TABLE 52.2381—EPA-APPROVED REGULATIONS—Continued

[Vermont SIP regulations 1972 to present]

| State citation, title and subject | Date adopted by State | Date approved by EPA | Federal Register citation | 52.2370 | Comments and unapproved sections |
|---|---|---|---|---|---|
| * * | | * | * * | | * * |
| Section 5–253.1 Petroleum Liquid Storage in Fixed Roof Tanks. | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.2 Bulk Gasoline Terminals | 10/29/92 | 4/22/98 | [Insert FR citation published date]. | (c)(25) ..... | |
| Section 5–253.3 Bulk Gasoline Plants | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.4 Gasline Tank Trucks. | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.5 Stage I vapor recovery controls at gasoline dispensing facilities. | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.10 Paper Coating. | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.12 Coating of Flatwood Paneling. | 10/29/92 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–252.13 Coating of Miscellaneous Metal Parts. | 7/29/93 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
| Section 5–253.14 Solvent Metal Cleaning. | 7/29/93 | 4/22/98 | [Insert *FR* citation from published date]. | (c) 25) ..... | |
| Section 5–253.15 Cutback and Emulsified Asphalt. | 8/2/94 | 4/22/98 | [Insert *FR* citation from published date]. | (c)(25) ..... | |
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[FR Doc. 98–10724 Filed 4–21–98; 8:45 am]

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## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Part 180

**[OPP–300635; FRL–5782–1]**

**RIN 2070–AB78**

### Fenoxaprop-ethyl; Pesticide Tolerance

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Final rule.

**SUMMARY:** This regulation establishes tolerances for combined residues of fenoxaprop-ethyl [ethyl 2-[4-[(6-chloro-2-benzoxazolyl) oxy]phenoxy] propanoate] and its metabolites [2-[4-] (6-chloro-2-benzoxazolyl)oxy]phenoxy] propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one in or on the following raw agricultural commodities (RACs): barley, grain at 0.05 parts per million (ppm), and barley straw at 0.1 ppm. AgrEvo USA Company requested these tolerances under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170).

**DATES:** This regulation is effective April 22, 1998. Objections and requests for hearings must be received by EPA on or before June 22, 1998.

**ADDRESSES:** Written objections and hearing requests, identified by the docket control number, [OPP–300635], 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–300635], must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number [OPP–300635]. 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: Joanne I. Miller, Product Manager (PM) 23, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–6224, e-mail: miller.joanne@epamail.epa.gov.

**SUPPLEMENTARY INFORMATION:** In the **Federal Register** of September 17, 1997 (62 FR 48837) (FRL–5741–1), EPA, issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) announcing the filing of a pesticide petition (PP) for tolerance by AgrEvo USA Company, Little Falls One, 2711 Centerville Road, Wilmington, DE 19808. This notice included a summary of the petition prepared by AgrEvo USA Company, the registrant. There were no

comments received in response to the notice of filing.

The petition requested that 40 CFR 180.430 (b) be amended by establishing tolerances for combined residues of the herbicide fenoxaprop-ethyl [ethyl 2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate] and its metabolites [2-[4-](6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one, in or on the following raw agricultural commodities: barley, grain at 0.05 ppm; and barley straw at 0.1 ppm.

## I. Risk Assessment and Statutory Findings

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

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

### *A. Toxicity*

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

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

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

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

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

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

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

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

subgroups including infants and children.

*B. Aggregate Exposure*

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

Percent of crop treated estimates are derived from federal and private market survey data. Typically, a range of estimates are supplied and the upper end of this range is assumed for the exposure assessment. By using this upper end estimate of percent of crop treated, the Agency is reasonably certain that exposure is not understated for any significant subpopulation group. Further, regional consumption information is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups, to pesticide residues. For this pesticide, the most highly exposed population subgroup was not regionally based.

## II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action, EPA has sufficient data to assess the hazards of fenoxaprop-ethyl and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances for combined residues of fenoxaprop-ethyl [ethyl 2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate] and its metabolites [2-[4-](6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one in or on the following raw agricultural commodities: barley, grain at 0.05 ppm; and barley straw at 0.1 ppm. EPA's assessment of the dietary exposures and risks associated with establishing these 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 fenoxaprop-ethyl are discussed below.

1. *Acute toxicity.* A battery of acute toxicity studies is available, places technical fenoxaprop-ethyl in Toxicity Category III for acute oral (rat) ( $LD_{50}$ = 2,357 milligram/kilograms (mg/kg) (M) and 2,500 mg/kg (F) ), and acute inhalation (rat) $LC_{50}$ = >0.511 mg/L; in Toxicity Category IV for acute dermal (rat) = >2,000 mg/kg and rabbit =>1,000 mg/kg and skin irritation (slight irritant) ; and in Toxicity Category I for eye irritation (rabbit) with non-reversible corneal opacity at day 21. Fenoxaprop-ethyl was determined to be a non-sensitizer in a dermal sensitization study (guinea pig).

