representatives of State, local, and tribal governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

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

C. Executive Order 13084

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

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

IV. Submission to Congress and the Comptroller General

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

List of Subjects in 40 CFR Part 180

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

Dated: January 28, 1999.

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.

§ 180.317 [Amended]

2. In § 180.317, by amending the table in paragraph (b) by changing the date ''12/31/99'' to read ''12/31/01''.

[FR Doc. 99–3250 Filed 2–9–99; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300782; FRL–6056–4]

RIN 2070–AB78

Cymoxanil; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of the fungicide, cymoxanil, [2-cyano-N-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide], in or on imported tomatoes and grapes. E. I. DuPont De Nemours and Co., Inc. requested this tolerance under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996.

DATES: This regulation is effective February 10, 1999. Objections and requests for hearings must be received by EPA on or before April 12, 1999.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300782], 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–300782], must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 file format or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number [OPP–300782]. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: Mary Waller, Product Manager (PM) 21, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 247, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703)-308-9354, waller.mary@epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of November 15, 1995 (60 FR 57419-57422) (FRL–4971–5), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Pub. L. 104–170) announcing the filing of a pesticide petition (PP) for tolerance by DuPont, DuPont Agricultural Products, P.O. Box 80038, Wilmington, DE 19880-0038. This notice included a summary

of the petition prepared by DuPont, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR 180.503 (e) be amended by establishing tolerances for residues of the fungicide, cymoxanil, [2-cyano-N-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide], in or on imported tomatoes and grapes at 0.1 part per million (ppm).

I. Background and Statutory Findings

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

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

II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of cymoxanil and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for residues of Cymoxanil on imported tomatoes and grapes at 0.1 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 cymoxanil are discussed in this unit.

1. *Acute toxicity*. A battery of acute toxicity studies resulted in an acute oral LD_{50} > 760 milligrams/kilograms (mg/kg) for males and LD_{50} > 1,200 mg/kg for females; an acute dermal LD_{50} > 2,000 mg/kg for both sexes; an acute inhalation LC_{50} > 5.06 mg/L for both sexes; no ocular irritation; slight dermal irritation and a finding that the cymoxanil is not a dermal sensitizer.

2. *Subchronic toxicity*. i. A subchronic oral toxicity/neurotoxicity study in rats fed cymoxanil at dose levels of 0, 100, 750, 1,500, or 3,000 ppm (0, 6.54, 47.6, 102, or 224 mg/kg/day for males, and 0, 8.0, 59.9, 137, or 333 mg/kg/day for females) for approximately 97 days. A group of 10 rats/sex/dose were evaluated for subchronic systemic toxicity and a group of 10 rats/sex/dose underwent neurobehavioral testing at pre-test, 5, 9, and 13 weeks. The control and high-dose groups were assessed for neuropathology. The lowest-observed-adverse-effect level (LOAEL) for subchronic systemic toxicity is 1,500 ppm based on decreases in body weights, body weight gains, and food efficiency in the females, and body weight decreases and testicular and epididymal changes in the males. The no-observed-adverse-effect level (NOAEL) for subchronic systemic toxicity is 750 ppm.

ii. A subchronic oral toxicity study in mice fed doses of 0, 50, 500, 1,750, 3,500, or 7,000 ppm (average 0, 8.25, 82.4, 294, 566, or 1,306 mg/kg/day, for males; 0, 11.3, 121, 433, 846, or 1,130 mg/kg/day, for females) for 98 days showed a decrease in body weight gains in males dosed at 500, 1,750, and 3,500 ppm. An increase in the absolute liver and spleen weights was seen in females fed doses of 1,750 and 3,500 ppm. The NOAEL was established at 50 ppm for males and 500 ppm for females; the LOAEL was 500 ppm for males and 1,750 ppm for females.

