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.

X. 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 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 27, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180–[AMENDED]

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

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

2. Section 180.507 is amended in paragraph (b) by alphabetically adding the following commodities to the table to read as follows:

§ 180.507 Azoxystrobin; tolerances for residues.

* * * * *

(b) * * *

Commodity	Parts per million	Expiration/Revocation Date
* * *	* * *	*
Cucurbits	1.0	6/30/99
* * *	* * *	*
Watercress	1.0	6/30/99

* * * * *

[FR Doc. 98–12578 Filed 5–11–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300647; FRL–5787–7]

RIN 2070–AB78

Myclobutanil; Pesticide Tolerance.

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for the fungicide myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) in or on bananas (post-harvest). Rohm and Haas Company requested this tolerance under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (FQPA) (Pub. L. 104-170).

DATES: This regulation is effective May 12, 1998. Objections and requests for hearings must be received by EPA on or before July 13, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300647], 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–300647], 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 or 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–300647]. 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 L. Waller, 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, Rm 247, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 308-9354, e-mail: waller.mary@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of August 1, 1997 (62 FR 41379)(FRL–5732–4), 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) 2E4141 for a tolerance by Rohm and Haas Company, 100 Independence Mall

West, Philadelphia, PA 19106-2399. This notice included a summary of the petition prepared by Rohm and Haas Company, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR 180.443 be amended by establishing a tolerance for combined residues of the fungicide myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) in or on bananas (post-harvest) at 4.0 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% or less of the RfD) is generally considered acceptable by EPA. EPA generally uses the RfD to evaluate the chronic risks posed by pesticide exposure. For shorter term risks, EPA calculates a margin of exposure (MOE) by dividing the estimated human exposure into the NOEL from the appropriate animal study. Commonly, EPA finds MOEs lower than 100 to be unacceptable. This 100-fold MOE is based on the same rationale as the 100-fold uncertainty factor.

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

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

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

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

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

Chronic risk assessment describes risk which could result from several months to a lifetime of exposure. For this assessment, risks are aggregated

considering average exposure from all sources for representative population subgroups including infants and children.

B. Aggregate Exposure

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

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

II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action. EPA has sufficient data to assess the hazards of myclobutanil and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a tolerance for myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) on bananas (post-harvest) at 4.0 ppm. EPA's assessment of the dietary exposures and risks associated with establishing the tolerance follows.

A. Toxicological Data Base

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 myclobutanil are discussed below.

1. *Acute studies.* The primary eye irritation for the technical is classified as toxicity category I. All other acute studies on the technical were classified as either toxicity category III or IV. There was a positive sensitizing reaction.

2. *Subchronic toxicity testing*— i. *Rats.* A subchronic feeding study in rats was conducted for 13 weeks. The NOEL was determined to be 1,000 ppm and the lowest observed effect level (LOEL) was 3,000 ppm based on increased liver and kidney weights, hypertrophy and necrosis in the liver, pigmentation in convoluted kidney tubules and vacuolated adrenal cortex.

ii. *Dogs.* A subchronic feeding study in dogs conducted for 13 weeks resulted in a NOEL of 10 ppm and an LOEL of 200 ppm. Technical myclobutanil was tested at 0, 10, 200, 800, and 1,600 ppm (0, 0.34, 7.26, 29.13, and 56.80 milligrams/kilogram (mg/kg)/day for males and 0, 0.42, 7.88, 32.43 and 57.97 mg/kg/day for females). At 200 ppm, and above, hepatocellular centrilobular or midzonal hypertrophy was observed in males. At 800 ppm and above, the same effect was observed in females. In addition, increases in alkaline phosphatase, in absolute liver weights in both sexes and in relative liver weights in males were observed. At 1,600 ppm, all the previous effects plus increases in relative liver weights in females, a suggestion of mild red cell destruction or mild anemia, and decreases in body weight and food consumption (possibly related to palatability) were observed.