2. *Genotoxicity* . A battery of genotoxicity studies, none of which indicated any genotoxic potential. The studies submitted included: *in vitro* human lymphocyte chromosomal aberration, mouse micronucleus, *in vitro* unscheduled DNA synthesis, Ames *Salmonella* bacterial point mutation and yeast DNA repair assays.

3. In a subchronic feeding study with rats (30/sex/dose), fenoxaprop-ethyl was administered at doses of 0, 1, 4 or 16 mg/kg/day for 90 days. The NOEL was 1 mg/kg/day and the lowest observed effect level (LOEL) was 4 mg/kg/day) based on relative organ weight changes. After the 4-week recovery period, significantly decreased liver weights were observed in males at the 1 mg/kg/day dose and in females at 4 mg/kg/day.

4. In a subchronic feeding study in dogs (6 dogs/sex/dose), fenoxaprop-ethyl was administered at doses of 0, 0.4, 2, or 10 mg/kg /day were fed for 90 days. The NOEL was 0.4 mg/kg/day) and the LOEL was 2 mg/kg/day based on histological changes of the kidneys. Inflammatory changes of the kidneys (interstitial pyelonephritis) were detected in the 2 mg/kg/day and in the 10 mg/kg/day dosed dogs.

5. In a 21–day dermal toxicity study, Wistar rats (10/sex/dose) received repeated dermal applications of fenoxaprop-ethyl (96.5%, moistened with sesame oil) at doses of 0, 5, 10, or 20 mg/kg, 6 hours/day, 5 days/week, for 21 total exposures. The LOEL was >5 mg/kg based on decreased liver weights. A NOEL was not established.

6. In a second 21–day dermal toxicity study, Wistar rats (10/sex/dose) received repeated dermal applications of fenoxaprop-ethyl (96.5%, vehicle not specified) at doses of 0, 5, or 20 mg/kg, 6 hours/day, 5 days/week, for 21 total exposures. The study author concluded that the NOEL was >20 mg/kg; a LOEL was not established.

7. In a subchronic inhalation toxicity study, Wistar rats (10-15/sex/concentration) were exposed by nose-only inhalation to fenoxaprop-ethyl (96.5%) at target concentrations of 0, 0.075, 0.250, or 0.750 mg/L (analytically -determined concentrations of 0, 0.073, 0.248, or 0.727 mg/L, respectively) for 6 hours/day, 5 days/week, for 6 weeks (28-29 total exposures). An unequivocal NOEL was not established in this study. A second study using the same protocol with target concentrations of 0 or 0.015 mg/L (analytically determined to be 0 or 0.0143 mg/L, respectively) was conducted. The exposure period was followed by a 4-week recovery period for animals in all but the 0.750 mg/L group. A NOEL for the repeated dose inhalation was 0.015 mg/L.

8. In a chronic toxicity study in beagle dogs (6/sex/dose) dogs were fed fenoxaprop-ethyl (94%) at doses of 0, 0.075, 0.375 or 1.875 mg/kg/day for two years. The NOEL was 0.375 mg/kg/day and the LOEL was 1.875 mg/kg/day based on decreases in body weight gain.

9. In a carcinogenicity study with groups of 50 male and 50 female NMRKF (SPF71) mice, fenoxaprop-ethyl (94%) was administered at dose levels of 0, 0.375, 1.5, or 6 mg/kg/day for 24 months. The NOEL was >6 mg/kg/day (HDT). A LOEL was not established.

10. In a second carcinogenicity study, fenoxaprop-ethyl (96.8%) was administered to groups of 50 male and 50 female NMRI mice at doses of 0, 5.7, 16.6 or 44.6 mg/kg/day in males and 0, 6.8, 19.4 or 53.7 mg/kg/day in females for 24 months. For chronic toxicity the NOEL was 5.7 mg/kg/day and the LOEL was 16.6 mg/kg/day based on histopathological findings in the liver. There was evidence of carcinogenicity at the highest dose tested (HDT). Statistically (p=0.05) significant increases were seen in liver and adrenal gland tumors. In males at the high dose, the incidence of hepatocellular adenomas (30%) and carcinomas (8%) were increased when compared to controls (2%, adenomas and 0%, carcinomas). Also at this dose in males, the incidence of subcapsular adenomas of the adrenal glands was 43% compared to 22% in controls. In addition, microscopic pathology indicated the hepatocellular hypertrophy was observed in the majority of all treated animals (both sexes). Dosing was considered adequate to assess the carcinogenic potential of fenoxaprop-ethyl based on clinical signs, increased liver weight, and histopathology.