iii. A subchronic oral toxicity study was conducted in dogs fed doses of 0, 100 or 200 ppm (0, 3 or 5 mg/kg/day) for 13 weeks, or at 250 ppm (5 mg/kg/day) for 2 weeks followed by 500 ppm (11 mg/kg/day) for 11 weeks. Reduced body weight gain and food consumption were observed in the 100 and 200 ppm females, and in both sexes from the 250/500 ppm groups and final body weights were reduced 32% for males (250 ppm group) and 42% for females (500 ppm group), compared to the controls. Both sexes in the 200 and 250/500 ppm treatment groups exhibited reduced red blood cell parameters, and an increased incidence of ketonuria. Red blood cell counts, hemoglobin and hematocrit values were lower in both sexes. Decreased calcium, total protein, albumin, phosphorus, and chloride concentrations, and A/G ratio were also observed in the blood serum of the 250/500 ppm males and females. Males in the 250/500 ppm group had lower epididymal and testicular weights, and aspermatogenesis was observed. The 250/500 ppm females had lower kidney, liver, and thyroid gland weights. No associated microscopic lesions or corresponding decreases in relative organ weights were observed. One euthanized female from the 250/500 ppm group had dark red contents and reddened mucosa throughout the gastrointestinal tract. The LOAEL is 3 mg/kg/day (100 ppm) for dogs based on decreased body weights and food consumption in females. The NOAEL was not established.

iv. In a 28-day dermal toxicity study, cymoxanil was applied to the shaved backs of rats, for 6 hrs/day at doses of 0, 50, 500, and 1,000 mg/kg/day. There were no demonstrated effects and no compound-related histopathology. The NOAEL for systemic toxicity and dermal irritation was 1,000 mg/kg/day, the highest dose tested (HDT).

3. *Chronic toxicity*. i. A combined chronic/carcinogenicity study was conducted in rats fed cymoxanil at doses of 0, 50, 100, 700, or 2,000 ppm (0, 1.98, 4.08, 30.3, and 90.1 mg/kg/day for males, and 0, 2.71, 5.36, 38.4, and 126 mg/kg/day for females) for 23 months. A satellite group was included and terminated at 52 weeks. Because of poor survival in controls and treated rats, the study was terminated after 23 months. Survival was 24-45% and 21-40% in the male and female groups, respectively.

Chronic toxicity observed at 126 mg/kg/day in females, included significant decreases in mean body weight and body weight gains, a decrease in food efficiency, and increased incidences of non-neoplastic lesions in several organ systems including the lungs, intestines, and mesenteric lymph nodes. In females receiving 38.4 mg/kg/day, chronic toxicity was characterized by increased incidences of non-neoplastic lesions of the lungs, liver, sciatic nerve, and eyes (retinal atrophy). Chronic toxicity in the males dosed at 30.3 or 90.1 mg/kg/day included aggressiveness and/or

hyperactivity, decreased mean body weight and body weight gain, decreased food efficiency, and increased incidence of elongate spermatid degeneration and retinal atrophy. No important effects were observed in the low- and low-mid-dose groups. No increases in the incidences of any neoplasm was observed in dosed animals. The chronic LOAEL was 30.3 mg/kg/day for males and 38.4 mg/kg/day for females based on histologic changes detected in several organs of the females and decreased body weight, body weight gains, and food efficiency observed in the males and females. The chronic NOAEL is 4.08 mg/kg/day for males and 5.36 mg/kg/day for females. Under the conditions of this study, there was no evidence of carcinogenic potential.

ii. A chronic toxicity study was conducted in dogs fed cymoxanil at doses of 0, 25, 50 or 100 ppm for females (0, 0.7, 1.6, or 3.1 mg/kg/day) and 0, 50, 100, or 200 ppm for males (0, 1.8, 3.0, or 5.7 mg/kg/day) for 52 weeks. The only effect seen in females in the 100 ppm treatment group was weight loss during the first week of the study. No effect was observed in females in the 25 or 50 ppm group, or in males in the 50 or 100 ppm group. The LOAEL was 200 ppm for males based on depressed weight gains through week 12 and changes in hematology and blood chemistry. No LOAEL was established for females. The NOAEL was 100 ppm.

4. *Carcinogenicity.* i. A combined chronic/carcinogenicity study, conducted in rats (described in the chronic toxicity section in Unit II.A.3.i in the preamble of this document) showed no evidence of carcinogenic potential.

ii. A carcinogenicity study was conducted in mice fed cymoxanil at doses of 0, 30, 300, 1,500, and 3,000 ppm (0, 4.19, 42.0, 216, and 446 mg/kg/day for males; 0, 5.83, 58.1, 298, and 582 mg/kg/day for females) for approximately 80 weeks. Two additional groups were sacrificed at 31-32 days for cell proliferation and biochemical evaluation.