Subchronic dermal studies using a 40% active ingredient (ai) formulation (40WP) and a 24.99% emulsifiable concentrate formulation (2EC) of myclobutanil conducted in rats resulted in a NOEL for systemic effects of ≤100 mg ai/kg/day, a NOEL for skin irritation of 10 mg ai/kg/day and an LEL of 100 mg ai/kg/day. The 2EC was applied at either 1, 10 or 100 mg ai/kg and the 40WP applied at 100 mg ai/kg once per day for a total of 19-20 treatments over a 4 week period. No systemic effects were observed at any dose level for either formulation. Microscopic changes, indicating irritation, were observed in the skin.

3. *Chronic toxicity studies.* A 1-year dog feeding study was conducted using doses of 0, 10, 100, 400 and 1,600 ppm (equivalent to doses of 0, 0.34, 3.09, 14.28 and 54.22 mg/kg body weight (bwt)/day in males and 0, 0.40, 3.83, 15.68 and 58.20 mg/kg bwt/day in females). The NOEL is 100 ppm (3.09 mg/kg/day for males and 3.83 mg/kg/day for females) based upon hepatocellular hypertrophy, increases in liver weights, ''ballooned'' hepatocytes and increases in alkaline phosphatase, SGPT and GGT, and possible slight hematological effects. The LOEL is 400 ppm (14.28 mg/kg/day for males and 15.68 mg/kg/day for females).

4. *Carcinogenicity*— i. *Mice.* A carcinogenicity study in mice was conducted by administering 90.4% ai test material in the diet at 0, 20, 100, or 500 ppm (0, 2.7, 13.7 or 70.2 mg/kg/day for males and 0, 3.2, 16.5, or 85.2 mg/kg/day for females) for 24 months. The NOEL was determined to be 100 ppm (systemic) and the LOEL was 500 ppm (systemic) based on increased MFO (male and female), increased SGPT (male) and increased absolute and relative liver weights (male and female, increased incidences and severity of centrilobular hepatocytic hypertrophy, Kupffer cell pigmentation, periportal punctate vacuolation and individual hepatocellular necrosis (male), and increased incidences of focal hepatocellular alterations and multifocal hepatocellular vacuolation (male and female). In this test, dose levels in females was not high enough. In the following test, higher doses were tested (2,000 ppm; 393.5 mg/kg/day). No carcinogenic effects were observed.

A carcinogenicity study in mice was conducted for 18 months in which myclobutanil technical (92.9% ai) was administered in the diet at 0 and 2,000 ppm (393.5 mg/kg/day). No NOEL was established. The LOEL was 2,000 ppm (393.5 mg/kg/day) based on decreases in body weight and body weight gain, increases in liver weights, hepatocellular vacuolation, necrosis of single hypertrophied hepatocytes, yellow-brown pigment in the Kupffer cells and cytoplasmic eosinophilia and hypertrophy of the cells of the zona fasciculata area of the adrenal cortex. Myclobutanil was not carcinogenic under the conditions of the study.

ii. *Rats.* A carcinogenicity study in rats was conducted by administering technical myclobutanil (92.9% ai) in the diet at doses of 0 and 2,500 ppm (125 mg/kg/day). No NOEL was established (refer to next study). The LOEL was 2,500 ppm based on testicular atrophy and decreases in testes weights, increases in the incidences of centrilobular to midzonal hepatocellular enlargement and vacuolization in the liver of both sexes, increases in bilateral aspermatogenesis in the testes, increases in the incidence of hypospermia and cellular debris in the epididymides, and increased incidence of arteritis/periarteritis in the testes. No carcinogenic effects were observed.

