ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–301009; FRL–6590–7]

RIN 2070–AB78

Clodinafop-propargyl; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for combined residues of clodinafop-propargyl and its acid metabolite in or on wheat, grain; wheat, forage; wheat, hay; and wheat, straw. Novartis Crop Protection, Inc. requested these tolerances under the Federal Food, Drug, and Cosmetic Act, as amended by the Food Quality Protection Act of 1996.

DATES: This regulation is effective June 22, 2000. Objections and requests for hearings, identified by docket control number OPP–301009, must be received by EPA on or before August 21, 2000.

ADDRESSES: Written objections and hearing requests may be submitted by mail, in person, or by courier. Please follow the detailed instructions for each method as provided in Unit VI. of the ''SUPPLEMENTARY INFORMATION.'' To ensure proper receipt by EPA, your objections and hearing requests must identify docket control number OPP–301009 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT By mail: Joanne I. Miller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW.,Washington, DC 20460; telephone number: 703–305–6224; and e-mail address: miller.joanne@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

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

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

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

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

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

2. *In person*. The Agency has established an official record for this action under docket control number OPP–301009. The official record consists of the documents specifically referenced in this action, and other information related to this action, including any information claimed as Confidential Business Information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

II. Background and Statutory Findings

In the **Federal Register** of April 26, 2000 (65 FR 24471–24477) (FRL– 6554–2), EPA issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a as amended by the Food Quality Protection Act of 1996 (FQPA) (Public Law 104–170) announcing the amended filing of a pesticide petition (PP) for tolerance by Novartis Crop Protection, Inc., P.O. Box 18300, Greensboro, NC 27419. This notice included a summary of the petition prepared by Novartis Crop Protection, Inc., the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR part 180 be amended by establishing tolerances for combined residues of the herbicide clodinafop-propargyl (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-,2-propynyl ester, (2R)-) and its acid metabolite, CGA–193469, (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-, (2R)-), in or on wheat, grain at 0.1 part per million (ppm); wheat, forage at 0.1 ppm; wheat, hay at 0.1 ppm; and wheat, straw at 0.5 ppm.

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

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

III. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances for

combined residues of clodinafop-propargyl and its acid metabolite on wheat, grain at 0.1 ppm; wheat, forage at 0.1 ppm; wheat, hay at 0.1 ppm; and wheat, straw at 0.5 ppm. EPA's assessment of 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 clodinafop-propargyl are discussed in this unit as well as the no observed adverse effect level (NOAEL) and the lowest observed adverse effect level (LOAEL) from the toxicity studies reviewed.

TABLE 1.—SUBCHRONIC, CHRONIC AND OTHER TOXICITY

Guideline No./ Study Type	Results
870.3100 28-Day Oral Gavage	NOAEL <5 mg/kg LOAEL = 5 mg/kg for M and F based on liver toxicity (enzyme changes),
870.3100 13-Week Oral Toxicity in Rodent.	NOAEL = M: 0.9 mg/kg; F: 8.2 mg/kg/day LOAEL = M: 120 ppm (8.2 mg/kg/day); F: 1000 ppm (71.1 mg/kg/day) decreased body weight; based on increased liver weights and enzymes (AlPtase); decreased thymus weight (atrophy). Reversed after 28 day recovery period.
870.3100 13-Week Oral Toxicity in Mice	NOAEL = M: 0.9 mg/kg/day; F: 1.1 mg/kg/day LOAEL = M: 7.3 mg/kg/day ; F: 8.6 mg/kg/day based on clinical chemistry; glucose, sodium, and chloride increases and hepatocellular hypertrophy in males and females.
870.3150 90-Day Oral Toxicity in Dogs ..	The NOAEL = M: 0.346 mg/kg/day, F: 1.89 mg/kg/day. The LOAEL = M: 1.73 mg/kg/day ; F: 7.16 mg/kg/day based on occurrence of skin lesions.
870.3200 28-Day Dermal Toxicity in Rats.	Systemic NOAEL = 50 mg/kg/day Systemic LOAEL = 200 mg/kg based on dose-related increases in liver weights and clinical signs (piloerection and hunched posture) in male rats. Dermal NOAEL = 1000 mg/kg/day.
870.3700a Prenatal Developmental in Rats.	Maternal NOAEL = 160 mg/kg/day Maternal LOAEL > 160 mg/kg/day based on lack of effect. Developmental NOAEL = 5 mg/kg/day Developmental LOAEL = 40 mg/kg/day based on increased incidences of bilateral distension and torsion of the ureters, unilateral 14th ribs, and incomplete ossification of the metacarpals and various cranial bones (parietals, interparietals, occipital, and squamosal).
870.3700b Prenatal Developmental in Rabbits.	Maternal NOAEL = 25 mg/kg/day pMaternal LOAEL = 125 mg/kg/day based on mortality, clinical signs and body weight loss Developmental NOAEL = 125 mg/kg/day Developmental LOAEL > 125 mg/kg/day
870.3800 Two Generation Reproduction	Parental/Systemic NOAEL = 3.2 mg/kg/day. Parental/Systemic LOAEL = 31.7 mg/kg/day based on decrease in body weight gain, reduced food consumption, increased liver and kidney weights and histopathological changes in the liver and renal tubules. Offspring NOAEL = 3.2 mg/kg/day Offspring LOAEL = 31.7 mg/kg/day based on reduced viability, decreased pup body weight and dilatation of renal pelvis. Reproductive NOAEL = 64.2 mg/kg/day. Reproductive LOAEL ≧ 64.2 mg/kg/day
870.4100b Chronic Toxicity Nonrodent ..	NOAEL = M: 3.38 mg/kg/day; F: 3.37 mg/kg/day LOAEL = M: 15.2 mg/kg/day; F: 16.7 mg/kg/day based on occurrence of skin lesions, clinical signs, and reduced body weight gain and food consumption.
870.4200b Carcinogenicity Mice	NOAEL = M: 1.10 mg/kg/day; F: 1.25 mg/kg/day LOAEL = M: 11.0 mg/kg/day; F: 12.6 mg/kg/day based on increase in liver enzyme activity and liver weights. Under the conditions of this study, clodinafop-propargyl induced hepatocellular tumors at 29.6 mg/kg. The chemical was tested at doses sufficient to measure its carcinogenic potential.
870.4300 Chronic/Oncogenicity in the Rat.	NOAEL = M:0.03 mg/kg/day ; F: 0.03 mg/kg/day LOAEL = M: 0.3 mg/kg/day; F: 0.4 mg/kg/day based on hepatocytic hypertrophy, chronic progressive nephropathy, and tubular pigmentation. Under the conditions of this study, treatment with clodinafop-propargyl increased the incidence of prostate and ovarian tumors in rats at 750 ppm. For males, an increased incidence of prostate adenoma was seen in the high-dose group. The chemical was administered at a dose sufficient to test its carcinogenic potential.

TABLE 1.—SUBCHRONIC, CHRONIC AND OTHER TOXICITY—Continued

Guideline No./ Study Type	Results
870.5100 Gene Mutation *Salmonella* and *Escherichia*/Liver Microsome Test.	Negative for mutagenicity.
870.5200 Gene Mutation Mutation Test with Chinese Hamster cells V79.	Negative for mutagenicity.
870.5315 Chromosome Studies; Human Lymphocytes *in vitro*.	Owing to the conflicting results from the cytotoxicity assessment and the presence of rare complex chromosome aberrations both with and without S9 activation, the study is considered inconclusive.
870.5395 Micronucleus Test (Chinese Hamster).	No clear evidence that clodinafop-propargyl induced a clastogenic or aneugenic effect in either sex at any dose or sacrifice time.
870.5550 DNA Repair Human Fibroblasts.	Compound precipitation was seen at doses ≧ 320 μg/mL: there was, however, no indication of a cytotoxic effect at any dose. The positive control induced the expected marked increases in unscheduled DNA synthesis (UDS). There was, however, no evidence that CGA–184927 in the absence of S9 activation induced a genotoxic response in either trial.
870.5550 DNA Repair Rat Hepatocytes	Compound precipitation was noted at levels ≧ 4000 μ/mL. Lethality was apparent in the preliminary cytotoxicity test at 94.8 μg/mL. The positive control induced the expected marked increases in UDS. There was, however, no evidence that clodinafop-propargyl induced a genotoxic response in either trial.
870.7485 Metabolism and Pharmacokinetics.	The main metabolite was CGA–193469 (76% in male urine). Additional 5% was in the form of taurine conjugate of CGA–193469. Similar distribution was found in feces.
870.7485 Metabolism and Pharmacokinetics.	The major metabolite in urine and feces was determined to be CGA–193469, accounting for about 36% to 47% of the administered dose (AD) for males, and 80% to 85% of the AD for females. In addition, 11 minor metabolite fractions were isolated from urine and feces. Three were further identified as reference materials CGA– 193468, CGA–214111 and unchanged clodinofop-propargyl.
Special Study: Determination Of Residues As CGA–193469 in Abdominal Fat After A 3-Month Oral Toxicity Study in Rat.	There was a dose-dependent increase in clodinofop-propargyl residues in fat samples from both sexes taken at the end of treatment (14 weeks) and after the 4-week recovery period (18 weeks). Concentrations of clodinofop-propargyl were higher in male rats at all dose levels tested. With the exception of low-dose group males, for all remaining groups, residues in the fat at 18 weeks had decreased by between 40%—51.5% of the 14 week value.
Special Study Determination of Residues as CGA–193469 in Abdominal Fat After 12 Months in Study.	1 ppm and 10 ppm, the concentration of CGA–184927 in the abdominal fat was higher in males when compared to females. At 300 and 750 ppm, the concentration of CGA–184927 in the abdominal fat was comparable between males and females. The results of this study also indicate that the clodinafop-propargyl residue in fat is reduced after 1 year of treatment compared to 3 month treatment.
Special Study: The Effect Of CGA–184927 on Selected Biochemical Parameters in the Rat Liver Following Subchronic Administration.	The effects of clodinafop-propargyl on selected liver enzymes in the rat were similar to the effects seen after subchronic treatment with known peroxisome proliferators (hypolipidemic compounds, phenoxyacetic acid derivatives). Hence, clodinafop-propargyl was considered to most likely be a peroxisome proliferator in the rat liver.
Special Study: Apparently Clonal Thyroid Adenomas May Contain Heterogeneously Growing and Functioning Cell Subpopulations. New Frontiers in Thyroidology, p. 901–905, 1986.	The asynchronous growth rate of subsets of cells within the old adenomas as well as the intercellular heterogeneity of the endocytotic response to TSH suggests that clonal thyroid adenomas may acquire new qualities and can modify gene expression via much debated mechanism. The author concludes that the growth of benign thyroid tumors and progression does not require a change in genomic expression in any cell. The apparent heterogeneity of a tumor does not necessarily exclude its monoclonal origin.
Special Study: Assessment of Hyperplastic and Neoplastic Lesions of the Thyroid Gland. TIPS, Vol. 8, p. 511–514.	In cell cultures, TSH does not induce proliferation of human thyroid cells, but does stimulate the growth of cells obtained from rat and dog thyroids. Conventional procedures of evaluating carcinogenicity tests by simply counting tumors in rodents treated with high doses, and by mathematical extrapolation to the low doses to which humans are exposed, are not suitable for the proliferative reactions of the thyroid gland. In assessing the human risk, relevant conclusions can only be drawn if the physiological factors of growth control are known, and if the biological mechanisms by which chemicals initiate focal proliferation and support their progression to tumors are considered.
Special Study: Stott, W.T. Chemically Induced Proliferation of Peroxisomes: Implications for Risk Assessment. Regulatory Toxicology and Pharmacology, Vol. 8, p. 125–159, 1988.	The author concludes that a more appropriate maximum tolerated dose (MTD) of a peroxisome proliferative agent in sensitive species would appear to be based upon evidence of the proliferation of peroxisomes and the induction of peroxisomal enzymes capable of producing an increased intracellular oxidative stress. Exceeding these dosages will only result in a predictable sequence of events leading, ultimately, to tumor formation due to physiological adaptation of the animal to the administered compound rather than from the direct effects of the compound itself.

TABLE 1.—SUBCHRONIC, CHRONIC AND OTHER TOXICITY—Continued

Guideline No./ Study Type	Results
Special Study Bieri, F. The Effect of CGA–193469, the Free Acid Derivative of CGA–184927, on Peroxisomal-oxidation in Primary Cultures of Rat, Mouse, Marmoset and Guinea Pig Hepatocytes.	This study characterized and compared the *in vitro*effects of clodinafop-propargyl on selected parameters (i.e., cytotoxicity and induction of peroxisomal beta-oxidation) in primary hepatocytes from various species. The monolayer cultures were treated with medium containing clodinafop-propargyl, CGA–193469 or propargyl alcohol at the appropriate concentrations (0.1 to 100 μg/mL), or solvent controls and incubated for three days. Hepatocytes were then examined for morphological alterations and cell viability. The lactate dehydrogenase (LDH) activity was measured as an indicator of cytotoxicity. In addition, protein content of hepatocytes were measured to determine the membrane damage. Peroxisomal beta-oxidation was measured in hepatocyte homogenates treated with [1-14]palmitoyl-CoA, a peroxisomal enzyme marker. Clodinafop-propargyl-induced cytotoxicity through propargyl alcohol.
Special Study Guyomard, C. (1992). Effects of CGA–193469, the Acid Derivative of CGA–184927, on the Peroxisomal Beta-oxidation in Human Hepatocytes.	Under the conditions of this study, neither CGA–193469 nor bezafibric acid induced peroxisomal beta-oxidation in human hepatocytes, *in vitro*. However, in the absence of a known concurrent human positive control to validate the test system, (i.e., a substance known to elicit peroxisomal beta-oxidation in human hepatocytes,) this cannot be definitely concluded.
Special Study: Trendelenburg, C. Effects on Selected Plasma Concentrations and Biochemical Parameters in the Liver upon Subchronic Administration to Male Adult Rats.	Clodinafop-propargyl may act as a peroxisomal proliferating agent and alters monooxygenase activity in subfamilies of cytochrome P450 which are known to be involved in the synthesis or catabolism of steroid hormones.

B. Toxicological Endpoints

The dose at which no adverse effects are observed (the NOAEL) from the toxicology study identified as appropriate for use in risk assessment is used to estimate the toxicological level of concern (LOC). However, the lowest dose at which adverse effects of concern are identified (the LOAEL) is sometimes used for risk assessment if no NOAEL was achieved in the toxicology study selected. An uncertainty factor (UF) is applied to reflect uncertainties inherent in the extrapolation from laboratory animal data to humans and in the variations in sensitivity among members of the human population as well as other unknowns. An UF of 100 is routinely used, 10X to account for interspecies differences and 10X for intra species differences.

For dietary risk assessment (other than cancer) the Agency uses the UF to calculate an acute or chronic reference dose (acute RfD or chronic RfD) where the RfD is equal to the NOAEL divided by the appropriate UF (RfD=NOAEL/UF). Where an additional safety factor is retained due to concerns unique to the FQPA, this additional factor is applied to the RfD by dividing the RfD by such additional factor. The acute or chronic Population Adjusted Dose (aPAD or cPAD) is a modification of the RfD to accommodate this type of FQPA Safety Factor.

For non-dietary risk assessments (other than cancer) the UF is used to determine the LOC. For example, when 100 is the appropriate UF (10X to account for interspecies differences and 10X for intraspecies differences) the LOC is 100. To estimate risk, a ratio of the NOAEL to exposures (margin of exposure (MOE) = NOAEL/exposure) is calculated and compared to the LOC.

The linear default risk methodology (Q^*) is the primary method currently used by the Agency to quantify carcinogenic risk. The Q^* approach assumes that any amount of exposure will lead to some degree of cancer risk. A Q^* is calculated and used to estimate risk which represents a probability of occurrence of additional cancer cases (e.g., risk is expressed as 1×10^{-6} or one in a million). Under certain specific circumstances, MOE calculations will be used for the carcinogenic risk assessment. In this non-linear approach, a ''point of departure'' is identified below which carcinogenic effects are not expected. The point of departure is typically a NOAEL based on an endpoint related to cancer effects though it may be a different value derived from the dose response curve. To estimate risk, a ratio of the point of departure to exposure (MOE_{cancer} = point of departure/exposures) is calculated.

TABLE 2. SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR CLODINAFOP-PROPARGYL FOR USE IN HUMAN RISK ASSESSMENT

Exposure Scenario	Dose Used in Risk Assessment, UF	FQPA SF [1] and Level of Concern for Risk Assessment	Study and Toxicological Effects
Acute Dietary females 13–50 years of age.	NOAEL = 5 mg/kg/day UF = 100 Acute RfD = 0.05 mg/kg/day.	FQPA SF = 10X aPAD = acute RfD ÷ FQPA SF = 0.005 mg/kg/day.	Developmental Toxicity Study in Rats LOAEL = 40 mg/kg/day based on increased incidences of bilateral distension and torsion of the ureters, unilateral 14th ribs, and incomplete ossification of the metacarpals and various cranial bones (parietals, interparietals, occipital, and squamosal) .

TABLE 2. SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR CLODINAFOP-PROPARGYL FOR USE IN HUMAN RISK ASSESSMENT—Continued

Exposure Scenario	Dose Used in Risk Assessment, UF	FQPA SF [1] and Level of Concern for Risk Assessment	Study and Toxicological Effects
Acute Dietary infants and children.	NOAEL = 25 mg/kg/day UF = 100 ..... Acute RfD = 0.25 mg/kg/day	FQPA SF = 3X aPAD = acute RfD ÷ FQPA SF = 0.083 mg/kg/day	Developmental Toxicity Study in Rabbits LOAEL = 125 mg/kg/day based on increased mortality, clinical signs and body weight loss
Acute Dietary general population.	NOAEL = 25 mg/kg/day UF = 100 ..... Acute RfD = 0.25 mg/kg/day	FQPA SF = 1X aPAD = acute RfD ÷ FQPA SF = 0.25 mg/kg/day.	Developmental Toxicity Study in Rabbits LOAEL = 125 mg/kg/day based on increased mortality, clinical signs and body weight loss
Chronic Dietary all populations.	NOAEL = 0.03 mg/kg/day UF = 100 ..... Chronic RfD = 0.0003 mg/kg/day	FQPA SF = 10X cPAD = chronic RfD ÷ FQPA SF = 0.00003 mg/kg/day.	Chronic Toxicity Study in Rats LOAEL = 0.3 mg/kg/day based on Hepatocytic hypertrophy, chronic progressive nephropathy, and tubular pigmentation

[1] The reference to the FQPA Safety Factor refers to any additional safety factor retained due to concerns unique to the FQPA.

Carcinogenicity. In accordance with the EPA *Proposed EPA Weight-of-the-Evidence Categories*, August 1999, the Agency's Cancer Assessment Review Committee (CARC) classified clodinafop-propargyl as ''likely to be carcinogenic to humans'' by the oral route based on the occurrence of prostate tumors in male rats, ovarian tumors in female rats, and liver tumors in both sexes of mice, as well as blood vessel tumors in female mice. For the quantification of human cancer risk, the CARC recommended a linear low-dose extrapolation approach based on the most potent of these tumor types. This approach is supported by possible genotoxic potential and the lack of confirmation of the mode of action of clodinafop-propargyl. The most potent unit risk, Q_1^*(mg/kg/day)$^{-1}$, of those calculated for clodinafop-propargyl is that for male mouse liver benign hepatoma and/or carcinoma combined tumor rates at 0.129 (mg/kg/day)$^{-1}$ in human equivalents.

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* No tolerances have previously been established for clodinafop-propargyl. Risk assessments were conducted by EPA to assess dietary exposures from clodinafop-propargyl in food as follows:

i. *Acute exposure.* 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 Dietary Exposure Evaluation Model (DEEM®) analysis evaluated the individual food consumption as reported by respondents in the USDA 1989–1992 nationwide Continuing Surveys of Food Intake by Individuals (CSFII) and accumulated exposure to the chemical for each commodity. The following assumptions were made for the acute exposure assessments: (1) residues of clodinafop-propargyl and its acid metabolite would be present in/on wheat at the tolerance level (0.1 ppm); and (2) 100% of the wheat crop would be treated.

ii. *Chronic exposure.* In conducting this chronic dietary risk assessment the Dietary Exposure Evaluation Model (DEEM®) analysis evaluated the individual food consumption as reported by respondents in the USDA 1989–1992 nationwide Continuing Surveys of Food Intake by Individuals (CSFII) and accumulated exposure to the chemical for each commodity. The following assumptions were made for the chronic exposure assessments: (1) residues of clodinafop-propargyl and its acid metabolite would be present in/on wheat at the anticipated residue level of 0.07 ppm; and (2) 4 percent of the wheat crop would be treated. The anticipated residue value of 0.07 ppm was derived from the sum of the limit of quantitation (LOQ) of clodinafop-propargyl (0.02 ppm) plus the LOQ of the acid metabolite (0.05 ppm) in/on wheat grain. The percent crop treated value of 4% assumes that the target pest, wild oats, occurs on 10% of the wheat acreage and that 40% of the affected acreage could be treated.

iii. *Cancer.* A lifetime cancer risk assessment was performed for the U.S. total population. Lifetime cancer risk was estimated by applying the Q_1^* value of 0.129 (mg/kg/day)$^{-1}$ to the chronic dietary exposure estimate.

iv. *Anticipated residue and percent crop treated information.* Section 408(b)(2)(E) authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide chemicals that have been measured in food. If EPA relies on such information, EPA must require that data be provided 5 years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the levels anticipated. Following the initial data submission, EPA is authorized to require similar data on a time frame it deems appropriate. As required by section 408(b)(2)(E), EPA will issue a data call-in for information relating to anticipated residues to be submitted no later than 5 years from the date of issuance of this tolerance.

Section 408(b)(2)(F) states that the Agency may use data on the actual percent of food treated for assessing chronic dietary risk only if the Agency can make the following findings: Condition 1, that the data used are reliable and provide a valid basis to show what percentage of the food derived from such crop is likely to contain such pesticide residue; Condition 2, that the exposure estimate does not underestimate exposure for any significant subpopulation group; and Condition 3, if data are available on pesticide use and food consumption in a particular area, the exposure estimate does not understate exposure for the population in such area. In addition, the Agency must provide for periodic evaluation of any estimates used. To provide for the periodic evaluation of the estimate of percent crop treated (PCT) as required by section 408(b)(2)(F), EPA may require registrants to submit data on PCT.

The Agency used PCT information as follows:

A routine chronic dietary exposure analysis for clodinafop-propargyl was based on 4% of the wheat crop treated, derived as follows. Of the approximately 63 to 70 million acres of wheat grown in the United States, about 6.5 million acres of wheat (or approximately 10% of the total) are treated to control the target pest, wild oats. The petitioner expects to capture up to 40% of the available market, or 2.5 million acres, representing 4% of the total U.S. wheat crop (40% × 10% = 4%).

The Agency believes that the three conditions previously discussed have been met. With respect to Condition 1, EPA finds that the PCT information described above for clodinafop-propargyl used on wheat is reliable and has a valid basis. The PCT information is based on reliable estimates of the potential market for clodinafop-propargyl and the petitioner's estimate of the market share it expects to capture. EPA believes the petitioner's estimate is an overestimate. At the present time, there are several competing products, making it very unlikely that the petitioner will gain 40% of the available market when it enters the market. The use of 4% in the chronic dietary exposure assessment is, therefore, considered conservative. As to Conditions 2 and 3, regional consumption information and consumption information for significant subpopulations is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Use of this consumption information in EPA's risk assessment process ensures that EPA's exposure estimate does not understate exposure for any significant subpopulation group and allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency. Other than the data available through national food consumption surveys, EPA does not have available information on the regional consumption of food to which clodinafop-propargyl may be applied in a particular area.

2. *Dietary exposure from drinking water.* The Agency lacks sufficient monitoring exposure data to complete a comprehensive dietary exposure analysis and risk assessment for clodinafop-propargyl and its acid metabolite in drinking water. Because the Agency does not have comprehensive monitoring data, drinking water concentration estimates are made by reliance on simulation or modeling, taking into account data on the physical characteristics of clodinafop-propargyl and its acid metabolite.

The Agency uses the Generic Estimated Environmental Concentration (GENEEC) or the Pesticide Root Zone/Exposure Analysis Modeling System (PRZM/EXAMS) to estimate pesticide concentrations in surface water and Screening Concentration in ground water (SCI-GROW), which predicts pesticide concentrations in groundwater. In general, EPA will use GENEEC (a tier 1 model) before using PRZM/EXAMS (a tier 2 model) for a screening-level assessment for surface water. The GENEEC model is a subset of the PRZM/EXAMS model that uses a specific high-end runoff scenario for pesticides. GENEEC incorporates a farm pond scenario, while PRZM/EXAMS incorporate an index reservoir environment in place of the previous pond scenario. The PRZM/EXAMS model includes a percent crop area factor as an adjustment to account for the maximum percent crop coverage within a watershed or drainage basin.

None of these models include consideration of the impact processing (mixing, dilution, or treatment) of raw water for distribution as drinking water would likely have on the removal of pesticides from the source water. The primary use of these models by the Agency at this stage is to provide a coarse screen for sorting out pesticides for which it is highly unlikely that drinking water concentrations would ever exceed human health levels of concern.

Since the models used are considered to be screening tools in the risk assessment process, the Agency does not use estimated environmental concentrations (EECs) from these models to quantify drinking water exposure and risk as a %RfD or %PAD. Instead, drinking water levels of comparison (DWLOCs) are calculated and used as a point of comparison against the model estimates of a pesticide's concentration in water. DWLOCs are theoretical upper limits on a pesticide's concentration in drinking water in light of total aggregate exposure to a pesticide in food, and from residential uses. Since DWLOCs address total aggregate exposure to clodinafop-propargyl they are further discussed in the aggregate risk sections below.

Based on the PRZM/EXAMS and SCI-GROW models the estimated environmental concentrations (EECs) of clodinafop-propargyl in surface water and ground water for acute exposures are estimated to be 0.23 parts per billion (ppb) for surface water and 5×10^{-6} ppb for ground water. The EECs for chronic exposures are estimated to be 0.0017 ppb for surface water and 5×10^{-6} ppb for ground water. The estimated environmental concentrations (EECs) of the acid metabolite, CGA–193496, in surface water and ground water for acute exposures are estimated to be 1.1 ppb for surface water and 0.044 ppb for ground water. The EECs for chronic exposures are estimated to be 0.11 ppb for surface water and 0.044 ppb for ground water.

3. *From non-dietary exposure.* The term ''residential exposure'' is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiticides, and flea and tick control on pets).

Clodinafop-propargyl is not registered for use on any sites that would result in residential exposure.

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

EPA does not have, at this time, sufficient data to determine whether clodinafop-propargyl has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. For the purposes of this tolerance action, therefore, EPA has not assumed that clodinafop-propargyl 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. Safety Factor for Infants and Children

1. *Safety factor for infants and children* i. *In general.* FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base on toxicity and exposure 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.

ii. *Prenatal and postnatal sensitivity*. The Agency concluded that there is concern for the increased susceptibility of infants and children to exposure to clodinafop-propargyl based on the developmental toxicity study in rats where increased skeletal effects were observed at doses much lower (LOAEL of 40 mg/kg/day) than the maternal NOAEL (160 mg/kg/day). Although there was no evidence of reproductive toxicity, a fetotoxic effect was noted in the two-generation reproduction study in rats since reduced fetal viability, decreased pup body weight, and dilatation of renal pelvis were observed in the offspring at doses that produced relatively minimal parental toxicity (decreased body weight gain, increased liver and kidney weights with histopathological changes).

iii. *Conclusion*. The toxicology database for clodinafop-propargyl is incomplete. Acute neurotoxicity, subchronic neurotoxicity, developmental neurotoxicity and *in vitro* cytogenetics studies are required. There is quantitative evidence of increased susceptibility of the young following *in utero* exposure to clodinafop-propargyl in the prenatal developmental study in rats, and there is concern for qualitative increased susceptibility in the 2-generation reproduction study in rats. A developmental neurotoxicity study has been required based on the evidence of potential endocrine disruption in the mechanism studies with clodinafop-propargyl.

For the reasons given above, the Agency concluded that the FQPA safety factor be retained at 10x. When assessing acute dietary exposure, the safety factor is retained at 10x for the females 13–50 years old population subgroup since there are data gaps in the toxicology database for clodinafop-propargyl including a developmental neurotoxicity study and there is quantitative evidence of increased susceptibility following *in utero* exposure to clodinafop-propargyl in the prenatal developmental study in rats.

The safety factor can be reduced to 3x for the infants and children population subgroups when assessing acute dietary exposure since the increased susceptibility observed following *in utero* exposure is only of concern for females of childbearing age leaving only the uncertainty due to the data gap for the developmental neurotoxicity study.

The safety factor can be reduced to 1x for all other populations subgroups not included in females 13-50 years old and infants and children when assessing acute dietary exposure. The increased susceptibility observed following *in utero* exposure is only of concern for females of childbearing age. The data gap for developmental neurotoxicity is of concern for infants and children.

When assessing the chronic dietary exposure, the safety factor should be retained at 10x for all population subgroups since there is concern for qualitative increased susceptibility of the young demonstrated after repeated oral exposures in the 2-generation reproduction study and since there are data gaps in the toxicology database including a developmental neurotoxicity study in rats.

E. Aggregate Risks and Determination of Safety

To estimate total aggregate exposure to a pesticide from food, drinking water, and residential uses, the Agency calculates DWLOCs which are used as a point of comparison against the model estimates of a pesticide's concentration in water (EECs). DWLOC values are not regulatory standards for drinking water. DWLOCs are theoretical upper limits on a pesticide's concentration in drinking water in light of total aggregate exposure to a pesticide in food and residential uses. In calculating a DWLOC, the Agency determines how much of the acceptable exposure (i.e., the population adjusted dose (PAD)) is available for exposure through drinking water e.g., allowable chronic water exposure (mg/kg/day) = cPAD—(average food + residential exposure). This allowable exposure through drinking water is used to calculate a DWLOC.

A DWLOC will vary depending on the toxic endpoint, drinking water consumption, and body weights. Default body weights and consumption values as used by the USEPA Office of Water are used to calculate DWLOCs: 2L/70 kg (adult male), 2L/60 kg (adult female), and 1L/10 kg (child). Default body weights and drinking water consumption values vary on an individual basis. This variation will be taken into account in more refined screening-level and quantitative drinking water exposure assessments. Different populations will have different DWLOCs. Generally, a DWLOC is calculated for each type of risk assessment used: acute, short-term, intermediate-term, chronic, and cancer.

When EECs for surface water and groundwater are less than the calculated DWLOCs, OPP concludes with reasonable certainty that exposures to the pesticide in drinking water (when considered along with other sources of exposure for which OPP has reliable data) would not result in unacceptable levels of aggregate human health risk at this time. Because OPP considers the aggregate risk resulting from multiple exposure pathways associated with a pesticide's use, levels of comparison in drinking water may vary as those uses change. If new uses are added in the future, OPP will reassess the potential impacts of residues of the pesticide in drinking water as a part of the aggregate risk assessment process.

1. *Acute risk*. Using the exposure assumptions discussed in this unit for acute exposure, the acute dietary exposure from food to clodinafop-propargyl will occupy <1.0% of the aPAD for the U.S. population, 7.5% of the aPAD for nursing females 13 years and older, the subgroup of adult females with the highest estimated exposure, and 1.0% of the aPAD for children 1 to 6 years old, the subgroup of infants and children with the highest estimated exposure. In addition, there is potential for acute dietary exposure to clodinafop-propargyl in drinking water. After calculating DWLOCs and comparing them to the EECs for surface and ground water, EPA does not expect the aggregate exposure to exceed 100% of the aPAD.

TABLE 3.—AGGREGATE RISK ASSESSMENT FOR ACUTE EXPOSURE TO CLODINAFOP-PROPARGYL

Population Subgroup	aPAD (mg/kg)	% aPAD (Food)	Surface Water EEC (ppb)	Ground Water EEC (ppb)	Acute DWLOC (ppb)
U.S. Population	0.25	<1.0	0.23 ppb clodinafop-propargyl; 1.1 ppb CGA–193469	5×10^{-6} ppb clodinafop-propargyl; 0.044 ppb CGA–193469	8.7×10^{3}
Females 13+ years old	0.005	7.5	Same as above	Same as above	1.4×10^{2}
Children, 1 to 6 years old	0.083	1.0	Same as above	Same as above	8.3×10^{2}

2. *Chronic risk*. Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that exposure to clodinafop-propargyl from food will utilize 14% of the cPAD for the U.S. population and 32% of the cPAD for children 1 to 6 years old, the subgroup of infants and children with the highest estimated exposure. There are no residential uses for clodinafop-propargyl that result in chronic residential exposure to clodinafop-propargyl.

TABLE 4.—AGGREGATE RISK ASSESSMENT FOR CHRONIC (NON-CANCER) EXPOSURE TO CLODINAFOP-PROPARGYL

Population Subgroup	cPAD mg/kg/day	% cPAD (Food)	Surface Water EEC (ppb)	Ground Water EEC (ppb)	Chronic DWLOC (ppb)
U.S. Population	0. 00003	14	0.0017 ppb clodinafop-propargyl; 0.11 ppb CGA–193469	5×10^{-6} ppb clodinafop-propargyl; 0.044 ppb CGA–193469	0.91
Children, 1 to 6 years old	0. 00003	32	Same as above	Same as above	0.21

3. *Short-term risk*. Short-term aggregate exposure takes into account residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

Clodinafop-propargyl is not registered for use on any sites that would result in residential exposure. Therefore, the aggregate risk is the sum of the risk from food and water, which do not exceed the Agency's level of concern.

4. *Intermediate-term risk*. Intermediate-term aggregate exposure takes into account residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

Clodinafop-propargyl is not registered for use on any sites that would result in residential exposure. Therefore, the aggregate risk is the sum of the risk from food and water, which do not exceed the Agency's level of concern.

5. *Aggregate cancer risk for U.S. population*. The DEEM® analysis using residues of 0.07 ppm for wheat and assuming 4% crop treated estimates that chronic exposure of the U.S. population to clodinafop-propargyl will be 0.000004 mg/kg/day. Applying the Q_1* value of 0.129 $(mg/kg/day)^{-1}$ results in a food only risk of 5.3×10^{-7}. Following an aggregate dietary (food + water) assessment for lifetime cancer risk, the resulting DWLOC is 0.13 μg/L or ppb. Using the models described above in section C.2, the largest EEC value is for surface water chronic exposure to the acid metabolite, CGA–193469 (0.11 ppb). The cancer DWLOC is slightly greater than the highest EEC.

Because the models used to obtain the EECs for clodinafop-propargyl and CGA–193469 are highly conservative screening models not designed specifically for estimating concentrations in drinking water and because of the conversative nature of the food exposure assessment (anticipated residues at LOQ for parent + metabolite), EPA believes this aggregate cancer dietary assessment will not underestimate exposure and that chronic dietary exposure from clodinafop-propargyl residues in food and drinking water will not exceed the Agency's level of concern for lifetime aggregate cancer risk.

6. *Determination of safety*. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to the general population, and to infants and children from aggregate exposure to clodinafop-propargyl residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

The petitioner has proposed residue analytical methods for tolerance enforcement that use both normal and reverse phase liquid chromatography with UV detection (HPLC-UV). These methods are currently being validated by the Analytical Chemistry Branch laboratories, BEAD (7503C), Office of Pesticide Programs. Upon successful completion of the EPA validation and the granting of this registration these methods will be forwarded to FDA for publication in a future revision of the Pesticide Analytical Manual, Vol-II (PAM–II). Prior to publication in PAM-II and upon request, the methods will be available prior to the harvest season from the Analytical Chemistry Branch (ACB), BEAD (7503C), Environmental Science Center, 701 Mapes Road, Fort George G. Meade, MD 20755–5350; contact Francis D. Griffith, Jr, telephone (410) 305–2905, e-mail griffith.francis@epa.gov. The analytical standards for these methods are also available from the EPA National Pesticide Standard Repository at the same location.

B. International Residue Limits

A default Maximum Residue Limit of 0.1 mg/kg has been established in Canada for residues of clodinafop-propargyl on wheat. A Mexican limit exists for clodinafop-propargyl on wheat at 0.050 ppm. There are no Codex tolerances for clodinafop-propargyl on wheat. Therefore, no compatibility issues exist with Codex in regard to the proposed U.S. tolerances discussed in this review.

C. Conditions

The registration of clodinafop-propargyl will be time-limited and conditioned upon submission of additional information/data to satisfy certain toxicology, residue chemistry, ecological effects and environmental fate data deficiencies. Several guideline requirements are either data gaps or are only partially fulfilled, and the additional information is required to confirm and/or refine the parameters of the Agency's risk assessment. Deficiencies exist in the following areas: toxicology (neurotoxicity and cytogenetics); residue chemistry (nature of the residue in plants and animals, analytical methods, storage stability, magnitude of the residue in wheat and processed commodities, and rotational crop data); ecological effects (avian reproduction and seedling emergence/vegetative vigor); and environmental fate (hydrolysis, photolysis in water, anaerobic and aerobic soil metabolism, adsorption/desorption and field dissipation). Because of these deficiencies, the Agency incorporated several conservative assumptions into the risk assessment for clodinafop-propargyl, including the use of the limit of quantitation (0.07 ppm) as the anticipated residue in wheat and the assumption of 4% crop treated in the chronic and cancer risk assessments. Therefore, despite the data deficiencies noted above, the Agency believes the available data and risk assessment support the determination that there is a reasonable certainty that no harm will result to the general population, and to infants and children, from aggregate exposure to clodinafop-propargyl residues.

V. Conclusion

Therefore, tolerances are established for combined residues of clodinafop-propargyl (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-,2-propynyl ester, (2R)-) and its acid metabolite (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-, (2R)-)], in or on wheat, grain at 0.1 ppm; wheat, forage at 0.1 ppm; wheat, hay at 0.1 ppm; and wheat, straw at 0.5 ppm.

VI. Objections and Hearing Requests

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

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

You must file your objection or request a hearing on this regulation in accordance with the instructions provided in this unit and in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket control number OPP–301009 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk on or before August 21, 2000.

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

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

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

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

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

3. *Copies for the Docket.* In addition to filing an objection or hearing request with the Hearing Clerk as described in Unit VI.A., you should also send a copy of your request to the PIRIB for its inclusion in the official record that is described in Unit I.B.2. Mail your copies, identified by docket control number OPP–301009, to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460. In person or by courier, bring a copy to the location of the PIRIB described in Unit I.B.2. You may also send an electronic copy of your request via e-mail to: opp-docket@epa.gov. Please use an ASCII file format and avoid the use of special characters and any form of encryption. Copies of electronic objections and hearing requests will also be accepted on disks in WordPerfect 6.1/8.0 file format or ASCII file format. Do not include any CBI in your electronic copy. You may also submit an electronic copy of your request at many Federal Depository Libraries.

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

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

VII. Regulatory Assessment Requirements

This final rule establishes a tolerance under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4). Nor does it require any prior consultation as specified by Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998); special considerations as required by Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994); or require OMB review or any Agency action under Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997). This action does not involve any

technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note). Since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. In addition, the Agency has determined that this action will not have a substantial direct effect on States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132, entitled *Federalism* (64 FR 43255, August 10, 1999). Executive Order 13132 requires EPA to develop an accountable process to ensure ''meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.'' ''Policies that have federalism implications'' is defined in the Executive Order to include regulations that have ''substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.'' This final rule directly regulates growers, food processors, food handlers and food retailers, not States. This action does not alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4).

VIII. Submission to Congress and the Comptroller General

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

List of Subjects in 40 CFR Part 180

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

Dated: June 6, 2000.

Susan B. Hazen,

Acting Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180—[AMENDED]

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

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

2. Section 180.559 is added to read as follows:

§ 180.559 Clodinafop-propargyl; tolerances for residues.

(a) *General.* Tolerances are established for combined residues of clodinafop-propargyl (propanoic acid, 2-[4-(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-,2-propynyl ester, (2R)-) and its acid metabolite (propanoic acid, 2-[4-[(5-chloro-3-fluoro-2-pyridinyl)oxy]phenoxy]-, (2R)-), in or on wheat, grain at 0.1 ppm ; wheat, forage at 0.1 ppm; wheat, hay at 0.1 ppm; and wheat, straw at 0.50 ppm.

Commodity	Parts per million
Wheat, forage	0.1
Wheat, grain	0.1
Wheat, hay	0.1
Wheat, straw	0.5

(b) *Section 18 emergency exemptions.* [Reserved]

(c) *Tolerances with regional registrations.* [Reserved]

(d) *Indirect or inadvertent residues.* [Reserved]

[FR Doc. 00–15715 Filed 6–21–00; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 300

[FRL–6718–4]

National Oil and Hazardous Substances Pollution Contingency Plan; National Priorities List

AGENCY: Environmental Protection Agency (EPA).

ACTION: Direct final rule and request for comments.

SUMMARY: EPA Region 5 announces the Partial Deletion of the Motor Wheel Disposal Superfund Site from the National Priorities List (NPL) and requests public comment on this action. Specifically, 3.45 acres of land would be deleted from the Site. The NPL constitutes Appendix B of 40 CFR part 300 to the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), which EPA promulgated pursuant to section 105 of the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), as amended. EPA is taking this action because the Remedial Investigation (RI) has shown that the release poses no significant threat to public health or the environment and, therefore, remedial measures are not appropriate for the 3.45 acres of land. EPA, in consultation with the State of Michigan, has determined that no further response is appropriate. Moreover, EPA and the State have determined that remedial activities conducted at the 3.45 acres of land to date have been protective of public health, welfare, and the environment.

DATES: This ''direct final'' action will be effective August 21, 2000 unless U.S. EPA receives dissenting comments by July 24, 2000. If written dissenting comments are received, EPA will publish a timely withdrawal of the rule in the **Federal Register** informing the public that the rule will not take effect.

ADDRESSES: Comments may be mailed to Gladys Beard, Associate Remedial Project Manager, Superfund Division, U.S. EPA, Region 5 77 W. Jackson Blvd. (SR–6J), Chicago, IL 60604. Comprehensive information on the site is available at U.S. EPA's Region 5 office and at the local information repository located at: The Lansing Public Library, Reference Section, 401 Capital Ave., Lansing, MI 48933. Requests for comprehensive copies of documents should be directed formally to the Region 5 Docket Office. The address and phone number for the Regional Docket Officer is Jan Pfundheller (H–7J), U.S. EPA, Region 5, 77 W. Jackson Blvd., Chicago, IL 60604, (312) 353–5821.

FOR FURTHER INFORMATION CONTACT: Heather Nelson, Remedial Project Manager, at (312) 353–0685 (SR–6J), or Gladys Beard, Associate Remedial Project Manager, Superfund Division (SR–6J), U.S. EPA, Region 5, 77 W. Jackson Blvd., Chicago, IL 60604, (312) 886–7253 or Jennifer Ostermeier (P–19J), Office of Public Affairs, U.S. EPA, Region 5 77 W. Jackson Blvd.,Chicago, IL 60604, (312) 353–0618.

SUPPLEMENTARY INFORMATION: