and the IPCB allows CILCO Edwards' variance to continue unamended through July 31, 2003, as stated in the Opinion and Order, then federal approval of the variance will continue until EPA approves alternate SO_2 limits for CILCO Edwards, or until July 31, 2003, whichever is earlier.

[FR Doc. 00–8952 Filed 4–12–00; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300991; FRL–6553–7]

RIN 2070–AB78

Fenhexamid; Pesticide Tolerances

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 almond, nutmeat at 0.02 parts per million (ppm), almond, hull at 2.0 ppm, stone fruit, except plum (fresh prune) at 6.0 ppm, plum (fresh prune) at 0.5 ppm, and prune, dried at 1.0 ppm. The TM-402 Fungicide Task Force which is 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 April 13, 2000. Objections and requests for hearings, identified by docket control number OPP–300991, must be received by EPA on or before June 12, 2000.

ADDRESSES: Written objections and hearing requests may be submitted by mail, in person, or by courier. Please follow the detailed instructions for each method as provided in Unit VI. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, your objections and hearing requests must identify docket control number OPP–300991 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Mary L. Waller, Product Manager 21, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW.,Washington, DC 20460; telephone number: (703) 308–9354; and e-mail address: waller.mary@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed under ''FOR FURTHER INFORMATION CONTACT.''

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**--Environmental Documents.'' You can also go directly to the**Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number OPP–300991. The official record consists of the documents specifically referenced in this action, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

II. Background and Statutory Findings

In the **Federal Register** of February 25, 2000 (65 FR 10078) (FRL–6494–2), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Public Law 104–170) announcing the filing of an amendment to pesticide petition (PP 7F4890) for tolerances, by the TM-402 Fungicide Task Force (Tomen Agro, Inc, 100 First Street, Suite 1610, San Francisco, CA 94105 and Bayer Corporation, 8400 Hawthorn Road, P.O. Box 4913, Kansas City, MO 64120–0013). This notice included a summary of the petition prepared by the TM-402 Fungicide Task Force. The registrant is Tomen Agro, Inc. There were no comments received in response to the notice of filing.

The amended petition requested that 40 CFR 180.553 be amended by establishing tolerances for the fungicide, fenhexamid in or on almond, nutmeat at 0.02 ppm, almond, hull at 2.0 ppm, stone fruit, except plum (fresh prune) at 6.0 ppm, plum (fresh prune) at 0.5 ppm, and prune, dried at 1.0 ppm.

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

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. For further discussion of the regulatory

requirements of section 408 and a complete description of the risk assessment process, see the final rule on Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997) (FRL–5754–7).

III. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of fenhexamid and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances in or almond, nutmeat at 0.02 ppm, almond, hull at 2.0 ppm, stone fruit, except plum (fresh prune) at 6.0 ppm, plum (fresh prune) at 0.5 ppm, and prune, dried at 1.0 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 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/kilogram (mg/kg) for both sexes. The acute inhalation LC_{50} for rats was > 5.06 milligrams/liter (mg/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 Alanine 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 developmental and maternal toxicity 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 mg/kg/day 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, that is, 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 postdose was 19.58%, 7.62%, and 2.63% and at 120 hours postdose 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 postdosing. 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 62–75% 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 a 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 (cPAD) 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. *Dietary*— i. *From food and feed uses*. Tolerances are currently established for fenhexamid at 40 CFR 180.553 for grapes at 4.0 ppm, strawberries at 3.0 ppm, and raisins at 6.0 ppm. Additional tolerances are being proposed as follows: almond, nutmeat at 0.02 ppm, almond, hull at 2.0 ppm, stone fruit, except plum (fresh prune) at 6.0 ppm, plum (fresh prune) at 0.5 ppm, and prune, dried at 1.0 ppm. Risk assessments were conducted by EPA to assess dietary exposures from fenhexamid as follows:

a. *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.

b. *Chronic exposure and risk*. The chronic risk analysis used the cPAD of 0.057 mg/kg/day which applies to all population 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 cPAD utilized is as follows: 15.7 for non-nursing infants; 14.2 for all infants (<1 year); 10.7 for nursing infants; 9.9 for children (1-6 years); 5.7 for non-Hispanic/non-white/non-black; 5.0 for children (7 to 12 years); 4.6 for U.S. population (summer season); 3.7 for U.S. population (total) and 2.6 for females (13-50 years).

ii. *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).

a. *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.

b. *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: 480 µg/L for infants/children; 1,700 µg/L for females 13-50 yrs.); and 1,900 µg/L for the U.S. population and all other subgroups. The EEC (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.

2. *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.

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

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 3.7% of the cPAD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non-nursing infants (< 1 year) discussed below. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to fenhexamid in drinking water, EPA does not expect the aggregate exposure to exceed 100% of the cPAD. 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 prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a 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. *Prenatal 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 two-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 day 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.

3. *Chronic risk*. Using the exposure assumptions described in this unit, EPA has concluded that the highest aggregate exposure to fenhexamid from food will utilize 15.7% of the cPAD for non nursing infants. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. 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 cPAD.

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.

IV. Other Considerations

A. Metabolism in Plants

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.

B. Metabolism in Animals

Almond hulls which are a livestock feed item contain 90% dry matter and its contribution to the livestock diet is a maximum of 10% each for beef and dairy cattle. Data from a study investigating the metabolism of ^{14}C fenhexamid in a lactating goat indicated that the metabolism of fenhexamid in the goat is similar to that in the rat, and based on the experimentally determined feeding level of 133 ppm in the feed, the Agency calculates that the dosage was equivalent to 605x the maximum theoretical dietary burden of 0.22 ppm for beef and dairy cattle. The total radioactive residues (TRR) were 0.045–0.212 ppm in milk, 4.682 ppm in liver, 3.267 ppm in kidney, 0.035 ppm in muscle, and 0.085 ppm in fat.

The qualitative nature of the residue in ruminants is adequately understood. Based on the goat metabolism study, the Agency concludes that there is no reasonable expectation of finite residues in milk or ruminant tissues as a result of the currently proposed uses on almonds and stone fruits, and ruminant commodity tolerances are not required.

C. 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, PIRIB, IRSD (7502C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460. Office location and telephone number: Rm 101FF, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–5229.

D. Magnitude of Residues

A total of five almond field trials were conducted in California. The almond field trial data are adequate in number and geographical representation. The data indicate that residues of fenhexamid will not exceed the proposed tolerances of 0.02 ppm in/on almond nutmeats and 2.0 ppm in/on almond hulls following applications of the proposed 50% WDG formulation according to the maximum proposed use patterns. Samples of almond RACs were harvested 142–173 days following the last of four sequential applications of the 50% WDG formulation at 0.73–0.76 lb ai/acre/application (3.0 lbs active ingredient acre/season (ai/acre/season); 1x the proposed maximum seasonal application rate). Residues of fenhexamid were non-detectable (<0.02 ppm) in/on all treated almond nutmeat samples.

Field trial data were submitted for cherries (sweet and tart), peaches, and plums, which are the three representative commodities of the stone fruits crop group (40 CFR 180.41, Crop Group 12). Samples (U.S. field trials) were harvested 0-days following the last of four sequential foliar applications of the 50% WDG formulation at 0.73–0.78 lb ai/acre/application (3.0 lbs ai/acre/season; 1x the proposed maximum seasonal application rate). Residues of fenhexamid (uncorrected for method recovery and storage stability data) in/on treated samples ranged from 0.844–1.826 ppm for sweet cherries, 1.049–4.950 ppm for tart cherries, 0.327-2.131 ppm for peaches, and <0.05–0.366 ppm for plums. The residue data for stone fruits indicates that the maximum residues for tart cherries (4.950 ppm) and plums (0.366 ppm) differ by a factor of 13.5. On this basis, the Agency concludes that plums should be excluded from the proposed stone fruits crop group tolerance, and an individual tolerance is being established for residues of fenhexamid in/on plums (fresh prunes) at 0.5 ppm.

No processing study data have been submitted for dried prunes. Based on the concentration factor which has previously been shown to occur in the processing of fenhexamid-treated grapes to raisins, it is probable that concentration of fenhexamid residues will occur in the processing of plums (fresh prunes) to dried prunes. The Agency concludes that the appropriate tolerance level for residues of fenhexamid *per se* in/on dried prunes is 1.0 ppm. This is based upon the highest average field trial (HAFT) residue value (0.264 ppm) for plums (fresh prunes) multiplied by the TMCF (3.4x) for dried prunes =0.90 ppm, which is rounded up to 1.0 ppm.

E. International Residue Limits

The Codex Alimentarius Commission has not established maximum residue limits (MRLs) for residues of fenhexamid or any of its metabolites in/on plant or animal commodities.

Harmonization is thus not an issue for this action.

F. Rotational Crop Restrictions

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

V. Conclusion

Therefore, tolerances are established for residues of fenhexamid in or on almond, nutmeat at 0.02 ppm, almond, hull at 2.0 ppm, stone fruit, except plum (fresh prune) at 6.0 ppm, plum (fresh prune) at 0.5 ppm and prune, dried at 1.0 ppm.

VI. Objections and Hearing Requests

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

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

You must file your objection or request a hearing on this regulation in accordance with the instructions provided in this unit and in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket control number OPP–300991 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk on or before June 12, 2000.

1. *Filing the request*. Your objection must specify the specific provisions in the regulation that you object to, and the grounds for the objections (40 CFR 178.25). If a hearing is requested, the objections must include a statement of the factual issues(s) on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the objector (40 CFR 178.27). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

Mail your written request to: Office of the Hearing Clerk (1900), Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460. You may also deliver your request to the Office of the Hearing Clerk in Rm. C400, Waterside Mall, 401 M St., SW., Washington, DC 20460. The Office of the Hearing Clerk is open from 8 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Office of the Hearing Clerk is (202) 260–4865.

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

EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding the waiver of these fees, you may contact James Tompkins by phone at (703) 305–5697, by e-mail at tompkins.jim@epa.gov, or by mailing a request for information to Mr. Tompkins at Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460.

If you would like to request a waiver of the tolerance objection fees, you must mail your request for such a waiver to: James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460.

3. *Copies for the Docket*. In addition to filing an objection or hearing request with the Hearing Clerk as described in Unit VI.A., you should also send a copy of your request to the PIRIB for its inclusion in the official record that is described in Unit I.B.2. Mail your copies, identified by docket control number OPP–300991, to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Ariel Rios Bldg., 1200 Pennsylvania Ave., NW., Washington, DC 20460. In person or by courier, bring a copy to the location of the PIRIB described in Unit I.B.2. You may also send an electronic copy of your request via e-mail to: opp-docket@epa.gov. Please use an ASCII file format and avoid the use of special characters and any form of encryption. Copies of electronic objections and hearing requests will also be accepted on disks in WordPerfect 6.1/8.0 file format or ASCII file format. Do not include any CBI in your electronic copy. You may also submit an electronic copy of your request at many Federal Depository Libraries.

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

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

VII. Regulatory Assessment Requirements

This final rule establishes a tolerance under 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) (Public Law 104–4). Nor does it require any prior consultation as specified by Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998); 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 or any Agency action under Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). Since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. In addition, the Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999). Executive Order 13132 requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.'' This final rule directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4).

VIII. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this final rule in the **Federal Register**. This final 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: March 30, 2000.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180–[AMENDED]

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

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

2. Section 180.553 is amended by alphabetically adding the following commodities to the table in paragraph (a) to read as follows:

§ 180.553 Fenhexamid; tolerances for residues.

(a) * * *

Commodity	Parts per million
Almond, hull	2.0
Almond, nutmeat	0.02
* * * * *	
Plum (fresh prune)	0.5
Prune, dried	1.0
* * * * *	
Stone fruit, except plum (fresh prune)	6.0
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[FR Doc. 00–9144 Filed 4–12–00; 8:45 am]

BILLING CODE 6560–50–F

DEPARTMENT OF DEFENSE

48 CFR Parts 213, 225, 242, and 252

[DFARS Case 98–D028]

Defense Federal Acquisition Regulation Supplement; Foreign Acquisition

AGENCY: Department of Defense (DoD).

ACTION: Final rule.

SUMMARY: The Acting Director of Defense Procurement has issued a final rule amending the Defense Federal Acquisition Regulation Supplement (DFARS). These amendments conform the DFARS to the Federal Acquisition Regulation (FAR) Amendments pertaining to foreign acquisition that were published in the **Federal Register** on December 27, 1999.

EFFECTIVE DATE: April 13, 2000.

FOR FURTHER INFORMATION CONTACT: Ms. Amy Williams, Defense Acquisition Regulations Council, PDUSD (AT&L) DP (DAR), IMD 3D139, 3062 Defense Pentagon, Washington, DC 20301–3062. Telephone (703) 602–0288; telefax (703) 602–0350. Please cite DFARS Case 98–D028.

SUPPLEMENTARY INFORMATION:

A. Background

This final rule amends DFARS Part 225, Foreign Acquisition, and updates related references, for conformance with the FAR Part 25 rewrite that was published at 64 FR 72416 on December 27, 1999 (Federal Acquisition Circular 97–15, Item II). The rule reorganizes the existing DFARS text to align it with the revised FAR text. The rule makes no substantive change to DFARS policy pertaining to foreign acquisition. The following list summarizes the reorganization of the DFARS text:

Text previously located at	Relocated to
225.000–70	225.003
225.000–71	225.001
225.102	225.103
225.105	225.502
Table 25–1	225.504
225.107	225.170
225.108	225.104
225.109(a)	225.1101(1)
225.109(b)	225.171(a)
225.109(d)	225.1101(2)
225.109–70(a)	225.1101(3)
225.109–70(b)	225.171(b)
225.303	225.304
225.305–70	225.1103(1)
225.401	225.003
225.402(c)	225.403
225.403	225.401
225.403–70	225.401–70
225.405	225.408
225.408	225.11
225.602	225.901
225.603	225.902
225.604	225.903
225.605	FAR 25.1101(e)(2)
225.605–70	225.11
225.702	225.701
225.970	225.1070
225.971	225.1103(2)
225.972	225.1103(3)

This rule was not subject to Office of Management and Budget review under Executive Order 12866, dated September 30, 1993.