A chronic feeding/carcinogenicity study was conducted in rats. Technical (90.4% and 91.4% pure) myclobutanil was administered in the diet for 24 months at 25/35/50, 100/140/200 and 400/560/800 ppm (2 weeks/2 weeks/to termination; 0, 2.49, 9.84 or 39.21 mg/kg/day for males; 0, 3.23, 12.86, or 52.34 mg/kg/day for females). The NOEL was 2.49 mg/kg/day and the LOEL was 9.84 mg/kg/day based on a decrease in testes weights and increase in testicular atrophy. Dosage rates were not high enough (refer to previous study). No carcinogenic effects were observed.

5. *Developmental toxicity*— i. *Rabbits.* A teratology study was conducted in rabbits at doses of 0, 20, 60 or 200 mg ai/kg/day (technical myclobutanil; 90.4% ai) administered by oral gavage on days 7-19 of gestation which resulted in a maternal NOEL of 60 mg/kg/day and a maternal LOEL of 200 mg/kg/day based on reduced body weight and body weight gain during the dosing period and clinical signs of toxicity and possibly abortions. The developmental NOEL was 60 mg/kg/day and the developmental LOEL was 200 mg/kg/day based on increases in number of resorptions, decreases in litter size and decrease in the viability index.

ii. *Rats.* In a teratology study, rats were treated with dosages of 0, 31.26, 93.77, 312.58 and 468.87 mg/kg/day by oral gavage from gestation days 6-15. The maternal NOEL was 93.8 mg/kg/day and the maternal LOEL was 312.6 mg/kg/day based on observation of rough hair coat and salivation at 312.6 mg/kg/day and salivation, alopecia, desquamation and red exudate around mouth at 468.87 mg/kg/day. The developmental NOEL was 93.8 mg/kg/day. The developmental LOEL was 312.6 mg/kg/day based on increased incidences of 14th rudimentary and 7th cervical ribs.

6. *Reproductive toxicity.* A 2-generation rat reproduction study was conducted with dosage rates of 0, 50, 200 and 1,000 ppm (equivalent to 0, 2.5, 10 and 50 mg/kg/day). The parental (systemic) NOEL was 50 ppm (2.5 mg/kg/day) and the parental (systemic) LOEL was 200 ppm (10 mg/kg/day) based on hepatocellular hypertrophy and increases in liver weights. The reproductive toxicity NOEL was 200 ppm (10 mg/kg/day) and reproductive toxicity LOEL was 1,000 ppm (50 mg/kg/day) based on an increased incidence in the number of stillborns and atrophy of the testes, epididymides and prostate. The developmental NOEL was 200 ppm (10 mg/kg/day) and the developmental LOEL was 1,000 ppm (50 mg/kg/day) based on a decrease in pup body weight gain during lactation.

7. *Mutagenicity.* A reverse mutation assay (Ames), point mutation in CHO/HGPRT cells, in vitro and in vivo (mouse) cytogenetic assays, unscheduled DNA synthesis and a dominant lethal mutation study in rats, were conducted, all of which were negative for mutagenic effects.

8. *Metabolism*— i. *Mice.* A metabolism study in mice demonstrated that myclobutanil was rapidly absorbed and excreted. It was completely eliminated by 96 hours. The chemical was extensively metabolized prior to excretion with metabolic patterns similar for both sexes. Disposition and metabolism after pulse administration is linear over the dose range.

ii. *Rats.* In a metabolism study in rats, myclobutanil was completely and rapidly absorbed. It was extensively metabolized and rapidly and essentially completely excreted. Elimination of label from plasma was biphasic and evenly distributed between urine and feces. There was no tissue accumulation after 96 hours.

In another metabolism study in rats, at least 7 major metabolites of myclobutanil were recovered and identified. The highest amounts of radioactivity were found in the liver, kidneys, and large and small intestines. There was no tissue accumulation.

9. *Neurotoxicity.* There have been no clinical neurotoxic signs or other types of neurotoxicity observed in any of the evaluated toxicology studies. The Hazard ID Assessment Review Committee did not recommend that a developmental neurotoxicity study be required for myclobutanil. The following information was considered in the weight-of-evidence evaluation:

i. Myclobutanil does not appear to be a neurotoxic chemical.

ii. The toxicology profile for this chemical did not indicate that there were any treatment-related effects on the central or peripheral nervous system. No acute or subchronic neurotoxicity studies in rats or delayed neuropathy studies in chickens were available for review so there was no evaluation of the nervous system following perfusion.

iii. 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 468.9 and 200 mg/kg/day, respectively.

10. *Other toxicological considerations.* Myclobutanil has a complete data base and no other toxicological concerns have been identified in the evaluated studies.

B. Toxicological Endpoints

1. *Acute toxicity.* EPA has determined that data do not indicate the potential for adverse effects after a single dietary exposure.

2. *Short - and intermediate - term toxicity.* EPA has determined that when short- and intermediate-term risk assessments are appropriate for occupational and residential routes of exposure, the following should be used. OPP recommended that the NOEL of 100 mg/kg/day, taken from the 28-day dermal toxicity study in rats, be used for the short-term dermal MOE calculations. This dose level was the highest tested in the study. For intermediate-term MOE calculations, OPP recommended using the NOEL of 10 mg/kg/day from the 2-generation rat study. Effects seen at the LOEL in this study (50 mg/kg/day) were decreases in pup body weight, an increased incidence in number of stillborns, and atrophy of the prostate and testes.

3. *Chronic toxicity.* EPA has established the RfD for myclobutanil at 0.025 mg/kg/day. This RfD is based on [the chronic feeding study in rats with a NOEL of 2.5 mg/kg/day and an uncertainty factor of 100. There was testicular atrophy at the lowest observed effect level (LOEL) of 9.9 mg/kg/day.

4. *Carcinogenicity*. Using its Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), EPA has classified myclobutanil as a Group E chemical--''no evidence of carcinogenicity for humans''--based on the results of carcinogenicity studies in two species. The doses tested are adequate for identifying a cancer risk.

B. Exposures and Risks

1. *From food and feed uses.* Tolerances have been established (40 CFR 180.443) for myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) in or on a variety of raw agricultural commodities. Commodities include: almonds, apples, cherries, cotton seed, grapes, stone fruits (except cherries) and tolerances for meat, milk, poultry and eggs. In today's action, a tolerance will be established for combined residues of myclobutanil and its metabolite in or on bananas (post-harvest) at 4.0 ppm. Risk assessments were conducted by EPA to assess dietary exposures and risks from myclobutanil as follows:

i. *Acute exposure and risk.* Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a one day or single exposure. The Toxicology Endpoint Selection Committee did not identify an acute dietary toxicological endpoint and stated that an acute dietary risk assessment is not required.

ii. *Chronic exposure and risk.* In conducting the chronic dietary (food only) risk assessment, EPA has made several very conservative assumptions. With the exceptions of bananas for which a level representing residues in pulp rather than the whole banana was used and selected commodities which were corrected for percent crop treated, all commodities having myclobutanil tolerances will contain myclobutanil and metabolite residues and those residues will be at the levels of the established tolerances. For bananas, the level of 0.8 ppm was used in the dietary risk assessment rather than the proposed tolerance of 4.0 ppm since residues in the pulp will not exceed 0.8 ppm. Percent crop-treated estimates were utilized for selected commodities included in the assessment. Thus, in making a safety determination for this tolerance, EPA is taking into account this partially refined exposure assessment.

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: (a) that the data used are reliable and provide a valid basis for showing the percentage of food derived from a crop that is likely to contain residues; (b) that the exposure estimate does not underestimate the exposure for any significant subpopulation and; (c) where data on regional pesticide use and food consumption are available, that the exposure estimate does not understate exposure for any regional population. In addition, the Agency must provide for periodic evaluation of any estimates used. To provide for the periodic evaluation of these estimates of percent food treated as required by the section 408(b)(2)(F), EPA may require registrants to submit data on percent crop treated.

As indicated above, the Agency is required to determine the reliability of the percent crop-treated data. Percent crop-treated estimates are derived from federal and private market survey data. Typically, a range is assumed for the exposure assessment. By using this upper end estimate, the Agency is reasonably certain that the exposure is not understated for any significant population sub-group. Additionally, the DRES (Dietary Risk Evaluation System) modeling used in estimating chronic dietary risk uses regional consumption groups that are geographically based regions of the United States. None of these subgroups exceeded the Agency's level of concern.

The existing myclobutanil tolerances (published, pending, and including the necessary Section 18 tolerances) for crops other than bananas and the anticipated residues on bananas result in an Anticipated Residue Contribution (ARC) that is equivalent to the following percentages of the RfD.

Population Subgroup	%RfD
U.S. Population (48 states)	17
Nursing Infants (<1 year old)	25
Non-nursing Infants (<1 year old)	75
Children (1-6 years old)	46
Children (7-12 years old)	28
Northeast Region	18
Western Region	19
Hispanics	20
Non-Hispanic Others	18

The subgroups listed above are: (a) the U.S. population (48 states), (b) those for infants and children, and (c) the other subgroups for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48 states).

2. *From drinking water.* Based on information in the EFED (Environmental Fate and Effects Division) One-Liner Database, myclobutanil is persistent and not considered mobile in soils with the exception of sandy soils. Data are not available for its metabolite. There is no established Maximum Contaminant Level for residues of myclobutanil in drinking water. No Health Advisory Levels for myclobutanil in drinking water have been established. The ''Pesticides in Groundwater Database'' has no information concerning myclobutanil. Estimates of ground and surface water concentrations for myclobutanil were determined based on the label rate of 0.65 lbs. a.i./acre and assuming 15 applications per season. Although the requested tolerance is for bananas, these estimates were based on turf since it would more realistically estimate the concentrations in water. The surface water numbers are based on the results of a Generic Environmental Concentration (GENEEC) model. The ground water numbers are based on a screening tool, SCI-GROW, which tends to overestimate the true concentration in the environment. For acute effects, the surface water EEC was determined to be 0.14596 ppm or mg/L (maximum initial concentration). For chronic effects the surface water EEC was 0.1186 ppm or mg/L (average 56-day concentration). Current policy allows the 90/56-day GENEEC value to be divided by 3 to obtain a value for chronic risk assessment calculations. Therefore, the surface water value for use in the chronic risk assessment would be 0.04 ppm or mg/L.

i. *Acute exposure and risk.* The Toxicology Endpoint Selection Committee did not identify an acute dietary toxicological endpoint and stated than an acute dietary risk assessment is not required.

ii. *Chronic exposure and risk.* Chronic exposure is calculated based on surface water. Chronic exposure from ground water is lower. Chronic exposure (mg/kg/day) is calculated by multiplying the concentration in water in mg/L by the daily consumption (2L/day for male and female adults and 1L/day for children) and dividing this figure by average weight (70 kg for males, 60 kg for females and 10 kg for children). For adult males, exposure is 1.1×10^{-3} mg/kg/day; for adult females, 1.3×10^{-3} mg/kg/day; and for children, 4.0×10^{-3} mg/kg/day. Chronic risk (non-cancer) from surface water was calculated to be 4.4% of the Rfd for males, 5.2% for females and 16% for children.

3. *From non-dietary exposure.* Myclobutanil is currently registered for use on the following non-food sites: outdoor residential and greenhouse use on annuals and perennials, turf, shrubs, trees and flowers.

i. *Acute exposure and risk.* An acute toxicological endpoint was not identified for myclobutanil.

ii. *Chronic exposure and risk.* HED has determined that these uses do not constitute a chronic exposure scenario, but may constitute a short- to intermediate-term exposure scenario.

iii. *Short- and intermediate-term exposure and risk.* The home use of myclobutanil on turf has the greatest potential for exposure and was used in estimating short-term risk. HED concluded that residential intermediate-term exposure is not expected for handlers or persons re-entering treated areas. Fungicide use on home lawns is limited, restricted to certain parts of the country, and considered to be a ''rare, extra treatment'' in homeowner Do-It-Yourself programs. The end-point selected for short-term risk assessment is from a 28-day dermal study in rats; this dosing duration is expected to adequately reflect the typical human exposures for this use. Maximum application rates are calculated from the use directions on the label. Typical lawn size of 13,000 ft^2 is used in place of the high-end lawn default value of 20,000 ft^2. Post-application exposure estimates assume that 10% of the application rate is available as dislodgeable residue since the label states that the product is not washed away by rain or sprinklers.

Currently there is no use/usage information source available to HED for residential end-use products. Therefore, pertinent information is unknown and assumptions are made for parameters such as: amount of product applied, how often treatment is actually required; the number of applications that are typically made; whether applications are generally spot or full lawn treatments, etc. Similarly, a number of assumptions and best estimates are made in assessing post-application exposure, including: the duration and degree of activity in the treated area by children and adults; the amount of product available to dislodge and transfer to the skin during activity; and the amount of product dissipation over time.

HED determined that there is potential for intermittent short-term exposures to homeowners associated with typical end-product use of myclobutanil. Three exposure scenarios with the greatest potential for exposure are considered for application to home lawns: (a) loading and application of granular product by hand held rotary granular spreader; (b) mixing, loading and application of a soluble concentrate product by low pressure handwand sprayer; and (c) mixing, loading, and application of a soluble concentrate product by garden hose-end sprayer. Short-term dermal exposure assessments using the ''Pesticide Handlers Exposure Database'' surrogate data and risk calculations for homeowners resulted in a short-term MOE of 460 for scenario 1, 260 for scenario 2 and 890 for scenario 3.

There is also the potential for post-application homeowner exposure following applications to lawn and garden sites. There are no chemical-specific data to use in assessing these potential exposures. Post-application exposure is estimated and risk assessments performed using typical transfer coefficients (Tc) and surrogate dislodgeable foliar residues (DFR) derived from the application rate. Short-term post-application exposure assessments and risk calculations for adults and toddlers re-entering treated areas on the day of application resulted in a short-term MOE of 350 for adult dermal exposure, 100 for toddler dermal exposure, 1,600 for toddlers for non-dietary ingestion and 100 for combined dermal and non-dietary ingestion for toddlers. Dietary ingestion is addressed in the discussion of aggregate risk.

Using these exposure assumptions for short-term risk assessments, it is concluded that the MOEs that will result from the residential use of myclobutanil do not exceed the level of concern.

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

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

EPA does not have, at this time, available data to determine whether myclobutanil has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, myclobutanil 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 myclobutanil has a common mechanism of toxicity with other substances.

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

1. *Acute risk.* No acute dietary risks were identified.

2. *Chronic risk.* Using the partially refined exposure assumptions described above, EPA has concluded that aggregate exposure to myclobutanil from food will utilize 17% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is non-nursing infants (<1 year old) which is discussed below. EPA generally has no concern for exposures below 100% of the RfD because the RfD

represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to myclobutanil in drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the RfD. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to myclobutanil residues.

3. *Short- and intermediate-term risk.* Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. Since short-term residential exposure scenarios are present, short-term aggregate MOEs for adults and children from the turf use were determined. The short-term aggregate MOE for adults was 150 and for children it was 94. Although an MOE of 94 was calculated, this MOE is acceptable based on conservative estimates of exposure. Since worst case estimates were used in the calculations, the MOE would be above 100 under usual conditions of use. It was concluded that short-term aggregate MOEs for both adults and children are acceptable. This is based on the consideration of the conservative nature of the default assumptions for duration and degree of activity in treated areas by children and adults, amount of product available to dislodge and transfer to skin during activity, and amount of product dissipation over time which were used in the derivation of exposure estimates. The estimates were calculated using the maximum application rate and the assumption that 10% of the application rate is available as dislodgeable residue. Both of these factors are likely overestimated. The fact that a LOEL was not identified in the 28-day rat dermal toxicity study used to determine the MOE indicates an overestimate since the level used was the highest dose tested. Additionally there are no indoor residential uses of myclobutanil; thus, indoor residential exposure is not a concern.

D. Aggregate Cancer Risk for U.S. Population

Myclobutanil is classified as Category E: not carcinogenic in two acceptable animal studies.

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

1. *Safety factor for infants and children— In general.* In assessing the potential for additional sensitivity of infants and children to residues of myclobutanil, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

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

2. *Developmental toxicity studies*— i. *Rats.* In the developmental study in rats, the maternal (systemic) NOEL was 93.8 mg/kg/day, based on rough hair coat and salivation at the LOEL of 312.6 mg/kg/day. The developmental (fetal) NOEL was 93.8 mg/kg/day based on incidences of 14th rudimentary and 7th cervical ribs at the LOEL of 312.6 mg/kg/day.

ii. *Rabbits.* In the developmental toxicity study in rabbits, the maternal (systemic) NOEL was 60 mg/kg/day, based on reduced weight gain, clinical signs of toxicity and abortions at the LOEL of 200 mg/kg/day. The developmental (fetal) NOEL was 60 mg/kg/day, based on increases in number of resorptions, decreases in litter size, and a decrease in the viability index at the LOEL of 200 mg/kg/day.

3. *Reproductive toxicity study— Rats.* In the 2-generation reproductive toxicity study in rats, the parental (systemic) NOEL was 2.5 mg/kg/day, based on increased liver weights and liver cell hypertrophy at the LOEL of 10 mg/kg/day. The developmental (pup) NOEL was 10 mg/kg/day, based on decreased pup body weight during lactation at the LOEL of 50 mg/kg/day. The reproductive NOEL was 10 mg/kg/day, based on the increased incidences of stillborns, and atrophy of the testes, epididymides, and prostate at the LOEL of 50 mg/kg/day.

4. *Pre- and post-natal sensitivity.* The pre- and post-natal toxicology data base for myclobutanil is complete with respect to current toxicological data requirements. Based on the developmental and reproductive toxicity studies discussed above, there does not appear to be an extra sensitivity for pre- or post-natal effects.

5. *Acute risk.* No acute dietary risk has been identified.

6. *Chronic risk.* Using the conservative exposure assumptions described above, EPA has concluded that exposure to myclobutanil from food will utilize 25% (nursing infants < 1 year old) and 75% (non-nursing infants < 1 year old) of the RfD. The percent of the RfD that will be used by the food and water exposure for children 1-6 years old is 62% and 21% for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to myclobutanil in drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the RfD. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to myclobutanil residues.

7. *Short- or intermediate-term risk.* Intermediate-term risk is not expected since there is no expectation of intermediate-term exposure. Short-term exposure scenarios are expected and the MOEs which were determined for aggregate short-term risk does not exceed HED's level of concern. It was concluded that there is a reasonable certainty that no harm will result from aggregate exposure to myclobutanil residues.

8. *Conclusion.* EPA concludes that reliable data support use of the 100-fold uncertainty factor and that an additional 10-fold factor is not needed to ensure the safety of infants and children from dietary exposure.

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 the 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. Based on the adverse testicular findings in the chronic toxicity and reproduction studies in rats, myclobutanil should be considered as a candidate for evaluation as an endocrine disrupter.

B. Metabolism In Plants and Animals

1. *Plants*. Based on the three metabolism studies on wheat, apples and grapes (which indicate a similar metabolic route for crops in three different crop groups), the nature of the residue in bananas is adequately understood. The residues of concern in bananas are myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound).

2. *Animals*. The nature of the residue in animals is adequately understood. The residues of concern in animal commodities except milk are myclobutanil and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free). The residues of concern in milk are myclobutanil and its metabolites alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) and alpha-(4-chlorophenyl)-alpha-(3,4-dihydroxybutyl)-1*H*-1,2,4-triazole-1-propanenitrile.

C. Analytical Enforcement Methodology

An adequate enforcement method, 34S-88-10, is available to enforce the tolerance on bananas. Quantitation is by GLC using a nitrogen/phosphorus detector for parent myclobutanil and an electron capture detector (Ni^{63}) for residues measured as the alcohol metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile. Enforcement methods for the established tolerances on animal commodities are Methods 34S-88-22, 34S-88-15, 31S-87-02, and 34S-88-21. These methods have been submitted for publication in PAM II. The methods are available to anyone who is interested in pesticide residue enforcement from: By mail, Calvin Furlow, Public Information and Records Intregrity Branch, 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. 119FF, 1921 Jefferson Davis Hwy., (703) 305–5229.

D. Magnitude of Residues

The combined residues of myclobutanil and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) resulting from the proposed use will not exceed 4.0 ppm in bananas (post-harvest). The tolerance on bananas is for the raw agricultural commodity as defined in 40 CFR 180.1(j)(1). Both peel and pulp are included. Crown tissue or stalk are excluded. For risk assessment purposes, it was concluded that residues resulting from the proposed use will not exceed 0.8 ppm in banana pulp.

E. Rotational Crop Restrictions.

Rotational crop studies are not required for uses of pesticides on bananas.

F. International Residue Limits

There are no Codex, Canadian or Mexican residue limits established for myclobutanil and its metabolites on bananas. Therefore, no compatibility problems exist for the proposed tolerance on bananas.

IV. Conclusion

Therefore, the tolerance is established for the combined residues of the fungicide myclobutanil [alpha-butyl-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile] and its metabolite alpha-(3-hydroxybutyl)-alpha-(4-chlorophenyl)-1*H*-1,2,4-triazole-1-propanenitrile (free and bound) in or on the raw agricultural commodity bananas (post-harvest) at 4.0 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 13, 1998, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33(i). If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

VI. Public Docket

EPA has established a record for this rulemaking under docket control number [OPP–300647] (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 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 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 23, 1998.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180–[AMENDED]

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

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

2. Section 180.443, is amended by adding and alphabetically inserting into the table of paragraph (a) the commodity bananas (Post-H) at 4.0 ppm to read as follows:

§ 180.443 Myclobutanil; tolerances for residues.

(a) *General.* * * *

Commodity	Parts per million
* * *	* *
Bananas (Post-H)	4.0
* * *	* *

* * * * *

[FR Doc. 98–12577 Filed 5–11–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300628A; FRL–5785–4]

RIN 2070–AB78

Imidacloprid; Pesticide Tolerance Correction

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule; correction.

SUMMARY: EPA is correcting the final rule issued in the **Federal Register** of March 25, 1998 (63 FR 14371)(FRL–5778–3), establishing permanent tolerances for residues of the insecticide 1-[(6-chloro-3-pyridinyl)methyl]-*N*-nitro-2-imidazolidinimine and its metabolites in or on sorghum grain at 0.05 parts per million (ppm), sorghum forage at 0.10 ppm, and sorghum stover at 0.10 ppm. Gustafson, Inc. submitted a petition to EPA under the Federal Food, Drug and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104-170) requesting these tolerances.

DATES: This correction is effective May 12, 1998.

FOR FURTHER INFORMATION CONTACT: By mail: Elizabeth T. Haeberer, 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) 308–2891, e-mail: haeberer.elizabeth@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

I. Regulatory Assessment Requirements

This final rule does not impose any requirements. It only implements a technical correction to the Code of Federal Regulations (CFR). As such, this action does not require review by the Office of Management and Budget (OMB) under Executive Order 12866, entitled Regulatory Planning and