report containing this action 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**. A major rule cannot take effect until 60 days after it is published in the **Federal Register**. This action is not a ''major rule'' as defined by 5 U.S.C. 804(2).

Under section 307(b)(1) of the Clean Air Act, petitions for judicial review of this action must be filed in the United States Court of Appeals for the appropriate circuit by September 12, 2017. Filing a petition for reconsideration by the Administrator of this final rule does not affect the finality of this action for the purposes of judicial review nor does it extend the time within which a petition for judicial review may be filed, and shall not postpone the effectiveness of such rule or action. This action may not be challenged later in proceedings to enforce its requirements. (See section 307(b)(2).)

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Carbon monoxide, Incorporation by reference, Intergovernmental relations, Lead, Nitrogen dioxide, Ozone, Particulate matter, Reporting and recordkeeping requirements, Sulfur oxides, Volatile organic compounds.

Dated: June 26, 2017.

Deborah A. Szaro,

Acting Regional Administrator, EPA New England.

Part 52 of chapter I, title 40 of the Code of Federal Regulations is amended as follows:

PART 52—APPROVAL AND PROMULGATION OF IMPLEMENTATION PLANS

■ 1. The authority citation for part 52 continues to read as follows:

Authority: 42 U.S.C. 7401 *et seq.*

Subpart U—Maine

■ 2. In § 52.1020:

■ a. In paragraph (c), the table titled ''EPA-Approved Maine Regulations'' is amended by revising the entry for ''Chapter 118.''

■ b. In paragraph (e), the table titled ''Maine Non Regulatory'' is amended by adding an entry for ''Demonstration of Compliance with the Comparable Measures Requirement of CAA section 184(b)(2)'' at the end of the table.

The revision and addition read as follows:

§ 52.1020 Identification of plan.

* * * * *

(c) * * *

EPA-APPROVED MAINE REGULATIONS

State citation	Title/subject	State effective date	EPA approval date EPA approval date and citation [1]	Explanations
*	* *	*	*	* *
Chapter 118	Gasoline Dispensing Facilities Vapor Control.	1/1/2012	7/14/2017, [Insert **Federal Register** citation].	Includes decommissioning of Stage II vapor recovery systems.
*	* *	*	*	* *

[1] In order to determine the EPA effective date for a specific provision listed in this table, consult the **Federal Register** notice cited in this column for the particular provision.

* * * * * (e) * * *

MAINE NON REGULATORY

Name of non regulatory SIP provision	Applicable geographic or nonattainment area	State submittal date/effective date	EPA approved date [3]	Explanations
*	* *	*	*	* *
Demonstration of Compliance with the Comparable Measures Requirement of CAA section 184(b)(2).	York, Cumberland, and Sagadahoc Counties.	4/13/2016	7/14/2017, [Insert **Federal Register** citation].	Emission calculations and narrative associated with Stage II Decommissioning SIP revision.

[3] In order to determine the EPA effective date for a specific provision listed in this table, consult the **Federal Register** notice cited in this column for the particular provision.

[FR Doc. 2017–14735 Filed 7–13–17; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2016–0254; FRL–9962–05]

Difenoconazole; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of difenoconazole in or on cottonseed subgroup 20C; rice, grain; and rice, wild, grain. It also amends the existing tolerance for cotton, gin byproducts, and removes the tolerance for cotton, undelinted seed. Syngenta Crop Protection, LLC requested these

tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective July 14, 2017. Objections and requests for hearings must be received on or before September 12, 2017, and must be filed in accordance with the instructions provided in 40 CFR part 178 (see also Unit I.C. of the **SUPPLEMENTARY INFORMATION**).

ADDRESSES: The docket for this action, identified by docket identification (ID) number EPA–HQ–OPP–2016–0254, is available at *http://www.regulations.gov* or at the Office of Pesticide Programs Regulatory Public Docket (OPP Docket) in the Environmental Protection Agency Docket Center (EPA/DC), West William Jefferson Clinton Bldg., Rm. 3334, 1301 Constitution Ave. NW., Washington, DC 20460–0001. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OPP Docket is (703) 305–5805. Please review the visitor instructions and additional information about the docket available at *http://www.epa.gov/dockets.*

FOR FURTHER INFORMATION CONTACT: Michael Goodis, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001; main telephone number: (703) 305–7090; email address: *RDFRNotices@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. The following list of North American Industrial Classification System (NAICS) codes is not intended to be exhaustive, but rather provides a guide to help readers determine whether this document applies to them. Potentially affected entities may include:

- Crop production (NAICS code 111).
- Animal production (NAICS code 112).
- Food manufacturing (NAICS code 311).
- Pesticide manufacturing (NAICS code 32532).

B. How can I get electronic access to other related information?

You may access a frequently updated electronic version of EPA's tolerance regulations at 40 CFR part 180 through the Government Printing Office's e-CFR site at *http://www.ecfr.gov/cgi-bin/text-idx?&c=ecfr&tpl=/ecfrbrowse/Title40/40tab_02.tpl.*

C. How can I file an objection or hearing request?

Under FFDCA section 408(g), 21 U.S.C. 346a, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. You must file your objection or request a hearing on this regulation in accordance with the instructions provided in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket ID number EPA–HQ–OPP–2016–0254 in the subject line on the first page of your submission. All objections and requests for a hearing must be in writing, and must be received by the Hearing Clerk on or before September 12, 2017. Addresses for mail and hand delivery of objections and hearing requests are provided in 40 CFR 178.25(b).

In addition to filing an objection or hearing request with the Hearing Clerk as described in 40 CFR part 178, please submit a copy of the filing (excluding any Confidential Business Information (CBI)) for inclusion in the public docket. Information not marked confidential pursuant to 40 CFR part 2 may be disclosed publicly by EPA without prior notice. Submit the non-CBI copy of your objection or hearing request, identified by docket ID number EPA–HQ–OPP–2016–0254, by one of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be CBI or other information whose disclosure is restricted by statute.
- *Mail:* OPP Docket, Environmental Protection Agency Docket Center (EPA/DC), (28221T), 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001.
- *Hand Delivery:* To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at *http://www.epa.gov/dockets/contacts.html.*

Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at *http://www.epa.gov/dockets.*

II. Summary of Petitioned-For Tolerance

In the **Federal Register** of February 7, 2017 (82 FR 9555) (FRL–9956–86), EPA issued a document pursuant to FFDCA section 408(d)(3), 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 6F8445) by Syngenta Crop Protection, LLC, P.O. Box 18300, Greensboro, NC 27419. The petition requested that 40 CFR 180.475 be amended by establishing tolerances for residues of the fungicide difenoconazole, 1-[2-[2-chloro-4-(4-chlorophenoxy)phenyl]-4-methyl-1,3-dioxolan-2-ylmethyl]-1H-1,2,4-triazole, in or on cottonseed subgroup 20C at 0.40 parts per million (ppm); rice, grain at 7 ppm; and rice, wild, grain at 7 ppm. In addition, the petition requested that the existing tolerance for cotton, gin byproducts be increased from 0.05 ppm to 15 ppm; and requested the tolerance in/on cotton, undelinted seed at 0.05 ppm as a seed treatment be removed from 40 CFR 180.475 because the proposed new tolerance in/on cottonseed subgroup 20C reflecting foliar uses will be adequate to support the seed treatment uses. That document referenced a summary of the petition prepared by Syngenta Crop Protection, LLC, the registrant, which is available in the docket, *http://www.regulations.gov.* There were no comments received in response to the notice of filing.

III. Aggregate Risk Assessment and Determination of Safety

Section 408(b)(2)(A)(i) of 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) of FFDCA 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) of FFDCA 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. . . .''

Consistent with FFDCA section 408(b)(2)(D), and the factors specified in FFDCA 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 for difenoconazole including exposure resulting from the tolerances established by this action. EPA's assessment of exposures and risks associated with difenoconazole 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.

Subchronic and chronic studies with difenoconazole in mice and rats showed decreased body weights, decreased body weight gains and effects on the liver (*e.g.* hepatocellular hypertrophy, liver necrosis, fatty changes in the liver). No systemic toxicity was observed at the limit dose in the most recently submitted rat dermal toxicity study.

The available toxicity studies indicated no increased susceptibility of rats or rabbits from *in utero* or postnatal exposure to difenoconazole. In prenatal developmental toxicity studies in rats and rabbits and in the 2-generation reproduction study in rats, fetal and offspring toxicity, when observed, occurred at equivalent or higher doses than in the maternal and parental animals.

