Requirements Applicable to the Air Toxics Program (California Code of Regulations Title 17, section 93109) pertains to the perchloroethylene dry cleaning source category in the State of California, and has been approved under the procedures in § 63.93 to be implemented and enforced in place of subpart M—National Perchloroethylene Air Emission Standards for Dry Cleaning Facilities, as it applies to area sources only, as defined in § 63.320(h).

(*1*) * * *

(*i*) California is not delegated the Administrator's authority to implement and enforce California Code of Regulations Title 17, section 93109, in lieu of those provisions of subpart M which apply to major sources, as defined in § 63.320(g). * * *

(*ii*) * * *

(B) * * *

(*1*) * * *

(*i*) San Luis Obispo County Air Pollution Control District is not delegated the Administrator's authority to implement and enforce Rule 432 in lieu of those provisions of subpart M which apply to major sources as defined in § 63.320(g). * * *

(*ii*) * * *

(C) * * *

(D) The material incorporated in Chapter 4 of the *California Regulatory Requirements Applicable to the Air Toxics Program* (Yolo-Solano Air Quality Management District Rule 9.7) pertains to the perchloroethylene dry cleaning source category in the Yolo-Solano Air Quality Management District, and has been approved under the procedures in § 63.93 to be implemented and enforced in place of subpart M—National Perchloroethylene Air Emission Standards for Dry Cleaning Facilities, as it applies to area sources only, as defined in § 63.320(h).

(*1*) Authorities not delegated.

(*i*) Yolo-Solano Air Quality Management District is not delegated the Administrator's authority to implement and enforce Rule 9.7 in lieu of those provisions of subpart M which apply to major sources, as defined in § 63.320(g). Dry cleaning facilities which are major sources remain subject to subpart M.

(*ii*) Yolo-Solano Air Quality Management District is not delegated the Administrator's authority of § 63.325 to determine equivalency of emissions control technologies. Any source seeking permission to use an alternative means of emission limitation, under sections 216, 301.3.a(v), 301.3.b(ii)(c), and 502 of Rule 9.7, must also receive approval from the Administrator before using such alternative means of emission limitation for the purpose of complying with section 112.

* * * *

[FR Doc. 99–1910 Filed 1–27–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300778; FRL 6053–8]

RIN 2070–AB78

Diflufenzopyr; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for combined 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(6*H*)-one) in or on field corn stover, forage and grain. BASF Corporation requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104–170).

DATES: This regulation is effective January 28, 1999. Objections and requests for hearings must be received by EPA on or before March 29, 1999.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300778], must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing requests shall be labeled ''Tolerance Petition Fees'' and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees), P.O. Box 360277M, Pittsburgh, PA 15251. A copy of any objections and hearing requests filed with the Hearing Clerk identified by the docket control number, [OPP–300778], must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number [OPP–300778]. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

FOR FURTHER INFORMATION CONTACT: By mail: Joanne I. Miller, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305–6224; e-mail: miller.joanne@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of November 21, 1997, (62 FR 62304) (FRL 5755–4), EPA, issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) announcing the filing of a pesticide petition (PP) for tolerance by BASF Corporation, P.O. Box 13528, Research Triangle Park, North Carolina 27709. This notice included a summary of the petition prepared by BASF Corporation, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR part 180 be amended by establishing tolerances for combined residues of the herbicide 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(6*H*)-one), in or on field corn fodder (stover), forage and grain at 0.05 part per million (ppm). Note that the scientific assessments relevant to establishing these tolerances for diflufenzopyr were conducted jointly between EPA and the Pest Management Regulatory Agency (PMRA) of Canada as a project under the North American Free Trade Agreement (NAFTA) and the Canadian United States Trade Agreement (CUSTA). Diflufenzopyr qualified as a candidate for such a program due to its classification as a reduced risk pesticide.

