

**PART 52—APPROVAL AND PROMULGATION OF IMPLEMENTATION PLANS**Authority: 42 U.S.C. 7401 *et seq.*

“202–0060”, “202–0090”, and “250–0030” to read as follows:

**Subpart MM—Oregon****§ 52.1970 Identification of plan.**

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

■ 2. In § 52.1970, table 2 in paragraph (c) is amended by revising the entries

\* \* \* \* \*  
(c) \* \* \*

TABLE 2—EPA-APPROVED OREGON ADMINISTRATIVE RULES (OAR)

State citation	Title/subject	State effective date	EPA approval date	Explanations
<b>CHAPTER 340—DEPARTMENT OF ENVIRONMENTAL QUALITY</b>				
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<b>Division 202—Ambient Air Quality Standards and PSD Increments</b>				
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<b>Ambient Air Quality Standards</b>				
* * * * *				
202–0060	Suspended Particulate Matter	10/16/2015	5/24/2018, [insert Federal Register citation].	Federal Reg-
* * * * *				
202–0090	Ozone	07/13/2017	5/24/2018, [insert Federal Register citation].	Federal Reg-
* * * * *				
<b>Division 250—General Conformity</b>				
* * * * *				
250–0030	Definitions	10/16/2015	5/24/2018, [insert Federal Register citation].	Federal Reg-
* * * * *				

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■ 3. Section 52.1991 is amended by adding paragraphs (f) and (g) to read as follows:

**§ 52.1991 Section 110(a)(2) infrastructure requirements.**

\* \* \* \* \*

(f) The EPA approves Oregon’s December 27, 2013, submission as meeting the following CAA section 110(a)(2) infrastructure elements for the 2010 nitrogen dioxide and 2010 sulfur dioxide NAAQS: (A), (B), (C), (D)(i)(II), (D)(ii), (E), (F), (G), (H), (J), (K), (L), and (M).

(g) The EPA approves Oregon’s October 20, 2015, submission as meeting the following CAA section 110(a)(2) infrastructure elements for the 2012 PM<sub>2.5</sub> NAAQS: (A), (B), (C), (D)(i)(II), (D)(ii), (E), (F), (G), (H), (J), (K), (L), and (M).

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**ENVIRONMENTAL PROTECTION AGENCY****40 CFR Part 180**

[EPA–HQ–OPP–2015–0775; FRL–9976–66]

**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 May 24, 2018. Objections and requests for hearings must be received on or before July 23, 2018, 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–2015–0775, 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: [RDfRNtices@epa.gov](mailto:RDfRNtices@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](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–2015–0775 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 23, 2018. 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–2015–0775, by one of the following methods:

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

##### II. Summary of Petitioned-For Tolerance

In the **Federal Register** of February 7, 2017 (82 FR 9555) (FRL–9956–86), EPA issued a document pursuant to FFDCA section 408(d)(3), 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 6F8474) by Syngenta Crop Protection, LLC, P.O. Box 18300, Greensboro, NC 27419. The petition requested to establish tolerances in 40 CFR part 180 for residues of the fungicide pydiflumetofen in or on barley, grain at 4.0 ppm; barley, hay at 30.0 ppm; barley, straw at 30.0 ppm; corn, field, grain at 0.015 ppm; corn, field, forage at 6.0 ppm; corn, field, stover at 15.0 ppm; corn, field, milled by products at 0.06 ppm; corn, pop, grain at 0.015 ppm; corn, pop, forage at 6.0 ppm; corn, pop, stover at 15.0 ppm; corn, sweet, ear at 0.01 ppm; corn, sweet, forage at 5.0 ppm; corn, sweet, stover at 9.0 ppm; corn, sweet, cannery waste at 2.0 ppm; crop subgroup 4–15A, leafy greens subgroup at 40.0 ppm; crop subgroup 22B, leaf petiole vegetable subgroup at 15.0 ppm; fruits, small vine climbing, except fuzzy kiwi subgroup 13–07F at 1.5 ppm; grape, raisin at 2.0 ppm; grape, wet pomace at 1.5 ppm; grain, aspirated fractions at 100.0 ppm; grain, cereal, forage, fodder and straw, group 16 at 50 ppm; oat, grain at 2.0 ppm; oat, forage at 10.0 ppm; oat, hay at 40.0 ppm; oat, straw at 20.0 ppm; peas and bean, dried shelled, except soybean, subgroup 6C at 0.4 ppm; peas, hay at 40.0 ppm; peas, vine at 6.0 ppm; peanut, nutmeat at 0.02 ppm; peanut, refined oil at 0.05 ppm; peanut, hay at 20.0 ppm; potato, wet peel at 0.03 ppm; potato, dried pulp at 0.05 ppm; potato,

processed waste at 0.03 ppm; quinoa, grain at 4.0 ppm; rapeseed, subgroup 20A at 0.9 ppm; rye, grain at 4.0 ppm; rye, hay at 50.0 ppm; rye, straw at 30.0 ppm; soybean, seed at 0.4 ppm; soybean, forage at 30.0 ppm; soybean, hay at 150 ppm; tomato, dried pomace at 15.0 ppm; tomato, wet pomace at 1.5 ppm; tomato, sun-dried at 3.0 ppm; vegetables, fruiting, crop group 8–10 at 0.6 ppm; vegetables, tuberous and corm subgroup 1C at 0.015 ppm; vegetables, cucurbit, crop group 9 at 0.5 ppm; wheat, grain at 0.3 ppm; wheat, forage at 15.0 ppm; wheat, hay at 50.0 ppm; and wheat, straw at 30.0 ppm.

Additionally, the petition requested to establish tolerances for residues of pydiflumetofen and 2,4,6-trichlorophenol in or on cattle, fat at 0.03 ppm; cattle, kidney at 0.02 ppm; cattle, liver at 0.04 ppm; cattle, meat at 0.02 ppm; cattle, byproducts at 0.04 ppm; goat, fat at 0.03 ppm; goat, kidney at 0.02 ppm; goat, liver at 0.04 ppm; goat, meat at 0.02 ppm; goat, meat byproducts at 0.04 ppm; horse, fat at 0.03 ppm; horse, kidney at 0.02 ppm; horse, liver at 0.04 ppm; horse, meat at 0.02 ppm; horse, meat byproducts at 0.04 ppm; milk at 0.02 ppm; milk, cream at 0.04 ppm; sheep, fat at 0.03 ppm; sheep, kidney at 0.02 ppm; sheep, liver at 0.04 ppm; sheep, meat at 0.02 ppm; and sheep, meat byproducts at 0.04 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.

Consistent with the authority in FFDCA section 408(d)(4)(A)(1), EPA is establishing tolerances as requested with some variations. The reasons for these changes are explained in Unit IV.D.

##### 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 mg/kg/day. 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 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 2000 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  $3.98 \times 10^{-9}$  mm Hg at 25 °C); (2) the use pattern and exposure scenarios; (3) the margins of exposure for the worst case scenarios are  $\geq 13,000$  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. The absorption,

distribution, metabolism and elimination (ADME) information available 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 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. Furthermore, direct exposure to 2,4,6-trichlorophenol is anticipated from dietary exposures only and the dietary PODs selected for pydiflumetofen are protective of all adverse effects reported in the 2,4,6-trichlorophenol literature.

The carcinogenic potential of 2,4,6-trichlorophenol 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 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 referenced in this section and the nature of the adverse effects caused by pydiflumetofen and its metabolite 2,4,6-trichlorophenol, as well as the no-observed-adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from the toxicity studies can be found at <http://www.regulations.gov> in the document titled "Pydiflumetofen. Human Health Risk Assessment for Foliar Uses on Cereals (Wheat, Triticale, Barley, Rye, and Oat), Quinoa, Corn (Field, Pop, and Sweet), Cucurbits Crop Group 9 (Including Greenhouse Use on Cucumber), Fruiting Vegetables Crop Group 8–10, Small Fruit Vine Climbing Subgroup 13–07F (Except Fuzzy Kiwifruit), Peas and Beans Dried Shelled Subgroup 6C, Leafy Greens Subgroup 4–16A, Leaf Petiole Vegetables Subgroup 22B, Peanuts, Rapeseed Subgroup 20A, Soybean, Tuberous and Corm Vegetable Subgroup 1C, Golf Course Turf, and Ornamentals (Including Greenhouse Use)" on pages 61–73 in docket ID number EPA–HQ–OPP–2015–0775.

#### *B. Toxicological Points of Departure/ Levels of Concern*

Once a pesticide's toxicological profile is determined, EPA identifies toxicological points of departure (POD) and levels of concern to use in evaluating the risk posed by human exposure to the pesticide. For hazards that have a threshold below which there is no appreciable risk, the toxicological POD is used as the basis for derivation of reference values for risk assessment.

PODs are developed based on a careful analysis of the doses in each toxicological study to determine the dose at which no adverse effects are observed (the NOAEL) and the lowest dose at which adverse effects of concern are identified (the LOAEL). Uncertainty/safety factors are used in conjunction with the POD to calculate a safe exposure level—generally referred to as a population-adjusted dose (PAD) or a reference dose (RfD)—and a safe margin of exposure (MOE). For non-threshold risks, the Agency assumes that any amount of exposure will lead to some

degree of risk. Thus, the Agency estimates risk in terms of the probability of an occurrence of the adverse effect expected in a lifetime. For more information on the general principles EPA uses in risk characterization and a complete description of the risk assessment process, see <http://www2.epa.gov/pesticide-science-and-assessing-pesticide-risks/assessing-human-health-risk-pesticides>.

A summary of the toxicological endpoints for pydiflumetofen used for human risk assessment is shown in Table 1 of this unit. Because the Agency

concludes that the pydiflumetofen toxicity database accounts for 2,4,6-trichlorophenol toxicity that would result from exposure to pydiflumetofen, that exposure to the metabolite 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-trichlorophenol.

TABLE 1—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR PYDIFLUMETOFE 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 (All populations including infants and children).	NOAEL = 100 mg/kg/day UF <sub>A</sub> = 10x UF <sub>H</sub> = 10x FQPA SF = 1x	Acute RfD = 1 mg/kg/day. aPAD = 1 mg/kg/day	Acute neurotoxicity study—rat. LOAEL = 300 mg/kg/day based on a decrease in locomotor activity (the number of rears and total distance traveled) in females.
Chronic dietary (All populations)	NOAEL = 9.2 mg/kg/day UF <sub>A</sub> = 10x UF <sub>H</sub> = 10x FQPA SF = 1x	Chronic RfD = 0.092 mg/kg/day. cPAD = 0.092 mg/kg/day	Carcinogenicity study—mouse. MRID 49557940. LOAEL = 45.4 mg/kg/day based on liver weight increase concordant with higher incidence of liver masses, eosinophilic foci of cellular alteration, and centrilobular hypertrophy.
Oral short-term (1 to 30 days) ..	NOAEL = 36.1 mg/kg/day UF <sub>A</sub> = 10x UF <sub>H</sub> = 10x FQPA SF = 1x	LOC for MOE = 100	2-generation reproduction study—rat. LOAEL = 116.2 mg/kg/day based on reduced pup weight in the F1 generation.
Dermal short-term (1 to 30 days).	NOAEL = 36.1 mg/kg/day (dermal absorption rate = 17%). UF <sub>A</sub> = 10x UF <sub>H</sub> = 10x FQPA SF = 1x	LOC for MOE = 100	2-generation reproduction study—rat. LOAEL = 116.2 mg/kg/day based on reduced pup weight in the F1 generation.
Cancer (Oral, dermal, inhalation).	Classification: “Not Likely to be Carcinogenic to Humans” at doses that do not induce a proliferative response in the liver. EPA has determined that a nonlinear approach is appropriate and that the cRfD will be protective of cancer effects.		

FQPA SF = Food Quality Protection Act Safety Factor. LOAEL = lowest-observed-adverse-effect-level. LOC = level of concern. mg/kg/day = milligram/kilogram/day. MOE = margin of exposure. NOAEL = no-observed-adverse-effect-level. PAD = population adjusted dose (a = acute, c = chronic). RfD = reference dose. UF = uncertainty factor. UF<sub>A</sub> = extrapolation from animal to human (interspecies). UF<sub>H</sub> = potential variation in sensitivity among members of the human population (intraspecies).

### 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. 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 using a total toxic residues approach. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of pydiflumetofen and degradate SYN545547. 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 Water Calculator (PWC) modeling, the estimated drinking water concentrations (EDWCs) of pydiflumetofen for acute exposures are estimated to be 17 parts per billion (ppb) for surface water and 95 ppb for ground water and for chronic exposures are estimated to be 3.62 ppb for surface water and 93.4 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 95 ppb was used to assess the contribution to drinking water.

For the chronic dietary risk assessment, the water concentration of value 93.4 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 proposed for the following uses that could result in residential exposures: Golf course turf and ornamentals in greenhouses, nurseries, fields, and outdoor residential landscapes. EPA assessed residential exposure using the following assumptions: Residential handler exposures are not expected since the proposed residential uses require that handlers wear specific clothing (e.g., long-sleeved shirt and long pants; shoes

plus socks) and/or personal protective equipment, and the turf and ornamental use labels will indicate that the product is intended for use by professional applicators, while the crop use labels will include the statement “Not for residential use.” As a result, a residential handler assessment was not conducted. 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 although pydiflumetofen metabolizes into 2,4,6-trichlorophenol, this metabolite does not appear to be produced by other registered pesticides. 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 FQPA Safety Factor (SF). In applying this provision, EPA either retains the default value of 10X, or uses a different additional safety factor when reliable data available to EPA support the choice of a different factor.

*2. Prenatal and postnatal sensitivity.* 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 2000 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 post-application exposure of children. 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 8.5% of the aPAD at the 95th percentile of exposure

for children 3–5 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 21% of the cPAD for children 3–5 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, 590 for children 6 to less than 11 years old, and 2,500 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).

Intermediate-term adverse effects were 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). QuEChERS has been proposed as the enforcement analytical method for plant commodities.

The livestock analytical method was derived from the QuEChERS (EN 15662:2009–02) multi-residue method. It is based on extraction and clean-up procedures, and subsequent LC–MS/MS determination.

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](mailto: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

The applicant requested a few tolerances on commodities that EPA does not consider to be food or feed items (“corn, sweet, cannery waste,” “grape, wet pomace,” “potato, dried pulp,” “tomato, dried pomace,” and “tomato, wet pomace”); therefore, tolerances are unnecessary. With respect to rye grain, the applicant proposed a tolerance based on barley residue data, but the Agency determined that translating the rye grain tolerance from wheat residue data was more appropriate. For the petitioner-proposed tolerances for soybean forage and hay, there is a feeding restriction on the label, which makes these tolerances unnecessary; therefore, the Agency is not establishing tolerances for those two commodities. The pop corn stover tolerance was revised due to only pop corn stover residues used. For the oat grain and peanut hay tolerances, the petitioner included residues from both formulations, whereas EPA assessed the emulsifiable concentrate (EC) and soluble concentrate (SC) separately to determine if there was a formulation difference and set the tolerance at the higher level to cover residues from either formulation.

Although the petitioner requested tolerances for livestock commodities based on the aggregate residues of the parent and metabolite, EPA is establishing tolerances for livestock commodities based only on measuring residues of the parent compound, in order to harmonize tolerances with Canada. EPA is establishing a meat byproduct tolerance, which covers residues found in liver and kidney, instead of separate liver and kidney tolerances since separate tolerances are not needed. A tolerance for Grain, Cereal, Forage, Fodder and Straw, Group 16 was not set since residue data among the representative commodities varied by more than a factor of five; instead, EPA is establishing individual tolerances. The Agency used the Langmuir model to determine the tolerances for livestock tissue and milk. The milk tolerance was raised to harmonize with Canada’s MRLs and in effect would cover the expected cream residues. With respect to wheat germ, milled byproducts, and field corn flour, the median concentration factor was used by the Agency which resulted in different tolerances than those proposed by the petitioner. In addition, EPA has modified some of the commodity definitions, and numerical expression of the tolerance values in order to conform

to current Agency policy on significant figures.

### 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]-1H-pyrazole-4-carboxamide) in or on the commodity: Barley, grain at 4.0 ppm; Barley, hay at 30 ppm; Barley, straw at 30 ppm; Cattle, fat at 0.03 ppm; Cattle, meat at 0.01 ppm; Cattle, meat byproducts at 0.03 ppm; Corn, field, flour at 0.02 ppm; Corn, field, forage at 6.0 ppm; Corn, field, grain at 0.015 ppm; Corn, field, milled byproducts at 0.06 ppm; Corn, field, stover at 15 ppm; Corn, pop, forage at 6.0 ppm; Corn, pop, grain at 0.015 ppm; Corn, pop, stover at 10 ppm; Corn, sweet, forage at 5.0 ppm; Corn, sweet, kernel plus cob with husks removed at 0.01 ppm; Corn, sweet, stover at 9.0 ppm; Fruit, small vine climbing, except fuzzy kiwifruit, subgroup 13–07F at 1.5 ppm; Goat, fat at 0.03 ppm; Goat, meat at 0.01 ppm; Goat, meat byproducts at 0.03 ppm; Grain, aspirated fractions at 100 ppm; Grape, raisin at 2.0 ppm; Horse, fat at 0.03 ppm; Horse, meat at 0.01 ppm; Horse, meat byproducts at 0.03 ppm; Leaf petiole vegetable subgroup 22B at 15 ppm; Leafy greens subgroup 4–16A at 40 ppm; Milk at 0.03 ppm; Oat, forage at 10 ppm; Oat, grain at 3.0 ppm; Oat, hay at 40 ppm; Oat, straw at 20 ppm; Pea, field, forage at 6.0 ppm; Pea, field, hay at 40 ppm; Peanut at 0.02 ppm; Peanut, hay at 30 ppm; Peanut, refined oil at 0.05 ppm; Peas and bean, dried shelled, except soybean, subgroup 6C at 0.40 ppm; Potato, processed potato waste at 0.03 ppm; Potato, wet peel at 0.03 ppm; Quinoa, grain at 4.0 ppm; Rapeseed subgroup 20A at 0.90 ppm; Rye, grain at 0.30 ppm; Rye, hay at 50 ppm; Rye, straw at 30 ppm; Sheep, fat at 0.03 ppm; Sheep, meat at 0.01 ppm; Sheep, meat byproducts at 0.03 ppm; Soybean, seed at 0.40 ppm; Tomato, dried at 3.0 ppm; Vegetable, cucurbit, group 9 at 0.50 ppm; Vegetable, fruiting, group 8–10 at 0.60 ppm; Vegetable, tuberous and corm subgroup 1C at 0.015 ppm; Wheat, forage at 15 ppm; Wheat, germ at 0.40 ppm; Wheat, grain at 0.30 ppm; Wheat, hay at 50 ppm; Wheat, milled byproducts at 2.0 ppm; and Wheat, straw at 30 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: May 17, 2018,

**Richard P. Keigwin, Jr.,**

Director, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

### PART 180—[AMENDED]

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

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

■ 2. Add § 180.699 to subpart C to read as follows:

#### § 180.699 Pydiflumetofen; Tolerances for residues.

(a) *General.* Tolerances are established for residues of pydiflumetofen, including its metabolites and degradates, in or on the commodities in the table below. 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]-1H-pyrazole-4-carboxamide) in or on the commodity:

Commodity	Parts per million
Barley, grain .....	4.0
Barley, hay .....	30
Barley, straw .....	30
Cattle, fat .....	0.03
Cattle, meat .....	0.01
Cattle, meat byproducts .....	0.03
Corn, field, flour .....	0.02
Corn, field, forage .....	6.0
Corn, field, grain .....	0.015
Corn, field, milled byproducts .....	0.06
Corn, field, stover .....	15
Corn, pop, forage .....	6.0
Corn, pop, grain .....	0.015
Corn, pop, stover .....	10
Corn, sweet, forage .....	5.0
Corn, sweet, kernel plus cob with husks removed .....	0.01
Corn, sweet, stover .....	9.0
Fruit, small vine climbing, except fuzzy kiwifruit, subgroup 13-07F .....	1.5
Goat, fat .....	0.03
Goat, meat .....	0.01
Goat, meat byproducts .....	0.03
Grain, aspirated fractions .....	100
Grape, raisin .....	2.0
Horse, fat .....	0.03
Horse, meat .....	0.01
Horse, meat byproducts .....	0.03

Commodity	Parts per million
Leaf petiole vegetable subgroup 22B .....	15
Leafy greens subgroup 4-16A ..	40
Milk .....	0.03
Oat, forage .....	10
Oat, grain .....	3.0
Oat, hay .....	40
Oat, straw .....	20
Pea, field, forage .....	6.0
Pea, field, hay .....	40
Peanut .....	0.02
Peanut, hay .....	30
Peanut, refined oil .....	0.05
Peas and bean, dried shelled, except soybean, subgroup 6C ..	0.40
Potato, processed potato waste ..	0.03
Potato, wet peel .....	0.03
Quinoa, grain .....	4.0
Rapeseed subgroup 20A .....	0.90
Rye, grain .....	0.30
Rye, hay .....	50
Rye, straw .....	30
Sheep, fat .....	0.03
Sheep, meat .....	0.01
Sheep, meat byproducts .....	0.03
Soybean, seed .....	0.40
Tomato, dried .....	3.0
Vegetable, cucurbit, group 9 .....	0.50
Vegetable, fruiting, group 8-10 ..	0.60
Vegetable, tuberous and corm subgroup 1C .....	0.015
Wheat, forage .....	15
Wheat, germ .....	0.40
Wheat, grain .....	0.30
Wheat, hay .....	50
Wheat, milled byproducts .....	2.0
Wheat, straw .....	30

(b) *Section 18 emergency exemptions.*

[Reserved]

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

(d) *Indirect or inadvertent residues.*

[Reserved]

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