paperboard products (21 CFR 182.90). An exemption from tolerance has been established by FDA under 21 CFR 182.99 and by EPA under 40 CFR 180.1001(c) and (e) for its use as a solvent and co-solvent in pesticide formulations and as an adjuvant when added to pesticide dilutions by growers or applicators prior to application. It is also deemed GRAS by the Expert Panel of the Flavor and Extract Manufacturers'Association of America.

Propylene glycol, a hydrolysis product of the propylene glycol esters, is affirmed as GRAS under 21 CFR 184.1666. It is used as an anticaking agent, antioxidant, dough strengthener, emulsifier, flavor agent, formulation aid, humectant, processing aid, solvent and vehicle, stabilizer and thickener, surface-active agent, and tenderizer in foods at levels not to exceed current good manufacturing practice. The approved uses result in maximum levels, as served of 5% in alcoholic beverages, 24% in confections and frostings, 2.55% in frozen dairy products, 97% in seasonings and flavoring, 5% in nuts and nut products, and 2% in all other food categories. Propylene glycol is also exempt from the requirement of tolerance by EPA under 40 CFR 180.1001(c) and (e), and has been deemed GRAS by the Expert Panel of the Flavor and Extract Manufacturers' Association of America.

I. International Tolerances

No international tolerances have been established for the active ingredients in the VWX–42 Technology system. The FAO and the WHO through the JECFA has reviewed mono and diacylglycerol and propylene glycol esters of fatty acids and determined that they may be used safely in foods at levels of 1–3 grams per day for an adult. It as observed that ''alterations in the fatty acid distribution or polyglycerol content of individual members of a group of diverse substances have no toxicological bearing and only affect the physical and emulsifying properties of each ester.'' The Committee concluded safety based upon the biochemical and metabolic evidence that the breakdown products of such additives are normal dietary constituents.

[FR Doc. 01–30371 Filed 12–11–01; 8:45 am]

BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[PF–1058; FRL–6812–7]

Notice of Filing a Pesticide Petition to Establish a Tolerance fora Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket control number PF–1058, must be received on or before January 11, 2002.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION**. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–1058 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Hoyt Jamerson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460; telephone number: (703) 308–9368; e-mail address: jamerson.hoyt@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS codes	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

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

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically*. You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations,'' ''Regulations and Proposed Rules,'' and then look up the entry for this document under the ''**Federal Register**—Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. *In person*. The Agency has established an official record for this action under docket control number PF–1058. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–1058 in the subject line on the first page of your response.

1. *By mail*. Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs

(OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460.

2. *In person or by courier*. Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically*. You may submit your comments electronically by e-mail to: opp-docket@epa.gov, or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 6.1/8.0 or ASCII file format. All comments in electronic form must be identified by docket control number PF–1058. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: November 27, 2001.

Peter Caulkins,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by BASF Corporation, Agricultural Products, 26 Davis Drive, Research Triangle Park, NC 27709 and represents the view of BASF Corporation. EPA is publishing the petition summary verbatim without editing it in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

Interregional Research Project Number 4

PP 0E6185

EPA has received a pesticide petition (0E6185) from the Interregional Research Project Number 4 (IR–4), 681 U.S. Highway #1 South, North Brunswick, NJ 08902–3390 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of diflufenzopyr, 2-(1-(3,5-difluorophenylamino]carbonyl)-hydrazono]ethyl)-3-pyridinecarboxylic acid, and its metabolites convertible to M1 (8-methylpyrido[2,3-d]pyridazin-5(6H)-one) in or on crop group 17 (grass forage, fodder, and hay group) including: Forage at 3.0 parts per million (ppm); hay at 1.5 ppm; and corn, sweet, fresh at 0.05 ppm; corn, sweet, forage at 0.05 ppm; corn, sweet, stover at 0.05 ppm, and corn, pop, stover at 0.05 ppm. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The nature of the residue in plants (field corn) is understood. In field corn, no diflufenzopyr was detected in any of the corn matrices; metabolites comprising approximately 10% total radioactive residue (TRR) include M1 (8-methylpyrido[2,3-d]pyridazin-5(6H)-one), M10 (8-hydroxymethyl-5(6H)-pyrido[2,3-d]pyridazone) and its glucose conjugate, and M9 (8-methylpyrido[2,3-d]pyridazine-2,5(1H,6H)-dione in forage and fodder, and 6–14% TRR lignin was found in fodder. Corn grain contained 3–4 discrete unknowns, all at less than 10% TRR or less than 0.05 ppm each. The residues of concern in plants are diflufenzopyr, 2-(1-[([3,5-difluorophenylamino]carbonyl)-hydrazono]ethyl)-3-pyridinecarboxylic acid, and its metabolites convertible to M1 (8-methylpyrido [2,3-d]pyridazin-5(6H)-one).

