and address the comment in the proposed rulemaking. This action may not be challenged in later proceedings to enforce its requirements (see section 307(b)(2)).

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Incorporation by reference, Intergovernmental relations, Ozone, Reporting and recordkeeping requirements, Volatile organic compounds.

Dated: March 8, 2012.

Jared Blumenfeld,

Regional Administrator, Region IX.

Part 52, Chapter I, Title 40 of the Code of Federal Regulations is amended as follows:

PART 52—[AMENDED]

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

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

Subpart F—California

■ 2. Section 52.220 is amended by adding paragraphs (c)(88)(iii)(C) and (c)(391) to read as follows:

§ 52.220 Identification of plan.

* * * * *

(c) * * *

(88) * * *

(iii) * * *

(C) In Resolution 11–04 dated January 18, 2011, Antelope Valley Air Quality Management District certified that no sources which would be subject to Rule 1119, ''Petroleum Coke Calcining Operations,'' exist in the AVAQMD. Therefore, Rule 1119 has been rescinded and is removed from the SIP.

* * * * *

(391) New and amended regulations were submitted on June 21, 2011 by the Governor's designee.

(i) Incorporation by reference.

(A) Eastern Kern Air Pollution Control District.

(*1*) Rule 102, ''Definitions,'' amended on January 13, 2011.

(B) Santa Barbara County Air Pollution Control District.

(*1*) Rule 102, ''Definitions,'' revised on January 20, 2011.

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[FR Doc. 2012–10734 Filed 5–3–12; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2011–0179; FRL–9345–6]

Metconazole; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for residues of Metconazole, including its metabolites and degradates in or on sugarcane, cane. BASF Corporation requested the tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective May 4, 2012. Objections and requests for hearings must be received on or before July 3, 2012, 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: EPA has established a docket for this action under docket identification (ID) number EPA–HQ–OPP–2011–0179. All documents in the docket are listed in the docket index available at *http://www.regulations.gov*. Although listed in the index, some information is not publicly available, e.g., Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available in the electronic docket at *http://www.regulations.gov,* or, if only available in hard copy, at the OPP Regulatory Public Docket in Rm. S–4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. The Docket Facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The Docket Facility telephone number is (703) 305–5805.

FOR FURTHER INFORMATION CONTACT: Tamue L. Gibson, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001; telephone number: (703) 305–9096; email address: *gibson.tamue@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. Potentially affected entities may include, but are not limited to those engaged in the following activities:

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

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

B. How can I get 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://ecfr.gpoaccess.gov/cgi/t/text/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–2011–0179 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 July 3, 2012. 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 that does not contain any 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 a copy of your non-CBI objection or hearing

request, identified by docket ID number EPA–HQ–OPP–2011–0179, by one of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov*. Follow the online instructions for submitting comments.
- *Mail:* Office of Pesticide Programs (OPP) Regulatory Public Docket (7502P), Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001.
- *Delivery:* OPP Regulatory Public Docket (7502P), Environmental Protection Agency, Rm. S–4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. Deliveries are only accepted during the Docket Facility's normal hours of operation (8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays). Special arrangements should be made for deliveries of boxed information. The Docket Facility telephone number is (703) 305–5805.

II. Summary of Petitioned-For Tolerance

In the **Federal Register** of April 20, 2011 (76 FR 22067) (FRL–8869–7), EPA issued a notice pursuant to section 408(d)(3) of FFDCA, 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 0F7807) by BASF Corporation, 26 Davis Drive, P.O. Box 13528, Research Triangle Park, NC 27709–3528. The petition requested that 40 CFR part 180 be amended by establishing tolerances for residues of the fungicide metconazole, 5-[(4-chlorophenyl)methyl]-2,2-dimethyl-1-(1*H*-1,2,4-triazol-1-ylmethyl)cyclopentanol, measured as the sum of cis- and trans-isomers, in or on sugarcane, cane at 0.06 parts per million (ppm); and sugarcane, molasses at 0.08 ppm. That notice referenced a summary of the petition prepared by BASF Corporation, the registrant, which is available in the docket, *http://www.regulations.gov*. There were no comments received in response to the notice of filing.

Based upon review of the data supporting the petition, tolerances for sugarcane, molasses are not being established. The reason for this change is explained in Unit IV.C.

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

Acute oral and dermal toxicities to metconazole are moderate, while acute inhalation toxicity is low. Metconazole is a moderate eye irritant and a mild skin irritant. It is not a skin sensitizer.

Metconazole was shown to affect the liver, kidney, spleen, and certain blood parameters in all the species tested. Dose levels at which these effects occur are similar across species with the rat and dog being slightly more sensitive than the mouse. Like other triazoles, a primary target organ in mammalian toxicity studies is the liver. Liver toxicity was seen in the mouse, rat and dog following oral exposure to metconazole via subchronic or chronic exposure durations. While liver effects have been reported consistently across multiple durations and species, these effects were considered slight and minimal in some studies and appeared to be ''adaptive'' responses. However, based on the weight of evidence from the consistency of these reported effects and evidence that these effects increase in severity with duration, and leading to liver tumors in the chronic mouse study, they were considered ''adverse'' and formed the basis of the study lowest observed adverse effect levels (LOAELs). Metconazole is considered nongenotoxic and the liver tumors appear to have been formed via a mitogenic mode of action and therefore, metconazole is classified as ''not likely to be carcinogenic to humans'' at levels that do not cause mitogenesis. There is evidence of liver effects (microsomal induction, liver weight increases, hypertrophy) at 47.6 milligrams/kilograms/day (mg/kg/day), but no effects at 4.5 mg/kg/day in the mode of action studies in the mouse. There is no concern for mutagenicity. The chronic Reference Dose of 0.04 mg/kg/day based on the 2-year chronic rat study with a no observed adverse effect level (NOAEL) of 4.3 mg/kg/day would be protective of early liver disturbances seen in the mouse studies. Therefore, the Agency has determined that the quantification of risk using a non-linear approach (i.e., Reference dose (RfD)) will adequately account for all chronic toxicity, including carcinogenicity, that could result from exposure to metconazole.

Other major critical effects observed in oral studies were decreased body weight, decreased body weight gains, and blood effects (reductions in erythrocyte and/or platelet parameters) in the mouse, rat, dog and/or rabbit. Splenic effects including increased spleen weight and hyperplasia were observed in the mouse, rat and dog at dose levels where liver effects were also observed. In dogs, lenticular degeneration (cataracts) was observed at the highest dose tested (HDT) (114 mg/kg/day). Furthermore, at high dietary levels, there is evidence that metconazole is a gastrointestinal irritant in the dog.

There was no evidence of immunotoxicity at dose levels that produced systemic toxicity. No immunotoxic effects are evident for metconazole at dose levels as high as 52 mg/kg/day in rats, which is 12 times higher than the chronic dietary point of departure (4.3 mg/kg/day).

Metconazole did not demonstrate neurotoxicity in the standard battery of tests submitted. Information available from the submitted studies including acute, subchronic and chronic studies in several species, developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat do not indicate any neurotoxic signs. No effects were noted on brain weights and no clinical signs possibly related to neurotoxicity were noted up to and including the high doses in all studies.

Specific information on the studies received and the nature of the adverse effects caused by metconazole as well as the NOAEL and the LOAEL from the toxicity studies can be found at *http://www.regulations.gov* in document ''Metconazole: Human Health Risk Assessment for Proposed Uses on Sugarcane,'' at page 36 in docket ID number EPA–HQ–OPP–2011–0179.

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://www.epa.gov/pesticides/factsheets/riskassess.htm*. A summary of the toxicological endpoints for metconazole used for human risk assessment is shown in the following Table.

TABLE—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR METCONAZOLE FOR USE IN HUMAN HEALTH RISK ASSESSMENT

Exposure/scenario	Point of departure and uncertainty/safety factors	RfD, PAD, LOC for risk assessment	Study and toxicological effects
Acute dietary (Females 13–50 years of age).	NOAEL = 12 mg/kg/day UF_A = 10x UF_H = 10x FQPA SF = 1x	Acute RfD = 0.12 mg/kg/day. aPAD = 0.12 mg/kg/day	Developmental toxicity in rats. LOAEL = 30 mg/kg/day based on increases in skeletal variations. At 75 mg/kg/day increased incidence of post-implantation loss, hydrocephaly and visceral anomaliea (cranial hemorrhage, dilated renal pelvis, dilated ureters, and displaced testis) were reported.
Acute dietary (General population including infants and children).	An appropriate dose/endpoint attributable to a single dose was not observed in the available oral toxicity studies reviewed.		
Chronic dietary (All populations)	NOAEL= 4.3 mg/kg/day UF_A = 10x UF_H = 10x FQPA SF = 1x	Chronic RfD = 0.04 mg/kg/day. cPAD = 0.04 mg/kg/day	Chronic oral toxicity study in rats. LOAEL = 13.1 mg/kg/day based on increased liver (M) weights and associated hepatocellular lipid vacuolation (M) and centrilobular hypertrophy(M). Similar effects were observed in females at 54 mg/kg/day, plus increased spleen weight.
Incidental oral short-term (1 to 30 days).	NOAEL= 9.1 mg/kg/day UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	28-Day oral toxicity study in rats. LOAEL = 90.5 mg/kg/day based on decreased body weight (M), increased liver and kidney weight and hepatocellular hypertrophy and vacuolation (M/F).
Incidental oral intermediate-term (1 to 6 months).	NOAEL= 6.4 mg/kg/day UF_A= 10x UF_H= 10x FQPA SF = 1x	LOC for MOE = 100	90-Day oral toxicity study in rats. LOAEL = 19.2 mg/kg/day based on increased spleen wt (F) and hepatic vacuolation (M).
Dermal short-term and intermediate-term.	Quantification of dermal risk is not needed due to lack of systemic or dermal toxicity at the Limit Dose in a 21-day dermal toxicity study in the rat, the lack of target organ toxicity or neurotoxicity, and the lack of developmental or reproductive toxicity in the absence of parental effects which were looked for in the dermal toxicity.		
Inhalation short-term (1 to 30 days)	Inhalation (or oral) study NOAEL= 9.1 mg/kg/day (inhalation absorption rate = 100%). UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	28-Day oral toxicity study in rats. LOAEL = 90.5 mg/kg/day based on decreased body weight (M), increased liver and kidney weight and hepatocellular hypertrophy and vacuolation (M/F).

TABLE—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR METCONAZOLE FOR USE IN HUMAN HEALTH RISK ASSESSMENT—Continued

Exposure/scenario	Point of departure and uncertainty/safety factors	RfD, PAD, LOC for risk assessment	Study and toxicological effects
Inhalation (1 to 6 months)	Inhalation (or oral) study NOAEL = 6.4 mg/kg/day (inhalation absorption rate = 100%). UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	90-Day oral toxicity study in rats. LOAEL = 19.2 mg/kg/day based on increased spleen wt (F) and hepatic vacuolation (M).
Cancer (Oral, dermal, inhalation)	Classification: ''Not likely to be Carcinogenic to Humans'' based on evidence that a non-genotoxic mode of action for mouse liver tumors was established and that carcinogenic effects were not likely below a defined dose that does not cause mitogenesis.		

UF_A = extrapolation from animal to human (interspecies). UF_H = potential variation in sensitivity among members of the human population (intraspecies). FQPA SF = Food Quality Protection Act Safety Factor. PAD = population adjusted dose (a = acute, c = chronic). RfD = reference dose. MOE = margin of exposure. LOC = level of concern. M = male animals. F= female animals. Mg/kg/day = milligrams per kilogram per day. LOAEL= lowest observed adverse effect level. NOAEL = no observed adverse effect level.

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to metconazole, EPA considered exposure under the petitioned-for tolerances as well as all existing metconazole tolerances in 40 CFR 180.617. EPA assessed dietary exposures from metconazole 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 metconazole. In estimating acute dietary exposure, EPA used food consumption information from the U. S. Department of Agriculture (USDA) 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA made the following assumptions for the acute exposure assessment: Tolerance-level residues and 100 percent crop treated (PCT). EPA used Dietary Exposure Evaluation Model (DEEM™) version 7.81 default processing factors.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the USDA 1994–1996 and 1998 CSFII. As to residue levels in food, EPA made the following assumptions for the chronic exposure assessment: Tolerance-level residues and 100 PCT. EPA used DEEM™ version 7.81 default processing factors.

iii. *Cancer.* Based on the data summarized in Unit III.A., EPA has determined that the quantification of risk using a non-linear approach will adequately account for all chronic toxicity, including carcinogenicity, that could result from exposure to metconazole. Therefore, the chonic RfD is expected to be protective of chronic toxicity including carcinogenicity. For the purpose of assessing cancer risk under this approach EPA relied upon the exposure estimate discussed in Unit III.C.1.ii.

iv. *Anticipated residue and PCT information.* EPA did not use anticipated residue and/or PCT information in the dietary assessment for metconazole. Tolerance level residues and/or 100 PCT were assumed for all food commodities.

2. *Dietary exposure from drinking water.* The Agency used screening level water exposure models in the dietary exposure analysis and risk assessment for metconazole in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of metconazole. Further information regarding EPA drinking water models used in pesticide exposure assessment can be found at *http://www.epa.gov/oppefed1/models/water/index.htm.*

Based on the Pesticide Root Zone Model/Exposure Analysis Modeling System (PRZM/EXAMS) and Screening Concentration in Ground Water (SCI–GROW) models, the estimated drinking water concentrations (EDWCs) of metconazole for acute exposures are estimated to be 45.48 parts per billion (ppb) for surface water and 0.38 ppb for ground water.

Chronic exposures for non-cancer assessments are estimated to be 38.16 ppb for surface water and 0.38 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 45.48 ppb was used to assess the contribution to drinking water.

For chronic dietary risk assessment, the water concentration of value 38.16 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).

Metconazole is currently registered for the following uses that could result in residential exposures: Turf and ornamentals. EPA assessed residential exposure using the following assumptions: Adults, adolescents and children may be exposed to metconazole from its currently registered turf and ornamental uses. Adults and adolescents may experience short- and intermediate-term dermal exposure from golfing and other activities on treated turf, as well as from tending treated ornamentals. Children may experience short- and intermediate-term dermal and incidental oral exposure from activities on treated turf. However, because dermal toxicity endpoints for the appropriate durations of exposure were not identified, and because inhalation exposure is considered to be insignificant for postapplication exposures, only children's incidental oral postapplication exposures have been assessed. Postapplication risks to children following the application of metconazole to home lawns were calculated for short- and intermediate-term incidental oral exposures. Further information regarding EPA standard assumptions and generic inputs for residential exposures may be found at

http://www.epa.gov/pesticides/trac/science/trac6a05.pdf.

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.''

Metconazole is a member of the triazole-containing class of pesticides. 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 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 conazoles share common mechanisms of toxicity and EPA is not following a cumulative risk approach based on a common mechanism of toxicity for the conazoles. 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://www.epa.gov/pesticides/cumulative.*

Triazole-derived pesticides can form the metabolite 1,2,4-triazole (T) and two triazole conjugates triazolylalanine (TA) and triazolylacetic acid (TAA). To support existing tolerances and to establish new tolerances for triazole-derivative pesticides, EPA conducted an initial human-health risk assessment for exposure to T, TA, and TAA resulting from the use of all current and pending uses of any triazole-derived fungicide as of September 1, 2005. The risk assessment was 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). In addition, the Agency retained the additional 10X Food Quality Protection Act (FQPA) safety factor (SF) for the protection of infants and children. The assessment included evaluations of risks for various subgroups, including those comprised of infants and children. The Agency's complete risk assessment can be found in the propiconazole reregistration docket at *http://www.regulations.gov,* Docket Identification (ID) Number EPA–HQ–OPP–2005–0497 and an update to the aggregate human health risk assessment for free triazoles and its conjugates may be found in Docket Identification (ID) Number EPA–HQ–OPP–2011–0179 entitled ''Common Triazole Metabolites: Updated Aggregate Human Health Risk Assessment to Address Tolerance Petitions for Metconazole.''

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 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.* Developmental studies in rats and rabbits show some evidence of developmental effects, but only at dose levels that are maternally toxic. There was no quantitative susceptibility to the fetuses of rats or rabbits following *in utero* exposure to metconazole. In the developmental toxicity study in rats, skeletal variations (predominantly lumbar ribs) occurred in the presence of maternal toxicity (decreased body weight gains). In the prenatal developmental toxicity study in rabbits, developmental effects (increased post-implantation loss and reduced fetal body weights) were observed at the same dose that caused maternal toxicity (decreased body weight gains, reduced food consumption and alterations in hematology parameters). In the 2-generation reproduction study in rats, offspring toxicity (reduced fetal body weights F2 offspring and decreased viability in F1 and F2 offspring) was observed only at the HDT, a dose which also resulted in parental toxicity as evidenced by reduced parental body weight and body weight gains, increased incidence of fatty hepatocyte changes in male parental animals and increased incidence of spleen congestion in F1 parental females. In the rat study, there is a concern for qualitative susceptibility (skeletal variation in the presence of minimal maternal toxicity) due to the presence of more severe effects at higher dose levels such as post-implantation loss, hydrocephaly and visceral anomalies. However, there is a clear NOAEL for these effects and the point of departure for this endpoint is based on skeletal variations. Therefore, it is concluded that there is no residual uncertainty for prenatal and/or postnatal toxicity.

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:

- The toxicity database is complete except for an acute neurotoxicity study.
- There is no concern for neurotoxicity with metconazole. However, in accordance with the revised 40 CFR part 158 data requirements, a neurotoxicity battery is required for risk assessment. The existing metconazole database does not include an acute neurotoxicity study, and thus remains a data deficiency. An acceptable subchronic neurotoxicity study showed no neurotoxic effects at levels that produced systemic toxicity in the study, as well as in other subchronic and chronic studies. Therefore, concern for potential neurotoxicity is low and the 10X FQPA factor is not retained.
- There is no evidence of susceptibility following *in utero* exposure in the rabbit developmental study. In the rat developmental study there is qualitative evidence of susceptibility, however the concern is low since the developmental effects occur in the presence of maternal toxicity, the NOAELs are well defined, and the dose/endpoint is used for acute dietary risk assessment for the sensitive population. There is no evidence of increased susceptibility in the offspring based on the result of the 2-generation reproduction study. Dietary exposure assessments were conducted using tolerance level residues and assumed 100 PCT. Therefore, the acute and chronic dietary (food only) exposure is considered an upper bound conservative estimate. The contribution from drinking water is minimal. The Agency concludes that the acute and

chronic exposure estimates in this analysis are unlikely to underestimate actual exposure. The drinking water component of the dietary assessment utilizes water concentration values generated by model and associated modeling parameters which are designed to provide conservative, health protective, high-end estimates of water concentrations which will not likely be exceeded. While there is potential for postapplication residential exposure, the Agency used the current conservative approaches for residential assessment. Exposures are unlikely to be under estimated because the assessment was a screening level assessment.

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 metconazole will occupy 3.8% of the aPAD for females 13–49 years old, the only population subgroup of concern.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to metconazole from food and water will utilize 12.6% of the cPAD for children 1–2 years 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 metconazole is not expected.

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

Using the exposure assumptions described in this unit for short-term exposures, EPA has concluded the combined short-term food, water, and non-occupational/residential post application exposures result in aggregate MOEs of 420 for children 1–2 years old and 1,700 for adults. Because EPA's level of concern for metconazole is a MOE of 100 or below, these MOEs are not of concern.

4. *Intermediate-term risk.* Intermediate-term risk takes into account intermediate-term residential exposure plus chronic exposure to food and drinking water (considered to be a background exposure level). Metconazole is currently registered for uses that could result in intermediate-term residential exposure, and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with intermediate-term residential exposures to metconazole.

Using the exposure assumptions described in this unit for intermediate-term exposures, EPA has concluded that the combined intermediate-term food, water, and non-occupational residential exposures result in aggregate MOEs of 460 for children 1–2 years old and 1,700 for adults. Because EPA's level of concern for metconazole is a MOE of 100 or below, these MOEs are not of concern.

5. *Aggregate cancer risk for U.S. population.* As explained in Unit III.A., the Agency has determined that the quantification of risk using a non-linear (i.e., RfD) approach will adequately account for all chronic toxicity, including carcinogenicity, that could result from exposure to metconazole. Therefore, based on the results of the chronic risk assessment discussed in Unit III.E.2., metconazole 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 metconazole residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (high performance liquid chromatography/tandem mass spectrometry (HPLC/MS/MS) method BASF D0604) is available to enforce the tolerance expression. 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 U.N. 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 metconazole on sugarcane.

C. Revisions to Petitioned-For Tolerances

Based on the results of the sugarcane crop field data and the tolerance calculation procedures, EPA has determined that separate tolerances for sugarcane, molasses are unnecessary. The highest metconazole residue from the sugarcane field trials is 0.036 ppm. This residue multiplied by the processing factor for molasses (0.036 × 1.2) yields 0.043 ppm. As this is less than the tolerance for sugarcane, cane at 0.06 ppm, the sugarcane, cane tolerance will cover molasses.

V. Conclusion

Therefore, tolerances are established for residues of metconazole, 5-[(4-chlorophenyl)methyl]-2,2-dimethyl-1-(1*H*-1,2,4-triazol-1-ylmethyl)cyclopentanol, including its metabolites and degradates in or on sugarcane, cane at 0.06 ppm.

VI. Statutory and Executive Order Reviews

This final rule establishes tolerances under section 408(d) of FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled *Regulatory Planning and Review* (58 FR 51735, October 4, 1993). Because this final rule has been exempted from review under Executive Order 12866, this final rule 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 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.,* 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 section 408(d) of FFDCA, 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 final rule 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 section 408(n)(4) of FFDCA. 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 final rule. In addition, this final rule does not 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).

This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note).

VII. Congressional Review Act

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

List of Subjects in 40 CFR Part 180

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

Dated: April 24, 2012.

Daniel J. Rosenblatt,

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. 321(q), 346a and 371.

■ 2. Section 180.617 is amended by alphabetically adding the following commodity to the table in paragraph (a) to read as follows:

§ 180.617 Metconazole; tolerances for residues.

(a) * * *

Commodity	Parts per million
* * * * *	
Sugarcane, cane	0.06
* * * * *	

* * * * *

[FR Doc. 2012–10689 Filed 5–3–12; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2011–0428; FRL–9346–5]

Carfentrazone-ethyl; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of carfentrazone-ethyl in or on crop group 18, non-grass animal feed (forage, hay, and seed). FMC Corporation requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective May 4, 2012. Objections and requests for hearings must be received on or before July 3, 2012, 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: EPA has established a docket for this action under docket identification (ID) number EPA–HQ–OPP–2011–0428. All documents in the docket are listed in the docket index available at *http://www.regulations.gov*. Although listed in the index, some information is not publicly available, e.g., Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available in the electronic docket at *http://www.regulations.gov*, or, if only available in hard copy, at the OPP Regulatory Public Docket in Rm. S–4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. The Docket Facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The Docket Facility telephone number is (703) 305–5805.

FOR FURTHER INFORMATION CONTACT: Bethany Benbow, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 347–8072; email address: *benbow.bethany@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. Potentially affected entities may include, but are not limited to those engaged in the following activities:

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

This listing is not intended to be exhaustive, but rather to provide a guide for readers regarding entities likely to be affected by this action. Other types of