ERP No. F–FHW–E54009–NC, US 117 Corridor Improvement Project, US 13/70 at Goldsboro, north to US 301 in Wilson, Funding and Section 404 Permit, Wayne and Wilson Counties, NC.

Summary: EPA continued to favor Alternative 1, the improvement of the present road, or Alternative 4, a new alignment outside the Little River critical watershed area. EPA is concerned that this project could directly or indirectly cause the degradation of the Little River and reduce the classified use of this river as a water supply. EPA would like to see commitments to long-term maintenance of measures to minimize the inflow of pollutants to the river.

ERP No. F–FHW–K50007–CA, Benicia-Martinez Bridge System Project, Construction/Reconstruction, Portions of I–680, I–780 and I–80 Corridors, Funding, U.S. CGD Bridge Permit and COE Section 10 and 404 Permits, Contra Costa and Solano Counties, CA.

Summary: EPA expressed continuing concerns on the placement of new toll booths in close proximity to an industrial facility using hazardous materials and requested FHWA to reconsider its decision. EPA also requested FHWA provide additional information on a contingency plan to guide travelers along the highway in the event of a chemical upset or accidental releases. EPA asked that the Record of Decision clarify what level of High Occupancy Vehicle (HOV) features would be part of the project, and strongly recommend that all feasible efforts to implement one or more dedicated HOV lanes for both north and southbound traffic in peak commute periods be part of the final project.

ERP No. F–NAS–A12041–00, X–33 Advanced Technology Demonstrator Vehicle Program, Final Design, Construction and Testing, Implementation, Approvals and Permits Issuance, CA, UT and WA.

Summary: EPA had no objection to proposed action.

ERP No. F–USN–L11031–WA, Puget Sound Naval Station, Sand Point, Disposal and Reuse, Implementation, King County, WA.

Summary: Review of the Final EIS was not deemed necessary. No formal comment letter was sent to the preparing agency.

ERP No. FA–DOE–K03007–CA, Petroleum Production at Maximum Efficient Rate, updated Information for the Sale of Naval Petroleum Reserve No. 1 (NPR–1 also called ''Elk Hills'') Amendment for Kern County General Plan, Elk Hills, Kern County, CA.

Summary: Review of the Final EIS was not deemed necessary. No formal comment letter was sent to the preparing agency.

Dated: November 18, 1997.

B. Katherine Biggs,

Associate Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 97–30696 Filed 11–20–97; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[PF–778; FRL–5755–4]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–778, must be received on or before December 22, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as ''Confidential Business Information'' (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment 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. All written comments will be available for public inspection in Rm. 1132 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Kerry Leifer	Rm. 4W17, CS #1, 703–308–8811, e-mail: leifer.kerry@epamail.epa.gov.	2800 Crystal Drive, Arlington, VA

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–778] including comments and data submitted electronically as described below). 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 official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number PF-778 and appropriate petition number. Electronic comments on notice may be filed online at many Federal Depository Libraries.

List of Subjects

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

Dated: November 10, 1997

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them 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.

1. BASF Corporation

PP 7F4848

EPA has received a pesticide petition (PP 7F4848) from BASF Corporation, P.O. Box 13528, Research Triangle Park, NC 27709-3528 proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing tolerances for residues of diflufenzopyr, (2-[1-[[[(3,5-difluorophenyl) amino]carbonyl]hydrazono] -ethyl]-3-pyridinecarboxylic acid), and its metabolites M1 (8-methylpyrido(2,3-d)pyridazin-5(6H)-one) and M5 (6-((3,5-Difluorophenyl-carbamoyl-8-methyl-pyrido (2,3-d)-5-pyridazinone) all as the M1 component in or on the raw agricultural commodities corn grain, corn forage and corn fodder at 0.05 parts per million (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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Analytical method*. The proposed analytical method involves extraction, partition, clean-up and detection of residues by gas chromatography/ nitrogen phosphorous detector (gc/npd).