I. Risk Assessment and Statutory Findings

Section 408(b)(2)(A)(i) of the 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) 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) 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.''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. For further discussion of the regulatory requirements of section 408 and a complete description of the risk assessment process, see the Final Rule on Bifenthrin Pesticide Tolerances, November 26, 1997, (62 FR 62961) (FRL 5754–7).

II. Aggregate Risk Assessment and Determination of Safety

Consistent with 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 diflufenzopyr and to make a determination on aggregate exposure, consistent with section 408(b)(2), for tolerances for combined 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(6*H*)-one) on field corn stover, forage and grain at 0.05 ppm. EPA's assessment of the dietary exposures and risks associated with establishing the tolerance 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 nature of the toxic effects caused by diflufenzopyr are discussed below.

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

2. 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 milligram/kilogram body weight/day (mg/kg bw/day) for males, and 0, 72.8, 431, 890 and 1,750 mg/kg bw/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 no observed adverse effect level (NOAEL) was set at 5,000 ppm (equal to 352 mg/kg bw/day for males, and 431 mg/kg bw/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 4-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.

3. 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 bw/day for males, and 0, 84, 369, 787 and 1,605 mg/kg bw/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.

4. In a subchronic toxicity study in dogs, diflufenzopyr (98% a.i.) 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).

5. In the subchronic 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 bw 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 bw/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).

6. In a chronic toxicity study in dogs, diflufenzopyr (98.1% a.i.) 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).

7. 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 bw/day for males, and 0, 98, 500 and 1,004 mg/kg bw/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 bw/day for males and 1,004 mg/kg bw/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 oncogenic potential of diflufenzopyr for male or female mice at any dose level tested.

8. 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 bw/day for males, and 0, 29, 93, 323 and 697 mg/kg bw/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 bw/day for males and 323 mg/kg bw/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 oncogenic potential of diflufenzopyr at any dose level tested. The incidences of benign and malignant tumors were comparable between control and treated groups.

9. In a developmental toxicity study, technical diflufenzopyr (98.1% a.i.) 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 NOAEL is 300 mg/kg/day and the maternal 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 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.

10. In a developmental toxicity study, technical diflufenzopyr (98.1% a.i.) 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.

11. In a 2-generation reproduction study, technical diflufenzopyr (98.1% a.i.) 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).

12. In an acute neurotoxicity study, diflufenzopyr (96.4% a.i.) was administered by gavage to Crl:CD BR 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. Mean locomotor activities for the 2,000 mg/kg female treatment groups were decreased on Days 7 (~27%, $p < 0.05$) and 14 (~15%, not significant) after dosing, but the pattern of activity for the individual animals was similar to the individual controls over time. 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).

13. In a subchronic neurotoxicity study, diflufenzopyr (96.4% a.i.) 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 mg/kg/day.

14. In a microbial mutagenicity assay, Salmonella typhimurium strains TA98, TA100, TA1535, TA1537, and TA1538 were exposed to diflufenzopyr (97.1%) in DMSO at concentrations of 333, 667, 1,000, 3,330, 6,670 and 10,000 µg/plate in the presence and absence of mammalian metabolic activation. Diflufenzopyr (97.1%) was tested to twice the limit concentration of 5,000 µg/plate and cytotoxicity was observed at 6,670 and 10,000 µg/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.

15. In a mammalian cell gene mutation assay at the thymidine kinase locus, heterozygous L5178Y (TK +/-) mouse lymphoma cells cultured *in vitro* were exposed in independent repeat assays to diflufenzopyr technical (97.1% a.i.) in dimethyl sulfoxide at dose levels ranging from 0.05 to 3.0 mg/mL (50 to 3,000 µg/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 µg/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 TK locus in mouse L5178Y cells in the presence and absence of S9 activation. The positive controls induced the appropriate responses.

16. 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.