2. *Analytical method*. BASF Corporation has provided suitable independently validated analytical methods for detecting and measuring levels of diflufenzopyr and its metabolites in or on food with a limit of detection that allows monitoring of food with residues at or above the levels described in these and the existing tolerances. Adequate enforcement

methodology (gas chromatography) is available to enforce the tolerance expression. The method may be requested from Calvin Furlow, PIRIB, IRSD (7502C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Avenue, NW., Washington, DC 20460. Office location and telephone number: Rm 101FF, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703) 305–5229.

3. *Magnitude of residues*. Residue trials have been conducted with dicamba/diflufenzopyr end use product distinct on pasture and rangeland grasses and the sweet corn crop for expanded use requested in the subject petition. The tolerances listed below are based on the maximum expected residue from geographically representative field trial data. Crop group 17 (grass, forage, fodder, and hay group) including: Forage at 3.0 ppm; hay at 1.5 ppm; and corn, sweet, fresh at 0.05 ppm; corn, sweet, forage at 0.05 ppm; corn, sweet, stover at 0.05 ppm, and corn, pop, stover at 0.05 ppm.

4. *Animal residue*. Data from metabolism studies in goat and poultry have established that the expected dietary burden from crops treated with diflufenzopyr will not result in quantifiable residues above the limits of the standard analytical method.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicology studies place technical-grade diflufenzopyr in Toxicity Category III or IV for all routes of exposure. It is not a dermal sensitizer.

i. *Acute oral toxicity (rat)*. LD_{50} = >5,000 milligrams/kilogram (mg/kg) in males and females.Toxicity Category IV.

ii. *Acute dermal toxicity (rabbit)*. LD_{50} = >5,000 mg/kg in males and females. Toxicity Category IV.

iii. *Acute inhalation toxicity (rat)*. LC_{50} = >3.14 mg/L in males and females. Toxicity Category IV.

iv. *Primary eye irritation (rabbit)*. Diflufenzopyr is minimally irritating. Toxicity Category III.

v. *Primary dermal irritation (rabbit)*. Diflufenzopyr is not a dermal irritant. Toxicity Category IV.

vi. *Dermal sensitization (guinea pig)*. Diflufenzopyr is not a dermal sensitizer.

2. *Genotoxicty*. Diflufenzopyr shows no signs of being genotoxic—i. In a microbial mutagenicity assay, *Salmonella typhimurium* strains TA98, TA100, TA1,535, TA1,537, and TA1,538 were exposed to diflufenzopyr (97.1%) in DMSO at concentrations of 333, 667, 1,000, 3,330, 6,670, and 10,000 microgram/plate in the presence and absence of mammalian metabolic activation. Diflufenzopyr (97.1%) was tested to twice the limit concentration of 5,000 microgram/plate and cytotoxicity was observed at 6,670 and 10,000 microgram/plate in the absence of activation (-S9) but not in its presence (+S9). The positive controls induced the appropriate responses in the corresponding strains. There was no evidence that the test article induced mutant colonies over background.