2. *Magnitude of residues*. Over 20 residue trials were conducted in 16 states. Residues of diflufenzopyr, M5 and M1 were measured as M1 by gc/ npd. The method of detection had a limit of detection of 0.01 parts per million (ppm). Residues ranged from non detectable (majority) to 0.02 ppm rt text.

B. Toxicological Profile

1. *Acute toxicity*. A battery of acute toxicity tests were conducted which place diflufenzopyr in acute oral toxicity category IV, acute dermal toxicity category IV, acute inhalation toxicity category IV, primary eye irritation category III, and primary dermal irritation category IV. Diflufenzopyr is not a dermal sensitizer. Diflufenzopyr is not a neurotoxin in males and females at 2,000 mg/kg (limit test).

2. *Genotoxicity*. Diflufenzopyr was found to be negative for mutagenicity in a battery of mutagenicity tests (Ames Testing, Mouse Lymphoma testing *In vivo* micronucleus assay (mouse) and Unscheduled DNA synthesis).

3. *Reproductive and developmental toxicity—i. Developmental toxicity (rat)*. Sprague-Dawley rats were dosed with 0, 100, 300 and 1,000 mg/kg/day diflufenzopyr in the diet from days 6 through 15 of gestation. The maternal no observed adverse effect level (NOAEL) was determined to be 300 mg/kg/day and the maternal lowest effect level (LEL) was determined to be 1,000 mg/ kg/day based on reduced body weight gain, and reduced absolute and relative feed consumption during the dosing period. The developmental NOAEL was determined to be 300 mg/kg/day and the developmental LEL was determined to be 1,000 mg/kg/day based on reduced fetal body weight and reversible delays in sternal and caudal vertebral ossification.

ii. *Developmental toxicity (rabbit)*. New Zealand white rabbits were dosed with 0, 30, 100, and 300 mg/kg/day diflufenzopyr in the diet from days 6 through 19 of gestation. The maternal NOEL was determined to be 30 mg/kg/ day and the maternal LEL was determined to be 100 mg/kg/day based on increased incidence of abnormal feces and weight loss for the entire dosage period. The developmental NOEL was determined to be 100 mg/kg/ day and the developmental LEL was determined to be 300 mg/kg/day based on increased incidences of supernumerary thoracic ribs, a variation in fetal ossification that commonly occurs at maternally toxic dosages. Only at the 300 dose level deaths and abortions were accompanied by gastric trichobezoars. Diflufenzopyr was not teratogenic to rabbit fetuses even at the higher of two dosages (100 and 300 mg/ kg/day) that were toxic to the does.

iii. *Reproductive toxicity testing*. In a 2-Generation Reproduction study, Wistar rats were dosed with 0, 500, 2,000 and 8,000 ppm diflufenzopyr in the diet. The parental: systemic NOAEL/ reproductive-developmental NOEL was determined to be 2,000 ppm in both sexes (averaging 600 mg/kg/day in females during gestation). The parental LEL was determined to be 8,000 ppm (averaging 2,500 mg/kg/day in females during gestation) based on weight gain deficits in males and females during premating and pregnancy phases. The developmental NOEL was determined to be 2,000 ppm (averaging 400 mg/kg/day in dams during lactation) and the LEL determined to be 8,000 ppm (averaging 1,500 mg/kg/day in dams during lactation) based on slightly lower live birth (93%) and viability (90%) indices.

4. *Subchronic toxicity*— i. *21-Day dermal (rabbit)*. Rabbits were repeatedly dosed with diflufenzopyr at 0, 100, 300 and 1,000 mg/kg/day for 21 days. The NOAEL for systemic toxicity and dermal irritation was determined to be 1,000 mg/kg/day.

ii. *90-Day rodent (rat)*. Wistar rats were dosed with diflufenzopyr at 0, 1,000, 5,000, 10,000 and 20,000 ppm in the diet for 90 days. The NOEL was determined to be 5,000 ppm (350 mg/ kg/day) for males and 430 mg/kg/day in females. The LEL was determined to be 10,000 ppm (720 mg/kg/day) for males and 890 mg/kg/day in females based on reduced body weight gains, impaired food utilization; disturbances in hematology values in males, clinical chemistry values in both sexes, values for urinalysis in females; with histopathology seen in both sexes as increased foamy macrophages in the lungs.

iii. *90-Day mouse*. CD-1 mice were dosed with diflufenzopyr at 0, 350, 1,750, 3,500 and 7,000 ppm in the diet for 13 weeks. The NOEL was determined to be 7,000 ppm (1,225 mg/ kg/day) in males and (1,605 mg/kg/day) in females as no clear toxic effects were observed.

iv. *90-Day non-rodent (dog)*. Beagle dogs were dosed with diflufenzopyr at

0, 1,500, 10,000, and 30,000 ppm in the diet for 13 weeks. The NOEL was determined to be 1,500 ppm (58 mg/kg/day) in males and (59 mg/kg/day) in females. The LEL was determined to be 10,000 ppm (403 mg/kg/day) in males and (424 mg/kg/day) in females based on histopathological disturbances seen as erythreoid hyperplasia in the bone marrow and extramedullary hemopoiesis in the liver of a few dogs and hemosiderin deposits in Kupffer cells in 1 female dog.

v. *90-Day neurotoxicity (rat).* Rats were dosed with diflufenzopyr at 0, 25, 75, and 1,000 mg/kg/day in the diet for 13 weeks. At the 1,000 mg/kg/day treatment there was associated weight gain and impaired efficiency of food utilization. Therefore the no adverse effect level was set at 75 mg/kg/day. The NOAEL for subchronic neurotoxicity was determined to be 1,000 mg/kg/day based on the absence of changes indicative of neurotoxicity.

5. *Chronic toxicity*—i. *1-Year non-rodent (dog).* Beagle dogs were dosed with diflufenzopyr at 0, 750, 7,500 and 15,000 ppm in the diet for one year. The NOEL was determined to be 750 ppm (26 mg/kg/day) in males and (28 mg/kg/day) in females. The LOAEL was 7,500 ppm (299 mg/kg/day) in males and (301 mg/kg/day) in females. This is based on an erythropoietic response in bone marrow and increased hemosiderin deposits in spleen, liver and kidneys. Peripheral hematology investigations revealed mild to moderate reticulocytosis at the 7,500 and 15,000 ppm dose levels, in the absence of any signs of anemia. The erythropoietic response of bone marrow is thought to compensate probable toxic effects to erythrocytes. Because of a similarity of NOEL levels from this dog study and the rat chronic/oncogenicity study a suggested risk assessment reference dose (Rfd) is calculated by using 25 as a Noel level with a 100 fold safety factor ending with 0.25 mg/kg/day.

ii. *Combined rodent chronic toxicity/oncogenicity (rat).* Wistar rats were fed 0, 500, 1,500, 5,000 and 10,000 ppm diflufenzopyr in the diet for 104 weeks. The NOEL was determined to be 500 ppm (22 mg/kg/day) in males and (29 mg/kg/day) in females. The NOAEL was determined to be 1,500 ppm (69 mg/kg/day) in males and (93 mg/kg/day) in females based on reduced body weight gains of 8 % in males and 7% in females. The LEL was determined to be 5,000 ppm (235 mg/kg/day) in males and(323 mg/kg/day) in females based on 9% reduced weight gain in females and 11% in males plus males showed lower triglyceride and higher phosphate levels. Diflufenzopyr was not carcinogenic under the conditions of the test.

iii. *Oncogenicity in the rodent (mouse).* CD-1 mice were fed 0, 700, 3,500 and 7,000 ppm diflufenzopyr in the diet for 78 weeks. The NOAEL was determined to be 7,000 ppm (1037 mg/kg/day) in males and (1,004 mg/kg/day) in females. There were no changes or histopathological findings attributed to the dietary inclusion of test material in the 52 (interim) or 78 (terminal) week animals. Diflufenzopyr was not carcinogenic under the conditions of the test.