Males and females dosed at 300 ppm and above exhibited alterations in organ weights and microscopic pathology. Affected organs were the testes and epididymis in males, the gastrointestinal tract in females, and the liver in both sexes. Male mice fed 300 ppm exhibited treatment-related increased frequency of sperm cyst/cystic dilation, tubular dilation, and increased lymphoid aggregate. Centrilobular apoptotic hepatocytes, pigment-containing macrophages, and granuloma were detected in males dosed with 300 ppm. Elevated centrilobular hepatocellular hypertrophy and associated significant increases in liver weight in males dosed with 300 ppm was considered a pharmacologic response to cymoxanil. Hyperplastic gastropathy increased significantly in 300 ppm female mice and cystic enteropathy of the small intestine showed a significant positive trend. At the 1,500 ppm dose, decreases in body weight, body weight gain, and food efficiencies were observed in males and females. In addition to the testicular and epididymal abnormalities observed at the lower dose, the 1,500 ppm males exhibited increased incidence of sperm granuloma and bilateral oligospermia. Females at 1,500 ppm exhibited the microscopic liver abnormalities seen in males at the lower dose. Cystic enteropathy was observed in males at 1,500 ppm. At 3,000 ppm, there were significant reductions in body weight, body weight gain, food consumption, and food efficiencies in males and females. Survival over 18 months was decreased in the 3,000 ppm females, 57% compared to 69% in controls. Early deaths among high-dose females were attributed to pancreatic acinar cell necrosis and/or stress, evidenced by splenal and thymic atrophy and bone marrow congestion. Females dosed with 3,000 ppm exhibited increased frequency of pallor, weakness, and hunching over. Male mice dosed with 3,000 ppm showed hematological signs of decreased circulating erythrocyte mass at the 12-month evaluation. The high dose also resulted in gross and microscopic pathology of the liver, gastrointestinal tract, and testes. Dosing was considered adequate based on decreased body weight gains and an increase in non-neoplastic lesions in both sexes relative to the controls at the highest dose level.

The LOAEL was 300 ppm based on toxicity to the testes and epididymides in males and toxicity to the gastrointestinal mucosa in females. The NOAEL was 30 ppm. Under the conditions of this study, there was no evidence of a carcinogenic effect.

5. *Developmental toxicity.* i. A prenatal developmental toxicity study was conducted in rats gavaged with cymoxanil on days 7-16 of gestation at dose levels of 0, 10, 25, 75, or 150 mg/kg/day. The maternal LOAEL was 25 mg/kg/day based upon reduced body weight, body weight change, and food consumption. The maternal NOAEL was 10 mg/kg/day. The developmental LOAEL was 25 mg/kg/day based upon a significant increase in overall malformations and a generalized dose-related delay in skeletal ossification. Fetal body weights were significantly decreased at 75 and 150 mg/kg/day. At 150 mg/kg/day, increased early resorptions resulted in reduced litter sizes. The developmental NOAEL was 10 mg/kg/day.

ii. A prenatal developmental toxicity study was conducted in rabbits gavaged with cymoxanil on days 6-18 of gestation at dose levels of 0, 4, 8, or 16 mg/kg/day. There was no evidence of treatment-related maternal or developmental toxicity. A maternal and developmental LOAEL was not determined. The maternal and developmental NOAEL was ≥ 16 mg/kg/day. When considered along with other prenatal developmental toxicity studies in rabbits, this study provides acceptable information that assists in determining the overall maternal and developmental NOAEL and LOAEL for cymoxanil in a nonrodent species.

iii. A prenatal developmental toxicity study was conducted in rabbits gavaged with cymoxanil on days 6-18 of gestation at dose levels of 0, 8, 16, or 32 mg/kg/day. Evaluation of litter data and assessment of embryonic and fetal development revealed treatment-related increases in the incidence of malformations in all treated groups. The maternal LOAEL was 32 mg/kg/day based upon increased incidences of cold ears, anorexia, and/or few feces and body weight loss during the first four days of treatment; the maternal NOAEL was 16 mg/kg/day. The developmental LOAEL was 8 mg/kg/day based upon an increase in skeletal malformations of the cervical and thoracic vertebrae and ribs. The developmental NOAEL was ≤ 8 mg/kg/day but could not be determined. Although the results of this study suggested an additional susceptibility of fetal rabbits to *in utero* exposure with cymoxanil, uncertainties regarding the source of the parental rabbits substantially reduce the confidence that the observed skeletal anomalies are solely related to treatment.