11. In a combined chronic/oncogenicity study, Wistar rats (116/sex/dose) were dosed with fenoxaprop-ethyl (95.8%) at 0. 0.25, 1.5 or 9 mg/kg/day for 28 months. For chronic toxicity, the NOEL was 1.5 mg/kg/day) and the LOEL was 9 mg/kg//day based on decreased serum lipids and cholesterol in the males. Under the conditions of this study, there was no evidence of carcinogenic potential.

12. In an oral developmental toxicity study, pregnant Wistar rats (20/dose) received fenoxaprop-ethyl (93% a.i.) in sesame oil at doses of 0, 10, 32, or 100 mg/kg/day from days 7 through 16 of gestation. For maternal toxicity, the NOEL was 32 mg/kg/day and the LOEL was 100 mg/kg/day, based on slight initial reduction in body weight and food consumption. There were no treatment-related effects or clinical signs, body weight gain, food consumption, or development of the conceptuses in the uterus at dose levels of less than 32 mg/kg/day. Developmental toxicity was demonstrated at 100 mg/kg/day as slightly impaired growth of the fetuses (reduced body weights and placental weights and reduced skeletal ossification). For developmental toxicity, the NOEL was 32 mg/kg/day and the LOEL was 100 mg/kg/day, based on reduced fetal body weights, reduced placental weights, retarded skeletal ossification of the cranium, sternebrae and 5th metacarpals.

13. In a second oral developmental toxicity study, pregnant Cr1:COBS CD (SD) BR rats were dosed with fenoxaprop-ethyl (96.2%) in corn oil at doses of 0, 10, 32, or 100 mg/kg/day from days 6 through 15 of gestation. For maternal toxicity, the NOEL was 32 mg/kg/day and the LOEL was 100 mg/kg/day, based on decreased body weight gain and increased liver weights. For developmental toxicity, the NOEL was 32 mg/kg/day and the LOEL was 100 mg/kg/day base on increase malformations, significant fetal weight reduction and increase total visceral and skeletal anomalies.

14. In an oral developmental toxicity study with groups of Himalayan [(Hoe:HIMK(SPFWiga)] rabbits were dosed at doses of fenoxaprop-ethyl (93%) in sesame oil at 0, 0.5, 12.5, 50.0 or 200 mg/kg/day from days 7 through 19 of gestation. For maternal toxicity, the NOEL was 12.5 mg/kg/day and the LOEL was 50.0 mg/kg/day, based on decreased food consumption and body weight gain. For developmental toxicity, the NOEL was 50 mg/kg/day and the LOEL was 200 mg/kg/day based on reduced fetal weights, placental weights, crown-rump lengths, and fetal survival, and increased litter and fetal incidence of rib anomalies and diaphragmatic hernias. No developmental toxicity was observed at doses of less than 50.0 mg/kg/day.

15. In a dermal developmental toxicity study, pregnant KFM-Han Wistar rats (25/dose) received repeated dermal applications of fenoxaprop-ethyl (96.5%) in sesame oil at doses of 0, 100, 300, or 1,000 mg/kg/day for 6 hours/day on days 6–15 of gestation. For maternal toxicity, the NOEL was >1,000 mg/kg/day (HDT); a LOEL was not observed. For developmental toxicity, the NOEL was 1,000 mg/kg/day; a LOEL was not observed. There were no treatment-related malformations or variations noted upon external, visceral, and skeletal examination of the fetuses.

16. In a dermal developmental toxicity study, fenoxaprop-ethyl (96.5%) in sesame oil was administered dermally to 16 Chinchilla rabbits (SPF quality) at dose levels of 0,100, 300, or 1,000 mg/kg/day for 6 hours/day on days 6–18 of gestation. For maternal toxicity, the NOEL was 1,000 mg/kg/day (HDT); a LOEL was not observed. There was no developmental toxicity demonstrated at any dose level. For developmental toxicity, the NOEL was 1,000 mg/kg/day; a LOEL was not observed.

17. In a 2-generation reproductive toxicity study, fenoxaprop-ethyl (97.2%) was administered to 30 WISTAR/HAN rats/sex/dose in their diet at doses of 0, 0.25, 1.5, or 9.0 mg/kg/day. Exposure to animals began at 7 weeks of age and lasted for 80 days prior to mating to produce F1a and F1b pups. At 21 days of age, F1b pups were selected to become the parents of the F2a and F2b litters. There were no treatment-related effects on mortality, clinical signs of toxicity, body weight, food consumption or reproductive parameters at any does level. For parental/systemic toxicity, the NOEL was 0.25 mg/kg/day) and the LOEL was 1.5 mg/kg/day based on decreased blood lipids. The NOEL for systemic toxicity was 0.25 mg/kg/day. For reproductive toxicity, the NOEL was 0.25 mg/kg/day and the LOEL was 1.5 mg/kg/day based on reduced pup body weights (F1a).