C. Aggregate Exposure

1. *Dietary exposure.* The potential aggregate dietary exposure is based on the Theoretical Maximum Residue Contribution (TMRC) from the tolerances for all crops on which diflufenzopyr is to be applied. The TMRC from the proposed use of diflufenzopyr of corn at the tolerance level of 0.05 ppm is 0.173168 ug/kg/day, and utilizes 0.069 percent of the RfD for the overall U.S. population. The exposure of the most highly exposed subgroup in the population, non-nursing infants, is 0.195424 ug/kg/day, and utilizes 0.078 percent of the RfD.

2. *Drinking water.* Based on the studies submitted to EPA for assessment of environmental risk, BASF does not anticipate exposure to residues of diflufenzopyr in drinking water. There is no established maximum concentration level for residues of diflufenzopyr in drinking water under the Safe Drinking Water Act.

3. *Non-dietary exposure.* BASF has not estimated non-occupational exposure for diflufenzopyr since the only pending registration for diflufenzopyr is limited to commercial crop production use. Diflufenzopyr products are not labeled for any residential uses therefore, eliminating the potential for residential exposure. The potential for non-occupational exposure to the general population is considered to be insignificant.

D. Cumulative Effects

BASF also considered the potential for cumulative effects of diflufenzopyr and other substances that have a common mechanism of toxicity. BASF has concluded that consideration of a common mechanism of toxicity is not appropriate at this time since there is no indication that toxic effects produced by diflufenzopyr would be cumulative with those of any other chemical compounds. Semicarbazone chemistry is new and diflufenzopyr has a novel mode of action compared to currently registered active ingredients.

E. Safety Determination

1. *U.S. population.* Dietary and occupational exposure will be the major routes of exposure to the U.S. population and ample margins of safety have been demonstrated for both situations. The TMRC from the proposed tolerance of 0.05 ppm is 0.173168 ug/kg/day and utilizes 0.0692 percent of the RfD for the overall U.S population. The MOEs for occupational exposure are greater than 7,000. Based on the completeness and reliability of the toxicity data and the conservative exposure assessments, there is a reasonable certainty that no harm will result from the aggregate exposure of residues of diflufenzopyr including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children.* Dietary exposure of the most highly exposed subgroup in the population, non-nursing infants, is 0.195424 ug/kg/day. This accounts for only 0.078 percent of the RfD. There are no residential uses of diflufenzopyr and contamination of drinking water is extremely unlikely. All chronic, lifespan and multigenerational bioassays in mammals plus tests in aquatic organisms and wildlife failed to reveal any endocrine effects. Based on the completeness and reliability of the toxicity data and the conservative exposure assessment, there is a reasonable certainty that no harm will result to infants and children from the aggregate exposure of residues of diflufenzopyr including all anticipated dietary exposure and all other non-occupational exposures.

F. International Tolerances

A maximum residue level has not been established for diflufenzopyr by the Codex Alimentarius Commission.

2. Novartis Crop Protection, Inc.

PP 7E3489

EPA has received a pesticide petition (PP 7E3489) from Novartis Crop Protection, Inc. (formerly Ciba Crop Protection), P.O. Box 18300, Greensboro, NC 27419. proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for combined residues of 4-(dichloroacetyl)-3,4-dihydro-3-methyl-2H-1,4-benzoxazine (benoxacor) when used as an inert ingredient (safener) in pesticide formulations containing metolachlor in or on raw agricultural commodities for which tolerances have been established for metolachlor. The proposed analytical method is capillary gas

chromatography using Nitrogen/Phosphorous (N/P) detection. 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 supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant/Animal metabolism*. Novartis Crop Protection, Inc. (Novartis) notes that the metabolism in plants and animals (goat, hen, and rat) is well understood. Identified metabolic pathways are similar in plants and animals.

2. *Analytical method*. Novartis Analytical Method AG536(C) is available and involves extraction, filtering, dilution, partitioning, and cleanup. Samples are then analyzed by capillary gas chromatography using Nitrogen/Phosphorous (N/P) detection. The limit of quantitation (LOQ) is 0.01 ppm.

3. *Magnitude of residues*. More than 30 residue trials were conducted in 19 states on a variety of agricultural crops [corn (field and sweet); soybeans, potatoes, green beans, radishes, sorghum, peanuts, head lettuce, peas]. There were no detectable residues of benoxacor at the limit of quantitation (LOQ) of 0.01 ppm (many samples were analyzed at an LOQ of 0.005 ppm and no residues were detected) in any raw agricultural commodity or processed commodity. No transfer of residue to animals is expected through their diet. Benoxacor is stable for a minimum of 12 months at temperatures down to -15°C.

B. Toxicological Profile

1. *Acute toxicity*. A rat acute oral study with an LD_{50} > 5,000 mg/kg, a rabbit acute dermal study with an LD_{50} > 2,010 mg/kg, a rat inhalation study with an LC_{50} > 2,000 mg/liter, a primary eye irritation study in the rabbit showing moderate eye irritation, a primary dermal irritation study in the rabbit showing benoxacor is not a skin irritant, and a skin sensitization study which showed benoxacor to be a skin sensitizer in the Guinea pig. Results of a dermal absorption study show a maximum of 55.7% of benoxacor is absorbed by the rat following a 24 hour dermal exposure. Benoxacor was applied to the shaved skin of 5 male and 5 female New Zealand white rabbits at dose levels of 0, 1,500, or 1,010 mg/kg for at least 22 consecutive days. This study showed benoxacor is not dermally toxic at doses greater than the limit dose of 1,000 mg/kg/day.

2. *Genotoxicty*. Benoxacor did not induce point mutations *in vitro* at limit (cytotoxic) concentrations in a *Salmonella/mammalian* microsome test or show any mutagenic activity in the Chinese hamster V79 mammalian point mutation test and is neither clastogenic nor aneugenic in the Chinese hamster at doses up to the limit dose of 5,000 mg/kg. Benoxacor did not induce unscheduled DNA synthesis in isolated rat hepatocytes at cytotoxic concentrations up to 20 micrograms/ml.

3. *Reproductive and developmental toxicity*. A 2-generation reproduction study in the rat at approximate doses of 0, 0.5, 2.5, 25 or 50 mg/kg/day. No effects on fertility, reproductive performance or development were seen in the rat at a maximally-tolerated dose of 50 mg/kg/day. Treatment related effects on body weight at feeding levels of > 25 mg/kg/day were accompanied by marginally reduced food intake only in the high dose group. The parental NOEL ranged from 3.4 to 4.8 mg/kg/day while the developmental NOEL was approximately 10-fold greater. A developmental toxicity study in the rat at doses of 0, 1, 100, or 400 mg/kg/day by gavage with maternal and developmental NOEL's of 1 and 100 mg/kg/day, respectively. Maternal, embryo, and fetal toxicity were observed at doses > 100 mg/kg/day. A developmental toxicity study in the rabbit at doses of 0, 0.5, 2.5, 12.5 or 62.5 mg/kg/day. Slight evidence of maternal and fetal toxicity was observed at 62.5 mg/kg/day. The maternal and developmental NOEL's were 12.5 mg/kg/day.

4. *Subchronic toxicity*. Six groups of 15 male and 15 female Sprague Dawley rats were fed benoxacor at dietary concentrations of approximately 0, 0.5, 5, 15, 50 or 300 mg/kg/day for 13 weeks. The liver (pigmentation, karyomegaly, cytomegaly, bile duct proliferation, portal mononuclear cell infiltration) and stomach (pyloric gland degeneration and necrosis) were identified as target organs in the 300 mg/kg/day group. Based on a significant depression of body weight gain at 50 and 300 mg/kg/day as well as hematology, clinical chemistry and pathology findings, the NOEL was determined to be 15 mg/kg/day.

A 90-day feeding study in the dog at approximate doses of 0, 0.25, 1, 5, 50, 150, or 400 mg/kg/day. Liver, kidney, stomach, and thymus were identified as target organs. The NOEL was 50 mg/kg/day. The maximum tolerated dose was exceeded at > 150 mg/kg/day.

A 90-day feeding study in CD-1 mice at dietary concentrations of approximately 0, 6.25, 62.5, 250, or 750 mg/kg/day for 90 days. Effects on survival, clinical signs, body weight, food consumption, the hematological system, and liver and kidney were seen at 750 mg/kg/day and to a lesser extent at 250 mg/kg/day. The NOEL was 62.5 mg/kg/day.

5. *Chronic toxicity*. A 52-week feeding study in the dog at doses of 0, 1, 5, 40, or 80 mg/kg. Liver and kidney were identified as target organs and the NOEL was established at 5 mg/kg.

An 18-month oncogenicity study in the mouse at approximate doses of 0, 1.4, 4.2, 84, or 168 mg/kg/day with a NOEL of 4.2 mg/kg/day for both chronic toxicity and tumors. Target organs were the liver and forestomach. A carcinogenic response was noted in the forestomach and is likely to be linked to a non-genotoxic mode of action involving direct irritation to the epithelial lining of the forestomach and limiting ridge between the non-glandular and glandular stomach.

A 24-month chronic feeding and oncogenicity study in the rat at approximate doses of 0, 0.5, 2.5, 25, or 50 mg/kg/day. Liver and forestomach were identified as target organs. A carcinogenic response was seen in the forestomach and is likely linked to a non-genotoxic mode of action involving direct irritation to the epithelial lining of the forestomach and the limiting ridge. The NOEL for tumors was 25 mg/kg/day and the NOEL for chronic toxicity was 0.5 mg/kg/day.

Based on the available chronic toxicity data, EPA has established the RfD for benoxacor at 0.004 mg/kg/day. This RfD is based on the 2 year feeding study in rats with a NOEL of 0.4 mg/kg/day and an uncertainty factor of 100. The uncertainty factor of 100 was applied to account for inter-species extrapolation (10) and intra-species variability (10).

Using the Guidelines for Carcinogenic Risk Assessment published September 24, 1986 (51 FR 33992), Novartis believes the Agency will classify benoxacor as a Group C carcinogen (possible human carcinogen) based on findings of a carcinogenicity effect in the non-glandular stomach of both rats and mice. Because this carcinogenic response was only observed at high doses in the non-glandular stomach of the rodent, an anatomical structure not found in humans, it is likely this response occurred via a non-genotoxic, threshold based mechanism. Novartis believes exposure to benoxacor should be regulated using a margin of exposure approach where the carcinogenic NOEL established in the most sensitive species, the mouse, was 4.2 mg/kg/day.

C. Aggregate Exposure

1. *Dietary exposure— Food*. For purposes of assessing the potential dietary exposure under the proposed tolerances, Novartis has estimated aggregate exposure based on the theoretical maximum residue contribution (TMRC) from the benoxacor tolerance of 0.01 ppm in or on raw agricultural commodities for which tolerances have been established for metolachlor. In conducting this exposure assessment, Novartis has made very conservative assumptions--100% of all raw agricultural products for which tolerances have been established for metolachlor will contain benoxacor residues and those residues would be at the level of the tolerance (0.01 ppm) which result in an overestimate of human exposure.

2. *Drinking water*. Although benoxacor is mobile and hydrolyzes slowly at low pHs, it rapidly degrades in the soil (half-life of 49 days under aerobic conditions and 70 days anaerobically). Based on this data, Novartis does not anticipate exposure to residues of benoxacor in drinking water. This is supported by extensive experience with metolachlor, where in large scale ground water monitoring studies, metolachlor has been detected in less than 4% of the samples with the typical value being 1 ppb or less. Since benoxacor is formulated as a 1 to 30 ratio with metolachlor, (maximum of 0.2 pounds benoxacor per acre) the presence of benoxacor in groundwater is highly unlikely. The EPA has not established a Maximum Concentration Level for residues of benoxacor in drinking water.

3. *Non-dietary exposure*. Novartis has evaluated the estimated non-occupational exposure to benoxacor and based on its low use rate concludes that the potential for non-occupational exposure to the general population is unlikely except for the potential residues in food crops discussed above. Benoxacor is used only on agricultural crops and is not used in or around the home.

D. Cumulative Effects

Novartis also considered the potential for cumulative effects of benoxacor and other substances that have a common mechanism of toxicity. Novartis concluded that consideration of a common mechanism of toxicity is not appropriate at this time. Novartis does not have any reliable information to indicate that toxic effects seen at high doses of benoxacor (generalized liver toxicity, nephrotoxicity and the occurrence of forestomach tumors in an organ not present in humans) would be cumulative with those of any other chemical compounds; thus Novartis is considering only the potential risks of benoxacor in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data base for benoxacor, Novartis has calculated that aggregate exposure to benoxacor will utilize 4.7% of the RfD for the U.S. population based on chronic toxicity endpoints and only 0.4% based on a margin of exposure assessment and a carcinogenic NOEL of 4.2 mg/kg/day. EPA generally has no concern for exposures below 100 percent 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. Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to benoxacor residues.

2. *Infants and children*. Using the same conservative exposure assumptions used for the determination in the general population, Novartis has concluded that the percent of the RfD that will be utilized by aggregate exposure to residues of benoxacor is 5.3% for nursing infants less than 1 year old, 20.2% for non-nursing infants, 11.9% for children 1-6 years old and 7.7% for children 7-12 years old. These worst case estimates are likely at least 4 times greater than actual values when considering that benoxacor residues have not been detected at the limit of quantitation of 0.005 ppm (tolerance is 0.01 ppm) and using a more realistic market share of 50% rather than the conservative 100%. Therefore, based on the completeness and reliability of the toxicity data base and the conservative exposure assessment, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to benoxacor residues.

F. International Tolerances

A maximum residue level has not been established for benoxacor by the Codex Alimentarius Commission.

[FR Doc. 97–30659 Filed 11–20–97; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[FRL–5925–9]

Availability of Draft Document on Information for States on Developing Affordability Criteria for Drinking Water

AGENCY: Environmental Protection Agency.

ACTION: Notice of document availability.

SUMMARY: The Environmental Protection Agency is making available for public comment a draft document entitled Information for States on Developing Affordability Criteria for Drinking Water. The Safe Drinking Water Act Amendments of 1996 require the Agency to publish information to assist states in developing affordability criteria. To meet the statutory schedule, this information must be published by February 6, 1998. The draft document being made available today was developed by a diverse working group of stakeholders under the auspices of the National Drinking Water Advisory Council (NDWAC). The full NDWAC reviewed this draft and recommended it to EPA as a draft to be made available for public comment. EPA invites interested members of the public to submit comments on the draft document. EPA will consider public comments and publish a final document by the February 6, 1998, statutory deadline.

DATES: Submit comments on or before December 31, 1997.

ADDRESSES: Address all comments concerning this draft document to Peter E. Shanaghan, Small Systems Coordinator, Office of Ground Water and Drinking Water, Mail Code 4606, 401 M Street SW., Washington, DC 20460.

FOR FURTHER INFORMATION CONTACT:

Peter E. Shanaghan, 202–260–5813 or shanaghan.peter@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: A copy of the draft document may be obtained by calling the Safe Drinking Water Hotline at 1–800–426–4791. The hotline operates Monday through Friday, 9:00 a.m.–5:30 p.m. (EST). The document may also be downloaded from EPA's homepage, http://www.epa.gov/OGWDW.

Elizabeth Fellows,

Acting Director, Office of Ground Water and Drinking Water.

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