iv. A prenatal developmental toxicity study was conducted in rabbits gavaged with cymoxanil on days 6-18 of gestation at dose levels of 0, 1, 4, 8, or 32 mg/kg/day. The females showed significant post-treatment increases in body weight gain at 8 and 32 mg/kg/day. The maternal LOAEL was 8 mg/kg/day based upon a significant dose-related rebound in maternal body weight. The maternal NOAEL was 4 mg/kg/day. The developmental LOAEL was 8 mg/kg/day based upon an increase in skeletal malformations of the cervical and thoracic vertebrae and ribs; and, at 32 mg/kg/day, cleft palate was observed. The developmental NOAEL was 4 mg/kg/day.

6. *Reproductive toxicity*. i. A 2-generation reproduction study was conducted in rats fed cymoxanil at doses of 0, 100, 500, or 1,500 ppm (equivalent to 0, 6.5, 32.1, or 97.9 mg/kg/day in males and 0, 7.9, 40.6, or 130 mg/kg/day in females), over two consecutive generations. No effects of treatment were observed at 100 ppm. The parental systemic NOAEL was 100 ppm. The parental systemic LOAEL was 500 ppm, based upon reduced pre-mating body weight, body weight gain, and food consumption for F1 males; and decreased gestation and lactation body weight for F1 females. The offspring NOAEL was 100 ppm and the offspring LOAEL was 500 ppm, based upon decreased F1 pup viability on postnatal days 0-4 and on a significant reduction in F2b pup weight.

7. *Neurotoxicity*. i. The neurotoxicity portion of the subchronic/neurotoxicity study in rats demonstrated no effects on the Functional Observation Battery or on motor activity after 5, 9, and 13 weeks at dietary doses of cymoxanil of 0, 100, 750, 1,500, or 3,000 ppm (0, 6.54, 47.6, 102, or 224 mg/kg/day for males, and 0, 8.0, 59.9, 137, or 333 mg/kg/day for females), for 97 days. There were no treatment-related gross or microscopic findings detected in the nervous system or skeletal muscles. Grip strength and foot splay measurements were decreased (non-significantly) in males at 224 mg/kg/day in the 13-week subchronic neurotoxicity study in rats, although these findings occurred in conjunction with decreased body weight. A LOAEL for neurobehavioral and neuropathic effects was not established. The NOAEL for neurotoxicity was 3,000 ppm.

ii. No evidence of developmental anomalies of the fetal nervous system were observed in the prenatal developmental toxicity studies in either rats, or rabbits at maternally toxic oral doses up to 25 and 32 mg/kg/day, respectively. In addition, there was no evidence of behavioral or neurological effects on the offspring in the 2-generation reproduction study in rats.

iii. There were no major data gaps for the assessment of potential neurotoxicological effects due to cymoxanil. However, following a weight-of-the evidence review of the database, which suggested that neuropathological lesions, changes in brain weight, axon/myelin degeneration, and retinal atrophy could result from long-term exposure to cymoxanil, the Agency will require a confirmatory developmental neurotoxicity study in rats.

8. *Mutagenicity*. Mutagenicity studies with cymoxanil included gene mutation assays in bacterial and mammalian cells, a mouse micronucleus assay and an *in vivo*/*in vitro* unscheduled DNA synthesis (UDS) assay in rats. These studies did not demonstrate mutagenicity. An *in vitro* unscheduled DNA synthesis assay-primary rat hepatocytes was positive from 5-500 µg/mL and cytotoxicity was seen at concentrations of ≥500 µg/mL. A chromosome aberrations in human lymphocytes assay was positive at 100 - 1,500 µg/mL, positive at 1,250 and 1,500 µg/mL -S9 and 850-1,500 µg/mL +S9.

9. *Metabolism*. A metabolism study was conducted by gavaging rats with single doses of radiolabeled cymoxanil at 2.5 or 120 mg/kg, or as a single dose (2.5 mg/kg) following a 14-day pretreatment with unlabeled cymoxanil (2.5 mg/kg/day). Radiolabeled cymoxanil was readily absorbed through the intestinal tract. Maximum plasma concentrations were attained within 3-5 hours of dosing, then declined steadily. Dose rate and pretreatment did not appear to affect absorption.

Elimination was not dependent on sex or dosing regimen; occurring predominantly in the urine (63.8-74.8%), during the first 24 hours (58-66%). Fecal excretion accounted for 15.7-23.6% of the dose, and radioactivity in the tissues and carcasses accounted for <1% of the dose at sacrifice for all three dosing regimens. A pilot study indicated that approximately 3% of the dose would be expected to be respired as $^{14}CO_2$.

For each dosing regimen, there was also no difference between male and female rats in the distribution of radioactivity in tissues. No accumulation of radioactivity was observed over time in any tissues. However, in comparison, concentrations of radioactivity were highest in liver and kidney and lowest in brain tissue at 96 hours post-dosing sacrifice.

Peak plasma concentrations for the low and high dose groups were attained within 3-5 hours of dosing, and both dose groups had similar elimination half-lives from plasma, suggesting that the metabolic process was not saturated by the high dose. In addition, there was a 40-fold difference in the area under the curve for plasma from the low and high dose groups, approximating the 48-fold difference in the dose levels.

The metabolite profile in urine and feces was similar between sexes and among dose groups. In the urine, the majority of the radioactivity (36.7-55% of the dose) was free and/or conjugated [^{14}C]glycine, and 2-cyano-2-methoxyiminoacetic acid (IN-W3595) (6.5-33% of the dose) was also found. Intact [^{14}C] cymoxanil was not detected. In feces, trace levels (<1% dose) of [^{14}C] cymoxanil and IN-W3595 were detected, but the majority of radioactivity was the free and conjugated [^{14}C] glycine (8.5-13.1% of the dose). The data indicate that the principal pathway for the elimination of cymoxanil from rats is via renal elimination.

Based on the data, the proposed metabolic pathway involves hydrolysis of cymoxanil to IN-W3595, which is then degraded to glycine. Subsequently, glycine is incorporated into natural constituents or further metabolized.

10. *Other toxicological considerations*. The available studies indicate that cymoxanil is not mutagenic in bacterial or cultured mammalian cells. There is, however, confirmed evidence of clastogenic activity and UDS induction *in vitro*. In contrast, cymoxanil was neither clastogenic nor aneurogenic in mouse bone marrow cells and did not induce a genotoxic response in rat somatic or germinal cells. Accordingly, the negative results from the mouse bone marrow micronucleus assay support the lack of carcinogenic effect in the rat and mouse long-term feeding study.

Similarity of clinical signs were observed in the micronucleus and *in vivo* UDS assay, but the confidence in the negative findings of the *in vivo* UDS assay was not high because of a failure to demonstrate that test material reached either target tissue. It was concluded that the test may have been inadequate because of the short interval between dosing and cell harvest. Therefore, the Agency will be requiring that a supplemental rat dominant lethal assay be conducted to determine if any effects are noted which are associated with genetic damage to male germinal cells.

B. Toxicological Endpoints

1. *Acute toxicity*. To assess acute dietary exposure in the subpopulation of concern (females 13+), the Agency used a NOAEL of 4 mg/kg/day from prenatal developmental toxicity studies in rabbits based on an increase in skeletal malformations of the cervical and thoracic vertebrae and ribs at 8 mg/kg/day. EPA determined that the 10x factor for infants and children (required by FQPA) should be reduced to 3x and an MOE of 300 is required because of neuropathological lesions observed in the chronic toxicity study in rats and the need for a developmental neurotoxicity study. A dose and endpoint were not selected for the general population (including infants and children) because there were no

effects observed in oral toxicology studies, including maternal toxicity in the developmental toxicity studies in rats or rabbits, that could be attributable to a single exposure.

2. *Short- and intermediate- term toxicity*. The Agency determined that this dose and endpoint was not applicable for risk assessment because no dermal or systemic toxicity was seen in a 28 day dermal toxicity study.

3. *Chronic toxicity*. i. EPA has established the Reference dose (RfD) for cymoxanil at 0.013 mg/kg/day. This RfD is based on a chronic feeding study in rats with a NOAEL of 4.08 mg/kg/day and an uncertainty factor of 300.

ii. Based on the use pattern, chronic dermal exposure is not anticipated; therefore, a long-term dermal risk assessment is not required.

4. *Carcinogenicity*. Based on the lack of evidence of carcinogenicity in mice and rats, EPA has classified cymoxanil as a ''not likely'' human carcinogen, according to EPA's Proposed Guidelines for Carcinogen Risk Assessment.

C. Exposures and Risks

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.503 (a)) for the residues of cymoxanil, in or on potatoes and for the Section 18 emergency exemption use of cymoxanil in or on tomatoes (40 CFR 180.503 (b)). Risk assessments were conducted by EPA to assess dietary exposures from cymoxanil 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 one day or single exposure. The acute exposure analysis for the females (13+) subgroup was performed using tolerance level residues and assuming 100% crop treated. The resulting MOE's are as follows: 2,100 for females (13+/ pregnant/not nursing), 2,200 for females (13+/nursing), 980 for females (13-19 yrs/not pregnant or nursing), 1,500 for females (20+ years/not pregnant or nursing), and 1,200 for females (13-50 years). The estimated acute MOEs of 980 or more (MOE of 300 required) demonstrate no acute dietary concern.

ii. *Chronic exposure and risk*. The chronic dietary risk analysis used the RfD of 0.013 mg/kg/day. Chronic dietary exposure estimates utilized tolerance level residues on potatoes, tomatoes, and grapes, and assumed 100% of the crops were treated. The risk assessment resulted in use of < 3% of the RfD for the general population, < 2% of the RfD for infants (< 1 year), and < 5% of the RfD for children (1-6 years). The chronic dietary risk does not exceed the Agency's level of concern.

2. *From drinking water.* Cymoxanil appears to be mobile in soils. However, the rapid dissipation of cymoxanil in the environment precludes the possibility of extensive leaching. No detections of cymoxanil were observed below the 0-15 cm soil depth. The degradates of cymoxanil are mobile but the aerobic soil metabolism study showed they are short-lived. Therefore, cymoxanil and its degradates should not pose a threat to ground water.

EPA 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 maximum label rate to estimate surface water concentration. In addition, the model contains a number of conservative underlying assumptions. Therefore, the drinking water concentrations derived from GENEEC for surface water are likely to be overestimated.

EPA uses drinking water levels of comparison (DWLOCs) as a surrogate measure to capture risk associated with exposure to pesticides in drinking water. A DWLOC is the concentration of a pesticide in drinking water that would be acceptable as an upper limit in light of total aggregate exposure to that pesticide from food, water, and residential uses. A DWLOC will vary depending on the residue level in foods, the toxicity endpoint and with drinking water consumption patterns and body weights for specific subpopulations

i. *Acute exposure and risk.* The acute DWLOC for cymoxanil was calculated for the subpopulation of concern, females (13+ years) to be 280 parts per billion (ppb). The estimated maximum concentration of cymoxanil in surface water (4.13 ppb) derived from GENEEC is much lower than EPA's DWLOC of 280 ppb. Therefore, EPA concludes with reasonable certainty that residues of cymoxanil in drinking water do not contribute significantly to the aggregate acute human health risk.

ii. *Chronic exposure and risk.* The chronic DWLOCs are 440 ppb for the U.S. population and 120 ppb for the most sensitive subgroup, children (1-6 years). The DWLOCs are substantially higher than the GENEEC 56-day estimated environmental concentration of 0.19 ppb for cymoxanil in surface water. Therefore, EPA concludes with reasonable certainty that residues of cymoxanil in drinking water do not contribute significantly to the aggregate chronic human health risk.

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

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

EPA does not have, at this time, available data to determine whether cymoxanil 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, cymoxanil 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 cymoxanil 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*. For the population subgroup of concern, females (13+ years old), the lowest calculated MOE for dietary (food only) exposure is 980. The acute DWLOC for cymoxanil in females (13+ years old) is 280 ppb. The Agency concludes that there is a reasonable certainty that no harm will result from acute aggregate exposure to cymoxanil residues for children.

2. *Chronic risk*. Using the TMRC exposure assumptions described in this unit, EPA has concluded that aggregate exposure to cymoxanil from food will utilize 2 % of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is children 1-6 years 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 cymoxanil 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 cymoxanil residues.

3. *Aggregate cancer risk for U.S. population*. EPA has classified cymoxanil as a ''not likely'' human carcinogen, based on the lack of evidence of carcinogenicity in mice and rats, and therefore has a reasonable certainty that no harm will result from exposure to residues of cymoxanil.

4. *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 cymoxanil 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 cymoxanil, EPA considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre-and 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 margin of exposure (MOE) analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor.

ii. *Pre- and post-natal sensitivity*. The Agency determined that a developmental neurotoxicity study is required for cymoxanil. Evidence that support requiring a developmental neurotoxicity study include (1) clinical neurotoxicity in the database, (2) clinical observations of hyperactivity and aggressiveness were reported in males at 700 and 2,000 ppm (30.3 and 90.1 mg/kg/day) in the chronic toxicity study in rats, (3) diarrhea was noted in males in the subchronic dog study at 5-11 mg/kg/day, (4) grip strength and foot splay measurements were decreased (non-significantly) in males at 224 mg/kg/day in the 13-week subchronic neurotoxicity study in rats, although it was noted that these findings occurred in conjunction with decreased body weight, (5) brain weight changes and/or neuropathology in the cymoxanil database, particularly following long-term exposure, (6) absolute brain weight was decreased in both sexes at 1,500 and 3,000 ppm (216/298 and 446/582 mg/kg/day for M/F, respectively) in the chronic toxicity study in mice, (7) equivocal incidences of myelin degeneration were observed in males at 3,000 ppm (224 mg/kg/day) in the 13-week neurotoxicity study in rats, (8) axon/myelin degeneration of the sciatic nerve was observed in females at 700 and 2,000 ppm (38.4 and 126 mg/kg/day) in the chronic toxicity study in rats, and (9) retinal atrophy was reported in both sexes at 700 and 2,000 ppm (30.3/38.4 and 90.1/126 mg/kg/day, respectively) in the chronic toxicity study in rats.

The developmental toxicity and multigeneration reproduction study data demonstrated no indication of increased susceptibility of rats or rabbits *in utero* and/or postnatal exposure to cymoxanil. Overall, in the developmental toxicity studies in rats and rabbits, and in the 2-generation reproductive toxicity study with cymoxanil in rats, offspring toxicity was observed only at treatment levels which were toxic to parental adults. Although increased fetal susceptibility was suggested by the results of one prenatal developmental toxicity study, in which the fetal NOAEL was lower than the maternal NOAEL, uncertainties regarding the source of the rabbits substantially reduced the confidence that observed skeletal anomalies were solely related to treatment, and the results of this study were not duplicated in other rabbit developmental studies.

iii. *Conclusion*. Except for the pending requirements for a developmental neurotoxicity study and a rat dominant lethal study (both in rebuttal), there is a complete toxicity database for cymoxanil and exposure data is complete or is estimated based on data that reasonably accounts for potential exposures. The Agency determined that for cymoxanil, the 10x factor should be reduced to 3x, taking into account all of the following information: no sensitivity to perinatal animals, no data gaps for evaluating potential effects on offspring following *in utero* and/or postnatal exposure to cymoxanil by standard required studies and a weight-of-evidence review indicating that neuropathological lesions could result from long-term exposure to cymoxanil. A developmental neurotoxicity study and a rat dominant lethal study (both in rebuttal) were required to resolve concerns for potential genetic damage to male germinal cells that may be associated with the effects noted in the reproduction, developmental subchronic and chronic studies.

2. *Acute risk*. The MOE for the acute dietary (food only) risk assessment for the population subgroup of concern, females 13+ years, not pregnant or nursing, was estimated at 980. This risk estimate does not exceed the Agency's level of concern. EPA has calculated drinking water levels of comparison (DWLOCs) for acute exposure to cymoxanil in drinking water for females (13+ years old) to be 280 ppb. Therefore, EPA concludes with reasonable certainty that the potential risks from aggregate acute exposure (food & water) would not exceed the Agency's level of concern.

