on decreased terminal body weights and body weight gains, increased food consumption, decreased food efficiency and increased Alanin amino-transferase (ALAT) levels. The LOAEL in females was 20,000 ppm based on increased food consumption, decreased food efficiency, decreased liver weights, and liver histopathology (Kupffer cell proliferation and altered hepatocyte morphology).

v. In a 90–day oral toxicity study, 4 dogs/sex were fed fenhexamid at dose levels of 0, 1,000, 7,000 or 50,000 ppm (0, 33.9, 239.1, or 1,747.7 mg/kg/day for males and 0, 37, 261, or 1,866.2 mg/kg/day for females). The NOAEL in males and females was 1,000 ppm. The LOAEL in males and females was 7,000 ppm based on significant increases in Heinz bodies in males and females and increased absolute and relative liver weights in females.

vi. In a 90–day oral toxicity study, 10 mice/sex/dose were fed fenhexamid at dose levels of 0, 100, 1,000 or 10,000 ppm (0, 26.5, 266.5 or 3,283.5 mg/kg/day in males and 0, 51.6, 453.9, or 5,151.1 mg/kg/day in females) for 14 weeks. The NOAEL in males and females was 1,000 ppm. The LOAEL in males and females was 10,000 ppm based on the observation in both sexes of: increased serum cholesterol, bilirubin and creatinine, decreased kidney weights, increased water consumption, increased food consumption (males), decreased food efficiency (males), renal cortical tubular basophilia (both sexes), renal protein casts and cellular detritus (males), and marginal alterations of liver function (increased serum cholesterol, bilirubin, decreased Aspartate amino-transferase (ASAT), ALAT), marginal increase in liver weights and reduced glycogen content of hepatocytes (males).

vii. In a 56–day oral toxicity study, 10 rats/sex/dose were fed fenhexamid at dose levels of 0, 1,000, 5,000, 10,000, 15,000, or 20,000 ppm (0, 57.5, 284.7, 575.7, 943.8, or 1,217.1 mg/kg/day for males and 0, 78, 407.1, 896.5, 1,492.5, or 1,896.7 mg/kg/day for females). At 20,000 ppm, rats had fenhexamid plasma levels below the level of detection. Urine samples showed measurable excretion of conjugated fenhexamid indicating intestinal absorption in the dose range examined. Males had a maximum excretion rate at 15,000 ppm indicating a saturation of intestinal absorption between 15,000 and 20,000 ppm. Urine excretion in females was somewhat lower than in males, at concentrations of 10,000 ppm and above. The highest value was determined at 20,000 ppm suggesting that saturation in intestinal absorption was not achieved with this dose level in females.

3. *Developmental toxicity*—i. In a developmental toxicity study, 30 rats/dose were gavaged at dose levels of 0 and 1,000 (1,044 determined analytically) mg/kg/day from days 6 through 15 of gestation. At 1,000 mg/kg/day, there were no treatment-related effects on maternal mortality, clinical signs, cesarean parameters, or gross pathology. No treatment-related effects were noted in any embryo/fetal parameters. Under the conditions of the study, fenhexamid was not embryotoxic, fetotoxic, or teratogenic at a dose of 1,044 mg/kg/day. The NOAEL for maternal toxicity was < 1,044 mg/kg/day. The developmental NOAEL was 1,044 mg/kg/day. The LOAEL for maternal toxicity was 1,044 mg/kg/day based on the decreased body weight gain (–12% of controls) during gestation days 6–16 and a decrease in food consumption (10% of controls) during gestation days 6–11.

ii. In a developmental toxicity study, 16 rabbits were gavaged with fenhexamid at dose levels of 0, 100, 300, or 1,000 mg/kg/day from days 6 through 18 of gestation. No treatment-related effects were seen on mortality, general appearance or behavior. The NOAEL for maternal toxicity was 100 mg/kg/day. The LOAEL for maternal toxicity was 300 mg/kg/day based on observations at this dose and above of alterations of excretory products (discolored urine, small scybala), decreased body weight gain and feed consumption (mainly during the first week of the treatment period) and decreased placental weights. One abortion at 300 mg/kg/day and one abortion and two total litter resorptions at 1,000 mg/kg/day were not considered to be treatment-related because the incidences fell within the ranges of historical control data submitted with the study. Reduced and/or light feces were also noted at 1,000 mg/kg/day. Pale livers were noted in the 2 dams that aborted. The NOAEL for developmental toxicity was 300 mg/kg/day. The LOAEL for developmental toxicity was 1,000 mg/kg/day based on marginally decreased male fetal body weights and evidence of delayed ossification. Fenhexamid did not induce any treatment-related fetal malformations or deviations at any of the doses tested under the conditions of this study. All effects on intrauterine development were correlated with maternal toxicity and, therefore, no primary developmental effect was evident. Fenhexamid was not teratogenic up to and including 1,000 mg/kg/day.

4. *Reproductive toxicity*. In 2–generation reproduction study, 30 rats/sex/dose were fed fenhexamid at dose levels of 0, 100, 500, 5,000 or 20,000 ppm (0, 7.6, 38.2, 406, or 1,814 for males and 0, 9.0, 44.8, 477, or 2,043 mg/kg/day for females determined for the 10–week premating period). There were no compound-related effects on mortality, clinical signs, behavior or reproductive parameters for adult animals. The NOAEL for reproductive toxicity was 20,000 ppm.

The neonatal NOAEL was 500 ppm and the neonatal LOAEL was 5,000 ppm based on significantly decreased pup body weights on lactation days 14 and 21 for the F_1 (6–11% < controls) and on lactation days 7, 14, and 21 for F_2 pups (9–11% < controls). At 20,000 ppm, significantly decreased pup body weights were observed on lactation days 7, 14, and 21 for F_1 pups (15–30% < controls) and for F_2 pups (11–19% < controls). Treatment-related decreased pup body weights were not observed at birth or on lactation day 4. An additional effect observed at 20,000 ppm was an increase in the number of pups among the post-weaning F_1 pups selected to be F_1 parents which died viz. 0/66, 2/68, 0/68, 0/68 and 10/78 for the control, 100, 500, 5,000, and 20,000 ppm dose groups, respectively. This effect was attributed to the small size of the pups at weaning (30% < controls).

The parental NOAEL was 500 ppm and the parental LOAEL in males was 5,000 ppm based on increased creatinine levels in P-generation (but not F_1 generation) males at premating (20%, $p<0.05$) and at termination (20%, not significant); slightly increased alkaline phosphatase levels in P-generation and F_1-generation males at premating and at termination (20–34%, not significant); decreased absolute liver weight in P-generation and F_1-generation males (11–12%, $p<0.05$) and decreased liver/body weight ratios in P-generation and F_1-generation males (8–9%, $p<0.05$ for P-generation and not significant for F_1-generation); decreased absolute kidney weights in F_1-generation (but not P-generation) males (12%, $p<0.05$); and decreased kidney/body weight ratios in F_1-generation (but not P-generation) males (8%, $p>0.05$). The parental LOAEL in females was based on increased alkaline phosphatase levels in F_1-generation) (but not P-generation) females at premating (43%, $p<0.05$) and at termination (63%, $p<0.05$); and on very small increases in gamma glutamyl transferase (GGT) (not considered to be biologically relevant). Overall, treatment-related effects observed at 5,000 ppm in males and females were also observed at 20,000

ppm, but were slightly increased in severity. Toxicologically relevant additional toxicological effects observed at 20,000 ppm were decreased body weights and increased food consumption in males and increased urea nitrogen and creatinine levels, decreased kidney weights, decreased body weights, and increased food consumption in females.

5. *Mutagenicity*. No mutagenicity was noted in the following assays: Reverse gene mutation, *S. typhimurium, E. coli*; Forward gene mutation - Hypoxanthine guanine phophoribosyl transferase (HGPRT) locus; Chromosome aberration, Chinese hampster ovary (CHO) cells; Unscheduled DNA synthesis, rat hepatocytes; and Micronucleus assay in mice.

6. *Chronic toxicity*—i. In a 1–year chronic oral toxicity study, dogs were fed dose levels of 0, 500, 3,500, or 25,000 ppm (0, 17.4, 124.3, or 917.8 mg/kg/day for males and 0, 19.2, 132.7, or 947.1 mg/kg/day for females). The NOAEL in males and females was 500 ppm. The LOAEL was 3,500 ppm in males and females based on decreases in red blood cells (RBC), hemoglobin (Hb), and hematocrit (Hct) and on significant increases in Heinz bodies in both sexes, increased adrenal weight parameters in females, and the presence of intracytoplasmic vacuoles in the adrenal cortex of 3/4 females.

ii. In a combined chronic toxicity/carcinogenicity study, 50 rats/sex/dose were fed fenhexamid at dose levels of 0, 500, 5,000 or 20,000 ppm (0, 28, 292, or 1,280 mg/kg/day for males and 0, 40, 415, 2,067 mg/kg/day for females) for 24 months. The NOAEL in males and females was 500 ppm. The LOAEL for chronic toxicity in males and females was 5,000 ppm based on observations of decreased body weight gain (–6.8%) and food efficiency (–11.8%) in females, increased incidence of cecal mucosal hyperplasia in males, increased cellularity (hyperplasia) of the bone marrow in females and the presence of splenic extramedullary hematopoiesis in males. At 20,000 ppm, observations were increased food consumption, increased numbers of circulating reticulocytes, enlarged spleens observed macroscopically, increased splenic weights, and thyroid colloid alterations (both sexes). Fenhexamid was non-oncogenic at doses up to and including 20,000 ppm in the diet. At doses tested, there were no treatment related increases in tumor incidence, tumor spectrum, or latency when compared to controls.

7. *Carcinogenicity*. In a carcinogenicity study, 50 mice/sex/dose were fed fenhexamid at dose levels of 0, 800, 2,400, or 7,000 ppm (0, 247.4, 807.4, or 2,354.8 mg/kg/day for males and 0, 364.8, 1,054.5, or 3,178.2 mg/kg/day for females) for 2 years. The NOAEL for males was 800 ppm and the NOAEL for females was 2,400 ppm. The LOAEL for males was 2,400 ppm based on the observation of decreased kidney weights and decreases in sex-specific vacuolation of the proximal tubules in the kidneys in males. A marginal decrease in body weights (up to 8%) and body weight gain (17%) was observed in males at 7,000 ppm. The LOAEL for females was 7,000 ppm based on significantly increased water consumption, decreased kidney weights, and renal histopathology (increased incidence of basophilic cortical tubules). Fenhexamid was not oncogenic in mice at doses up to and including 7,000 ppm. There were no treatment-related increases in tumor incidence, tumor spectrum, or latency when compared to controls.

8. *Dermal absorption*. In a dermal absorption study, radiolabeled fenhexamid (50% formulation) was applied to the shaved skin of male rats at dose levels of 0.00138, 0.0147, or 0.148 mg/cm^2. A volume of 100 µL was applied to a skin area of approximately 12.5 cm^2 on each rat. Four rats/dose level were sacrificed at 0.5, 1, 2, 4, 10, 24, and 120 hours postdose. Mean total recovery of radioactivity ranged from 90.3% to 97.6% of the applied dose. The majority of radioactivity was recovered from the skin wash (69.9% to 96.1%). Radioactivity in the skin test site ranged from 0.44% to 10.2%; in the urine from ''not detectable'' to 3.34%; and in the feces from ''not detectable'' to 11.6% of the applied dose. Radioactivity in blood did not exceed 0.03% and in the carcass did not exceed 9.37%. Estimates of dermal absorption were based on the sum of radioactivity (as test material) in the skin test site, urine, feces, blood and carcass. The percentage dermal absorption decreased with increasing dose levels. The percentage dermal absorption at 10 hours post-dose was 19.58%, 7.62%, and 2.63% and at 120 hours post-dose was 21.0%, 6.91%, and 2.13% for the low, mid and high dose levels respectively.

9. *Metabolism*. In a metabolism study, rats were administered radiolabeled fenhexamid (a single oral low dose of 1 mg/kg, a single oral high dose of 100 mg/kg, or 15 repeated low doses of 1 mg/kg/day). Radiolabeled fenhexamid was rapidly absorbed from the gastrointestinal (GI) tract in all dose groups. After single and repeated administration of the low dose, the plasma concentration peaked within 5 to 10 minutes. After administration of the high dose, the maximum was detected 40 to 90 minutes post-dosing. The absorption of the test compound was shown to be almost complete in a bile-cannulation experiment, as more than 97% of the administered dose was absorbed from the GI tract 48 hours after intra-duodenal administration. These results are indicative of a pronounced first pass effect and enterohepatic circulation. Tissue residues declined rapidly and after 48 hours the total radioactivity residue in the body excluding the GI tract, was < 0.3% of the administered dose in all dose groups. Liver and kidney were the organs with the highest concentrations of radioactivcity in all dose groups. Excretion was rapid and almost complete with feces as the major route of excretion. Approximately 62–81% of the recovered radioactivity was found in feces, and 15–36% in urine within 48 hours post-dosing. More than 90% of the recovered radioactivity was eliminated with bile in the bile cannulation experiment. Only 0.02% of the administered radioactivity was recovered in exhaled air. Radioactive residues in rat bodies (excluding GI tract) were significantly lower in females after a single high dose. There was significantly higher renal excretion for females in comparison with males after 15 repeated low doses. In both sexes renal excretion was significantly higher after a single low dose when compared with a single high dose. Metabolite characterization studies showed that the main component detected in excreta was the unchanged parent compound which accounted for 62 to 75% of the dose independent of the dosing regime and sex. Metabolite 1, the glucuronic acid conjugate of the parent compound, ranged from 4 to 23% of the dose. Metabolite fractions 2 and 3 accounted for up to 3 and 7% of the dose, respectively. The proposed major pathway for biotransformation is via conjugation of the aromatic hydroxyl group with glucuronic acid. Prior to fecal excretion, hydrolysis in the intestine converts the conjugate back to the parent compound giving rise to enterohepatic circulation. Identification of radioactive residues ranged from 88% to 99% and was independent of dose and sex.

B. Toxicological Endpoints

1. *Acute toxicity*. An acute toxicological endpoint was not identified resulting from a single oral exposure, and therefore, an acute Reference Dose (RfD) was not selected.

2. *Short- and intermediate-term toxicity*. A short- and intermediate-term

dermal endpoint of 1,000 mg/kg/day from the 21–day dermal toxicity study in rabbits was selected for occupational exposure. No short- and intermediate-term endpoint was selected for non-occupational exposure as there are no residential uses of fenhexamid.

3. *Chronic toxicity*. EPA has established the RfD for fenhexamid at 0.17 mg/kg/day. This RfD is based on 1–year feeding study in dogs with a NOAEL = 17 mg/kg/day. An additional 3x FQPA safety factor was added and applies to all population subgroups resulting in a chronic population-adjusted dose (chronic PAD) of 0.057 mg/kg/day.

4. *Carcinogenicity*. Fenhexamid was classified as a ''not likely'' human carcinogen based on the lack of evidence of carcinogenicity in mice and rats and the lack of genotoxicity in a battery of mutagenicity studies.

C. Exposures and Risks

1. *From food and feed uses*. Fenhexamid is a new chemical and no tolerances are currently established. In today's action, tolerances are being established at 40 CFR 180.553 for grapes at 4.0 ppm, strawberries at 3.0 ppm, and raisins at 6.0 ppm. Risk assessments were conducted by EPA to assess dietary exposures from fenhexamid 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. No toxicological endpoint attributable to a single (acute) dietary exposure was identified.

ii. *Chronic exposure and risk*. The chronic risk analysis used the chronic PAD of 0.057 mg/kg/day which applies to all populations subgroups. The Dietary Exposure Evaluation Model (DEEM) which is a exposure analysis system that estimates exposure to a pesticide chemical in food comprising the diets of the U.S. population, including population subgroups was used to conduct the chronic (food) risk analysis. DEEM contains food consumption data as reported by respondents in the USDA Continuing Surveys of Food Intake by Individuals conducted in 1989–1992. The chronic food exposure was calculated assuming theoretical maximum residue contribution (TMRC) values and 100% crop treated estimates. The percent of the chronic PAD utilized is as follows: 6.6% for nursing infants (< 1 year); 4.8% for children (1–6 years); 3.6% for females (13+/nursing) and for all infants (< 1 year); 2.7% for the Pacific regions; 2.4% for non-nursing infants (< 1 year), Western region, and non-Hispanic other than black or white; and 1.8% for the U.S. population (48 states-all seasons).

2. *From drinking water*. In soil, fenhexamid is relatively immobile (K_{oc} = 446) and non-persistent ($t_{1/2}$ = ≧ 1 day). Fenhexamid is not expected to be a ground water contaminant, but has some potential to reach surface water on eroded soil particles. In surface water, fenhexamid would be expected to photodegrade rapidly ($t_{1/2}$ = ≧ 0.2 days).

No monitoring data are available to perform a quantitative drinking water assessment. The Agency estimated 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 the maximum label rate to estimate surface water concentration. GENEEC contains a number of conservative underlying assumptions. Therefore, the drinking water concentrations derived from GENEEC for surface water are likely to be overestimated. The modeling was conducted based on the environmental profile and the maximum seasonal application rate proposed for fenhexamid: 0.75 lb. active ingredient/acre x 4 applications/acre/year. The estimated environmental concentrations (EECs) derived from GENEEC are 17 µg/L (peak value) and 4.8 µg/L (56–day average).

The Agency used the Screening Concentration in Ground Water (SCI-GROW) model to estimate pesticide levels in ground water. The SCI-GROW model is based on actual monitoring data collected for a number of pesticides that serve as benchmarks to predict EECs in ground water. Using SCI-GROW, the EEC calculated for fenhexamid is 0.0007 µg/L (acute and chronic).

i. *Acute exposure and risk*. Drinking water levels of comparison (DWLOCs) for acute exposure were not calculated as there was no appropriate toxicological endpoint attributable to a single (acute) dietary exposure.

ii. *Chronic exposure and risk*. Chronic (non-cancer) DWLOCs were calculated for the U.S. population and the population subgroups with the highest (chronic) food exposure. The DWLOCs are as follows: 530 µg/L for infants/children; 1,700 µg/L for females 13+; 1,900 µg/L for the U.S. population - pacific region; and 2,000 µg/L for U.S. population (48 states, all seasons). The EECs (0.0007 µg/L from SCI-GROW, and 4.8 µg/L from GENEEC) for fenhexamid are well below the DWLOCs and therefore, are below the Agency's level of concern. Therefore, the Agency concludes with reasonable certainty that residues of fenhexamid in drinking water do not contribute significantly to the aggregate chronic human health risk.

3. *From non-dietary exposure*. Fenhexamid is not registered for use on residential non-food sites. Therefore, no non-occupational, non-dietary exposure and risk are 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.'

EPA does not have, at this time, available data to determine whether fenhexamid 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, fenhexamid 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 fenhexamid has a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the final rule for Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997).

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

1. *Acute risk*. Acute aggregate risk is the sum of exposures resulting from acute dietary food + acute drinking water. The Agency did not identify an appropriate toxicological endpoint attributable to a single (acute) dietary exposure.

2. *Chronic risk*. Using the TMRC, exposure assumptions described in this unit, EPA has concluded that aggregate exposure to fenhexamid from food will utilize 1.8% of the chronic PAD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is nursing infants (< 1 year) discussed below. EPA generally has no concern for exposures below 100% of the chronic PAD because the chronic PAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the

potential for exposure to fenhexamid in drinking water, EPA does not expect the aggregate exposure to exceed 100% of the chronic PAD. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fenhexamid residues.

3. *Short- and intermediate-term risk*. Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. Although short- and intermediate-term endpoints were identified, there are no residential uses for fenhexamid.

4. *Aggregate cancer risk for U.S. population*. Fenhexamid was classified as ''not likely'' to be a human carcinogen.

5. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to Fenhexamid residues.

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

1. *Safety factor for infants and children*—i. *In general*. In assessing the potential for additional sensitivity of infants and children to residues of fenhexamid, EPA considered data from developmental toxicity studies in the rat and rabbit and a 2–generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. 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 postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure (MOE) analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor.

ii. *Pre- and postnatal sensitivity*. Qualitatively, there is evidence of increased susceptibility in rat pups compared to adults, based on the relative severity of effects in the 2–generation reproduction study in rats. The effects on pups were of concern because: significant pup body weight decreases were observed in both the F_1 and the F_2 generations; the pup body weight decreases in the F_2 generation were observed during early lactation (lactation days 7 through day 21) when the pups are exposed to the test material primarily through the mother's milk; the pup body weight decreases in the F_1 generation were observed during late lactation (lactation days 14 through 21) when the pups are exposed to the test material through the mother's milk and through the feed; and, in the metabolism study on fenhexamid, glucuronidation of fenhexamid was clearly demonstrated to be the single major route of metabolism, detoxification and excretion of fenhexamid in adult male and female rats. The demonstrated poor glucuronidation capacity of rat pups between days 7 and 21 indicates a possibly increased sensitivity of pups and serves to support a concern for neonatal toxicity.

iii. *Conclusion*. There is a complete toxicity data base for fenhexamid and exposure data are complete or are estimated based on data that reasonably accounts for potential exposures. Although there is qualitative evidence of increased susceptibility, the Agency decided that an additional safety factor of 3x would be appropriate based on the following reasons: the increased susceptibility demonstrated in the 2–generation reproduction study was only qualitative (not quantitative) evidence and was observed only in the presence of parental toxicity; the qualitative offspring effect was limited to decreased body weight and no other adverse effects (e.g., decreased pup survival, behavioral alterations, etc.) were observed; and there is no indication of increased susceptibility of rat or rabbit fetuses to *in utero* exposure in the prenatal developmental toxicity studies with fenhexamid.

2. *Acute risk*. An acute endpoint was not identified and this risk assessment was not required.

3. *Chronic risk*. Using the exposure assumptions described in this unit, EPA has concluded that highest aggregate exposure to fenhexamid from food will utilize 6.6% of the chronic PAD for all infants (< 1 year). EPA generally has no concern for exposures below 100% of the chronic PAD because the chronic PAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to fenhexamid in drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the chronic PAD.

4. *Short- or intermediate-term risk*. There are no residential uses and thus these risks are not presented.

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

III. Other Considerations

A. Metabolism In Plants and Animals

The parent compound, fenhexamid, is the only compound of concern. Radiolabeled fenhexamid plant metabolism studies were conducted on grapes, tomatoes, and apples. The qualitative nature of fenhexamid residues in plants is adequately understood. The data indicate very little translocation of residues, i.e., residues of fenhexamid are non-systemic and are thus primarily surface residues. There are no animal feedstuffs associated with the uses of fenhexamid on grapes, strawberries, and ornamentals. Therefore, no animal metabolism data were submitted or required.

B. Analytical Enforcement Methodology

Adequate enforcement methodology (a high performance liquid chromotography method with electrochemical detection) is available to enforce the tolerance expression. The method may be requested from: Calvin Furlow, PRRIB, IRSD (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 101FF, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–5229.

C. Magnitude of Residues

An adequate number of geographically representative field trials were submitted to support the proposed uses on grapes and strawberries. These studies were conducted via use patterns approximating those proposed by the petition requesting these tolerances. The data indicate that residues of fenhexamid will not exceed the proposed tolerances. Residues concentrated an average of 1.9x in raisins. Multiplying 1.9x by the highest

average field trial residue value in grapes (2.3 ppm), yields 5.3 ppm as the maximum residue expected in raisins which is below the proposed tolerance of 6.0 ppm. The concentration factor was ≤ 0.25x in juice and ≤ 0.5x in wine grapes based on data from red and white wine grapes.

D. International Residue Limits

There are no codex, Canadian or Mexican maximum residue limits established for this chemical. This petition was jointly reviewed with Canada's Pest Management and Regulatory Agency and the tolerances proposed have been harmonized with Canada.

E. Rotational Crop Restrictions

The Agency concluded that a 30–day plantback interval is required for all crops without a fenhexamid tolerance.

IV. Conclusion

Therefore, tolerances are established for residues of fenhexamid in or on grapes at 4.0 ppm, strawberries at 3.0 ppm, and raisins at 6.0 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 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 July 27, 1999, 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 under the ''ADDRESSES'' section (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 regulation. 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). EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding tolerance objection fee waivers, contact James Tompkins, 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: Rm. 239, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–5697, tompkins.jim@epa.gov. Requests for waiver of tolerance objection fees should be sent to James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

VI. Public Record and Electronic Submissions

EPA has established a record for this regulation under docket control number [OPP–300866] (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 Rm. 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA.

Objections and hearing requests may be sent by e-mail directly to EPA at:

opp-docket@epa.gov

E-mailed objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption.

The official record for this regulation, as well as the public version, as described in this unit 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 record which will also include all comments submitted directly in writing. The official record is the paper record maintained at the Virginia address in ''ADDRESSES'' at the beginning of this document.

VII. Regulatory Assessment Requirements

A. Certain Acts and Executive Orders

This final rule establishes tolerances under section 408(d) of the FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any 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 tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. Nevertheless, the Agency 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.

B. Executive Order 12875

Under Executive Order 12875, entitled *Enhancing the Intergovernmental Partnership* (58 FR 58093, October 28, 1993), EPA may not issue a regulation that is not required by statute and that creates a mandate upon a State, local or tribal government, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by those governments. If the mandate is unfunded, EPA must provide to OMB a description of the extent of EPA's prior consultation with representatives of affected State, local, and tribal governments, the nature of their concerns, copies of any written communications from the governments, and a statement supporting the need to issue the regulation. In addition, Executive Order 12875 requires EPA to develop an effective process permitting elected officials and other representatives of State, local, and tribal governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

Today's rule does not create an unfunded Federal mandate on State, local, or tribal governments. The rule does not impose any enforceable duties on these entities. Accordingly, the requirements of section 1(a) of Executive Order 12875 do not apply to this rule.

C. Executive Order 13084

Under Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998), EPA may not issue a regulation that is not required by statute, that significantly or uniquely affects the communities of Indian tribal governments, and that imposes substantial direct compliance costs on those communities, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by the tribal governments. If the mandate is unfunded, EPA must provide OMB, in a separately identified section of the preamble to the rule, a description of the extent of EPA's prior consultation with representatives of affected tribal governments, a summary of the nature of their concerns, and a statement supporting the need to issue the regulation. In addition, Executive Order 13084 requires EPA to develop an effective process permitting elected officials and other representatives of Indian tribal governments ''to provide meaningful and timely input in the development of regulatory policies on matters that significantly or uniquely affect their communities.''

Today's rule does not significantly or uniquely affect the communities of Indian tribal governments. This action does not involve or impose any requirements that affect Indian tribes. Accordingly, the requirements of section 3(b) of Executive Order 13084 do not apply to this rule.

VIII. Submission to Congress and the Comptroller General

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

List of Subjects in 40 CFR Part 180

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

Dated: May 19, 1999.

Susan B. Hazen,

Acting Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180–[AMENDED]

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

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

2. Section 180.553, is added to subpart C to read as follows:

§ 180.553 Fenhexamid; tolerances for residues.

(a) *General.* Tolerances are established for the residues of the fungicide fenhexamid (N-2,3-dichloro-4-hydroxyphenyl)-1-methyl cyclohexanecarboxamide) in or on the following commodities:

Commodity	Parts per million
Grapes	4.0
Raisins	6.0
Strawberries	3.0

(b) Section 18 emergency exemptions. [Reserved]

(c) Tolerances with regional registrations. [Reserved]

(d) Indirect or inadvertent residues. [Reserved]

[FR Doc. 99–13656 Filed 5–27–99; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300862; FRL–6080–5]

RIN 2070–AB78

Terbacil; Extension of Tolerance for Emergency Exemptions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This rule extends a time-limited tolerance for residues of the herbicide terbacil and its metabolites in or on watermelon at 0.4 part per million (ppm) for an additional 2–year period, to May 30, 2001. 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 watermelons. Section 408(l)(6) of the Federal Food, Drug, and Cosmetic Act (FFDCA) requires EPA to establish a time-limited tolerance or exemption from the requirement for a tolerance for pesticide chemical residues in food that will result from the use of a pesticide under an emergency exemption granted by EPA under section 18 of FIFRA.

DATES: This regulation becomes effective May 28, 1999. Objections and requests for hearings must be received by EPA, on or before July 27, 1999.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP-300862], must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing