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 address in ''ADDRESSES'' at the beginning of this document.

III. Regulatory Assessment Requirements

This final rule extends time-limited tolerances that were previously extended by EPA 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). In addition, this final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 19885, April 23, 1997).

Since this extension of existing time-limited tolerances does 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.

IV. Submission to Congress and the General Accounting Office

Under 5 U.S.C. 801(a)(1)(A), as added by the Small Business Regulatory Enforcement Fairness Act of 1996, EPA submitted a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the General Accounting Office prior to publication of this rule in today's **Federal Register**. This is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

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

Dated: April 22, 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.

§ 180.509 [Amended]

2. In § 180.509, the table in paragraph (b) is amended by changing the date ''August 1, 1998'' to read ''2/1/00'', wherever it appears.

[FR Doc. 98–11763 Filed 5–5–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300653; FRL–5788–5]

RIN 2070–AB78

Cymoxanil; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for residues of the fungicide, cymoxanil, 2-cyano-*N*-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide, in or on potatoes. E.I. DuPont de Nemours & Company submitted a petition under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170) requesting this tolerance.

DATES: This regulation is effective May 6, 1998. Objections and requests for hearings must be received by EPA on or before July 6, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300653], 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–300653], 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 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–300653]. 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, Acting 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: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 308–9354, e-mail: waller.mary@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of (July 25, 1997, 62 FR 40075)(FRL–5726–4), 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 pesticide petition (PP 7F4805) for a tolerance by E.I. DuPont de Nemours and Company, E. I. DuPont Agricultural Products, Walker's Mill,

Barley Mill Plaza, P.O. Box 80038, Wilmington, Deleware, 19880–0038. This notice included a summary of the petition prepared by E.I. DuPont de Nemours & Company, the registrant. No comments were received in response to the notice of filing.

The petition requested that 40 CFR 180.503 be amended by establishing a tolerance for residues of the fungicide cymoxanil, 2-cyano-*N*-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide, in or on potatoes at 0.05 parts per million (ppm).

I. Risk Assessment and Statutory Findings

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

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

A. Toxicity

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

Once a study has been evaluated and the observed effects have been determined to be threshold effects, EPA generally divides the NOEL from the study with the lowest NOEL by an uncertainty factor (usually 100 or more) to determine the Reference Dose (RfD). The RfD is a level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. An uncertainty factor (sometimes called a ''safety factor'') of 100 is commonly used since it is assumed that people may be up to 10 times more sensitive to pesticides than the test animals, and that one person or subgroup of the population (such as infants and children) could be up to 10 times more sensitive to a pesticide than another. In addition, EPA assesses the potential risks to infants and children based on the weight of the evidence of the toxicology studies and determines whether an additional uncertainty factor is warranted. Thus, an aggregate daily exposure to a pesticide residue at or below the RfD (expressed as 100 percent 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 hundredfold MOE is based on the same rationale as the hundredfold 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 percent 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 (children 1 to 6 years old) was not regionally based.

II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action, EPA has sufficient data to assess the hazards of cymoxanil to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for residues of cymoxanil 2-cyano-*N*-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide in or on potatoes. EPA's assessment of the dietary exposures and risks associated with establishing this 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 is discussed below.

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 for both sexes; no ocular irritation; slight dermal irritation and a finding that the cymoxanil is not a dermal sensitizer.

2. *Subchronic toxicity.* a. 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 LOEL 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-effect level (NOEL) for subchronic systemic toxicity is 750 ppm.

b. A subchronic oral study in mice fed doses of 50, 500, 1,750, 3,500, or 7,000 ppm (average 8.25, 82.4, 294, 566, or 1,306 mg/kg/day, for males; 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 NOEL was established at 50 ppm for males and 500 ppm for females; the LOEL was 500 ppm for males and 1,750 ppm for females.

c. A subchronic oral toxicity study was conducted in dogs fed doses of 100 or 200 ppm (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. The 250/500 ppm males had lower epididymal and testicular weights, and aspermatogenesis was observed. The LOEL is 3 mg/kg body weight/day (100 ppm) for dogs based on decreased body weights and food consumption in females. The NOEL was not established.

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

3. *Chronic toxicity.* a. 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 percent and 21–40 percent 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 LOEL was 30.3 mg/kg/day for males and 38.4 mg/kg/day 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 NOEL 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.

b. A chronic toxicity study was conducted in dogs fed cymoxanil at doses of 25, 50, or 100 ppm for females (0.7, 1.6, or 3.1 mg/kg/day) and 50, 100, or 200 ppm for males (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 LOEL was 200 ppm for males, based on depressed weight gains through week 12 and changes in hematology and blood chemistry. No LOEL was established for females. The NOEL was 100 ppm.

4. *Carcinogenicity.* a. A combined chronic/carcinogenicity study, conducted in rats (described in the Chronic Toxicity Section, above, Unit II.A.3.) showed no evidence of carcinogenic potential.

b. A carcinogenicity study was conducted in mice fed cymoxanil at doses of 30, 300, 1,500, and 3,000 ppm (4.19, 42.0, 216, and 446 mg/kg/day for males; 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 percent compared to 69 percent 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. The 3,000 ppm females exhibited increased frequency of pallor, weakness, and hunching over. Male mice fed 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 LOEL was 300 ppm, based on toxicity to the testes and epididymides in males and toxicity to the gastrointestinal mucosa in females. The NOEL was 30 ppm. Under the conditions of this study, there was no evidence of a carcinogenic effect.

5. *Developmental toxicity.* a. 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 LOEL was 25 mg/kg/day, based upon reduced body weight, body weight change, and food consumption. The maternal NOEL was 10 mg/kg/day. The developmental LOEL 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, 150 and 150 mg/kg/day. Increased early resorptions resulted in reduced litter sizes. The developmental NOEL was 10 mg/kg/day.

b. 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 LOEL was not determined; the maternal and developmental NOEL was $\geqq$ 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 NOEL and LOEL for cymoxanil in a nonrodent species.

c. A prenatal developmental toxicity study was conducted in rabbits gavaged with cymoxanil on days 6–18 of gestation at dose levels of 8, 16, or 32 mg/kg/day. Uncertainties regarding the source of the parental rabbits substantially reduced the confidence that any observed skeletal effects were solely related to treatment.

d. 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 posttreatment increases in body weight gain at 8 and 32 mg/kg/day. The maternal LOEL was 8 mg/kg/day, based upon a significant dose-related rebound in maternal body weight. The maternal NOEL was 4 mg/kg/day. The developmental LOEL 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 NOEL was 4 mg/kg/day.

6. *Reproductive toxicity.* A two-generation reproduction study was conducted in rats fed cymoxanil at doses of 100, 500, or 1,500 ppm (equivalent to 6.5, 32.1, or 97.9 mg/kg/day in males and 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 LOEL was 500 ppm based upon reduced pre-mating body weight, body weight gain, and food consumption for F_1 males; and decreased gestation and lactation body weight for F_1 females. The parental systemic NOEL was 100 ppm. The offspring LOEL was 500 ppm based upon decreased F_1 pup viability on postnatal days 0–4 and on a significant reduction in F_{2b} pup weight. The offspring NOEL was 100 ppm.

7. *Neurotoxicity.* a. 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 of dietary doses of cymoxanil at 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 LOEL for neurobehavioral and neuropathic effects was not established. The NOEL for neurotoxicity was 3,000 ppm.

b. In the combined chronic toxicity/carcinogenicity study in rats, increased incidence of sciatic nerve axon/myelin degeneration was observed in females fed cymoxanil at doses of 38.4 and 126 mg/kg/day for 104 weeks. Also, increased incidence and severity of retinal atrophy was observed in males at 30.3 and 90.1 mg/kg/day as well as in females at 38.4 and 126 mg/kg/day. These two findings demonstrated a dose-related effect. In addition, clinical observations of hyperactivity and aggressiveness were reported in males at 700 and 2,000 ppm (30.3 and 90.1 mg/kg/day).

c. In the carcinogenicity study in mice, absolute brain weight was decreased in both sexes at 1,500 and 3,000 ppm (216/298 mg/kg/day and 446/582 mg/kg/day for males/females, respectively).

d. 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 two-generation reproduction study in rats.

e. 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 also 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 percent), during the first 24 hours (58–66 percent). Fecal excretion accounted for 15.7–23.6 percent of the dose, and radioactivity in the tissues and carcasses accounted for <1 percent of the dose at sacrifice for all three dosing regimens. A pilot study indicated that approximately 3 percent 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 fortyfold 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 percent of the dose) was free and/or conjugated [^{14}C]glycine, and 2-cyano-2-methoxyiminoacetic acid (IN-W3595) (6.5–33 percent of the dose) was also found. Intact [^{14}C]cymoxanil was not detected. In the feces, trace levels (<1 percent of the 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 percent 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 submitted mutagenicity test battery satisfied the new mutagenicity initial testing battery guidelines and 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-females 13+*. To assess acute dietary exposure, the Agency used a NOEL 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 to account for enhanced sensitivity of infants and children (required by FQPA) should be reduced to 3x. An MOE of 300 is required for the acute dietary assessment to protect the sub-population of concern, ''Females 13+,'' due to neuropathological lesions seen in the chronic toxicity study in rats and the need for an additional developmental neurotoxicity study.

Acute toxicity-general population. An acute dose and endpoint was not selected for the general population and

the sub-population including ''infants and children'' because there were no observable effects in oral toxicology studies, and no maternal toxicity in the developmental toxicity studies in rats or rabbits attributable to a single dose.

2. *Short- and intermediate-term residential 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, at the limit dose.

3. *Chronic residential toxicity*. Based on the use pattern, chronic dermal exposure is not anticipated and long-term dermal risk assessment is not required.

4. *Chronic dietary toxicity*. An RfD of 0.013 mg/kg/day was established based on a chronic feeding study in rats with a NOEL of 4.08 mg/kg/day and an uncertainty factor of 300.

5. *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 (April 10, 1996).

C. Exposures and Risks

1. *From food and feed uses*. Time-limited tolerances of 0.05 ppm have been established in the 40 CFR 180.503(b) for residues of cymoxanil in or on potatoes and tomatoes under section 18 of FIFRA. In today's action, a tolerance will be established for residues of cymoxanil in or on potatoes at 0.05 ppm under section 3 of FIFRA in 40 CFR 180.503(a) and the section 18 tolerance for potatoes will be removed. Risk assessments were conducted by EPA to assess dietary exposures and risks from cymoxanil as follows:

a. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study indicates an effect of concern may occur as a result of a 1–day or single exposure. For the subpopulation of concern, females 13+, the estimated acute MOE of 5,000 demonstrates no acute dietary concern.

b. *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 and tomatoes and assumed 100 percent of the crops were treated. The risk assessment resulted in use of <1 percent of the RfD for the general population, including infants (< 1 year old), and < 2 percent of the RfD for children (1–6 or 7–12 years old).

2. *From drinking water*. No monitoring data are currently available to perform a quantitative drinking water risk assessment. Cymoxanil appears to be mobile in soils, although its rapid environmental dissipation precludes extensive leaching. Cymoxanil was not detected below 0–15 cm of soil. Degradates of cymoxanil are mobile, but short-lived, and are not expected to 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. Surface water estimates derived from GENEEC assumed 7 applications of 0.12 lbs. active ingredient/acre would be applied. The model indicated that cymoxanil in surface water could reach 4.13 parts per billion (ppb) (peak concentration) and 0.19 ppb (average 56 day concentration).

a. *Acute exposure and risk*. EPA calculated drinking water levels of concern (DWLOC) for acute exposure by using the acute toxicity endpoint. The acute dietary food exposure (from the DRES analysis) was subtracted from the ratio of the acute NOEL (used for acute dietary assessments) to the ''acceptable'' MOE for aggregate exposure to obtain the acceptable acute exposure to cymoxail in drinking water.

EPA has calculated DWLOCs for acute exposure to cymoxanil in drinking water for females (13+ years old) to be 380 ppb. The maximum estimated concentrations of cymoxanil in surface and ground water are below EPA's levels of concern for cymoxanil in drinking water as a contribution to acute aggregate exposure. Therefore, EPA concludes with reasonable certainty that residues of cymoxanil in drinking water do not contribute significantly to the aggregate acute human health risk.

b. *Chronic exposure and risk*. Chronic (non-cancer), drinking water levels of concern are 450 ppb for the U.S. population and 130 ppb for children (1–6 years old). The estimated average concentrations of cymoxanil in surface and ground water are less than EPA's levels of concern for cymoxanil in drinking water as a contribution to chronic aggregate exposure. 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.'' 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).

At this time, EPA does not have 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. Cymoxanil is structurally related to metazachlor, dimethenamid and amiphos. Of these pesticides, only dimethenamid is currently registered for use in the United States. 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 and that structurally-related chemicals will not have common toxic metabolites to cymoxanil.

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

1. *Acute risk.* The MOE for the acute dietary (food only) risk assessment for the population subgroup of concern, females 13+ years, was estimated at 5,000. This risk estimate does not exceed the Agency's level of concern. EPA has calculated drinking water levels of concern (DWLOCs) for acute exposure to cymoxanil in drinking water for females (13+ years old) to be 380 ppb. Chronic (non-cancer), drinking water levels of concern are 450 ppb for the U.S. population and 130 ppb for children (1–6 years old). 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.

2. *Chronic risk.* Using the TMRC exposure assumptions described above, EPA has concluded that aggregate exposure to cymoxanil from food will utilize <1 percent of the RfD. The estimated average concentrations of cymoxanil in surface and ground water are less than EPA's levels of concern for cymoxanil in drinking water as a contribution to chronic aggregate exposure. Therefore, EPA concludes with reasonable certainty that residues of cymoxanil in drinking water do not contribute significantly to the potential aggregate chronic human health risk at the present time, considering the present uses and those proposed in this action.

E. 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.

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

Safety factor for infants and children - 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 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 during 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.

The Agency determined that for cymoxanil, the 10x factor for the protection of infants and children (as required by FQPA) should be reduced to 3x, based on the following weight of the evidence considerations: (1) No increased sensitivity in fetuses as compared to maternal animals was observed following *in utero* exposures in developmental studies in rats and rabbits; (2) no increased sensitivity in pups when compared to adults was seen in the two-generation reproduction study in rats; (3) the toxicology data base is complete except for the requirement to submit a developmental neurotoxicity study; and (4) no frank neurotoxicity was seen in the 90–day neurotoxicity study. The Agency has determined that a MOE of 300 is required because of the observance of neuropathological lesions in the chronic toxicity study in rats and the need for a developmental neurotoxicity study.

III. Other Considerations

A. Endocrine Disrupter Effects

EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect....'' The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3 years from the passage of FQPA (August 3, 1999) to implement this program. At that time, EPA may require further testing of this active ingredient and end use products for endocrine disrupter effects.

B. Metabolism in Plants and Animals

Plants. Based on a metabolism study on potatoes, the nature of the residue is adequately understood. Only the parent cymoxanil compound is of regulatory concern.

Animals. Based on a metabolism study in lactating goats, the nature of the residue in animals is adequately understood. Only the parent cymoxanil compound is of regulatory concern.

C. Analytical Enforcement Methodology

An adequate enforcement method, AMR 3705–95, is available to enforce the tolerance on potatoes. 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, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Crystal Mall #2, Rm 101FF, 1921 Jefferson Davis Hwy., Arlington, VA (703) 305–5229.

D. Magnitude of Residues

Residues of cymoxanil resulting from the proposed use will not exceed 0.05 ppm in potatoes. The tolerance on potatoes is for the raw agricultural commodity as defined in 40 CFR 180.1(j)(1). For risk assessment purposes, it was concluded that

residues resulting from the proposed use will not exceed 0.05 ppm in potatoes.

E. International Residue Limits

There are no Codex or Canadian residue limits established for cymoxanil on potatoes but a Mexican maximum residue limit (MRL) of 0.05 ppm is established for potatoes. Therefore, no compatibility problems exist for the proposed tolerance on potatoes.

F. Rotational Crop Restrictions

The confined rotational crop studies provided adequate results to conclude that a 30–day plant back interval is sufficient for all crops.

IV. Conclusion

Therefore, the tolerance is established for residues of cymoxanil, 2-cyano-*N*-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide, in or on the raw agricultural commodity, potatoes, at 0.05 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 July 6, 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 EPA docket for this rule making. 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 Confidential Business Information (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 and Electronic Submissions

EPA has established a record for this rulemaking under docket control number [OPP–300653] (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 a tolerance under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled Regulatory Planning and Review (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 19885, April 23, 1997).

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

VIII. Submission to Congress and the General Accounting Office

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 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: April 22, 1998.

Stephen L. Johnson,

Acting Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180— [AMENDED]

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

Authority: 21 U.S.C. 346a and 371.

2. Section 180.503 is amended by adding text to paragraph (a) to read as follows and by removing the entry for ''potatoes'' in paragraph (b) .

§ 180.503 Cymoxanil; tolerances for residues.

(a) *General* . A tolerance is established for residues of the fungicide, cymoxanil, 2-cyano-*N*-[(ethylamino)carbonyl]-2-(methoxyimino) acetamide, in or on the following food commodity.

Commodity	Parts per million
Potatoes	0.05

* * * * *

[FR Doc. 98–11764 Filed 5–5–98; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300654; FRL–5789–3]

RIN 2070–AB78

Peroxyacetic Acid; Exemption From the Requirement of a Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This document establishes an exemption from the requirement of a tolerance for residues of the antimicrobial pesticide peroxyacetic acid up to 100 ppm, in or on raw agricultural commodities, in processed commodities, when such residues result from the use of peroxyacetic acid as an antimicrobial agent on fruits, tree nuts, cereal grains, herbs, and spices. Ecolab, Inc. requested this exemption under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170).

DATES: This regulation is effective May 6, 1998. Objections and requests for hearings must be received by EPA on or before July 6, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300654], 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–300654], 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 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–300654]. 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: Marshall Swindell, Product Manager 33, Antimicrobials Division (7510W), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: 2800 Crystal Drive, 6th Floor, Arlington, VA, 22202, 703–308–6341, e-mail: swindell.marshall@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of January 14, 1998 (63 FR 2232) (FRL–5759–6), 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) 7F4808 for tolerance by Ecolab, Inc., 370 Wabasha Street, St. Paul, MN 55102. This notice included a summary of the petition prepared by Ecolab, Inc., the registrant. There were no comments received in response to the notice of filing.

Subsequently, the proposed tolerance exemption was amended to delete meat, meat by-products, poultry, milk, and eggs. This was done because at the low proposed use concentrations, no residues of toxicological concern are expected on any animal feeds that may be exposed to peroxyacetic acid. Therefore, no residues of toxicological concern are anticipated either in animals that may consume these feeds, or in associated animal by-products.

In addition, the proposed tolerance exemption was amended to include a maximum residue limit of 100 ppm for peroxyacetic acid. This limitation was added because of Agency concerns that a high use concentration could result in measurable residues of peroxyacetic acid. Residue data will be needed to increase or remove this limitation.

I. Risk Assessment and Statutory Findings

New section 408(b)(2)(A)(i) of the FFDCA allows EPA to establish a tolerance or an exemption from the requirement of a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance or exemption from the requirement of a 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