18. No developmental neurotoxicity data are required for fenoxaprop-ethyl. No effects on histopathology of the brain were observed in any of the studies in which these parameters were measured. There no evidence of developmental anomalies of the fetal nervous system in the prenatal developmental toxicity studies with rats or rabbits or in the 2-generation reproduction study in rats.

19. Studies on metabolism: In a rat metabolism study fenoxaprop-ethyl(U-14C-chlorophenyl; 98% radiochemical purity) was administered to male and female Wistar HOE: Wiskf (SPF 71) strain of rats (10–15 animals/dose/sex) by gavage as a single dose at levels of 2 or 10 mg/kg, or at a single dose at 2 mg/kg following a 4–day pretreatment with unlabeled fenoxaprop-ethyl at 2 mg/kg/day. Within 6 hours of dosing 83–109% of the administered radioactivity was recovered in the urine and feces, with a majority of the dose (51–65%) being recovered within 24 hours of dosing. Within 24 hours of dosing, urinary excretion accounted for 39–48% of the dose for females and 22–311% of the dose for males. The primary metabolite in urine of both sexes in each dose group was 6-chlorobenzoxazole-2-mercapturic acid, accounting for 22–50% of the total radioactivity in the urine (15–26% of the dose) The urine of female rats dosed either once at 10 mg/kg or repeatedly at 2 mg/kg also contained high levels (23–28% of dose) of 2-(4-(6-chloro-2-benzoxazolyloxy)-phenoxy)-propionic acid (the free acid of fenoxaprop-ethyl). At the 10 mg/kg dose, unchanged parent accounted for 24% of the fecal radioactivity (15% of dose) for male rats and 6% for female rats (1.7% of dose).

In a second rat metabolism study, fenoxaprop-ethyl (1–14C-dioxyphenyl; 96% radiochemical purity) was administered by gavage as a single dose

to male and female SPF Wistar strain rats (10 animals /sex) at 10 mg/kg body weight and to 15 females at 2 mg/kg body weight. Within 96 hours of dosing, 101.3% and 87.4% of the 10 gm/kg dose was recovered from male and female rats, respectively, and 108.8% of the 2 mg/kg dose was recovered from female rats. There were sex- and dose-related differences in excretion. In the 0- to 24-hour urine of male rats dosed at 10 mg/kg, 99% of the radioactivity was identified as 2-(4-hydroxyphenoxy)-propionic acid (HPP-acid), accounting for 47.5% of the administered dose. In female rats dosed at 10 mg/kg, the primary urinary metabolites were identified as HPP acid (27.5% of dose) and 2-(4-6-chloro-2-benzoxazolyloxy)-phenoxy)-propionic acid (the free acid of fenoxaprop-ethyl; 27% of dose). In feces of the 10 mg/kg dose groups, fenoxaprop-ethyl and its free acid accounted for 20.1 and 16.6% of the dose for males and 9.0 and 11.3% of the dose for females.

*B. Toxicological Endpoints*

1. *Acute toxicity*. EPA has selected for acute dietary risk assessment the NOEL of 32 mg/kg/day from the rat developmental toxicity study. The effects were increased incidence of fetuses with malformations (including skeletal defects, eye defects, absent innominate artery, diaphragmatic hernia and umbilical hernia at 100 mg/kg/day (LOEL). Population subgroup of concern is females 13+ years old.

An acute dietary risk assessment for the general population, including infants and children, (excluding the subgroup, females 13+ years old) is not required because no treatment-related effects attributable to a single exposure (dose) were seen in oral studies conducted with fenoxaprop-ethyl. A MOE of 100 is adequate to ensure protection for females 13+ years old.

2. *Short - and intermediate - term toxicity*. No dermal or systemic toxicity was seen in a dermal developmental toxicity study in rats and rabbits following repeated dermal applications of fenoxaprop-ethyl at 1,000 mg/kg/day (Limit-Dose). Also, no dermal or systemic toxicity was seen at the HDT (20 mg/kg/day) in a 21–day dermal toxicity study in rats.

3. *Intermediate-term inhalation toxicity*. A 6-week rat inhalation toxicity study demonstrated a NOEL = 0.015 mg/L based on decreases in total lipids, increased triglycerides, increased alkaline phosphatase, increased liver and kidney weights, and liver hypertrophy at 0.075 mg/L (LOEL).

*C. Cancer Dietary/Inhalation*

1. *Chronic toxicity*. EPA has established the RfD for fenoxaprop-ethyl at 0.0025 milligrams/kilogram/day (mg/kg/day). This RfD is based on reduced pup weights observed in a 2-generation rat reproductive toxicity study with a NOEL of 0.25 mg/kg/day. An uncertainity factor of 100 was used in calculating the RfD to account for both inter- and intra-species variations.

2. *Carcinogenicity*. Characterization of the carcinogenicity of fenoxaprop-ethyl has been referred to EPA Health Effects Division's Cancer Peer Review Committee. For the interim, a worst case and protective risk assessment was carried out by use of a linear low dose extrapolation method (Q1*) based on the increases in adrenal tumors in male mice. The Q1* for the adrenal tumors is $9.1 \times 10^{-2}$.

*D. Exposures and Risks*

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.430 (a)) for the combined residues of fenoxaprop-ethyl [ethyl 2-[4-[(6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoate] and its metabolites [2-[4-](6-chloro-2-benzoxazolyl)oxy]phenoxy]propanoic acid, and 6-chloro-2,3-dihydrobenzoxazol-2-one in or on a variety of raw agricultural commodities. Risk assessments were conducted by EPA to assess dietary exposures and risks from fenoxaprop-ethyl as follows:

i. *Acute exposure and risk.* Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1 day or single exposure. The NOEL for the acute dietary exposure was 32 mg/kg/day from a rat study. The Agency has determined that the uncertainty factor of 10 to account for enhanced sensitivity of infants and children should be removed for fenoxaprop-ethyl, and that the MOE of 100 to account for inter (10) and intra (10) species variation is adequate to insure protection for this population from exposure to fenoxaprop-ethyl, because in the rat developmental toxicity study, the fetal effects (malformations) were seen at maternally toxic doses (i.e., the LOEL was the same for both adults and fetuses).

From the acute dietary (food only) risk assessment a high-end exposure estimate of 0.001 mg/kg/day was calculated. This exposure yields a dietary (food only) MOE of 32,000 for females 13+ years, the population subgroup of concern. This risk estimate was highly conservative because it assume that 100% of barley and all other commodities having tolerances for residues of fenoxaprop-ethyl will contain residues at tolerance levels. Therefore, this is an overestimation of human dietary exposure. Use of anticipated residue values and percent crop-treated data will result in a lower acute dietary exposure estimate if estimated by probabilistic techniques.

ii. *Chronic exposure and risk*. The anticipated residues for existing fenoxaprop-ethyl uses (including the use on barley) result in Anticipated Residue Contribution that varies between 0.000009 and 0.000023 mg/kg/day for the population subgroups ( the U.S. Population, Nursing Infants (<1 year old), Non-Nursing Infants (> year old), Children (1–6 years old) , Children (7–12 years old) and Non-Hispanic Others); and occupied between 0.4% and 0.9% of the RfD.

Section 408(b)(2)(E) authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide chemicals that have been measured in food. If EPA relies on such information, EPA must require that data be provided five years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the levels anticipated. Following the initial data submission, EPA is authorized to require similar data on a time frame it deems appropriate. Section 408(b)(2)(F) states that the Agency may use data on the actual percent of crop treated for assessing chronic dietary risk only if the Agency can make the following findings: (1) that the data used are reliable and provide a valid a basis for showing the percentage of food derived from a crop that is likely to contain residues; (2) that the exposure estimate does not underestimate the exposure for any significant subpopulation and; (3) where data on regional pesticide use and food consumption are available, that the exposure estimate does not understate exposure for any regional population. In addition the Agency must provide for periodic evaluation of any estimates used.

The percent of crop treated estimates for fenoxaprop-ethyl were derived from federal and market survey data. EPA considers these data reliable. A range of estimates are supplied by this data and the upper end of this range was used for the exposure assessment. By using this upper end estimate of percent crop treated, the Agency is reasonably certain that exposure is not underestimated for any significant subpopulation. Further, regional consumption information is taken into account through EPA's

computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Review of this regional data allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency. To provide for the periodic evaluation of these estimates of percent crop treated as required by the section 408(b)(2)(F), EPA may require fenoxaprop-ethyl registrants to submit data on percent crop treated. As required by section 408(b)(2)(E), EPA will issue a data call-in for information relating to anticipated residues to be submitted no later than five years from the date of issuance of this tolerance.

In the absence of an Agency Cancer Assessment Review, the Health Effects Division of the Office of Pesticide Programs recommended a worst case and protective risk assessment using a linear low dose extrapolation method (Q1*) based on the increases in adrenal tumors in mice. The Q1* for the adrenal tumors was determined to be $9.1 \times 10^{-2}$. Based on the US population chronic dietary exposure of 0.00001 mg/kg/day, this results in a cancer risk estimate of $9.1 \times 10^{-7}$.

2. *From drinking water.* Based on the acute and chronic dietary (food) exposure and using default body weights and water consumption figures, acute and chronic drinking water levels of concern (DWLOC) for drinking water were calculated. To calculate the DWLOC, the acute or chronic dietary food exposure (from the DRES analysis) was subtracted from the acute toxicity NOEL or RfD, as appropriate. DWLOCs were then calculated using the default bodyweights and drinking water consumption figures.

For acute drinking water exposure for both adults and children, the level of concern was 960 ppm. For chronic and cancer exposure in drinking water the levels of concern were 80 ppb and 110 ppt, respectively. For adults, the estimate was based on a body weight of 70 kg and consumption of 2 liters of water per day; for children, a body weight of 10 kg and a consumption of 1 liter of water per day. Agency estimates for contamination of drinking water from the registered uses of fenoxaprop-ethyl is less than 1 ppb for acute exposure and less than 100 ppt for chronic exposure. These levels are not greater than levels of EPA concern.

3. *From non-dietary exposure.* Fenoxaprop-ethyl is currently registered for use on turfgrass including sod farms, commercial and residential turf and ornamentals. Applications to residential turf are done by professional applicators. There are no homeowner uses. There is a potential dermal exposure to infants to fenoxaprop-ethyl from the registered uses for lawn and turfgrass weed control but no dermal toxicity has been shown in animal studies. Potentially, infants and children may have some inhalation exposure due to residual residues of the pesticide on lawns but such exposure would be very low. Currently there are no inhalation exposure data required for post-application of pesticides to lawns and turf uses. As inhalation exposure for mixer/loaders is acceptable, the risk to children and infants from inhalation exposure under a much lower exposure scenario is characterized qualitatively as being extremely low. Exposure data are required for hand to mouth movements of infants and children; however, no acute dietary toxicity endpoints have been identified for fenoxaprop-ethyl for infants and children. There is an acute dietary toxicity endpoint; however, the only population subgroup of concern is females 13+ for developmental effects. The risk to this subgroup will be addressed later in this document under ''Aggregate Risk and Determination of Safety for Infants and Children.'' No acute dietary toxicity endpoints have been identified for the general populations.

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

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

EPA does not have, at this time, available data to determine whether fenoxaprop-ethyl 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, fenoxaprop-ethyl 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 fenoxaprop-ethyl has a common mechanism of toxicity with other substances.

### *E. Aggregate Risks and Determination of Safety for U.S. Population*

1. *Acute risk.* From the acute dietary (food use) risk assessment a high-end exposure estimate of 0.001 mg/kg/day was determined for females 13+ years, the population subgroup of concern for acute toxicity. This exposure yields a dietary MOE of 32,000. The potential contribution to acute exposure from residues in drinking water is minimal (1,000-fold less than EPA's level of concern) and would not result in an aggregate acute exposure that exceeds EPA's level of concern. EPA concludes there is a reasonable certainty that no acute harm will result from aggregate exposure to fenoxapro-ethyl residues.

2. *Chronic risk.* Using the ARC exposure assumptions described above, EPA has concluded that aggregate exposure to fenoxaprop-ethyl from food

will utilize less than 0.4% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non-nursing infants less than one year old. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to fenoxaprop-ethyl in drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the RfD. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fenoxaprop-ethyl residues.

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.

Fenoxaprop-ethyl is currently registered for use on turfgrass including sod production, commercial and residential turf and landscape ornamentals. No short- or intermediate-term dermal toxicity endpoints have been identified for fenoxaprop-ethyl. An inhalation endpoint has been identified, however, as the uses are outdoors, exposure from inhalation route should be considerable less than that determined for worker mixer/loaders, who have an MOE of 2,800. Additionally, based on the low level of chronic dietary exposure, the Agency concludes that aggregate short- and intermediate-term exposure is at a level below EPA's level of concern.

*F. Aggregate Cancer Risk for U.S. Population*

Based on a upper bound potency factor (Q1*) of 9.1 x $10^{-2}$ $(mg/kg/day)^{-1}$, the lifetime cancer risk from residues of fenoxaprop-ethyl in food commodities is 9.1 x $10^{-7}$. Taking into accoutn the exposure from residues in food, EPA has caluculated a drinking water level of concern which would not result in a greater negligible total cancer risk from chronic exposure to fenoxaprop-ethyl residues in food and water. The Agency's GENEEC screening model was then used to estimate maximum residues in surface water. This model estimates potential residues in surface water for use in ecological risk assessment. As such, it provides high-end values on the concentrations of pesticides that might be found in ecologically sensitive environments. The residue levels obtained for fenoxaprop-ethyl plus its acid metabolite in water using GENEEC do not exceed the drinking water level of concern. Therefore, EPA doen not expect there to be a greater than negligible cancer risk from chronic exposures to fenoxaprop-ethyl in drinking water and food.

*G. Aggregate Risks and Determination of Safety for Infants and Children*

1. *Safety factor for infants and children*— a. *In general.* In assessing the potential for additional sensitivity of infants and children to residues of fenoxaprop-ethyl, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from 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 post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard 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.

b. *Developmental toxicity studies.* In a oral developmental toxicity study with Wistar rats, the maternal NOEL was 32 mg/kg/day, based on reduction in body weight and food consumption. The developmental NOEL was 32 mg/kg/day, based on reduced fetal body weights, reduced placental weights and retarded skeletal ossification of the cranium, sternebrae and 5th metacarpals.

In a second oral developmental toxicity study with Crl:COBS CD (SD) BR rats, the maternal NOEL was 32 mg/kg/day, based on decreased body weight gain and increased liver weights. The developmental NOEL was 32 mg/kg/day, based on increase malformations, significant fetal weight reduction and increase total visceral and skeletal anomalies.

In a oral developmental toxicity study with Himalayan rabbits, the maternal NOEL was 12.5 mg/kg/day, based on decreased food consumption and body weight gain. The developmental NOEL was 50mg/kg/day, based on reduced fetal weights, placental weights, crown-rump lengths, fetal survival and increased litter and fetal incidence of rib anomalies and diaphragmatic hernias.

c. *Reproductive toxicity study.* In a rat reproduction study, the parental (systemic) NOEL was 0.25 mg/kg/day, based on decreased blood lipids. The reproductive (pup) NOEL was 0.25 mg/kg/day, based on reduced pup body weights.

d. *Pre- and post-natal sensitivity.* The toxicological data base is complete and adequate to determine pre- and post-natal toxicity. The prenatal developmental toxicity data demonstrated no indication of increased sensitivity of rats or rabbits to *in utero* exposure or repeated dermal applications of fenoxaprop-ethyl. The rat reproduction study did not identify any increased sensitivity of rats to *in utero* or postnatal exposure. Maternal and parental NOELs were equivalent to developmental or offspring NOELs.

e. *Conclusion.* Based on the above data, EPA determined that the standard safety factor would be adequate to protect the safety of infants and children thus the additional children's safety factor was removed.

2. *Acute risk.* The acute dietary (food only) MOE for females 13+ years old (accounts for both maternal and fetal exposure) was determined to be 32,000. This MOE was based on the developmental NOEL in rats of 32 mg/kg/day. This risk assessment assumed 100% crop-treated and tolerance level residues on all treated crops consumed, resulting in a significant over-estimate of dietary exposure. Despite the potential for exposure to fenoxaprop-ethyl in drinking water, EPA does not expect the acute aggregate exposure to exceed level of concern. The large acute dietary MOE determined for females 13+ years old provides assurance that there is a reasonable certainty of no harm from both females 13+ years and the pre-natal development of infants.

3. *Chronic risk.* Using the exposure assumptions described in this rule, the percentage of the RfD that will be utilized by chronic dietary (food only) exposure to residues of fenoxaprop-ethyl ranges from 0.4% for nursing infants less than one year old, up to 0.9% for non-nursing infants less than

one year old. Despite the potential for exposure to fenoxaprop-ethyl in drinking water, EPA does not expect the chronic aggregate exposure to exceed 100% of the RfD. Based on the nature of the residential uses, no chronic residential exposure is anticipate. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from chronic aggregate exposure to fenoxaprop-ethyl regulable residues.

**III. Other Considerations**

*A. Metabolism In Plants and Animals*

The metabolism of fenoxaprop-ethyl in plants and animals is adequately understood for purposes of these tolerances.

*B. Analytical Enforcement Methodology*

An adequate analytical method for determining the magnitude of residues in the raw agricultural commodities listed in this Final Rule has been evaluated by EPA and is published in the Pesticide Analytical Manual (PAM II). The method may be requested from: Calvin Furlow, Public Information Branch, Field Operations Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 1130A, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703–305–5937).

*C. Magnitude of Residues*

The nature of the residue in plants is adequately understood for the purposes of these tolerances.

*D. International Residue Limits*

No CODEX Maximum Residue Levels (MRLs) have been established for fenoxaprop-ethyl. Canadian MRLs for combined residues of fenoxaprop-ethyl, its free acid metabolite [2-[4-[(6-chloro-2-benzoxazolyl)oxy]propanoic acid] and 6-chloro-2,3-dihydrobenzoxazol-2-one have been established at 0.02 ppm for milk. This tolerance expression and level for milk is in harmony with subject tolerances of the final rule.

**IV. Conclusion**

Therefore, the tolerances are established for combined residues of fenoxaprop-ethyl [ethyl 2-[4-[(6-chloro-2-benzoxazolyl) oxy]phenoxy] propanoate] and its metabolites [2-[4-] (6-chloro-2-benzoxazolyl)oxy]phenoxy] propanoic acid and 6-chloro-2,3-dihydrobenzoxazol-2-one, in or on the following raw agricultural commodities: barley, grain at 0.05 ppm; and barley straw at 0.1 ppm.

**V. Objections and Hearing Requests**

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

Any person may, by June 22, 1998, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33(i). If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

**VI. Public Docket**

EPA has established a record for this rulemaking under docket control number [OPP–300635] (including any comments and data submitted electronically). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in Room 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments may be sent directly to EPA at:
opp-docket@epamail.epa.gov.

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

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

**VII. Regulatory Assessment Requirements**

This final rule establishes tolerances under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled Regulatory Planning and Review (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the

Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 1985, April 23, 1997).

In addition, since these tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. Nevertheless, the Agency has previously assessed whether establishing tolerances, exemptions from tolerances, raising tolerance levels or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950) and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

### VIII. Submission to Congress and the 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 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: April 8, 1998.

**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. 346a and 371.

2. In § 180.430, in paragraph (a) by alphabetically adding the following commodities to the table to read as follows:

**§ 180.430 Fenoxaprop-ethyl; tolerances for residues.**

(a) * * *

| Commodity | Parts per million |
|---|---|
| Barley, grain | 0.05 |
| Barley, straw | 0.1 |
| * * * * * | |

* * * * *

[FR Doc. 98–10395 Filed 4–21–98; 8:45 am]

BILLING CODE 6560–50–F

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Parts 264 and 265

**[FRL–6001–8]**

### Removal of Direct Final Rule for XL Project at OSi Specialties, Inc., Sistersville, WV

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Removal of direct final rule amendments.

**SUMMARY:** On March 6, 1998, EPA published a direct final rule entitled ''Project XL Site-Specific Rulemaking for OSi Specialties, Inc., Sistersville, West Virginia'' at 63 FR 11124–11147. That **Federal Register** notice provided that the direct final rule would be withdrawn if relevant adverse comments were received by March 27. Because EPA received such comments, EPA is now removing the amendments made by the direct final rule.

**EFFECTIVE DATE:** April 22, 1998.

**ADDRESSES:** *Docket.* A docket containing documents relevant to this action is available for public inspection and copying at the EPA's docket office located at Crystal Gateway, 1235 Jefferson Davis Highway, First Floor, Arlington, Virginia. The public is encouraged to phone in advance to review docket materials. Appointments can be scheduled by phoning the Docket Office at (703) 603–9230. Refer to RCRA docket number F–98–MCCP–FFFFF.

A duplicate copy of the docket is available for inspection and copying at U.S. EPA, Region 3, 841 Chestnut Street, Philadelphia, PA 19107–4431, during normal business hours. Persons wishing to view the duplicate docket at the Philadelphia location are encouraged to contact Mr. Tad Radzinski in advance, by telephoning (215) 566–2394.

**FOR FURTHER INFORMATION CONTACT:** Mr. Tad Radzinski, U.S. Environmental Protection Agency, Region 3 (3WC11), Waste Chemical Management Division, 841 Chestnut Street, Philadelphia, PA 19107–4431, (215) 566–2394.

**SUPPLEMENTARY INFORMATION:** A companion proposal to the direct final rule was published in the March 6, 1998 **Federal Register** (63 FR 11200–11202). EPA will address the comments received in response to that proposal but will not institute a second comment period.

However, persons who stil wish to comment on the companion proposal may do so by attending the hearing announced in the proposed rule section of today's **Federal Register**. In a subsequent final rule EPA will address the comments received on the proposed rule.

The Congressional Review Act, 5 U.S.C. Section 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. Section 804, however, exempts from Section 801 the following types of rules: rules of particular applicability; rules relating to Agency management or personnel; and rules of Agency organization, procedure, or practice that do not substantially affect the rights or obligations of non-Agency parties, 5 U.S.C. Section 804(3). EPA is not required to submit a rule report regarding today's action under Section 801 because this is rule of particular applicability.

Because the rule now being withdrawn is currently effective, EPA also finds that good cause exists under section 3010(b)(3) of RCRA (42 U.S.C. 6903(b)(3)) to publish this removal of the site-specific regulation with an immediate effective date, rather than having it take effect in 30 days. See 5 U.S.C. 553(d)(3). Today's removal of the direct final rule affects only one facility, and is limited in its scope to a removal of a temporary conditional deferral of a relatively narrow set of RCRA regulations. As such, the deferral was