In a rat developmental toxicity study, developmental effects were observed at doses higher than those which caused maternal toxicity. Developmental effects in the rat included increased incidence of ossification of the thoracic vertebrae and thyroid, decreased number of sternal centers of ossification, increased number of ribs and thoracic vertebrae, and decreased number of lumbar vertebrae. In the rabbit study, developmental effects (increases in post-implantation loss and resorptions and decreases in fetal body weight) were also seen at maternally toxic doses (decreased body weight gain and food consumption). Since the developmental effects are more severe than the maternal effects, qualitative susceptibility is indicated in the rabbit developmental study; however, the selected POD is protective of this effect. In the 2-generation reproduction study in rats, toxicity to the fetuses and offspring, when observed, occurred at equivalent or higher doses than in the maternal and parental animals.

In an acute neurotoxicity study in rats, reduced fore-limb grip strength was observed on day one in males at the lowest-observed-adverse-effect-level (LOAEL), and clinical signs of neurotoxicity were observed in females only at the highest dose tested. In a subchronic neurotoxicity study in rats, decreased hind limb strength was observed in males only at the mid- and high-doses. The effects observed in acute and subchronic neurotoxicity studies were considered transient.

Although there is some evidence that difenoconazole affects antibody levels at doses that cause systemic toxicity, there are no indications in the available studies that organs associated with immune function, such as the thymus and spleen, are affected by difenoconazole.

Difenoconazole is not mutagenic, and no evidence of carcinogenicity was seen in rats. Evidence for carcinogenicity was seen in mice (liver tumors), but statistically significant carcinoma tumors were only induced at excessively-high doses. Adenomas (benign tumors) and liver necrosis only were seen at 300 ppm (46 and 58 milligram/kilogram/day (mg/kg/day) in males and females, respectively); the NOAEL in that study was 30 ppm. EPA has concluded that the chronic point of departure (POD) for assessing chronic risk (0.96 mg/kg/day) will be protective of any cancer effects for the following reasons: (1) Tumors were seen in only one species; (2) carcinoma tumors were observed only at the two highest doses (2,500 and 4,500 ppm) in the mouse carcinogenicity study; (3) benign tumors and necrosis were observed at the mid-dose (300 ppm) ; (4) the absence of tumors at the study's lower doses (30 ppm); (5) the absence of genotoxic or mutagenic effects. The cRfD of 0.96 mg/kg/day is well below the no-observed-adverse-effect-level (NOAEL) of the mouse carcinogenicity study of 30 ppm (4.7 and 5.6 mg/kg/day in males and females, respectively), at which no effects on the biological endpoints relevant to tumor development (*i.e.,* hepatocellular hypertrophy, liver necrosis, fatty changes in the liver and bile stasis) were seen. As a result, EPA has concluded that a nonlinear RfD approach is appropriate for assessing cancer risk to difenoconazole and a separate quantitative cancer exposure assessment is unnecessary.

Specific information on the studies received and the nature of the adverse effects caused by difenoconazole as well as the no-observed-adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from the toxicity studies can be found at *http://www.regulations.gov* in document ''Difenoconazole: Human Health Risk Assessment for Proposed New Foliar Uses on Cotton, Rice and Wild Rice'' at pp. 20–21 in docket ID number EPA–HQ–OPP–2016–0254.

B. Toxicological Points of Departure/Levels of Concern

Once a pesticide's toxicological profile is determined, EPA identifies toxicological points of departure (POD) and levels of concern to use in evaluating the risk posed by human exposure to the pesticide. For hazards that have a threshold below which there is no appreciable risk, the toxicological POD is used as the basis for derivation of reference values for risk assessment. PODs are developed based on a careful analysis of the doses in each toxicological study to determine the dose at which no adverse effects are observed (the NOAEL) and the lowest dose at which adverse effects of concern are identified (the LOAEL). Uncertainty/safety factors are used in conjunction with the POD to calculate a safe exposure level—generally referred to as a population-adjusted dose (PAD) or a reference dose (RfD)—and a safe margin of exposure (MOE). For non-threshold risks, the Agency assumes that any amount of exposure will lead to some degree of risk. Thus, the Agency estimates risk in terms of the probability of an occurrence of the adverse effect expected in a lifetime. For more information on the general principles EPA uses in risk characterization and a complete description of the risk assessment process, see *http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/assessing-human-health-risk-pesticides.*

A summary of the toxicological endpoints for difenoconazole used for human risk assessment is discussed in Unit III.B. of the final rule published in the **Federal Register** of April 2, 2015 (80 FR 17697) (FRL–9923–82).

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to difenoconazole, EPA considered exposure under the petitioned-for tolerances as well as all existing difenoconazole tolerances in 40 CFR 180.475. EPA assessed dietary exposures from difenoconazole in food as follows:

i. *Acute exposure.* Quantitative acute dietary exposure and risk assessments are performed for a food-use pesticide, if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1-day or single exposure.

Such effects were identified for difenoconazole. In estimating acute dietary exposure, EPA used food consumption information from the United States Department of Agriculture (USDA) National Health and Nutrition

Examination Survey, What We Eat in America, (NHANES/WWEIA). This dietary survey was conducted from 2003 to 2008. As to residue levels in food, EPA assumed tolerance-level residues, 100 percent crop treated (PCT), and available empirical or DEEM (ver. 7.81) default processing factors.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the USDA National Health and Nutrition Examination Survey, What We Eat in America, (NHANES/WWEIA). This dietary survey was conducted from 2003 to 2008. As to residue levels in food, EPA used tolerance-level residues for some commodities, average field trial residues and USDA Pesticide Data Program monitoring samples for the remaining commodities, available empirical or DEEM (ver.7.81) default processing factors, and average PCT assumptions for some commodities.

iii. *Cancer.* Based on the data summarized in Unit III.A., EPA has concluded that a nonlinear RfD approach is appropriate for assessing cancer risk to difenoconazole. Therefore, a separate quantitative cancer exposure assessment is unnecessary since the chronic dietary risk estimate will be protective of potential cancer risk.

iv. *Anticipated residue and percent crop treated (PCT) information.* Section 408(b)(2)(E) of FFDCA authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide residues that have been measured in food. If EPA relies on such information, EPA must require pursuant to FFDCA section 408(f)(1) 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. For the present action, EPA will issue such data call-ins as are required by FFDCA section 408(b)(2)(E) and authorized under FFDCA section 408(f)(1). Data will be required to be submitted no later than 5 years from the date of issuance of these tolerances.

Section 408(b)(2)(F) of FFDCA states that the Agency may use data on the actual percent of food treated for assessing chronic dietary risk only if:

- Condition a: 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 the pesticide residue.
- Condition b: The exposure estimate does not underestimate exposure for any significant subpopulation group.
- Condition c: 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 PCT as required by FFDCA section 408(b)(2)(F), EPA may require registrants to submit data on PCT.

For the chronic dietary exposure assessment, the Agency used average PCT estimates for existing uses as follows: Almond 10%, apple 20%, apricot 10%, broccoli 2.5%, Brussels sprouts 2.5%, cabbage 5%, cantaloupe 2.5%, carrot 5%, cauliflower 2.5%, cherry 2.5%, cucumber 5%, garlic 5%, grape 10%, grapefruit 2.5%, hazelnut 1%, nectarine 2.5%, onions 5%, orange 2.5%, peach 2.5%, pear 10%, pecan 2.5%, pepper 5%, pistachio 5%, plum/prune 10%, potato 20%, pumpkin 2.5%, soybean 2.5%, squash 5%, strawberry 2.5%, sugar beet 15%, tangerine 2.5%, tomato 25%, walnut 1%, watermelon 5%, and wheat (seed treatment) 10%.

In most cases, EPA uses available data from United States Department of Agriculture/National Agricultural Statistics Service (USDA/NASS), proprietary market surveys, and the National Pesticide Use Database for the chemical/crop combination for the most recent 6–7 years. EPA uses an average PCT value for chronic dietary risk analysis. The average PCT value for each existing use is derived by combining available public and private market survey data for that use and averaged across all observations and is rounded up to the nearest multiple of 5%, for use in the analysis unless the average PCT value is estimated at less than 2.5% or 1%, in which case the Agency uses 2.5% or 1%, respectively, as the average PCT value in the analysis. EPA uses a maximum PCT value for acute dietary risk analysis. The maximum PCT value is the highest observed maximum value reported within the recent 6 years of available public and private market survey data for the existing use and rounded up to the nearest multiple of 5% for use in the analysis, unless the maximum PCT value is estimated at less than 2.5%, in which case the Agency uses 2.5% as the maximum PCT value in the analysis.

The Agency believes that the three conditions discussed in Unit III.C.1.iv. have been met. With respect to Condition a, PCT estimates are derived from Federal and private market survey data, which are reliable and have a valid basis. The Agency is reasonably certain that the percentage of the food treated is not likely to be an underestimation. As to Conditions b and c, 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 reliable information on the regional consumption of food to which difenoconazole may be applied in a particular area.

2. *Dietary exposure from drinking water.* The drinking water assessment was performed using a total toxic residue method, which considers both parent difenoconazole and its major metabolite, CGA 205375, in surface and groundwater. Therefore, the Agency used screening-level water exposure models in the dietary exposure analysis and risk assessment for difenoconazole and its major metabolite in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of difenoconazole and CGA 205375. Further information regarding EPA drinking water models used in pesticide exposure assessment can be found at *http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/about-water-exposure-models-used-pesticide.*

Based on the Tier II Pesticide in Water Calculator, the Revised Tier 1 Rice Model, the Surface Water Concentration Calculator, and Pesticide Root Zone Model Ground Water (PRZM GW), the estimated drinking water concentrations (EDWCs) of total toxic residues of difenoconazole for acute exposures are estimated to be 33.4 parts per billion (ppb) for surface water and 2.0 ppb for ground water. For chronic exposures estimated drinking water concentrations (EDWCs) of total toxic residues of difenoconazole for non-cancer assessments are estimated to be 27.8 ppb for surface water and 0.60 ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For acute dietary risk assessment, the water concentration value of 33.4 ppb was used to assess the contribution to drinking water. For chronic dietary risk assessment, the water concentration of

value 27.8 ppb was used to assess the contribution to drinking 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).

Difenoconazole is currently registered for the following uses that could result in residential exposures: Treatment of ornamental plants in commercial and residential landscapes and interior plantscapes. EPA assessed residential exposure using the following assumptions: For residential handlers, adult short-term dermal and inhalation exposure is expected from mixing, loading, and applying difenoconazole on ornamentals (gardens and trees). For residential post-application exposures, short-term dermal exposure is expected for both adults and children from post-application activities in treated residential landscapes.

The scenarios used in the aggregate assessment were those that resulted in the highest exposures. The highest exposures consist of the short-term dermal exposure to adults from post-application activities in treated gardens and short-term dermal exposure to children 6 to 11 years old from post-application activities in treated gardens. Further information regarding EPA standard assumptions and generic inputs for residential exposures may be found at *http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/standard-operating-procedures-residential-pesticide.*

4. *Cumulative effects from substances with a common mechanism of toxicity.* Section 408(b)(2)(D)(v) of FFDCA 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.''

Difenoconazole is a member of the conazole class of fungicides containing the 1,2,4-triazole moiety. Although conazoles act similarly in plants (fungi) by inhibiting ergosterol biosynthesis, there is not necessarily a relationship between their pesticidal activity and their mechanism of toxicity in mammals. Structural similarities do not constitute a common mechanism of toxicity. Evidence is needed to establish that the chemicals operate by the same, or essentially the same, sequence of major biochemical events (EPA, 2002).

In the case of conazoles, however, a variable pattern of toxicological responses is found; some are hepatotoxic and hepatocarcinogenic in mice. Some induce thyroid tumors in rats. Some induce developmental, reproductive, and neurological effects in rodents. Furthermore, the conazoles produce a diverse range of biochemical events including altered cholesterol levels, stress responses, and altered DNA methylation. It is not clearly understood whether these biochemical events are directly connected to their toxicological outcomes. Thus, there is currently no evidence to indicate that difenoconazole shares a common mechanism of toxicity with any other conazole pesticide, and EPA is not following a cumulative risk approach for this tolerance action. For information regarding EPA's procedures for cumulating effects from substances found to have a common mechanism of toxicity, see EPA's Web site at *http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/cumulative-assessment-risk-pesticides.*

This class of compounds can form the common metabolite 1,2,4-triazole and two triazole conjugates (triazolylalanine and triazolylacetic acid). To support existing tolerances and to establish new tolerances for triazole-containing pesticides, including difenoconazole, EPA conducted a human health risk assessment for exposure to 1,2,4-triazole, triazolylalanine, and triazolylacetic acid resulting from the use of all current and pending uses of any triazole-containing fungicide. The risk assessment is a highly conservative, screening-level evaluation in terms of hazards associated with common metabolites (*e.g.,* use of a maximum combination of uncertainty factors) and potential dietary and non-dietary exposures (*i.e.,* high end estimates of both dietary and non-dietary exposures). The Agency retained a 3X for the LOAEL to NOAEL safety factor when the reproduction study was used. In addition, the Agency retained a 10X for the lack of studies including a developmental neurotoxicity (DNT) study. The assessment includes evaluations of risks for various subgroups, including those comprised of infants and children. The Agency's complete risk assessment is found in the propiconazole reregistration docket at *http://www.regulations.gov,* Docket ID Number EPA–HQ–OPP–2005–0497.

The Agency's latest updated aggregate risk assessment for the triazole-containing metabolites was finalized on November 15, 2016 and includes the new uses in this rule. It is titled, ''Common Triazole Metabolites: Updated Aggregate Human Health Risk Assessment to Address the New Section 3 Registrations for Use of Difenoconazole on Rice and Cotton.'' Aggregate risk estimates associated with 1,2,4-triazole (T) and the conjugated triazole metabolites (*i.e.,* combined residues of triazolylalanine (TA) and triazolylacetic acid (TAA)), are below the Agency's level of concern. There are no human health risk issues for these metabolites that would preclude the new uses of difenoconazole. The assessment may be found at *http://www.regulations.gov* in document in docket ID number EPA–HQ–OPP–2016–0254.

D. Safety Factor for Infants and Children

1. *In general.* Section 408(b)(2)(C) of FFDCA provides that EPA shall apply an additional tenfold (10X) 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 database on toxicity and exposure unless EPA determines based on reliable data that a different margin of safety will be safe for infants and children. This additional margin of safety is commonly referred to as the FQPA Safety Factor (SF). In applying this provision, EPA either retains the default value of 10X, or uses a different additional safety factor when reliable data available to EPA support the choice of a different factor.

2. *Prenatal and postnatal sensitivity.* The prenatal and postnatal toxicology database for difenoconazole includes rat and rabbit prenatal developmental toxicity studies and a 2-generation reproduction toxicity study in rats. The available Agency guideline studies indicated no increased qualitative or quantitative susceptibility of rats to *in utero* and/or postnatal exposure to difenoconazole. In the prenatal developmental toxicity studies in rats and rabbits and the 2-generation reproduction study in rats, toxicity to the fetuses/offspring, when observed, occurred at equivalent or higher doses than in the maternal/parental animals. In a rat developmental toxicity study developmental effects were observed at doses higher than those which caused maternal toxicity. In the rabbit study, developmental effects (increases in post-implantation loss and resorptions and decreases in fetal body weight) were also seen at maternally toxic doses (decreased body weight gain and food consumption). Since the developmental effects are more severe than the maternal effects, qualitative susceptibility is indicated in the rabbit developmental study; however, the selected POD is protective of this effect. In the 2-generation reproduction study

in rats, toxicity to the fetuses/offspring, when observed, occurred at equivalent or higher doses than in the maternal/parental animals.

3. *Conclusion.* EPA has determined that reliable data show the safety of infants and children would be adequately protected if the FQPA SF were reduced to 1X. That decision is based on the following findings:

i. The toxicity database for difenoconazole is complete.

ii. There are no clear signs of neurotoxicity following acute, subchronic or chronic dosing in multiple species in the difenoconazole database. The effects observed in acute and subchronic neurotoxicity studies are transient and showed in one sex (males as reduced fore-limb grip strength with no histologic findings), and the selected endpoints of toxicity for risk assessment are protective of any potential neurotoxicity. Based on the toxicity profile, and lack of concern for neurotoxicity, there is no need for a developmental neurotoxicity study or additional uncertainty factors (UFs) to account for neurotoxicity.

iii. There is no evidence that difenoconazole results in increased susceptibility in *in utero* rats or rabbits in the prenatal developmental studies or in young rats in the 2-generation reproduction study. The qualitative susceptibility seen in the rabbit developmental study is adequately protected by the selected POD.

iv. There are no residual uncertainties identified in the exposure databases. The dietary risk assessment utilized tolerance-level residues and 100 PCT for the acute assessment; the chronic assessment was refined by using USDA PDP monitoring data, average field-trial residues for some commodities, tolerance-level residues for remaining commodities, and average PCT for some commodities. These assumptions will not underestimate dietary exposure to difenoconazole. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to difenoconazole in drinking water. EPA used similarly conservative assumptions to assess postapplication exposure of children. These assessments will not underestimate the exposure and risks posed by difenoconazole.

E. Aggregate Risks and Determination of Safety

EPA determines whether acute and chronic dietary pesticide exposures are safe by comparing aggregate exposure estimates to the acute PAD (aPAD) and chronic PAD (cPAD). For linear cancer risks, EPA calculates the lifetime probability of acquiring cancer given the estimated aggregate exposure. Short-, intermediate-, and chronic-term risks are evaluated by comparing the estimated aggregate food, water, and residential exposure to the appropriate PODs to ensure that an adequate MOE exists.

1. *Acute risk.* Using the exposure assumptions discussed in this unit for acute exposure, the acute dietary exposure from food and water to difenoconazole will occupy 53% of the aPAD for all infants less than 1 year old, the population group receiving the greatest exposure.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to difenoconazole from food and water will utilize 50% of the cPAD for all infants less than 1 year old the population group receiving the greatest exposure. Based on the explanation in Unit III.C.3., regarding residential use patterns, chronic residential exposure to residues of difenoconazole is not expected.

3. *Short-term risk.* Short-term aggregate exposure takes into account short-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level). Difenoconazole is currently registered for uses that could result in short-term residential exposure, and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with short-term residential exposures to difenoconazole.

Using the exposure assumptions described in this unit for short-term exposures, EPA has concluded the combined short-term food, water, and residential exposures result in aggregate MOEs of 250 for children and 180 for adults. Because EPA's level of concern for difenoconazole is a MOE of 100 or below, these MOEs are not of concern.

4. *Intermediate-term risk.* Intermediate-term aggregate exposure takes into account intermediate-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level). An intermediate-term adverse effect was identified; however, difenoconazole is not registered for any use patterns that would result in intermediate-term residential exposure. Intermediate-term risk is assessed based on intermediate-term residential exposure plus chronic dietary exposure. Because there is no intermediate-term residential exposure and chronic dietary exposure has already been assessed under the appropriately protective cPAD (which is at least as protective as the POD used to assess intermediate-term risk), no further assessment of intermediate-term risk is necessary, and EPA relies on the chronic dietary risk assessment for evaluating intermediate-term risk for difenoconazole.

5. *Aggregate cancer risk for U.S. population.* Based on the data summarized in Unit III.A., the chronic dietary risk assessment is protective of any potential cancer effects. Based on the results of that assessment, EPA concludes that difenoconazole is not expected to pose a cancer risk to humans.

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, or to infants and children from aggregate exposure to difenoconazole residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (gas chromatography with nitrogen phosphorus detection (GC/NPD) method AG–575B) is available for the determination of residues of difenoconazole in or on plant commodities. Liquid chromatography with tandem mass spectrometry (LC/MS/MS) method REM 147.07b is available for the determination of residues of difenoconazole and CGA–205375 in livestock commodities. Adequate confirmatory methods are also available.

The method may be requested from: Chief, Analytical Chemistry Branch, Environmental Science Center, 701 Mapes Rd., Ft. Meade, MD 20755–5350; telephone number: (410) 305–2905; email address: *residuemethods@epa.gov.*

B. International Residue Limits

In making its tolerance decisions, EPA seeks to harmonize U.S. tolerances with international standards whenever possible, consistent with U.S. food safety standards and agricultural practices. EPA considers the international maximum residue limits (MRLs) established by the Codex Alimentarius Commission (Codex), as required by FFDCA section 408(b)(4). The Codex Alimentarius is a joint United Nations Food and Agriculture Organization/World Health Organization food standards program, and it is recognized as an international food safety standards-setting organization in trade agreements to which the United States is a party. EPA may establish a tolerance that is different from a Codex MRL; however,

FFDCA section 408(b)(4) requires that EPA explain the reasons for departing from the Codex level.

The Codex has not established a MRL for difenoconazole in or on cottonseed subgroup 20C; cotton gin byproducts; rice, grain; and rice, wild, grain.

V. Conclusion

Therefore, tolerances are established for residues of difenoconazole, 1-[2-[2-chloro-4-(4-chlorophenoxy)phenyl]-4-methyl-1,3-dioxolan-2-ylmethyl]-1H–1,2,4-triazole, in or on cottonseed subgroup 20C at 0.40 ppm; rice, grain at 7.0 ppm; and rice, wild, grain at 7.0 ppm. Additionally, this regulation amends the current tolerance for cotton, gin byproducts from 0.05 ppm to 15 ppm. Finally, EPA is removing the established tolerance for residues of difenoconazole in or on cotton, undelinted seed at 0.05 ppm because residues on cotton, undelinted seed are covered by the new tolerance for cottonseed subgroup 20C.

VI. Statutory and Executive Order Reviews

This action establishes tolerances under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled ''Regulatory Planning and Review'' (58 FR 51735, October 4, 1993). Because this action has been exempted from review under Executive Order 12866, this action is not subject to Executive Order 13211, entitled ''Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use'' (66 FR 28355, May 22, 2001) or Executive Order 13045, entitled ''Protection of Children from Environmental Health Risks and Safety Risks'' (62 FR 19885, April 23, 1997). This action does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA) (44 U.S.C. 3501 *et seq.*), nor does it require any special considerations under Executive Order 12898, entitled ''Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations'' (59 FR 7629, February 16, 1994).

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.

This action directly regulates growers, food processors, food handlers, and food retailers, not States or tribes, nor does this action alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4). As such, the Agency has determined that this action will not have a substantial direct effect on States or tribal governments, on the relationship between the national government and the States or tribal governments, or on the distribution of power and responsibilities among the various levels of government or between the Federal Government and Indian tribes. Thus, the Agency has determined that Executive Order 13132, entitled ''Federalism'' (64 FR 43255, August 10, 1999) and Executive Order 13175, entitled ''Consultation and Coordination with Indian Tribal Governments'' (65 FR 67249, November 9, 2000) do not apply to this action. In addition, this action does not impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act (UMRA) (2 U.S.C. 1501 *et seq.*).

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 (NTTAA) (15 U.S.C. 272 note).

VII. Congressional Review Act

Pursuant to the Congressional Review Act (5 U.S.C. 801 *et seq.*), 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 action 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 1, 2017.

Michael L. Goodis,

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. 321(q), 346a and 371.

■ 2. In § 180.475:

■ i. Remove the entry ''Cotton, undelinted seed'';

■ ii. Revise the entry for ''Cotton, gin byproducts''; and

■ iii. Add alphabetically the entries ''Cottonseed subgroup 20C'', ''Rice, grain'', and ''Rice, wild, grain'' to the table in paragraph (a)(1) to read as follows:

§ 180.475 Difenoconazole; tolerances for residues.

(a) * * * (1) * * *

Commodity	Parts per million
* * * * *	
Cotton, gin byproducts	15
Cottonseed subgroup 20C ...	0.40
* * * * *	
Rice, grain	7.0
Rice, wild, grain	7.0
* * * * *	

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[FR Doc. 2017–14105 Filed 7–13–17; 8:45 am]

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DEPARTMENT OF HOMELAND SECURITY

Coast Guard

46 CFR Chapter I

[Docket No. USCG–2016–0669]

Marine Safety Manual, Volume III, Parts B and C, Change–2

AGENCY: Coast Guard, DHS.

ACTION: Availability of updated Marine Safety Manual.

SUMMARY: The Coast Guard announces the availability of Change–2 to the Marine Safety Manual (MSM), Volume III, Marine Industry Personnel, and the corresponding Commandant Change Notice that highlights the changes made to that manual. MSM Volume III provides information and interpretations on international conventions and U.S. statutory and regulatory issues relating to marine industry personnel. This Commandant Change Notice discusses the substantive changes to Parts B and C of MSM Volume III. All changes are underlined in the final version and each changed page is annotated with CH–2 in the footer. The date of each change since