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EPA Registration Division contact: Mindy Ondish, (202) 566-2857

Nufarm Limited

PP2F8995

EPA has received a pesticide petition (2F8995) from Nufarm Americas Inc., Agent for Nufarm Limited, 4020 Aerial Center Parkway, Morrisville, NC 27560, requesting, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180.

1. by establishing a tolerance for residues of

(R)-2-(2,4-dichlorophenoxy)propionic acid (dichlorprop-p; 2,4-DP-p), both free and conjugated, determined as the acid in or on the food commodities Amaranth, grain, forage at 50 parts per million (ppm); Amaranth, grain, hay at 80 ppm; Amaranth, grain, straw at 40 ppm; Amaranth, purple, forage at 50 ppm; Amaranth, purple, hay at 80 ppm; Amaranth, purple, straw at 40 ppm; Baby corn, forage at 0.06 ppm; Baby corn, stover at 0.15 ppm; Barley, hay at 40 ppm; Barley, straw at 15 ppm; Barley, subgroup 15-22B at 0.3 ppm; Buckwheat, hay at 40 ppm; Buckwheat, straw at 15 ppm; Buckwheat, tartary, hay at 40 ppm; Buckwheat, tartary, straw at 15 ppm; Canarygrass, annual, hay at 40 ppm; Canarygrass, annual, straw at 15 ppm; Cañihua, forage at 50 ppm; Cañihua, hay at 80 ppm; Cañihua, straw at 40 ppm; Chia, forage at 50 ppm; Chia, hay at 80 ppm; Chia, straw at 40 ppm; Corn, field, forage at 0.01 ppm; Corn, field, stover at 0.01 ppm; Corn, field, subgroup 15-22C at 0.01 ppm; Corn, pop, stover at 0.01 ppm; Corn, sweet, forage at 0.06 ppm; Corn, sweet, stover at 0.15 ppm; Corn, sweet, subgroup 15-22D at 0.01 ppm; Cram-cram, forage at 50 ppm; Cram-cram, hay at 80 ppm; Cram-cram, straw at 40 ppm; Fonio, black, forage at 0.02 ppm; Fonio, black, stover at 0.09 ppm; Fonio, white, forage at 0.02 ppm; Fonio, white, stover at 0.09 ppm; Huauzontle, grain, forage at 50 ppm; Huauzontle, grain, hay at 80 ppm; Huauzontle, grain, straw at 40 ppm; Inca wheat, forage at 50 ppm; Inca wheat, hay at 80 ppm; Inca wheat, straw at 40 ppm; Job's tears, forage at 0.02 ppm; Job's tears, stover at 0.09 ppm; Millet, barnyard, forage at 0.02 ppm; Millet, barnyard, hay at 0.02 ppm; Millet, barnyard, straw at 0.09 ppm; Millet, finger, forage at 0.02 ppm; Millet, finger, hay at 0.02 ppm; Millet, finger, straw at 0.09 ppm; Millet, foxtail, forage at 0.02 ppm; Millet, foxtail, hay at 0.02 ppm; Millet, foxtail, straw at 0.09 ppm; Millet, little, forage at 0.02 ppm; Millet, little, hay at 0.02 ppm; Millet, little, straw at 0.09 ppm; Millet, pearl, forage at 0.02 ppm; Millet, pearl, hay at 0.02 ppm; Millet, pearl, straw at 0.09 ppm; Millet, proso, forage at 0.02 ppm; Millet, proso, hay at 0.02 ppm; Millet, proso, straw at 0.09 ppm; Oat, forage at 40 ppm; Oat, hay at 40 ppm; Oat, straw at 15 ppm; Oat, Abyssinian, forage at 40 ppm; Oat, Abyssinian, hay at 40

ppm; Oat, Abyssinian, straw at 15 ppm; Oat, common, forage at 40 ppm; Oat, common, hay at 40 ppm; Oat, common, straw at 15 ppm; Oat, naked, forage at 40 ppm; Oat, naked, hay at 40 ppm; Oat, naked, straw at 15 ppm; Oat, sand, forage at 40 ppm; Oat, sand, hay at 40 ppm; Oat, sand, straw at 15 ppm; Prince's-feather, forage at 50 ppm; Prince's-feather, hay at 80 ppm; Prince's-feather, straw at 40 ppm; Psyllium, forage at 50 ppm; Psyllium, hay at 80 ppm; Psyllium, straw at 40 ppm; Psyllium, blond, forage at 50 ppm; Psyllium, blond, hay at 80 ppm; Psyllium, blond, straw at 40 ppm; Quinoa, forage at 50 ppm; Quinoa, hay at 80 ppm; Quinoa, straw at 40 ppm; Rye, forage at 50 ppm; Rye, hay at 80 ppm; Rye, straw at 40 ppm; Sorghum, grain, and millet, subgroup 15-22E at 0.01 ppm; Sorghum, grain, forage at 0.02 ppm; Sorghum, grain, stover at 0.09 ppm; Soybean, forage at 0.05 ppm; Soybean, hay at 0.03 ppm; Soybean, seed at 0.01 ppm; Teff, forage at 0.02 ppm; Teff, hay at 0.02 ppm; Teff, straw at 0.09 ppm; Teosinte, forage at 0.01 ppm; Teosinte, stover at 0.01 ppm; Triticale, forage at 50 ppm; Triticale, hay at 80 ppm; Triticale, straw at 40 ppm; Wheat, forage at 50 ppm; Wheat, germ at 0.2 ppm; Wheat, hay at 80 ppm; Wheat, straw at 40 ppm; Wheat, subgroup 15-22A at 0.1 ppm; Wheat, club, forage at 50 ppm; Wheat, club, hay at 80 ppm; Wheat, club, straw at 40 ppm; Wheat, common, forage at 50 ppm; Wheat, common, hay at 80 ppm; Wheat, common, straw at 40 ppm; Wheat, durum, forage at 50 ppm; Wheat, durum, hay at 80 ppm; Wheat, durum, straw at 40 ppm; Wheat, einkorn, forage at 50 ppm; Wheat, einkorn, hay at 80 ppm; Wheat, einkorn, straw at 40 ppm; Wheat, emmer, forage at 50 ppm; Wheat, emmer, hay at 80 ppm; Wheat, emmer, straw at 40 ppm; Wheat, macha, forage at 50 ppm; Wheat, macha, hay at 80 ppm; Wheat, macha, straw at 40 ppm; Wheat, oriental, forage at 50 ppm; Wheat, oriental, hay at 80 ppm; Wheat, oriental, straw at 40 ppm; Wheat, Persian, forage at 50 ppm; Wheat, Persian, hay at 80 ppm; Wheat, Persian, straw at 40 ppm; Wheat, Polish, forage at 50 ppm; Wheat, Polish, hay at 80 ppm; Wheat, Polish, straw at 40 ppm; Wheat, poulard, forage at 50 ppm; Wheat, poulard, hay at 80 ppm; Wheat, poulard, straw at 40 ppm; Wheat, shot, forage at 50 ppm; Wheat, shot, hay at 80 ppm; Wheat, shot, straw at 40 ppm; Wheat, spelt, forage at 50 ppm; Wheat, spelt, hay at 80 ppm; Wheat, spelt, straw at 40 ppm; Wheat, timopheevi, forage at 50 ppm; Wheat, timopheevi, hay at 80 ppm; Wheat, timopheevi, straw at 40 ppm; Wheat, vavilovi, forage at 50 ppm; Wheat, vavilovi, hay at 80 ppm; Wheat, vavilovi, straw at 40 ppm; Wheat, wild einkorn, forage at 50 ppm; Wheat, wild einkorn, hay at 80 ppm; Wheat, wild einkorn, straw at 40 ppm; Wheat, wild emmer, forage at 50 ppm; Wheat, wild emmer, hay at 80 ppm; Wheat, wild emmer, straw at 40 ppm; Wheatgrass, intermediate, forage at 50 ppm; Wheatgrass, intermediate, hay at 80 ppm; Wheatgrass, intermediate, straw at 40 ppm; Cattle, fat at 0.15 ppm; Cattle, kidney at 1.0 ppm; Cattle, liver at 0.05 ppm; Cattle, meat at 0.01 ppm; Cattle, meat byproducts at 1.0 ppm; Egg at 0.01 ppm; Goat, fat at 0.15 ppm; Goat, kidney at 1.0 ppm; Goat, liver at 0.05 ppm; Goat, meat at 0.01 ppm; Goat, meat byproducts at 1.0 ppm; Hog, fat at 0.15 ppm; Hog, kidney at 1.0 ppm; Hog, liver at 0.05 ppm; Hog, meat at 0.01 ppm; Hog, meat byproducts at 1.0 ppm; Horse, fat at 0.15 ppm; Horse, kidney at 1.0 ppm; Horse, liver at 0.05 ppm; Horse, meat at 0.01 ppm; Horse, meat byproducts at 1.0 ppm; Milk at 0.01 ppm; Poultry, fat at 0.01 ppm; Poultry, liver at 0.01 ppm; Poultry, meat at 0.01 ppm; Poultry, meat byproducts at 0.01 ppm; Sheep, fat at 0.15 ppm; Sheep, kidney at 1.0 ppm; Sheep, liver at 0.05 ppm; Sheep, meat at 0.01 ppm; Sheep, meat byproducts at 1.0 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408 (d)(2) of

FDDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The metabolism of dichlorprop-p (2,4-DP-p) in wheat and oranges was investigated. Results of these studies indicated that the majority of the residues in both crops at harvest consisted of various 2,4-DP-p conjugates, plus smaller amounts of parent 2,4-DP-p, along with low concentrations of minor, mostly unidentified metabolites.

2. *Analytical method.* Residue analysis is performed using a hydrolysis reaction, QuEChERS extraction and quantitation of residues by high performance liquid chromatography employing tandem mass spectrometric detection (LC-MS/MS). The method determines the total 2,4-DP-p acid present in food commodities whether in the form of the acid, 2,4-DP-p 2-ethylhexyl ester, or conjugate. Successful validation and independent laboratory validation of analytical methods for determination of total 2,4-DP-p acid in crop and animal commodities were carried out to a limit of quantitation (LOQ) of 0.01 mg/kg.

3. *Magnitude of residues.* Studies were conducted to determine the magnitude and decline of the residue in or on barley, field corn, grain sorghum, soybean, sweet corn, and wheat. Field trials were carried out at the maximum label rates and number of applications. Each field trial included separate plots treated with 2,4-DP-p 2-EHE and 2,4-DP-p DMA salt. Trials for barley, wheat, field corn and soybean were also conducted at an exaggerated application rate (3x or 5x) to determine the magnitude of the residue in/on processed commodities. Processing studies were conducted for barley and wheat commodities. Processing studies were not conducted for field corn or soybean commodities because residues trials conducted at exaggerated rates resulted in no detectable residues in grain or seed samples from these trials. The residue data support the proposed tolerances for these commodities. Nufarm also proposes to rely on these data to establish tolerances on related crops. The data for barley support tolerances for canarygrass (annual), oat, and oat (common); data for field corn support popcorn and teosinte; data for grain sorghum support millet (barnyard, finger, foxtail, little, pearl, and proso) and teff; and data for wheat support triticale, rye, wheat (common), wheat (durum) and wheatgrass (intermediate).

B. Toxicological Profile

1. *Acute toxicity.* The available toxicity data indicate low acute oral, dermal, inhalation, and primary dermal irritation toxicity for 2,4-DP-p acid, 2,4-DP-p 2-EH ester, and 2,4-DP-p DMA salt (toxicity categories III and IV). 2,4-DP-p acid is a severe eye irritant (toxicity category I). 2,4-DP-p acid is not a dermal sensitizer; 2,4-DP-p 2-EH ester and 2,4-DP-p dimethylamine salt are dermal sensitizer.

2. *Genotoxicity.* 2,4-DP-p did not induce gene mutation in bacteria or a reproducible positive clastogenic response in an *in vitro* mammalian cell cytogenetic assay. Although 2,4-DP-p is considered clastogenic in an *in vitro* Chinese hamster ovary (CHO) chromosomal aberration assay, the response was confined to S-9 activated cytotoxic concentration and not confirmed in two additional *in vitro* chromosomal aberration assays conducted with human lymphocytes.

In vivo, 2,4-DP was neither clastogenic nor aneugenic in mouse bone marrow, did not induce structural chromosomal aberration in Chinese bone marrow assay, or cause unscheduled DNA synthesis in rat livers. However, there was evidence that test material was reactive with DNA as indicated by increased incidences of sister chromatid exchange (SCEs) in Chinese hamster bone marrow cells. In the absence of DNA damage manifested as gene mutation or chromosomal aberration, the positive increase in SCEs is not sufficient to conclude a mutagenic response and suggest that the damage to DNA is repaired. Available data support the conclusion that 2,4-DP-p is not mutagenic *in vitro* or *in vivo*.

3. *Reproductive and developmental toxicity.* No evidence of increased quantitative or qualitative susceptibility or sensitivity was observed in prenatal developmental studies in two species or the 2-generation reproductive study in rats. Forms of dichlorprop tested include 2,4-DP-EH ester, 2,4-DP-DMA salt, and 2,4-DP-p acid**.**

In the rat prenatal developmental study, 2,4-DP-p acid caused maternal compound-related clinical signs and mortality. Developmental effects were observed only in the presence of maternal toxicity and included decreased fetal body weight (↓12-13%), increase in the number of sternebrae not ossified, sternebrae incompletely ossified or reduced in size, and an increase in the number of rudimentary cervical ribs (NOAEL=80mg/kd/day). In the rabbit developmental study, maternal clinical signs resulting in death were similar to those observed in the rat prenatal developmental study. Developmental effects occurred at the same dose level as maternal effects, and included an increased incidence of the 13th accessary ribs (NOAEL=50 mg/kg/day). Early and late resorptions also occurred in rabbits, and due to the unknown etiology, are considered both a maternal and developmental effect.

In the 2-generation reproduction study, 2,4-DP-p acid caused mortality and clinical signs, decreased body weights and food consumption, increased water consumption, and effects on the liver and kidneys. The reproductive and offspring effects occurred at the same dose as maternal effects, and included decreased mating, decreased fertility, decreased gestation indices, decreased viability indices and retarded growth and development.

4. *Subchronic toxicity.* Following subchronic oral exposure to 2,4-DP-p, the liver, kidney, and male reproductive tract were target organs. All subchronic effects in the rat were observed at or near the same dose level. Two subchronic studies in the rat and one in the mouse are available. Effects noted in the first rat study at 125 mg/kg/day were adverse liver histopathology, changes in blood chemistry parameters (total bilirubin, triglycerides, cholesterol), increased liver enzymes (ALT, AST [males only], ALP), and increased absolute and relative liver weight (NOAEL=25 mg/kg/day). At the same dose

level in the second subchronic rat study, adverse male reproductive tract histopathology was noted (reduced epididymal size, spermatocele of the epididymides, testes reduced in size, calcification of the testes, degeneration of the testes and, Leydig cell hyperplasia of the testes) (NOAEL=25 mg/kg/day). Following subchronic exposure in the mouse, adverse effects included decreased body weight, alterations in clinical chemistry (ALP and CIPCO), changes in liver and kidney weights, and histopathological alterations in the liver (dark-brown in color and eosinophilic hepatocytes) and kidney (marked cytoplasmic eosinophilia in renal tubule cells) (NOAEL=224 mg/kg/day).

No evidence of adverse neuropathology was observed in a subchronic neurotoxicity study conducted with 2,4-DP-p. Effects included decreased fore- and hindlimb grip strength, in addition to decreased body weight, decreased food consumption, alterations in clinical chemistry, hematology, and urinalysis parameters, and changes in liver weight and histopathology. The liver histopathology was similar to that seen in the rat subchronic study, but also included the absence of fat storage and slight central hypertrophy.

In 28-day dermal toxicity studies conducted with 2,4-DP-p acid, 2,4-DP-p EH ester, and 2,4-DP-p DMA salt, no systemic toxicity was noted up to the limit dose (1000 mg/kg/day). 2,4-DP-p EH ester and 2,4-DP-p DMA salt caused sporadic skin irritation at the application site, which was resolved after the cessation of treatment. 2,4-DP-p acid induced slight erythema and minimal diffuse skin thickening accompanied by minimal diffuse inflammatory cells in the superficial dermis.

A 28-day inhalation toxicity study in rats conducted with 2,4-DP-p EH ester is available. Although portal-of-entry effects, including minimal squamous metaplasia of the anterior laryngeal respiratory epithelium (Level 1) and mononuclear inflammatory cell infiltration of the larynx were observed at 0.20 mg/L (the highest dose tested), these effects were not considered adverse because there was no dose response observed for mononuclear inflammatory cell infiltration (there was a higher incidence observed in the control group compared to the dosed groups), and all effects were resolved by the end of the 4-week recovery period.

5. *Chronic toxicity.* 2,4-DP-p is classified as "*Not Likely to Be a Human Carcinogen*". No treatment related increases in tumor incidences were observed in rodent carcinogenicity studies.

A 2,4-DP-p acid combined chronic toxicity/carcinogenicity study in rats is available. Chronic toxicity consisted of changes in absolute body weight, histopathological findings in the liver (darkened liver, diffuse hepatocellular swelling, increased brown pigment deposition in hepatic cells) and kidney (darkened kidneys, kidney mineralization, increased brown pigment deposition in proximal tubular cells), changes in clinical chemistry parameters, and decreased specific gravity of the urine (NOAEL=37 mg/kg/day). In the mouse carcinogenicity study with 2,4-DP-p acid, the LOAEL of 59 mg/kg/day was based on decreased absolute body weight, food consumption, and food efficiency in males, histopathology of the kidney in males (chronic nephropathy, calcification, tubuli pigmentation) and females (chronic nephropathy and calcification), and increased absolute kidney weight in females (NOAEL=6 mg/kg/day).

6. *Animal metabolism.* Oral metabolism studies in rats showed that 2,4-DP-p acid was absorbed rapidly and the majority of the administered dose was excreted in urine within 5 days after dosing. No significant tissue accumulation was observed. The major component in the urine and feces was the unchanged 2,4-DP-p acid parent compound.

Oral metabolism studies on 2,4-DP-p acid, 2,4-DP-p EH ester and 2,4-DP-p DMA salt showed similar pharmacokinetic parameters. These studies showed that both 2,4-DP-p EH ester and 2,4-DP-p DMA salt were quantitatively converted *in vivo* to the free acid 2,4-DP-p and absorbed, distributed and metabolized. The major component in the urine was 2,4-DP-p acid, and neither 2,4-DP-p EH ester nor 2,4-DP-p DMA salt was detected in urine or feces.

In a goat metabolism study, 2,4-DP-p was found to be well absorbed and rapidly excreted, predominantly in the urine. Unchanged parent 2,4-DP-p acid was the major component recovered indicating that 2,4-DP-p acid was not extensively metabolized. There was no evidence for the accumulation of radioactivity in milk or edible tissues.

In a laying hen metabolism study, the majority of the administered dose was recovered in the excreta. Unchanged 2,4-DP-p acid was the major component in eggs and tissues, with no significant metabolism observed.

7. *Metabolite toxicology*. The only significant residue of concern in crops, animal tissues, milk, or eggs is parent 2,4-DP-p acid. EPA has previously indicated that 2,4-DP-p residues of concern for drinking water are parent 2,4-DP-p, 2,4-dichlorophenol (2,4-DCP) and 2,4-dichloroanisole (2,4-DCA). EPA-HED considers each of these two degradates to be no more or less toxic than parent 2,4-DP-p.

8. *Endocrine disruption*. The potential of 2,4-DP-p to induce estrogenic or other endocrine effects has not been fully investigated.

C. Aggregate Exposure

1. *Dietary exposure.* Conservative acute and chronic dietary risk assessments were conducted based on proposed tolerances for crop and animal commodities, assuming 100% crop treated. The DEEM model (DEEM-FCID) was used to conduct these assessments. Toxicity endpoints for the acute and chronic dietary assessments were the same as those selected by EPA for its water-only dietary analyses. The acute assessment was based on the RfD of 0.5 mg/kg/day derived from a NOEL of 50 mg/kg/day in a rabbit developmental study. The chronic assessment was based on the RfD of 0.06 mg/kg/day derived from a NOEL of 6 mg/kg/day in a carcinogenicity study conducted with mice.

i. *Drinking water*. Unrefined acute and chronic dietary risk assessments were performed by EPA for drinking water only during Registration Review. For acute exposure, the most highly exposed population subgroup is all infants (<1 year old) occupying <1% of the acute population adjusted dose (aPAD). For chronic exposure, the

most highly exposed population subgroup is non-nursing infants occupying 1.5% of the chronic population adjusted dose (cPAD). These results are below HED's level of concern.

ii. *Food.* A conservative, unrefined dietary risk assessment was conducted for 2,4-DP-p based on proposed tolerance levels and assuming 100% crop treated. Acute exposure (99.9^{th} percentile) for food only utilizes <3.5% of the aPAD for the US population and <7.3% for nursing infants, the most highly exposed subpopulation. Chronic exposure utilizes <4% of the cPAD for the US population and <17% of the cPAD for non-nursing infants, the most highly exposed subpopulation.

2. *Non-dietary exposure*. Residential handler and post-application exposures were assessed for the registered uses of 2,4-DP-p during Registration Review. Dermal risks were not quantitatively assessed since there is no dermal hazard identified. There were no residential handler inhalation risk estimates of concern (i.e., margins of exposure (MOEs) ≥ the level of concern (LOC) of 30). Short-term residential incidental risk estimates for children (1 to <2 years old) reflect post-application hand-to-mouth exposures to turf treated with a liquid formulation and result in a margin of exposure (MOE) of 1,600. Short-term residential risk exposure for children is therefore not of concern.

D. Cumulative Effects

EPA has not made a common mechanism of toxicity finding as to 2,4-DP-p and any other substances.

E. Safety Determination

1. *U.S. population*. Short-term residential exposure for adults is not expected to be of concern since no dermal hazard was identified. Only negligible handler inhalation exposure is anticipated for adults and since the selected endpoints for the oral and inhalation routes are based upon different effects and/or target organs, these routes of exposure should not be combined. The acute aggregate risk estimates for 2,4-DP-p therefore include only food and drinking water and are equivalent to the acute dietary risk estimates. Acute and chronic dietary risk assessments were conducted based on EPA's unrefined dietary risks for drinking water and conservative risk estimates for food based on proposed tolerance levels and 100% crop treated. Results indicate that only 3.72% of the aPAD is used for the general population and potential acute aggregate risks are therefore well below HED's level of concern. The chronic aggregate risk estimates are equivalent to the chronic dietary risks since there are no long-term residential uses of 2,4-DP-p. The conservative chronic dietary risk assessment conducted for 2,4-DP-p indicates that 4% of the cPAD is used for the U.S. general population. Potential chronic aggregate risks are therefore well below HED's level of concern.

2. *Infants and children*. The acute aggregate risk estimates for 2,4-DP-p include only food and drinking water and are equivalent to the acute dietary risk estimates. Results of a conservative acute dietary risk assessment show that exposure from all

proposed food uses and drinking water would result in 7.31% of the aPAD for the most sensitive population subgroup, nursing infants. A residential exposure assessment conducted by EPA for children 1 to <2 years old for use in a short-term aggregate assessment resulted in a margin of exposure (MOE) of 1,600 and is not of concern. When considered together with risks estimates for food and drinking water for children 1 to <2 years old, the resulting short-term aggregate risks are well below EPA's level of concern. Based on the registered uses of 2,4-DP-p, neither intermediate- nor long-term combined exposures are expected. The chronic aggregate risk is therefore equivalent to the chronic dietary risk (food and drinking water only). Conservative risk estimates indicate that 17.5% of the cPAD is used for the most exposed subpopulation, non-nursing infants. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to 2,4-DP-p residues.

F. International Tolerances

MRLs for total 2,4-DP-p acid are established in Canada for several food crops: barley, buckwheat, millet (proso and pearl), oats, popcorn, rice, rye, sorghum, sweet corn, teosinte, triticale, wheat, wild rice. MRLs are also established for eggs, milk, and meat, meat byproducts, and fat of cattle, goats, hogs, sheep, and poultry. In the European Union, MRLs are established for barley, rye, and wheat (including durum wheat and spelt). MRLs for citrus fruits are established in South Africa.