3. *Chronic risk*. Using the exposure assumptions described above, EPA has concluded that aggregate exposure to cymoxanil from food will utilize less than 5% of the RfD for infants and children. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to cymoxanil in drinking water, EPA does not expect the aggregate exposure to exceed 100% of the RfD.

4. *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 cymoxanil residues.

III. Other Considerations

A. Metabolism In Plants and Animals

The nature of the residue in plants and animals is adequately understood. Only the parent cymoxanil compound is of regulatory concern. There are no animal feed items currently associated with grapes and tomatoes.

B. Analytical Enforcement Methodology

An adequate enforcement method, AMR 3060-94, is available to enforce the tolerance on grapes and tomatoes. Quantitation is by HPLC/UV. These methods have been submitted for publication in PAM I. The methods are available to anyone who is interested in pesticide residue enforcement from: Calvin Furlow, Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., S.W., Washington, DC 20460. Office location and telephone number: Crystal Mall #2, Rm 101FF, 1921 Jefferson Davis Hwy., (701) 305-5229.

C. Magnitude of Residues

EPA has concluded that residue data submitted in support of the tolerances for grapes and tomatoes at 0.1 ppm, indicate that the tolerances requested by the petitioner are adequate.

D. International Residue Limits

There are no Codex or Canadian residue limits established for cymoxanil on grapes or tomatoes. Therefore, no compatibility problems exist for the proposed tolerances.

E. Rotational Crop Restrictions

Rotational crop restrictions are not an issue as there are no U.S. registrations associated with imported tolerances.

IV. Conclusion

Therefore, tolerances are established for residues of cymoxanil, [2-cyano-N-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide] in or on the raw agricultural commodities, imported grapes and tomatoes 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 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 April 12, 1999, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given under the **ADDRESSES** section (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this regulation. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33(i). EPA is authorized to waive any fee requirement ''when in the judgement of the Administrator such a waiver or refund is equitable and not contrary to the purpose of this subsection.'' For additional information regarding tolerance objection fee waivers, contact James Tompkins, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 239, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305-5697, tompkins.jim@epa.gov. Requests for waiver of tolerance objection fees should be sent to James Hollins, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

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

VI. Public Record and Electronic Submissions

EPA has established a record for this regulation under docket control number [OPP–300782] (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 Hwy., Arlington, VA.

Objections and hearing requests may be sent by e-mail directly to EPA at: opp-docket@epa.gov.

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

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

VII. Regulatory Assessment Requirements

A. Certain Acts and Executive Orders

This final rule establishes a tolerance under section 408(d) of the FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104-4). Nor does it require any prior consultation as specficed by Executive Order 12875, entitled *Enhancing the Intergovernmental Partnership* (58 FR 58093, October 28, 1993), or special considerations as required by Executive

Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997).

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

B. Executive Order 12875

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

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

C. Executive Order 13084

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

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

VIII. Submission to Congress and the Comptroller General

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

List of Subjects in 40 CFR Part 180

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

Dated: January 25, 1999.

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 § 180.503 by adding paragraph (e) to read as follows:

§ 180.503 Cymoxanil; tolerance for residues.

* * * * *

(e) *Import.* Import tolerances are established for residues of the fungicide [2-cyano-N-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide] expressed as cymoxanil in or on the following food commodities:

Commodity	Parts per million
Grapes, imported	0.1
Tomatoes, imported	0.1

[FR Doc. 99–3249 Filed 2–9–99; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300790; FRL–6059–8]

RIN 2070–AB78

Tebufenozide; Extension of Tolerance for Emergency Exemptions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation extends a time-limited tolerance for residues of the insecticide tebufenozide and its metabolites in or on turnip tops at 5.0 part per million (ppm) for an additional 18–month period. This tolerance will expire and is revoked on August 31, 2000. This action is in response to EPA's granting of an emergency exemption under section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) authorizing use of the pesticide on turnip tops. Section 408(l)(6) of the Federal Food, Drug, and Cosmetic Act requires EPA to establish a time-limited tolerance or exemption from the requirement for a