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

Subpart I—Delaware

■ 2. In § 52.420, the table in paragraph (e) is amended by adding the entry ''2008 8-Hour Ozone Certification for Nonattainment New Source Review (NNSR)'' at the end of the table to read as follows:

§ 52.420 Identification of plan.

* * * * *

(e) * * *

Name of non-regulatory SIP revision	Applicable geographic area	State submittal date	EPA approval date	Additional explanation
*	* *	*	*	* *
2008 8-Hour Ozone Certification for Nonattainment New Source Review (NNSR).	Delaware portion of the Philadelphia-Wilmington-Atlantic City, nonattainment area and the Seaford, Delaware nonattainment area.	06/29/2018	8/12/2019, [insert **Federal Register** citation].	

[FR Doc. 2019–17128 Filed 8–9–19; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2018–0688; FRL–9997–09]

Pydiflumetofen; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of pydiflumetofen in or on multiple commodities which are identified and discussed later in this document. Syngenta Crop Protection requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective August 12, 2019. Objections and requests for hearings must be received on or before October 11, 2019 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–2018–0688, 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–2018–0688 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 October 11, 2019. 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–2018–0688, 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 April 19, 2019 (84 FR 16430) (FRL–9991–14), 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 8F8696) by Syngenta Crop Protection, LLC, P.O. Box 18300, Greensboro, NC 27419. The petition requested that 40 CFR part 180 be amended by establishing tolerances for residues of the fungicide, pydiflumetofen, in or on root vegetable crop subgroup 1A at 0.30 parts per million (ppm); bulb vegetable crop subgroup 3–07A at 0.20 ppm; bulb vegetable crop subgroup 3–07B at 2 ppm; *brassica* leafy greens subgroup 4–16B at 50 ppm; *brassica* head and stem crop group 5–16 at 3 ppm; leaves of root and tuber vegetables, crop group 2 at 15.0 ppm; edible-podded legume vegetables subgroup 6A at 1.0 ppm; succulent shelled pea and bean subgroup 6B at 0.09 ppm; citrus fruit crop group 10–10 at 0.90 ppm; citrus oil at 15 ppm; pome fruit crop group 11–10 at 0.20 ppm; apple, wet pomace at 1.0 ppm; stone fruit, cherry subgroup 12–12A at 2.0 ppm; stone fruit, peach subgroup 12–12B at 1.0 ppm; stone fruit, plum subgroup 12–12C at 0.6 ppm; plum, prune at 1.5 ppm; bushberry crop subgroup 13–07B at 5 ppm; berries, low growing crop subgroup 13–07G, except cranberry and blueberry, at 1 ppm; tree nuts crop group 14–12, nutmeat at 0.05 ppm; almond hull at 9.0 ppm; cottonseed subgroup 20C, cotton undelinted seed at 0.4 ppm; cotton gin by-products at 7.0 ppm; sunflower subgroup 20B at 0.60 ppm; sorghum grain at 3.0 ppm; sorghum forage at 1.5 ppm; and sorghum stover at 10 ppm. That document referenced a summary of the petition prepared by Syngenta Crop Protection, 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, EPA has modified the levels at which some of the commodities are being set as well as some of the commodity definitions. The reasons for these changes are 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 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 pydiflumetofen including exposure resulting from the tolerances established by this action. EPA's assessment of exposures and risks associated with pydiflumetofen follows.

A. Toxicological Profile

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children.

The liver was a common target across species tested, likely in part due to the extensive first pass metabolism of absorbed pydiflumetofen. Liver effects were either concurrent with body weight depression and other target organ toxicity as in rats, or the first symptoms of treatment-related toxicity as in mice and dogs. Liver toxicity commonly manifested as increased liver weight concordant with hepatocyte hypertrophy in all species and was accompanied by increased cholesterol and triglyceride serum levels and a higher incidence of liver masses and eosinophilic foci of cellular alteration in mice and increased serum levels of liver enzymes and triglycerides in dogs. Male mice further exhibited a dose-dependent increase in the incidence of hepatocellular adenomas and carcinomas (accounted for separately and combined) and in the frequency of individual mice exhibiting multiple liver adenomas following chronic exposure. Treatment-related liver tumors were not observed in female mice nor in rats of either sex.

Body weight effects were also observed in rodents in response to treatment. Adult rats experienced depressed body weight following both subchronic (concurrent with liver toxicity) and chronic oral exposure (in isolation) and mice exhibited body weight depression following chronic exposure concurrent with symptoms of liver toxicity. A dose-dependent increase in the incidence and severity of thyroid gland follicular cell hypertrophy was also noted in rats following subchronic dietary exposure at doses greater than or equal to 587 milligrams/kilogram/day (mg/kg/day). The isolated thyroid findings occurred at a dose level over an order of magnitude above the subchronic and chronic point of departures (PODs) selected for risk assessment. In general, short and intermediate duration repeat dose oral exposures were well tolerated by adult rodents and dogs. Rodents were, however, considerably less tolerant of long-term exposure. Liver and body weight effects manifested at doses 25 and 12 times lower in chronic studies as compared to subchronic studies in mice and rats, respectively. A similar progression of toxicity was not evident in dogs.

The database does not support a conclusion that the pesticide is a neurotoxicant. Although a dose-dependent decrease in two locomotor activity parameters, number of rears and total distance traveled, was observed in female adult rats only within 6 hours of exposure following acute gavage oral exposure to doses greater than or equal to 300 milligrams/kilogram (mg/kg) in the acute neurotoxicity study, there were no neuropathology lesions or consistent evidence of other behavioral changes accompanying the depressed locomotor activity up to acute doses of 2,000 mg/kg. Detailed functional observations of rats and dogs following repeat dose dietary exposure did not identify similar changes in locomotor activity or any other behavioral changes indicative of neurotoxicity.

Body weight toxicity was not a unique observation in adults; it was also observed in rat offspring. In the two-generation reproduction study, rat pups exhibited significantly reduced weight during lactation that persisted through weaning and into adulthood. The pup body weight decrements were observed in the absence of parental toxicity indicating post-natal susceptibility to pydiflumetofen exposure. There was no evidence of enhanced fetal susceptibility following gestational exposure to pregnant rats or rabbits in the developmental studies.

Although there is some evidence of carcinogenicity in the database (*i.e.,* hepatocellular adenomas and carcinomas in male mice), the Agency

has concluded that pydiflumetofen is not likely to be carcinogenic to humans at doses that do not induce a proliferative response in the liver. This conclusion is based on the limited nature of tumors seen in the available data (liver tumors found only in male mice), the fact that pydiflumetofen is not a mutagenic concern *in vivo,* and available mode of action data. The available mode of action data supports the Agency's conclusion that liver tumors are likely induced via activation of the constitutive androstane receptor (CAR) and subsequent stimulation of hepatocellular proliferation, and that hepatocellular proliferation is not likely to occur at the doses at which EPA is regulating exposure to pydiflumetofen. As a result, a non-linear approach using the chronic reference dose would adequately account for chronic toxicity, including carcinogenicity.

Pydiflumetofen exhibited low acute toxicity via the dermal and inhalation route. Acute dermal exposure to dermal doses of 5000 mg/kg elicited reduced activity in rats similar to observations following acute oral exposure, but it did not incur mortality. Acute exposure did not irritate the skin nor did it elicit dermal sensitization. No dermal or systemic toxicity was observed following repeat-dose dermal exposures up to 1000 mg/kg/day. Acute lethality from inhalation exposure was limited to high inhalation concentrations and it was a mild acute eye irritant. The requirement for the subchronic inhalation toxicity study was waived for the pydiflumetofen risk assessment based on a weight of evidence (WoE) approach that considered all of the available hazard and exposure information for pydiflumetofen, including: (1) the physical-chemical properties of pydiflumetofen indicated low volatility (vapor pressure is 3.98×10^{-9} mm Hg at 25 °C); (2) the use pattern and exposure scenarios; (3) the margins of exposure for the worst case scenarios are ≥13,000 using an oral point of departure and assuming inhalation and oral absorption are equivalent; (4) pydiflumetofen exhibits low acute inhalation toxicity (Category IV); and (5) the current endpoints selected for risk assessment, liver toxicity and pup body weight decrements, were the most sensitive effects identified in the database and an inhalation study is not likely to identify a lower POD or more sensitive endpoint for risk assessment.

The toxicity of 2,4,6-trichlorophenol—a pydiflumetofen metabolite and residue of concern in livestock commodities—was evaluated based on studies from the open literature that were provided by the registrant, identified in a previous EPA review of 2,4,6-trichlorophenol (*https://www.epa.gov/sites/production/files/2016-09/documents/2-4-6-trichlorophenol.pdf*) and the Agency for Toxic Substance and Disease Registry (ATSDR) review of chlorophenols (*https://www.atsdr.cdc.gov/toxprofiles/tp107.pdf*), or retrieved in a search of the literature conducted for this risk assessment. Based on available information, the absorption, distribution, metabolism and elimination (ADME) for 2,4,6-trichlorophenol is similar to the ADME profile for pydiflumetofen: Near complete absorption and extensive metabolism followed by rapid excretion without appreciable tissue accumulation. Oral exposure to 2,4,6-trichlorophenol elicited effects in the liver, kidneys, and hematopoietic system as well as body weight depression. Subchronic oral exposure in rats elicited an increase in liver, kidney (males only), and spleen weight, an increase in total protein and albumin serum levels, a moderate to marked increase in splenic hematopoiesis, and an increased incidence of hepatocyte vacuolation.

Following chronic dietary exposure, male rats exhibited an increased incidence of leukemias, lymphomas, and nephropathy, and both sexes exhibited an increased incidence of bone marrow hyperplasia, leukocytosis, fatty metamorphosis in the liver, and chronic inflammation of the kidney. Tissue specific toxicity in mice was limited to the liver and manifest as an increased incidence of liver adenomas and carcinomas following chronic exposure. Adult body weight depression was observed in both rodent species. Mortality also occurred with greater frequency in both species at or above the limit dose. The few studies that examined developmental and offspring effects presented equivocal evidence of offspring toxicity following exposure to 2,4,6-trichlorophenol. Prenatal subchronic drinking water exposure in female rats led to a reduction in litter size and perinatal drinking water exposure in rats elicited changes in offspring spleen and liver weight; however, the health of the dams and its potential contribution to the manifestation of the offspring effects was not discussed in this study so it is unclear whether the offspring toxicity is a direct result of exposure or secondary to maternal toxicity. In a separate study, pup body weight decrements were observed in the presence and absence of parental toxicity following subchronic exposure, but the body weight effect was considered a consequence of the larger litter size rather than treatment. In any event, the effects seen in these studies occurred at doses above the endpoints selected for regulation of pydiflumetofen exposure.

These studies illustrate a spectrum of responses to increasing oral 2,4,6-trichlorophenol exposure: Isolated organ weight changes and a reduction in litter size were observed at doses as low as 30 mg/kg/day with adverse effects in the target tissues and significant body weight depression in adult animals manifesting when the oral dose exceeded 200 mg/kg/day. However, the 2,4,6-trichlorophenol doses that elicited the subchronic and chronic toxicity described above were not below the empirical no-observed-adverse-effect-levels (NOAELs) established in comparable pydiflumetofen guideline studies (after converting both to millimoles/kg/day) suggesting that direct exposure to 2,4,6-trichlorophenol is not more toxic than direct exposure to pydiflumetofen. Direct exposure to 2,4,6-trichlorophenol is anticipated from dietary exposures only. The PODs selected for pydiflumetofen are protective of the adverse effects reported in the 2,4,6-trichlorophenol literature and, therefore, are adequate for assessing direct dietary exposure to 2,4,6-trichlorophenol.

The carcinogenic potential of 2,4,6-tricholorophenol was assessed in 1990 by EPA and classified as a B2-probable human carcinogen in accordance with the 1986 cancer classification guidance based on an increased incidence of combined lymphomas and leukemias in male F344 rats and hepatocellular adenomas or carcinomas in male and female mice. Since that evaluation of 2,4,6-trichlorophenol, new literature has been published on the human relevance of leukemias in the F344 rat. The EPA re-evaluated the 2,4,6-trichlorophenol carcinogenicity literature and the broader scientific literature on rodent leukemia to determine if the data supported conducting a separate cancer assessment for 2,4,6-trichlorophenol. The rodent leukemia literature indicated that the leukemia finding in male F344 rats is common for this strain of rat, is highly variable, and lacks a direct human correlate. Although treatment-related, the EPA concluded the leukemia incidence in rats did not support a linear approach to cancer quantification given its questionable relevance to human health risk assessment. Furthermore, the incidence of lymphomas was not remarkable when examined independently from the leukemias and thus not evidence of carcinogenicity in isolation. The liver

tumors observed in male and female mice were considered treatment-related; however, the tumors could not be solely attributed to 2,4,6-trichlorophenol exposure because the investigators did not account for known carcinogenic contaminants of commercial 2,4,6-trichlorophenol solutions that may have contributed to the induction of the liver tumors. These carcinogenic contaminants would not be present when 2,4,6-trichlorophenol is formed through metabolism; therefore, these data were not considered strong evidence of carcinogenicity and did not support a linear approach to 2,4,6-trichlorophenol cancer quantification for exposure resulting from pydiflumetofen use. The literature also did not suggest 2,4,6-trichlorophenol was a mutagenic concern *in vivo.*

Based on the limited evidence of carcinogenicity and mutagenicity for the metabolite, the EPA concluded that using the reference dose (RfD) approach with the chronic dietary POD selected for the pydiflumetofen dietary assessment would be adequate for assessing direct dietary exposure to 2,4,6-trichlorophenol from the proposed pydiflumetofen uses. Because the chronic POD selected for pydiflumetofen is 66 and 165x lower than the 2,4,6-trichlorophenol dose (on a molar basis) that elicited tumors in rats and mice, respectively, this approach will be protective of potential carcinogenicity from exposure to the metabolite. Consequently, a separate cancer dietary assessment for 2,4,6-trichlorophenol is not warranted at this time.

Specific information on the studies received and the nature of the adverse effects caused by pydiflumetofen as well as the NOAEL and the lowest-observed-adverse-effect-level (LOAEL) from the toxicity studies can be found at *http://www.regulations.gov* in the document titled, ''*Pydiflumetofen. Human Health Risk Assessment for New Foliar Uses on Berries, Low Growing, Crop Subgroup 13–07G; Brassica Head and Stem Crop Group 5–16; Brassica Leafy Greens Subgroup 4–16B; Bulb Vegetable Crop Subgroup 3–07A; Green Onion Crop Subgroup 3–07B; Bushberry Crop Subgroup 13–07B; Citrus Fruit Crop Group 10–10; Cottonseed Subgroup 20C; Edible-podded Legume Vegetables Subgroup 6A; Succulent Shelled Pea and Bean Subgroup 6B; Pome Fruit Crop Group 11–10; Root Vegetable Crop Subgroup 1A; Sorghum; Stone Fruit Crop Subgroups 12–12A, 12–12B, and 12–12C; Sunflower Subgroup 20B; Tree Nut Crop Group 14–12; Leaves of Root and Tuber Vegetable Crop Group 2; and New Seed Treatment Uses on Rapeseed Crop Subgroup 20A and Soybean; and Registration of a New Seed Treatment End-Use Product*'' on pages 56–69 in docket ID number EPA–HQ–OPP–2018–0688.

B. Toxicological Points of Departure/ Levels of Concern

Once a pesticide's toxicological profile is determined, EPA identifies toxicological 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 the NOAEL and the LOAEL are identified. 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 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-pesticide.*

A summary of the toxicological endpoints for pydiflumetofen used for human risk assessment is discussed in Unit III.B. of the final rule published in the **Federal Register** of May 24, 2018 (83 FR 24036) (FRL–9976–66). Because the available data indicate that exposure to 2,4,6-trichlorophenol is not more toxic than direct exposure to pydiflumetofen and that there is insufficient information to warrant a separate cancer assessment of the metabolite at this time, EPA concludes that the endpoints for pydiflumetofen will be protective of effects from exposure to the metabolite 2,4,6-triclorophenol.

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to pydiflumetofen, EPA considered exposure under the petitioned-for tolerances as well as all existing pydiflumetofen tolerances in 40 CFR 180.699. EPA assessed dietary exposures from pydiflumetofen 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 pydiflumetofen. In estimating acute dietary exposure, EPA used 2003–2008 food consumption data from the US Department of Agriculture's (USDA's) National Health and Nutrition Examination Survey, What We Eat in America (NHANES/WWEIA). As to residue levels in food, EPA assumed tolerance-level residues and 100 percent crop treated (PCT).

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used 2003–2008 food consumption data from USDA's NHANES/WWEIA. As to residue levels in food, EPA assumed tolerance-level residues and 100 PCT.

iii. *Cancer.* As discussed in Unit III.A., the Agency has determined that a separate cancer assessment is not necessary for assessing exposure to pydiflumetofen. Because the chronic reference dose (cRfD) is below 10 mg/kg/day, *i.e.,* the lowest dose known to induce hepatocellular proliferation based on available MOA data, the chronic assessment will be protective for assessing direct dietary exposure to pydiflumetofen. Also discussed in Unit II.A. is the Agency's conclusion that a separate cancer assessment is not required for assessing exposure to 2,4,6-trichlorophenol (free and conjugated) and the cRfD will be protective of potential carcinogenic effects.

iv. *Anticipated residue and PCT information.* EPA did not use anticipated residue or PCT information in the dietary assessment for pydiflumetofen. Tolerance-level residues and 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 pydiflumetofen and its degradate SYN545547 in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of pydiflumetofen. 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 Pesticides in Water Calculator (PWC) the estimated drinking water concentrations (EDWCs) of

pydiflumetofen for acute exposures are estimated to be 10.4 parts per billion (ppb) for surface water and 113.3 ppb for ground water and for chronic exposures are estimated to be 3.37 ppb for surface water and 101 ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For the acute dietary risk assessment, the water concentration value of 113.3 ppb was used to assess the contribution to drinking water. For the chronic dietary risk assessment, the water concentration of value 101 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).

Pydiflumetofen is registered for the following uses that could result in residential exposures: Golf course turf; and ornamentals grown in greenhouses, nurseries, and fields for residential planting. EPA assessed residential exposure using the following assumptions: Residential handler exposures are not expected since the turf and ornamental use labels indicate that the product is intended for use by professional applicators, while the crop use labels include the statement ''Not for residential use.'' As a result, residential handler exposures are not expected. There is the potential for residential short-term post-application exposure for individuals exposed as a result of being in an environment that has been previously treated with pydiflumetofen.

The quantitative exposure/risk assessment for residential post-application exposures is based on the short-term dermal exposure from contact with residues on treated golf course turf while golfing for adults, children 6 to less than 11 years old, and children 11 to less than 16 years old, and short-term dermal exposure from post-application activities with treated ornamental plants for adults and for children ages 6 to less than 11. Intermediate-term exposures are not expected.

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.''

EPA has not found pydiflumetofen to share a common mechanism of toxicity with any other substances, and pydiflumetofen does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has assumed that pydiflumetofen does not have a common mechanism of toxicity with other substances. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see EPA's website at *http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/cumulative-assessment-risk-pesticides.*

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 Food Quality Protection Act Safety Factor (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.* There was no evidence of fetal sensitivity or toxicity in rat and rabbit developmental studies; however, quantitative offspring sensitivity was noted in the 2-generation reproduction study. Pup body-weight depression starting on day 4 of lactation and persisting into adulthood was observed at doses that did not elicit an adverse response in the parental rats. Although body weight was depressed in these animals after maturity and during the mating and post-mating period (specifically in males), it was considered evidence of offspring susceptibility because the lower body weight was a result of impaired growth in the pups. Reduced pup weight, reduced litter size, and increased liver and spleen weight in offspring was also noted following prenatal and perinatal exposure to the pydiflumetofen metabolite, 2,4,6-trichlorophenol. PODs were selected for each exposure scenario to be protective of the parent and metabolite offspring toxicity and offspring susceptibility in the risk evaluation.

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 pydiflumetofen is complete.

ii. Regarding neurotoxicity, evidence of behavioral changes in the pydiflumetofen toxicity database was limited to adult rats in the acute neurotoxicity study (ACN). Female rats exhibited depressed locomotor activity in the form of fewer number of rears and less distance traveled following acute exposure to doses of pydiflumetofen ≥ 300 mg/kg (3x to 30x higher than the PODs selected for risk assessment). Male rats did not exhibit any symptoms of neurotoxicity following acute exposure up to 2,000 mg/kg/day. No evidence of neurotoxicity was observed in the subchronic rat and dog dietary studies that included additional detailed functional observations to identify neurological impairment nor in the routine clinical observations of the chronic studies and the guideline requirement for a subchronic neurotoxicity (SCN) study was waived. The concern for neurotoxicity in sensitive populations is low because the behavioral effects observed in the acute neurotoxicity studies have well-defined NOAEL/LOAELs, the PODs selected for risk assessment are protective of the acute behavioral change observed in females, there were no corresponding neuropathology changes in females exhibiting decreased locomotor activity, and there was no evidence of neurotoxicity following repeat-dose exposure.

iii. There was evidence of quantitative offspring sensitivity in the 2-generation reproduction study; however, as noted in Section D.2., PODs were selected for each exposure scenario to be protective of the offspring susceptibility in the risk evaluation.

iv. There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessments were performed based on 100 PCT and tolerance-level residues. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to pydiflumetofen in drinking water. EPA used similarly conservative assumptions

to assess residential post-application exposure. These assessments will not underestimate the exposure and risks posed by pydiflumetofen.

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 pydiflumetofen will occupy 9.5% of the aPAD for children 3 to 5 years 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 pydiflumetofen from food and water will utilize 29% of the cPAD for children 1 to 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 pydiflumetofen 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).

Pydiflumetofen 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 pydiflumetofen.

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 400 for adults, 560 for children 6 to less than 11 years old, and 2400 for children 11 to less than 16 years old. Because EPA's level of concern for pydiflumetofen 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, pydiflumetofen 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 pydiflumetofen.

5. *Aggregate cancer risk for U.S. population.* As discussed in Unit III., the Agency has concluded that regulating on the chronic reference dose will be protective of potential carcinogenicity from exposure to pydiflumetofen. Because the chronic risk assessment did not exceed the Agency's level of concern, the Agency concludes there is not an aggregate cancer risk from exposure to pydiflumetofen.

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 pydiflumetofen residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Analytical multi-residue method QuEChERS (Quick, Easy, Cheap, Effective, Rugged, and Safe) as described in Eurofins validation study S14–05402 was independently validated in the following crop matrices: Lettuce (high water content), wheat grain (high starch content), oil seed rape (high oil content) and coffee bean (difficult commodity).

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 any MRLs for pydiflumetofen at this time.

C. Revisions to Petitioned-for Tolerances

EPA has modified several of the commodity definitions to be consistent with Agency nomenclature as well as the numerical expression of many of the proposed tolerance values to conform to current EPA policy on trailing zeroes.

For the tolerance in or on berries, low growing crop subgroup 13–07G, the proposed exceptions to the tolerance for lowbush blueberry and for cranberry are not appropriate, since use on both lowbush blueberry and cranberry are included on the proposed label 100–1601 and listed under directions for use on strawberry and low growing berry crop subgroup 13–07G.

EPA has modified several of the petitioned-for tolerances for the following reasons. For the tolerances in/on vegetable, root, subgroup 1A; nut, tree, group 14–12; pea and bean, succulent shelled, subgroup 6B; and fruit, citrus, group 10–10, the petitioner combined the individual commodities together in one calculator analysis when it is Agency practice to separate commodities. For the tolerances in/on vegetable, leaves of root and tuber, group 2 and sunflower subgroup 20B, the petitioner used U.S. residue data only where the Agency used both U.S. and Canadian residue data for harmonization purposes. For the tolerance in prune, the petitioner used the highest residue (HR) value from the field trials while the Agency's practice is to use the highest average field trial (HAFT) value from the field trials. For the tolerance in citrus oil, the Agency's practice is to use the HAFT and median concentration factor, and based on these data, the appropriate tolerance in citrus oil is 30 ppm; hence, the petitioned-for tolerance (15 ppm), the basis for which was not explained in the petition, is too low. As a result, several of the tolerance levels being established are different than those proposed by the petitioner.

V. Conclusion

Therefore, tolerances are established for residues of pydiflumetofen including its metabolites and degradates, in or on the following commodities. Compliance with the tolerance levels specified below is to be determined by measuring only pydiflumetofen (3-(difluoromethyl)-*N*-methoxy-1-methyl-*N*-[1-methyl-2-(2,4,6-trichlorophenyl)ethyl]-1*H*-pyrazole-4-carboxamide) in or on the commodity: Almond, hulls at 9 ppm; apple, wet pomace at 1 ppm; berry, low growing, subgroup 13–07G at 1 ppm; *brassica,* leafy greens, subgroup 4–16B at 50 ppm; bushberry subgroup 13–07B at 5 ppm; cherry subgroup 12–12A at 2 ppm; cotton, gin byproducts at 7 ppm; cottonseed subgroup 20C at 0.4 ppm; fruit, citrus, group 10–10 at 1 ppm; fruit, citrus, group 10–10, oil at 30 ppm; fruit, pome, group 11–10 at 0.2 ppm; nut, tree, group 14–12 at 0.07 ppm; onion, bulb, subgroup 3–07A at 0.2 ppm; onion, green, subgroup 3–07B at 2 ppm; pea and bean, succulent shelled, subgroup 6B at 0.1 ppm; peach subgroup 12–12B at 1 ppm; plum, prune, dried at 1 ppm; plum subgroup 12–12C at 0.6 ppm; sorghum, grain, forage at 1.5 ppm; sorghum, grain, grain at 3 ppm; sorghum, grain, stover at 10 ppm; sunflower subgroup 20B at 0.5 ppm; vegetable, *brassica,* head and stem, group 5–16 at 3 ppm; vegetable, leaves of root and tuber, group 2 at 10 ppm; vegetable, legume, edible podded, subgroup 6A at 1 ppm; and vegetable, root, subgroup 1A at 0.5 ppm.

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), nor is it considered a regulatory action under Executive Order 13771, entitled ''Reducing Regulations and Controlling Regulatory Costs'' (82 FR 9339, February 3, 2017). 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: 7/26/2019.

Daniel 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. In § 180.699, add alphabetically the commodities almond, hulls; apple, wet pomace; berry, low growing, subgroup 13–07G; *Brassica,* leafy greens, subgroup 4–16B; bushberry subgroup 13–07B; cherry subgroup 12–12A; cotton, gin byproducts; cottonseed subgroup 20C; fruit, citrus, group 10–10; fruit, citrus, group 10–10, oil; fruit, pome, group 11–10; nut, tree, group 14–12; onion, bulb, subgroup 3–07A; onion, green, subgroup 3–07B; pea and bean, succulent shelled, subgroup 6B; peach subgroup 12–12B; plum, prune, dried; plum subgroup 12–12C; sorghum, grain, forage; sorghum, grain, grain; sorghum, grain, stover; sunflower subgroup 20B; vegetable, *Brassica,* head and stem, group 5–16; vegetable, leaves of root and tuber, group 2; vegetable, legume, edible podded, subgroup 6A; and vegetable, root, subgroup 1A to the table in paragraph (a) to read as follows:

§ 180.699 Pydiflumetofen; tolerances for residues.

(a) * * *

Commodity	Parts per million
Almond, hulls	9
Apple, wet pomace	1

Commodity	Parts per million
* * * * * * *	
Berry, low growing, subgroup 13–07G	1
Brassica, leafy greens, subgroup 4–16B	50
Bushberry subgroup 13–07B	5
* * * * * * *	
Cherry subgroup 12–12A	2
* * * * * * *	
Cotton, gin byproducts	7
Cottonseed subgroup 20C	0.4
Fruit, citrus, group 10–10	1
Fruit, citrus, group 10–10, oil	30
Fruit, pome, group 11–10	0.2
* * * * * * *	
Nut, tree, group 14–12	0.07
* * * * * * *	
Onion, bulb, subgroup 3–07A	0.2
Onion, green, subgroup 3–07B	2
Pea and bean, succulent shelled, subgroup 6B	0.1
Peach subgroup 12–12B	1
* * * * * * *	
Plum, prune, dried	1
Plum subgroup 12–12C	0.6
* * * * * * *	
Sorghum, grain, forage	1.5
Sorghum, grain, grain	3
Sorghum, grain, stover	10
* * * * * * *	
Sunflower subgroup 20B	0.5
* * * * * * *	
Vegetable, *Brassica,* head and stem, group 5–16	3
* * * * * * *	
Vegetable, leaves of root and tuber, group 2	10
Vegetable, legume, edible podded, subgroup 6A	1
* * * * * * *	
Vegetable, root, subgroup 1A	0.5
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[FR Doc. 2019–17144 Filed 8–9–19; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2018–0127; FRL–9997–00]

Propiconazole; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of propiconazole in or on multiple commodities which are identified and discussed later in this document. Interregional Research Project No. 4 (IR–4) requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective August 12, 2019. Objections and requests for hearings must be received on or before October 11, 2019, 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–2018–0127, 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.*