ii. In a mammalian cell gene mutation assay at the *thymidine kinase* locus, heterozygous L5178Y (TK +/-) mouse lymphoma cells cultured *in vitro* were exposed to diflufenzopyr technical (97.1% active ingredient) in dimethyl sulfoxide at dose levels ranging from 0.05 to 3.0 mg/mL (50 to 3,000 microgram/mL) in the presence and absence of S9 mammalian metabolic activation in the first trial, and 0.05 to 2.0 mg/mL (50 to 2,000 microgram/mL) in the second. Diflufenzopyr was tested up to cytotoxic dose levels and mutation frequencies were determined for dose levels selected on the basis of relative growth. Although initially declared positive by the then study director, application of more recent criteria for mutagenic responses has rendered the test article negative for forward gene mutation at the *thymidine kinase* locus in mouse L5178Y cells in the presence and absence of S9 activation. The positive controls induced the appropriate responses.

iii. In an *in vivo* mouse bone marrow micronucleus assay, groups of 15 male and female ICR mice were dosed by oral gavage with diflufenzopyr (technical, 97.1%) in corn oil at 500, 1,667, and 5,000 mg/kg. Bone marrow cells were harvested at 24, 48, or 72 hours and scored for micronucleated polychromatic erythrocytes (MPCEs). No mortalities or adverse clinical signs were observed at any dose including the limit dose of 5,000 mg/kg, and there were no changes in the PCE/NCE ratios (an indirect measure of cytotoxicity). The positive control induced significant increases in MPCEs, also in the absence of any target cell cytotoxicity. No significant increase in the frequency of MPCEs in bone marrow cells after any treatment time were recorded; therefore, the test article is considered negative in this micronucleus assay.

iv. In an unscheduled DNA synthesis (UDS) assay, primary rat hepatocyte cultures were exposed to diflufenzopyr (97.1% active ingredient) in dimethylsulfoxide (DMSO) at 15 concentrations ranging from 0.0250 to 1,000 microgram/mL in the presence of 10 microCi/mL (42 Ci/mmole) for approximately 19 hours. Mutagenicity, as measured by UDS, was determined for 6 concentrations selected on the basis of cytotoxicity. The concentrations selected were 5.00, 10.0, 25.0, 50.0, 100, and 250 microgram/mL. The highest concentration selected for UDS evaluation, 250 microgram/mL, was moderately toxic (50.8% survival). There was no evidence that unscheduled DNA synthesis, as determined by radioactive tracer procedures (nuclear silver grain counts) was induced. The positive control induced the appropriate response.

3. *Reproductive and developmental toxicity*—i. In a rat developmental toxicity study, technical diflufenzopyr (98.1% active ingredient) in 0.5% aqueous methyl cellulose was administered by gavage to 25 female Crl: CD BR VAF/Plus (Sprague Dawley) rats/dose at dose levels of 0, 100, 300, or 1,000 mg/kg/day from days 6 through 15 of gestation. The maternal no observed adverse effect level (NOAEL) is 300 mg/kg/day and the maternal lowest observed adverse effect level (LOAEL) is 1,000 mg/kg/day based on decreases in food consumption and weight gain. Developmental effects, characterized as significantly lower fetal body weights (bwts) in males (5%) and skeletal variations, exhibited as incompletely ossified and unossified sternal centra and reduced fetal ossification sites for caudal vertebrae, were observed at 1,000 mg/kg/day. The developmental LOAEL is 1,000 mg/kg/day, based on decreased fetal body weights and skeletal variations. The developmental NOAEL is 300 mg/kg/day.

ii. In a rabbit developmental toxicity study, technical diflufenzopyr (98.1% active ingredient) in 0.5% aqueous methyl cellulose was administered by gavage to 20 female New Zealand White Hra: (NZW)SPF rabbits/dose at dose levels of 0, 30, 100, or 300 mg/kg/day from days 6 through 19 of gestation. The maternal LOAEL is 100 mg/kg/day, based on minimal reductions in body weight gain with no reduction in food consumption and clinical signs of toxicity (abnormal feces). The maternal NOAEL is 30 mg/kg/day. Developmental effects, characterized as significant increases ($p \leq 0.01$) in the incidence of supernumerary thoracic rib pair ossification sites (12.74 vs. 12.54 for controls) occurred at the 300 mg/kg/day dose. No treatment-related developmental effects were noted at the low- or mid-doses. The developmental LOAEL is 300 mg/kg/day based on increased skeletal variations (supernumerary rib ossification sites). The developmental NOAEL is 100 mg/kg/day.

iii. In a 2–generation rat reproduction study, technical diflufenzopyr (98.1% active ingredient) was administered continuously in the diet to 26 Wistar rats/sex/dose at dose levels of 0, 500, 2,000 or 8,000 ppm in the diet (0, 27.3–42.2, 113.1–175.9, or 466.2–742.0 mg/kg/day). The systemic LOAEL is 2,000 ppm (113.1–175.9 mg/kg/day) based on reduced body weight gain, increased food consumption, and increased seminal vesicle weights. The systemic NOAEL is 500 ppm (27.3–42.2 mg/kg/day). The reproductive LOAEL is 8,000 ppm (466.2–742.0 mg/kg/day) based on lower live birth and viability indices, total pre-perinatal loss, reduced body weights and body weight gain during lactation, a higher proportion of runts, and a higher percentage of offspring with no milk in the stomach. The reproductive NOAEL is 2,000 ppm (113.1–175.9 mg/kg/day).

iv. In an acute rat neurotoxicity study, diflufenzopyr (96.4% active ingredient) was administered by gavage to Crl:CD BRR rats (10/sex/group) at dose levels of 0, 125, 500 or 2,000 mg/kg. The rats were evaluated for reactions in functional observations and motor activity measurements at 3 hours, 7 days, and 14 days postdosing. Histopathological evaluation on the brain and peripheral nerves was assessed after day 14. Diflufenzopyr had no definite impact on neurotoxic responses, although a few abnormalities were observed in the functional battery on the day of dosing. A decrease in immediate righting responses that was observed in several males in all treatment groups was not concentration-dependent. Nasal staining was observed in more rats in the 2,000 mg/kg treatment groups (6 males, 3 females), but was not considered a definite or significant response to treatment. Lower mean brain weights in all female treatment groups lacked associated macroscopic and microscopic histopathological changes, and were only 4–5% lower than the control brain weight. There were no definite treatment-related differences in body weights or food consumption in any of the treatment groups. There was no evidence of treatment-related neuropathology in the 2,000 mg/kg treatment group. A LOAEL was not established. The NOAEL for acute neurotoxicity is 2,000 mg/kg (the limit dose).

v. In a subchronic rat neurotoxicity study, diflufenzopyr (96.4% active ingredient) was administered in the diet to Crl: CD BR rats (10/sex/group) at dose levels of 0, 25, 75, or 1,000 mg/kg/day for 13 weeks. The rats were evaluated for reactions in functional observations and motor activity testing at 4 hours and during weeks 4, 8, and 13 of treatment. No treatment-related neurotoxicological effects were observed at any treatment level. A LOAEL for neurotoxicological effects was not established; the NOAEL was 1,000 mg/kg/day for both sexes. Treatment-related toxic effects were observed at the 1,000 mg/kg/day treatment level. The toxicological LOAEL for this study is 1,000 mg/kg/day, based on decreased body weight gains for both sexes. The toxicological NOAEL is 75 milligram/kilogram/day (mg/kg/day).

4. *Subchronic toxicity*—i. In a subchronic feeding study in rats, male and female Wistar rats were fed test diets containing technical diflufenzopyr, purity 96%, at dose levels of 0, 1,000, 5,000, 10,000, and 20,000 ppm (equal to 0, 60.8, 352, 725, and 1,513 mg/kg body weight/day (mg/kg bw/day) for males, and 0, 72.8, 431, 890, and 1,750 mg/kg bwt/day for females) for a period of 13 weeks, 10 rats per sex per group. An additional 10 rats per sex were assigned to the 0 and 20,000 ppm groups for a 4–week recovery period following treatment. The NOAEL was set at 5,000 ppm (equal to 352 mg/kg bwt/day for males, and 431 mg/kg bwt/day for females) based on lower mean body weight gain and decreased food efficiency in the 10,000 and 20,000 ppm groups, both sexes. Additional findings were decreased food intake (20,000 ppm, males only); slight increases in cholesterol (20,000 ppm, both sexes, and 10,000 ppm, males only) and ALAT (10,000 and 20,000 ppm, both sexes); and slightly lower chloride (20,000 ppm, both sexes). Histopathological findings were an increased incidence of foamy macrophages in the lungs in the 10,000 and 20,000 ppm groups, both sexes, and testicular atrophy in the 20,000 ppm group. Following the 47–week recovery period, the only treatment-related effects which showed partial or no evidence of recovery were foamy macrophages in the lungs and testicular atrophy.

ii. In a 13–week feeding study, male and female CD–1 mice were fed test diets containing technical diflufenzopyr, purity 97.1%, at dietary concentrations of 0, 350, 1,750, 3,500, and 7,000 ppm (equal to 0, 58, 287, 613 and 1,225 mg/kg bwt/day for males, and 0, 84, 369, 787 and 1,605 mg/kg bwt/day for females) for a period of 13 weeks, 10 mice per sex per group. The NOAEL was determined to be 7,000 ppm (equal to 1,225 mg/kg bw/day for males and 1,605 mg/kg bw/day for females) since there were no treatment-related effects observed in male or female mice at any dose level tested.

iii. In a subchronic toxicity study in dogs, diflufenzopyr (98% active ingredient) was administered to beagle dogs (4/sex/dose) by feeding at dose levels of 0, 1,500, 10,000, or 30,000 ppm (0, 58, 403, or 1,131 mg/kg/day for males; 0, 59, 424, or 1,172 mg/kg/day for females) for 13 weeks. The lowest adverse effect level LOAEL for this study is 10,000 ppm (403 mg/kg/day in males and 424 mg/kg/day in females), based on the occurrence of erythroid hyperplasia in the bone marrow, extramedullary hemopoiesis in the liver, and hemosiderin deposits in Kupffer cells. The NOAEL is 1,500 ppm (58 mg/kg/day in males and 59 mg/kg/day in females).

iv. In the subchronic rabbit dermal toxicity study, technical diflufenzopyr, purity 96.4%, was moistened with distilled water and administered by dermal application to male and female New Zealand white rabbits, 5/sex/dose, at dose levels of 0, 100, 300, and 1,000 mg/kg bwt per application. Duration of application was 6 hours a day, daily for 21 to 24 consecutive days. The NOAEL for systemic toxicity was determined to be 1,000 mg/kg bwt/day, since there were no apparent signs of treatment-related systemic effects observed in male or female rabbits at any dose level tested. A NOAEL for dermal effects could not be determined since local dermal irritation was observed at all dose levels tested (there were no corresponding findings upon histopathological examination).

5. *Chronic toxicity*—i. In a chronic toxicity study in dogs, diflufenzopyr (98.1% active ingredient) was administered to Beagle dogs (4/sex/dose) by feeding at dose levels of 0, 750, 7,500, or 15,000 ppm (0, 26, 299, or 529 mg/kg/day for males; 0, 28, 301, or 538 mg/kg/day for females) for 52 weeks. The LOAEL for this study is 7,500 ppm (299 mg/kg/day for males and 301 mg/kg/day for females), based on erythroid hyperplasia in the bone marrow in bone sections, reticulocytosis, and increased hemosiderin deposits in the liver, kidneys, and spleen. The NOAEL is 750 ppm (26 mg/kg/day for males and 28 mg/kg/day for females).

ii. In a mouse carcinogenicity study, male and female CD–1 mice were fed test diets containing technical diflufenzopyr, purity 98.1%, at dietary concentrations of 0, 700, 3,500 and 7,000 ppm (equal to 0, 100, 517, and 1,037 mg/kg bwt/day for males, and 0, 98, 500, and 1,004 mg/kg bwt/day for females), 60 mice per sex per group, for a period of 78 weeks. The NOAEL for systemic toxicity was determined to be

7,000 ppm (equal to 1,037 mg/kg bwt/day for males and 1,004 mg/kg bwt/day for females). There were no treatment-related effects observed at any dose level tested in male rats. There was a slight, but statistically significantly lower mean overall body weight gain for females in the 7,000 ppm group, due primarily to decreased gain/increased weight loss during the second year of the study. In the absence of any other treatment-related findings, this was not considered to be an adverse, toxicologically significant finding. There was no evidence of carcinogenic potential of diflufenzopyr for male or female mice at any dose level tested.

iii. In a combined chronic toxicity/carcinogenicity study, male and female Wistar rats were fed test diets containing technical diflufenzopyr, purity 97.1% to 99.6%, at dietary concentrations of 0, 500, 1,500, 5,000, and 10,000 ppm (equal to 0, 22, 69, 236, and 518 mg/kg bwt/day for males, and 0, 29, 93, 323, and 697 mg/kg bwt/day for females), 72 rats per sex per group, for a period of 104 weeks. The NOAEL for systemic toxicity was set at 5,000 ppm (equal to 236 mg/kg bwt/day for males and 323 mg/kg bwt/day for females). Treatment-related effects in the 10,000 ppm group were significantly lower body weight and body weight gains throughout the study period and decreased food efficiency. There was no evidence of carcinogenic potential of diflufenzopyr at any dose level tested. The incidences of benign and malignant tumors were comparable between control and treated groups.

6. *Animal metabolism*. In rats, goats, and hens the majority (greater than 90%) of diflufenzopyr was excreted. In the ruminant, major metabolites include M1, M5 (6-((3,5-difluorophenylcarbamoyl-8-methyl-pyrido[2,3-d]-5-pyridazinone) and M19 (8-hydroxymethylpyrido[2,3-d]pyridazine-2,5(1H,6H)-dione. In poultry, diflufenzopyr was not detected, and M1 was the only significant metabolite identified, and in egg white only. Transfer of secondary residues to livestock is not expected.

7. *Metabolite toxicology*. Toxicity of the metabolites of diflufenzopyr to humans is concurrently evaluated during toxicity testing because both plant and animal metabolites are formed during the course of toxicity tests. Both plant and animal major metabolites are considered not of toxicological concern and have been identified in the rat metabolism study.

8. *Endocrine disruption*. No specific tests have been conducted with diflufenzopyr to determine whether this active ingredient may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, chronic, lifespan, and multigenerational bioassays in mammals and acute aquatic organisms and wildlife did not reveal endocrine effects. It is expected that these studies would reveal endocrine disrupting activity of this active ingredient if it existed.

C. Aggregate Exposure

1. *Dietary exposure*. EPA has established the reference dose (RfD) for diflufenzopyr at 0.26 milligrams/kilogram/day (mg/kg/day). This RfD is based on bone marrow compensated hemolytic anemia observed in the 1–year dog feeding study with a NOAEL of 26 mg/kg/day and an uncertainty factor of 100.

Cancer classification and risk assessment. Based on the lack of evidence of carcinogenicity in mice and rats at doses that were judged to be adequate to assess the carcinogenic potential, diflufenzopyr has been characterized as ''not likely'' to be a human carcinogen.

i. *Food—chronic dietary exposure*. A chronic dietary risk assessment was performed for diflufenzopyr and its metabolites characterized as M1. The analysis used the RfD of 0.26 mg/kg bwt/day and assumed that 100% of corn-derived foods contain residues at the tolerance level (0.05 ppm). These assumptions result in a theoretical maximum residue contribution (TMRC) that is less than or equal to 0.1% of the RfD for the overall U.S. population (48 states) and all population subgroups.

ii. *Drinking water*. There are no established maximum contaminant levels or health advisory levels for residues of diflufenzopyr or its metabolites in drinking water. EPA used the screening concentration in ground water (SCI-GROW) model to estimate residues of diflufenzopyr in ground water and the generic expected environmental concentration (GENEEC) model to estimate diflufenzopyr residue levels in surface water. Estimated maximum concentrations of diflufenzopyr in surface and ground water are 3.80 parts per billion (ppb) and 0.006 ppb, respectively. The estimated maximum concentrations in water are less than EPA's level of comparison (29,970 ppb) for diflufenzopyr residues in drinking water as a contribution to acute aggregate exposure. Therefore, taking into account the uses proposed in this action, BASF Corporation concludes with reasonable certainty that residues of diflufenzopyr in drinking water (when considered along with other sources of exposure for which EPA has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

iii. *Acute exposure and risk*. An acute dietary risk assessment was performed for diflufenzopyr and its metabolites. The analysis was conducted using the acute RfD of 1.0 mg/kg/day, based on developmental findings (increased skeletal variations) observed in the rabbit developmental study. For the population subgroup of concern, females 13 years and older, the estimated 95th percentile of exposure is equal to 0.01% of the acute RfD. The analysis is conservative since it assumes that 100% of corn-derived foods contain residues at the tolerance level (0.05 ppm).

iv. *Chronic exposure and risk*. Using TMRC exposure assumptions, EPA has concluded that aggregate exposure to diflufenzopyr from food will utilize less than 0.1% of the RfD for the U.S. population. Despite the potential for exposure to diflufenzopyr in drinking water and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the RfD. BASF Corporation concludes that there is a reasonable certainty that no harm will result from aggregate exposure to diflufenzopyr residues.

2. *Non-dietary exposure*. There are no registered or proposed residential uses for diflufenzopyr.

D. Cumulative Effects

EPA does not have, at this time, available data to determine whether diflufenzopyr has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, diflufenzopyr does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, BASF Corporation has not assumed that diflufenzopyr has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. Using TMRC exposure assumptions EPA has concluded that aggregate exposure to diflufenzopyr from food will utilize less than 0.1% of the RfD for the U.S. population.

2. *Infants and children*. There is a complete toxicity data base for diflufenzopyr and exposure data are

complete or are estimated based on data that reasonably account for potential exposures. Taking into account the completeness of the data base and the toxicity data regarding prenatal and postnatal sensitivity, BASF Corporation concludes, based on reliable data, that use of the standard margin of safety will be safe for infants and children without addition of another ten-fold factor. Using the standard exposure assumptions EPA has concluded that aggregate exposure to diflufenzopyr from food will utilize 0.1% of the RfD for infants and children. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to diflufenzopyr in drinking water, BASF Corporation does not expect the aggregate exposure to exceed 100% of the RfD. Based on these risk assessments, BASF Corporation concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to diflufenzopyr residues.

F. International Tolerances

There are no CODEX or Mexican residue limits established for diflufenzopyr or its metabolites.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–7117–3]

Regional Haze Regulations; Availability of Draft Guidance Documents

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: We, the EPA are announcing today the availability of draft guidance to assist State and tribal air pollution control agencies in the implementation of regulations governing regional haze which were published in the **Federal Register** on July 1, 1999. These draft documents address the establishment of natural visibility conditions and the tracking of progress under the regional haze program.

DATES: Comments should be submitted on or before January 11, 2002.

ADDRESSES: Comments should be submitted to Lara Autry, U.S. Environmental Protection Agency (MD–14), Research Triangle Park, NC 27711; E-mail *autry.lara@epa.gov.* An electronic copy of the draft guidance can be accessed at: *http://www.epa.gov/ttn/amtic/visinfo.html.*

FOR FURTHER INFORMATION CONTACT: Lara Autry at the same address; E-mail *autry.lara@epa.gov;* telephone (919) 541–5544.

SUPPLEMENTARY INFORMATION: In section 169A of the 1977 Amendments to the Clean Air Act, Congress established a national visibility goal as the ''prevention of any future, and the remedying of any existing, impairment of visibility in mandatory Federal Class I areas which impairment results from manmade air pollution.'' 42 U.S.C. 7491. These provisions were further supplemented by section 169B of the Clean Air Act Amendments of 1990. 42 U.S.C. 7492. States are required to develop implementation plans that make ''reasonable progress'' toward this goal.

EPA issued initial visibility regulations in 1980 [1] that addressed visibility impairment in a specific mandatory Federal Class I area that is determined to be ''reasonably attributable'' to a single source or small group of sources. Regulations to address regional haze were deferred until improved techniques could be developed in monitoring, modeling, and in understanding the effects of specific pollutants on visibility impairment. EPA issued regional haze regulations in 1999.[2]

The overall framework of the regional haze rule requires States to develop SIPs that include (1) reasonable progress goals for improving visibility in each mandatory Federal Class I area, and (2) set of emission reduction measures to meet these goals. Specifically, States will set progress goals for each mandatory Federal Class I area to:

- provide for an improvement in visibility for the 20% most impaired (i.e., worst visibility) days over the period of the implementation plan, and
- ensure no degradation in visibility for the 20% least impaired (i.e., best visibility) days over the same period.

Baseline visibility conditions for the 20% worst and 20% best days are to be determined using monitoring data collected during calendar years 2000–2004. Baseline conditions for 2000–2004, progress goals, and tracking changes over time are to be expressed in terms of the deciview index.[3]

Most States (and Tribes as appropriate [4]) participating in regional planning organizations will submit regional haze implementation plans, including estimates of natural conditions and proposed progress goals, in the 2008 time frame. The regional haze SIP deadlines are linked to the dates when $PM_{2.5}$ designations are finalized. For states that choose to participate in a regional planning organization, the initial (committal) SIP is due within one year of the $PM_{2.5}$ designation and the full control strategy SIP is due within three years of the $PM_{2.5}$ designation, but not later than December 31, 2008. For states that choose not to participate in a regional planning organization, regional haze SIPs are due within one year of the $PM_{2.5}$ designation (for geographic areas designated as attainment or unclassifiable) and within three years of the $PM_{2.5}$ designation (for geographic areas designated as nonattainment), which is the same time that control strategies to attain the $PM_{2.5}$ standard are due. In developing any progress goal, the State will need to analyze and consider in its set of options the rate of improvement between 2004 (when 2000–2004 baseline conditions are set) and 2018 that, if maintained in subsequent implementation periods, would result in achieving estimated natural conditions in 2064.

The purpose of the draft documents announced in today's notice, when completed, will be to provide guidance to the States in implementing the regional haze program and to explain how EPA intends to exercise its discretion in implementing Clean Air Act provisions and EPA regulations concerning the estimation of natural visibility under the Regional Haze program. The guidance is designed to implement national policy on these issues. Sections 169A and 169B of the Clean Air Act and implementing regulations at 40 CFR 51.308 and 51.309 contain legally binding requirements. When completed and issued, these draft guidance documents will not substitute for those provisions or regulations, nor will they constitute regulations themselves. Thus, they will not impose binding, enforceable requirements on any party, and may not apply to a particular situation based upon the circumstances. We and State decision

[1] See 45 FR 80084 (December 2, 1980).

[2] See 64 FR 35713 (July 1, 1999). See also 40 CFR 51.300–51.309.

[3] The deciview is a haze index derived from calculated light extinction, such that uniform changes in haziness correspond to uniform incremental changes in visual perception across the entire range of conditions, from pristine to highly impaired. Deciview = 10 $\ln(b_{ext}/10)$.

[4] Under the Tribal Air Rule (63 FR 7254; February 12, 1998; 40 CFR part 49), Tribal governments may elect to implement air programs in much the same way as states, including development of Tribal implementation plans.