17. In an unscheduled DNA synthesis assay, primary rat hepatocyte cultures were exposed to diflufenzopyr (97.1% a.i.) in dimethylsulfoxide (DMSO) at 15 concentrations ranging from 0.0250 to 1,000 µg/mL in the presence of 10µCi/ml[3] HtdR (42 Ci/mmole) for approximately 19 hours. Mutagenicity, as measured by unscheduled DNA synthesis (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 µg/mL. The highest concentration selected for UDS evaluation, 250 µg/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.

18. In a rat metabolism study, (phenyl-U-^{14}C) or (pyridinyl-4,6-^{14}C) diflufenzopyr was administered to five Wistar rats/sex/dose group as a single intravenous dose at 1 mg/kg/day, a single oral dose (gavage) at 10 or 1,000 mg/kg or a single dose at 10 mg/kg following a 14-day pretreatment with unlabeled diflufenzopyr at 10 mg/kg. Bile-duct cannulated rats from each dose group were sacrificed at 48 hours post-dose (Subgroup 2). Non-cannulated rats from each dose group were sacrificed at 72 hours (Subgroup 1) or 24 hours (Subgroup 3) post-dose. (^{14}C) Diflufenzopyr was only partially absorbed from the GI tracts of orally dosed rats as indicated by the levels of excretion in urine and bile. In all orally dosed groups, 20–44% of the dose was excreted in the urine and 3–11% was excreted in the bile. In contrast, intravenously dosed rats excreted 61–89% of the dose in urine and 4–19% of the dose in bile. For all orally dosed groups, the level of absorption was similar between sexes. Dose level and pretreatment had little effect on the proportion of the dose excreted in urine following oral administration. Enterohepatic circulation plays a role in the elimination of ^{14}C diflufenzopyr in rats. 3–19% of the dose was recovered in the bile of all dose groups. Within 72 hours of dosing, intravenously-dosed rats excreted the majority of radioactivity in urine (61–89%), whereas orally-dosed rats excreted most of the radioactivity in feces (49–79%), regardless of radiolabel or sex. Pretreatment did not appear to affect the pattern of excretion. Bile-cannulated rats excreted lesser amounts in feces compared to non-cannulated rats; 3–19% of the dose was excreted in bile. The estimated half-lives of radiocarbon eliminated in urine and feces was 5.3-6.9 hours for all single intravenous and oral dose groups, and 7.7-10.8 hours for all repeat oral dose groups. Total radioactive residues in tissues from rats in all dose groups were <3% of the administered dose. Total tissue residue levels were highest in rats sacrificed at 24 hours post-dose; residue levels were highest in blood, blood cell, and serum for the phenyl label groups, and were highest in liver and kidney for the pyridinyl label groups. Blood residue levels for all dose groups were <1% of the administered dose at all sampling intervals through 72 hours post-dose. TLC and HPLC analyses were conducted on 0-72 and 0-48 urine and feces samples, and on 0-48 hour bile samples from each treatment regimen. The structures of the metabolites were confirmed using 2-D TLC, HPLC, LC/MS, DIP/MS, FAB/MS, and proton NMR. For each dose group, the metabolic profile was similar between sexes, except for differences in metabolite levels. Unchanged diflufenzopyr was identified as the major component in urine, feces, and bile from all dose groups using either radiolabel. Urinary metabolites identified in the ^{14}C-phenyl labeled dose groups included: 3,5-difluoroaniline (M2; aniline) and 6-((3,5-difluorophenyl) carbomyl)-8-methyl-pyrido (2,3-*d*)-5-pyridazinone (M5; carbamoyl phthalazinone). Urinary metabolites identified in the ^{14}C-pyridinyl labeled dose groups included: 8-methyl-5-hydroxy-pyrido(2,3-*d*)-pyridazine (M1; phthalazinone); carbamoyl phthalazinone (M5); 2-acetyl nicotinic acid (M6; 2-acetyl nicotinic acid); 8-methylpyrido[2,3-d]pyridazine-2,5(1*H*, 6*H*)-dione (M9; 2-keto-M1); 8-hydroxymethyl-5(6*H*)-pyrido[2,3-d]pyridazinone (M10; 8-hydroxymethyl-M1); and, 8-hydroxymethylpyrido[2,3-d]pyridazine-2,5(1*H*,6*H*)-dione (M19; 2-keto-8-hydroxymethyl-M1 or Metabolite E). Fecal metabolites identified in the phenyl label groups included: methyl *N*-(3,5-difluorophenyl)carbamate (M8) and M5. Fecal metabolites identified in the pyridinyl label groups included: M1, M5, M6, M9, and M10. Besides parent, bile samples also contained minor amounts of M5 (both labels) and M1 (pyridinyl label only). The data indicate that diflufenzopyr is excreted primarily as unchanged parent in urine, feces, and bile. Minor amounts of hydrolysis products (M1, M5, and M6) and hydroxylation products (M9, M10, and M19) were identified in excreta.

B. Toxicological Endpoints

1. *Acute toxicity*. For acute dietary risk assessment, an acute Reference Dose (RfD) of 1.0 mg/kg/day has been selected, based on the developmental NOAEL of 100 mg/kg/day from the Rabbit Developmental Study and an uncertainty factor of 100 (10x for interspecies differences and 10x for intraspecies variations). The endpoint is based on developmental findings (increased skeletal variations) in rabbits which can be attributed to a single gavage dose during gestation and which occurred at a maternally toxic dose. The population subgroup at risk for this developmental effect is females of child-bearing age (13+ years). No appropriate toxicological endpoint is available in the data base for other subgroups of the population including infants and children.

2. *Short - and intermediate - term toxicity.* Since there was no observed dermal or systemic toxicity in a rabbit 21-day dermal study with diflufenzopyr, short- and intermediate-term toxicity endpoints are not being established.

3. *Chronic toxicity*. EPA has established the 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.

4. *Carcinogenicity*. 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.

C. Exposures and Risks

1. *From food and feed uses.* No previous tolerances have been established for the combined 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(6*H*)-one). Risk assessments were conducted by EPA to assess dietary exposures from diflufenzopyr as follows:

i. *Acute exposure and risk.* Acute dietary 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 one day or single exposure. An acute dietary risk assessment was performed for diflufenzopyr, its metabolites characterized as M1, and M10. 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).

ii. *Chronic exposure and risk.* A chronic dietary risk assessment was performed for diflufenzopyr, its metabolites characterized as M1, and M10. 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.

2. *From 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 SCI-GROW (Screening Concentration in Ground Water) model to estimate residues of diflufenzopyr in ground water and the GENEEC (Generic Expected Environmental Concentration) model to estimate diflufenzopyr residue levels in surface water. Estimated environmental concentrations (EECs) in ground water reflecting an application rate of 0.12 pounds of active ingredient per acre were 0.006 parts per billion (ppb) for acute and chronic exposure scenarios. EECs in surface water were 3.8 ppb for acute exposure scenarios and 1.95 ppb for chronic exposure scenarios. The computer generated EECs represent conservative estimates and should be used only for screening.

i. *Acute exposure and risk.* EPA has calculated a drinking water level of comparison (DWLOC) for acute exposure to diflufenzopyr in drinking water for the relevant population subgroup, females 13 + years of age. THE DWLOC is 29,970 ug/L.

To calculate the DWLOCs for acute exposure relative to an acute toxicity endpoint, the acute dietary food exposure from the DEEM (Dietary Exposure Evaluation Model) analysis was subtracted from the ratio of the acute NOAEL (used for acute dietary assessments) to the acceptable margin of exposure (MOE) for aggregate exposure to obtain the acceptable acute exposure to diflufenzopyr in drinking water. DWLOCs were then calculated using default body weights and drinking water consumption figures.

Estimated maximum concentrations of diflufenzopyr in surface and ground water are 3.80 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 use proposed in this action, EPA 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.

ii. *Chronic exposure and risk.* EPA has calculated drinking water levels of comparison (DWLOCs) for chronic exposure to diflufenzopyr in drinking water. For chronic (non-cancer) exposure to diflufenzopyr in surface and ground water, the drinking water levels of comparison are 9,100 ug/L and 2,600 ug/L for the U.S. population and the subgroup children (1–6 years old), respectively.

To calculate the DWLOCs for chronic (non-cancer) exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure (from the DEEM analysis) and residential exposure were subtracted from the RfD to obtain the acceptable chronic (non-cancer) exposure to diflufenzopyr in drinking water. DWLOCs were then calculated using default body weights and drinking water consumption figures.

Estimated average concentrations of diflufenzopyr in surface and ground water are 1.95 ppb and 0.006 ppb, respectively. The DWLOCs are 9,100 ppb for the U.S. population and 2,600 ppb for the subgroup, children (1–6 years old). The estimated average concentrations of diflufenzopyr in surface and ground water are less than EPA's levels of comparison for diflufenzopyr in drinking water as a contribution to chronic aggregate exposure.

3. *From non-dietary exposure.* There are no registered or proposed residential uses for diflufenzopyr.

4. *Cumulative exposure to substances with common mechanism of toxicity.* Section 408(b)(2)(D)(v) 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 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, EPA has not assumed that diflufenzopyr has 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 the Final Rule for Bifenthrin Pesticide Tolerances (62 FR 62961, November 26, 1997).

D. Aggregate Risks and Determination of Safety for U.S. Population

1. *Acute risk.* For the population subgroup of concern, females 13+ years old, the acute dietary (food) exposure does not exceed 0.02% of the acute RfD. The drinking water level of comparison (DWLOC) for acute exposure to diflufenzopyr residues is 29,970 ug/L for females (13+ years). The maximum concentration of diflufenzopyr in drinking water (3.80 ug/L) is less than EPA's level of comparison for diflufenzopyr in drinking water as a contribution to acute aggregate exposure. EPA concludes with reasonable certainty that residues of diflufenzopyr in drinking water will not contribute significantly to the aggregate acute human health risk and that the

acute aggregate exposure from diflufenzopyr in food and water will not exceed the Agency's level of concern for acute dietary exposure.

2. *Chronic risk.* Using the TMRC exposure assumptions described above, EPA has concluded that aggregate exposure to diflufenzopyr from food will utilize less than 0.1% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure, children 1–6 years old, is ''discussed below.'' 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 and from non-dietary, non-occupational exposure, EPA does not expect the aggregate exposure to exceed 100% of the RfD. EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to diflufenzopyr residues.

3. *Short- and intermediate-term risk.* Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. There are no established or proposed residential uses for diflufenzopyr. Therefore, the short and intermediate aggregate risks are adequately addressed by the chronic aggregate dietary risk assessment.

4. *Aggregate cancer risk for U.S. population.* Diflufenzopyr has been classifiedd as ''not likely'' to be a human carcinogen.

5. *Determination of safety.* Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result from aggregate exposure to diflufenzopyr residues.

E. Aggregate Risks and Determination of Safety for Infants and Children

1. *Safety factor for infants and children—* i. *In general.* In assessing the potential for additional sensitivity of infants and children to residues of diflufenzopyr, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure (MOE) analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor.

ii. *Pre- and post-natal sensitivity.* There is no indication of increased sensitivity of rats or rabbits to *in utero* and/or early postnatal exposure to diflufenzopyr in the developmental and reproductive toxicity studies.

iii. *Conclusion.* There is a complete toxicity database for diflufenzopyr and exposure data is complete or is estimated based on data that reasonably accounts for potential exposures. Taking into account the completeness of the database and the toxicity data regarding pre- and post-natal sensitivity, EPA concludes, based on reliable data, that use of the standard margin of safety will be safe for infants and children without addition of another tenfold factor.

2. *Acute risk.* No appropriate acute toxicological endpoint has been identified for infants and children.

3. *Chronic risk.* Using the exposure assumptions described above, 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, EPA does not expect the aggregate exposure to exceed 100% of the RfD.

4. *Determination of safety.* Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to diflufenzopyr residues.

III. Other Considerations

A. Metabolism In Plants and Animals

The nature of the residue in plants (field corn) and animals is understood. In field corn, the urea bond is cleaved to yield metabolites containing a new bicyclic ring system. No diflufenzopyr was detected in any of the corn matrices; metabolites comprising >10% total radioactive residue (TRR) include M1 (8-methylpyrido[2,3-*d*]pyridazin-5(6*H*)-one), M10 (8-hydroxymethyl-5(6*H*)-pyrido[2,3-*d*]pyridazone) and its glucose conjugate, and M9 (8-methylpyrido[2,3-*d*]pyridazine-2,5(1*H*,6*H*)-dione in forage and fodder, and 6–14% TRR lignin was found in fodder. Corn grain contained 3–4 discrete unknowns, all at <10% TRR or <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(6*H*)-one).

In livestock, the majority (≧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(1*H*,6*H*)-dione. A substantial amount (8–50%) of diflufenzopyr was also found in milk, kidney, and liver. 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 .

B. Analytical Enforcement Methodology

Adequate enforcement methodology (gas chromatography) is available to enforce the tolerance expression. The method may be requested from: Calvin Furlow, PRRIB, IRSD (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm 101FF, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703–305–5229).

C. Magnitude of Residues

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(6*H*)-one) are not expected to exceed 0.05 ppm in field corn grain, forage and stover.

D. International Residue Limits

There are no CODEX or Mexican residue limits established for diflufenzopyr or its metabolites. As part of the joint review, Canada will be setting an equivalent Maximum Residue Level (MRL) for corn grain. Therefore, no compatibility problems exist for the proposed tolerances.

E. Rotational Crop Restrictions

The end-use product, which contains the active ingredients diflufenzopyr and dicamba (sodium salts), will contain a statement limiting the planting of rotational crops for a least 120 days after application. This restriction is based on rotational crop data for dicamba. The rotational crop study submitted for diflufenzopyr was not conducted in accordance with EPA guidelines. However, based on the results of this study, the low residues in the treated corn crop and diflufenzopyr's lack of persistence in soil, EPA does not expect residues of diflufenzopyr and its metabolites to occur in rotational crops at the 120–day plant-back interval, when corn is treated at the label rate of up to 0.125 pounds active ingredient per acre per season.

IV. Conclusion

Therefore, tolerances are established for combined 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(6*H*)-one) in field corn stover, forage and grain at 0.05 ppm ppm.

V. Objections and Hearing Requests

The new FFDCA section 408(g) provides essentially the same process for persons to ''object'' to a tolerance regulation as was provided in the old section 408 and in section 409. However, the period for filing objections is 60 days, rather than 30 days. EPA currently has procedural regulations which govern the submission of objections and hearing requests. These regulations will require some modification to reflect the new law. However, until those modifications can be made, EPA will continue to use those procedural regulations with appropriate adjustments to reflect the new law.

Any person may, by March 29, 1999, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33. If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential 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. A copy of the information that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice.

VI. Public Record and Electronic Submissions

EPA has established a record for this rulemaking under docket control number [OPP–300778] (including any comments and data submitted electronically). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The public record is located in Room 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA.

The official record for this rulemaking, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer any copies of objections and hearing requests received electronically into printed, paper form as they are received and will place the paper copies in the official rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the Virginia address in ''ADDRESSES'' at the beginning of this document.

VII. Regulatory Assessment Requirements

A. Certain Acts and Executive Orders

This final rule 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). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled *Enhancing the Intergovernmental Partnership* (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled *Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations* (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled *Protection of Children from Environmental Health Risks and Safety Risks* (62 FR 19885, April 23, 1997).

In addition, since tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances 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. Nevertheless, the Agency has previously assessed whether establishing tolerances, exemptions from tolerances, raising tolerance levels or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950) and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

B. Executive Order 12875

Under Executive Order 12875, entitled *Enhancing the Intergovernmental Partnership* (58 FR 58093, October 28, 1993), EPA may not issue a regulation that is not required by statute and that creates a mandate upon a State, local, or tribal government, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by those governments. If the mandate is unfunded, EPA must provide to OMB a description of the extent of EPA's prior consultation with representatives of affected State, local, and tribal governments, the nature of their concerns, copies of any written communications from the governments, and a statement supporting the need to issue the regulation. In addition, Executive Order 12875 requires EPA to develop an effective process permitting elected officials and other representatives of State, local, and tribal governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

Today's rule does not create an unfunded Federal mandate on State, local, or tribal governments. The rule does not impose any enforceable duties on these entities. Accordingly, the requirements of section 1(a) of Executive Order 12875 do not apply to this rule.

C. Executive Order 13084

Under Executive Order 13084, entitled *Consultation and Coordination with Indian Tribal Governments* (63 FR 27655, May 19, 1998), EPA may not issue a regulation that is not required by statute, that significantly or uniquely affects the communities of Indian tribal governments, and that imposes substantial direct compliance costs on those communities, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by the tribal governments. If the mandate is unfunded, EPA must provide to OMB, in a separately identified section of the preamble to the rule, a description of the extent of EPA's prior consultation with representatives of affected tribal governments, a summary of the nature of their concerns, and a statement supporting the need to issue the regulation. In addition, Executive Order 13084 requires EPA to develop an effective process permitting elected officials and other representatives of Indian tribal governments ''to provide meaningful and timely input in the development of regulatory policies on matters that significantly or uniquely affect their communities.''

Today's rule does not significantly or uniquely affect the communities of Indian tribal governments. This action does not involve or impose any requirements that affect Indian tribes. Accordingly, the requirements of section 3(b) of Executive Order 13084 do not apply to this rule.

VIII. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. This rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

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

Dated: January 14, 1999.

Marcia E. Mulkey,

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. 346a and 371.

2. By adding §180.549 to read as follows:

§180.549 Diflufenzopyr; tolerances for residues.

(a) *General.* Tolerances are established for combined 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(6*H*)-one) in or on the following raw agricultural commodities.

Commodity	Parts per million
Field corn, forage	0.05
Field corn, grain	0.05
Field corn, stover	0.05

(b) *Section 18 emergency exemptions.* [Reserved]

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

(d) *Indirect or inadvertent residues.* [Reserved]

[FR Doc. 99–1901 Filed 1–27–99; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300788; FRL–6058–7]

RIN 2070–AB78

Partial Withdrawal of Cryolite Tolerance Revocations

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule; partial withdrawal.

SUMMARY: This final rule and order withdraws the revocation of tolerances for residues of cryolite (fluorine compounds) on apricots, blackberries, boysenberries, dewberries, kale, loganberries, nectarines, and youngberries made in a final rule entitled ''Revocation of Tolerances for Canceled Food Uses'', (October 26, 1998; (63 FR 57067) (FRL–6035–6) which had an effective date of January 25, 1999. EPA is withdrawing the revocation of those specific tolerances because comments from Gowan Company made to the proposed rule (63 FR 5907, February 5, 1998) (FRL–5743–9) concerning cryolite were inadvertently not addressed.

DATES: This rule is effective on January 25, 1999.

FOR FURTHER INFORMATION CONTACT: For technical information contact: Joseph Nevola, Special Review Branch, (7508C), Special Review and Reregistration Division, Office of Pesticide Programs, U.S. Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location: Special Review Branch, CM #2, 6th floor, 1921 Jefferson Davis Hwy., Arlington, VA. Telephone: (703) 308–8037; e-mail: nevola.joseph@epa.gov.

SUPPLEMENTARY INFORMATION: