information recommended in the CSO control policy that will be developed by municipalities with combined sewer systems that have combined sewer overflows (CSOs). Specifically, the information is the documentation that the municipalities have implemented the nine minimum controls specified in the CSO policy, the long-term control plan that the municipalities must develop and implement to achieve compliance with the requirements of the Clean Water Act and applicable State water quality standards (WQS), and compliance monitoring data for demonstrating compliance with applicable WQS and National Pollutant Discharge Elimination System (NPDES) permit conditions. The first two information submittals are one-time submittals; the last element will be submitted semi-annually as part of the municipalities' Discharge Monitoring Reports (DMRs). EPA will use this information to determine how well the CSO control policy is being implemented at the State and local level and to prepare the performance reports required under the Government Performance and Results Act (GPRA). Under the GPRA, EPA selected the CSO Control Program as a pilot program for FY 1997 and FY 1998. As such, EPA developed a FY 1997 Performance Plan that includes performance goals and associated performance measures for determining how well the program is achieving these goals. The information to be collected under this information collection is necessary to determine the program's achievement of the performance measures. An Agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR part 9 and 48 CFR Chapter 15. The **Federal Register** Notice required under 5 CFR 1320.8(d), soliciting comments on this collection of information was published on February 25, 1997 (62 FR 8445). No comments were received.

*Burden Statement:* The annual public reporting and recordkeeping burden for this collection of information is estimated to average 680 hours per response. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

*Respondents/Affected Entities:* Municipalities with combined sewer overflow systems that have combined sewer overflows (CSOs).

*Estimated Number of Respondents:* 980.

*Frequency of Response:* One time for selected items and semi-annually for other items.

*Estimated Total Annual Hour Burden:* 622,777.

*Estimated Total Annualized Cost Burden:* $73,900.

Send comments on the Agency's need for this information, the accuracy of the provided burden estimates, and any suggested methods for minimizing respondent burden, including the use of automated collection techniques, to the following addresses. Please refer to EPA ICR No. 1680.02 and OMB Control No. 2040–0170 in any correspondence.

Ms. Sandy Farmer, U.S. Environmental Protection Agency, OPPE Regulatory Information Division (2137), 401 M Street, SW., Washington, DC 20460
and
Office of Information and Regulatory Affairs, Office of Management and Budget, Attention: Desk Officer for EPA, 725 17th Street, NW., Washington, DC 20503.

Dated: June 26, 1997.

**Joseph Retzer,**

*Director, Regulatory Information Division.*

[FR Doc. 97–17371 Filed 7–1–97; 8:45 am]

**BILLING CODE 6560–50–P**

## ENVIRONMENTAL PROTECTION AGENCY

**[FRL–5851–9]**

### Environmental Statistics Subcommittee of the National Advisory Council for Policy and Technology; Public Meeting

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Cancellation of notice of public meeting.

**SUMMARY:** This is a cancellation notice for the July 22, 1997, Environmental Statistics Subcommittee (of the Environmental Information, Economics and Technology Committee) of the National Advisory Council on Environmental Policy and Technology (NACEPT) meeting.

The Environmental Statistics Subcommittee was formed to provide key recommendations and strategic advice on the statistical products and activities necessary to enhance the Agency's knowledge about environmental statistics and trends, and to explore information gaps from the perspective of the users/products of these data products. The meeting was being held to discuss and offer critical advice on initiatives of the Office of Strategic Planning and Environmental Data.

**DATES:** The public meeting was to be held on July 22, 1997, from 9 a.m. to 5 p.m. The meeting was to be held at Loews L'Enfant Plaza Hotel, 480 L'Enfant Plaza, SW, 2nd Floor Renoir Conference Room, Washington, DC 20024. This meeting was open to the public.

**ADDRESSES:** Written comments should be sent to: N. Phillip Ross, Office of Strategic Planning and Environmental Data, U.S. Environmental Protection Agency, Mail Code 2161, 401 M Street, SW., Washington, DC 20460.

**FOR FURTHER INFORMATION CONTACT:** N. Phillip Ross, Designated Federal Official, Direct Line (202) 260–0250, General Line (202) 260–5244; FAX (202) 260–8550.

**N. Phillip Ross,**

*Designated Federal Official.*

[FR Doc. 97–17373 Filed 7–1–97; 8:45 am]

**BILLING CODE 6560–50–P**

## ENVIRONMENTAL PROTECTION AGENCY

**[PF–740; FRL–5722–9]**

### Notice of Filing and Withdrawal 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, as well as the withdrawal of a pesticide petition.

**DATES:** Comments, identified by the docket control number PF–740, must be received on or before August 1, 1997.

**ADDRESSES:** By mail submit written comments to: Public Response and Program Resources Branch, Field Operations Divison (7505C), 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 by following 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 |
|---|---|---|
| James Tompkins, (PM 25). | Rm. 237, CM #2, 703–305–7740; e-mail: Tompkins.James@epamail.epa.gov. | 1921 Jefferson Davis Hwy, Arlington, VA |
| Mary L. Waller, (PM 21) | Rm. 265, 703 308–9354; e-mail: waller.mary@epamail.epa.gov. | Do. |
| George LaRocca (PM 13). | Rm. 204, 703–305–5540, e-mail: LaRocca.george@epamail.epa.gov. | Do. |

**SUPPLEMENTARY INFORMATION:** EPA has received pesticide petitions as follows proposing the establishment, amendment and/or withdrawal of regulations for residues of certain pesticide chemicals in or on various raw 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 grantinig of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under document control number PF–740 (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''.

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 document control number (insert docket number) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

**Authority: 21 U.S.C. 346a.**

## List of Subjects

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

Dated: June 23, 1997.

**James Jones,**

*Acting Director, Registration Division, Office of Pesticide Programs.*

## Summaries of Petitions

Below summaries of the pesticide petitions are printed. The summaries of the petitions were prepared by the petitioners. 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. Bayer Corporation Withdrawal Of Pesticide Petition

*PP 6E3182*

On November 8, 1984 Bayer Corporation, P.O. Box 4913, Kansas City, MO 64120, filed an import petition on behalf of the Ministry of Agriculture, Fisheries and Forestry in Japan, requesting establishment of a permanent tolerance (0.1 ppm) for the insecticide prothiophos (Tokuthion) in/on Japanese sand pears being imported from Japan. On March 27, 1997 Bayer notified EPA that it requests that the petition be withdrawn without prejudice to future filing. The Agency has withdrawn the subject petition. (PM 13).

### 2. Merck Research Laboratories, Inc.

*PP 6F4628*

EPA has received pesticide petition 6F4628 from Merck Research Laboratories, Inc, P.O. Box 450, Hillsborough Road, Three Bridges, NJ 08887-0450, proposing pursuant to section 408 of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C section 346a (d), to amend 40 CFR part 180 by establishing tolerances for residues of the insecticide Emamectin Benzoate, 4′-epi-methylamino-4′-deoxyavermectin B1 benzoate [a mixture of a minimum of 90% 4′-epi-methylamino-4′-deoxyavermectin B1a and a maximum of 10% 4′-epi-methylamino-4′-deoxyavermectin B1b benzoate] and it degradates (with Merck research numbers in parentheses) 8,9-isomer of the B1a and of the B1b component of the parent insecticide (C-695,638); 4′-deoxy-4′-epi-amino-avermectin B1 (L-653,64); 4′-deoxy-4′-epi-(*N*-formyl-*N*-methyl)amino-avermectin B1 (L-660,599); and 4′-deoxy-4′-epi(*N*-formyl)amino-avermectin B1 (L-657,831) in or on the raw agricultural commodities cole crops vegetables (cabbage, broccoli, cauliflower and brussels sprouts) at 0.025 parts per million (ppm) and leafy vegetables (celery and head lettuce) at 0.025 ppm. The proposed analytical method is high performance liquid chromatography (HPLC).

*A. Residue Chemistry*

1. *Plant metabolism*. The metabolism of emamectin benzoate in plants has been studied in lettuce, cabbage, and sweet corn. The major portion of the residue is parent compound and its delta 8,9-photoisomer. Studies of the metabolism of emamectin in animals are not required because the commodities

that are the subject of the petition are not significant animal feed items.

2. *Analytical method*. Adequate analytical method (HPLC-fluorescence methods) are available for enforcement purposes.

3. *Magnitude of residues*. Eighteen field trials have been conducted: 10 on cabbage, 4 on broccoli, and 4 on cauliflower. These trials were conducted in the major U.S. growing areas for these crops. In samples taken after passage of the proposed interval between last treatment and harvest, the highest combined residue of emamectin benzoate and the degradates, which occurred in one cabbage sample, was 0.020 ppm (actually quantified) of the main component, an unquantifiable amount that could be almost as high as the 0.005 limit of quantification or as low as the 0.001 ppm limit of detection, and undetectable amounts of the other two components, for a total somewhere between 0.021 part per million (ppm) and 0.027 ppm (total of actually quantified residues plus maximum possible levels of detectable but nonquantifiable residues between 0.001 and 0.005 ppm). In all other samples taken the combined measurable and nonquantifiable residues were well below the 0.025 ppm level.

*B. Toxicological Profile*

The primary toxic effect seen in animal studies of emamectin benzoate is neurotoxicity. No-observed-effect-levels (NOELs) for this effect have been well-characterized in multiple studies. Emamectin benzoate has not been shown to be oncogenic or teratogenic in animal studies, it lacks mutagenic activity, and it is not selectively developmentally toxic. The petition refers to toxicity data that establish the following information about the toxicity of emamectin benzoate:

1. *Acute toxicity*. Acute oral $LD_{50}$: rat, 76–89 mg/kg; CD-1 mouse 107-120 mg/kg; CF-1 mouse, 22-31 mg/kg. Acute oral neurotoxicity: rat, No observed effect level (NOEL) = 5 mg/kg, Lowest observed effect level (LOEL) = 10 mg/kg. Acute dermal $LD_{50}$: rat and rabbit, >2,000 mg/kg. Dermal irritation: rabbit, not irritating to skin. Eye irritation: rabbit, severe eye irritant. Acute inhalation 4-hour $LC_{50}$: rat, 2.12-4.44 mg/l.

2. *Reproductive/developmental toxicity*. Developmental toxicity: rat, maternal NOEL = 2 mg/kg/day, developmental NOEL = 4 mg/kg/day, developmental LOEL = maternally toxic 8 mg/kg/day (HDT) for developmental delay; rabbit, maternal NOEL = 3 mg/kg/day, developmental NOEL = 6 mg/kg/day (maternally toxic HDT). Developmental neurotoxicity: rat, maternal NOEL = 3.6/2.5 mg/kg/day (HDT), developmental NOEL = 0.6 mg/kg/day, developmental LOEL = 3.6/2.5 mg/kg/day for signs of neurotoxicity in pups. Two-generation reproductive toxicity: rat, parental and reproductive NOEL = 0.6 mg/kg/day, parental LOEL = 3.6/1.8 mg/kg/day (for decreased weight gain and neuronal lesions); reproductive toxicity LOEL = 3.6/1.8 mg/kg/day (for decreased fecundity and signs of neurotoxicity in pups).

3. *Subchronic And chronic toxicity and oncogenicity*. With the single exception of the chronic rat study, LOELS for the following studies are based on clinical signs and/or histopathological evidence of neurotoxicity (described further below). Subchronic (90-day) toxicity: rat, NOEL = 0.5 mg/kg/day, LOEL = 2.5 mg/kg/day; CD-1 mouse, NOEL = 5.4 mg/kg/day (TWA), LOEL = 0.5 mg/kg/day; dog, NOEL = 0.25 mg/kg/day, LOEL = 0.5 mg/kg/day Subchronic (90-day) neurotoxicity; rat, NOEL = 1 mg/kg/day, LOEL = 5 mg/kg/day. Chronic (105-week) toxicity/oncogenicity, rat: NOEL = 0.25 mg/kg/day, LOEL = 1 mg/kg/day (based on decreased body weight and clinical chemistry changes), neurotoxicity NOEL = 1 mg/kg/day, not oncogenic. Chronic (79-week) toxicity/oncogenicity, CD-1 mouse: NOEL = 2.5 mg/kg/day, LOEL = 5 mg/kg (males), 7.5 mg/kg/day (females), not oncogenic. Chronic (53-week) toxicity, dog: NOEL = 0.25 mg/kg/day, LOEL= 0.5 mg/kg./day.

Exposure to sufficiently high doses of emamectin benzoate may be associated with clinical signs of central nervous system (CNS) toxicity and microscopic evidence of CNS/peripheral nervous system (PNS) damage. Neurotoxicity has generally been the most sensitive endpoint for toxicity in oral animal studies with emamectin benzoate. Clinical signs of CNS toxicity resulting from emamectin benzoate exposure include tremors, mydriasis, and changes in motor activity (e.g., lethargy, hyperactivity, and/or ataxia). Nervous system lesions (generally focal and of a low degree of severity) have been observed microscopically in white and gray matter in the brain stem, spinal cord, and peripheral nerves. Sporadic lesions of the optic nerve and/or retina have also been seen at higher dose levels. NOELs have been determined in all studies. The lowest toxic dose level of emamectin benzoate for CNS/PNS lesions (0.5 mg/kg/day) was identified in a 1-year study in dogs (NOEL of 0.25 mg/kg/day).

The CF-l mouse is uniquely sensitive to emamectin benzoate-induced neurotoxicity. Studies have shown that a significant fraction of the members of this strain inherit an inability to produce a P-glycoprotein one that most strains and species do produce that functions to resist the entrance of avermectin-type compounds into the central nervous system. P-glycoprotein is also present in the gut of most species and limits absorption of avermectin-type compounds following oral exposure. In a 16-day feeding study in the CF-1 mouse, tremors were seen at 0.3 mg/kg/day of emamectin benzoate with a NOEL of 0.1 mg/kg/day. No histopathologic evidence of neurotoxicity was seen in this study up to the highest dose tested (0.9 mg/kg/day).

Emamectin benzoate photodegrades on plants and in soil. The major photodegradates that are not animal metabolites were tested in a 15-day neurotoxicity study in CF-1 mice. Only one photodegradate showed neurotoxicity (Merck research number L-660,599, the *N*-formyl-*N*-methyl degradate). Its NOEL was found to be 0.075 mg/kg/day, slightly lower than the value for the parent compound in the same kind of study, and both clinical signs and peripheral nerve lesions were observed at levels of 0.1 mg/kg/day and higher.

4. *Mutagenicity*. Emamectin benzoate was tested in a battery of *in vitro* and *in vivo* mutagenicity assays and showed no evidence of mutagenic potential.. The photodegradates have also been tested in the Ames bacterial mutagenicity assay and show no mutagenic potential in this test system.

5. *Endpoint selection*. Merck is proposing that the 0.075 mg/kg/day NOEL from the CF-1 mouse 15-day neurotoxicity study with the L-660,599 photodegradate be used as the basis for acute dietary risk assessment. For evaluation of chronic dietary risks, Merck is proposing that the one-year dog chronic study NOEL of 0.25 mg/kg/day be used. The dog appears to be the most sensitive species to long-term exposure to emamectin benzoate. Accordingly, chronic exposure is compared against a RfD of 0.0025 mg/kg/day, based on the dog study results and an uncertainty factor of 100.

*C. Aggregate Exposure*

1. *Dietary exposure*. Except for a temporary tolerance associated with an experimental use permit, no tolerances for residues of emamectin benzoate have been established. Merck projects that by the year 2001, emamectin benzoate will be used on approximately 17% of the acreage for the six crops covered by this petition. Chronic dietary exposure analyses were conducted for the overall

U.S. population and 26 population subgroups. Assuming 100% of the crop treated, chronic exposure for the overall U.S. population was estimated to be 0.000003 mg/kg BW/day, and for the most highly exposed subgroup, nursing females 13 years and older, 0.000004 mg/kg BW/day.

2. *Nondietary exposure.* No products containing emamectin benzoate have yet been registered under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) for any food or nonfood use. The environmental fate of emamectin has been evaluated, and the compound is not expected to contaminate groundwater or surface water to any measurable extent. No significant nondietary, nonoccupational exposure is anticipated.

*D. Cumulative Effects*

Emamectin is a member of the avermectin family of natural and synthetic compounds that includes the Merck products abamectin (a naturally occurring compound that is the active ingredient of several insecticides registered under FIFRA) and ivermectin (a human and animal drug made from abamectin). Emamectin is made from abamectin but is less similar to abamectin than is ivermectin. Other companies produce certain other drugs that are members of the avermectin family. Some of the effects seen in toxicity studies of abamectin and ivermectin are similar to some of the effects seen in toxicity studies of emamectin. See the discussion of abamectin and ivermectin in 61 FR 65043 (Dec. 10, 1996). Merck is not aware of any information indicating what, if any, cumulative effect would result from exposure to two or more of these compounds.

*E. Safety Determination*

1. *U.S. population chronic risk.* Chronic exposures were analyzed with reference to the chronic effects referenced dose (RfD) NOEL of 0.0025 mg/kg/day. Assuming 100% of the crop treated, the chronic exposure estimate was 0.1% of the RfD for the overall U.S. population, and 0.2% of the RfD for the most highly exposed subgroup, nursing females 13 years and older. If 25% crop treatrment is assumed, exposure estimates were less than 0.1% of the RfD for all population groups.

2. *U.S. population acute risk.* Acute dietary exposures were analyses for the overall U.S. population, and the population subgroups (1) women 13 years and older, (2) infants, and (3) children. In addition, Tier 2 and Tier 3 acute analyses were conducted assessing acute exposures against the 0.075 mg/kg/day NOEL. These analyses showed that the margins of exposure (MOEs) calculated from the proposed uses of emamectin benzoate are acceptable whether using a highly conservative approach (Tier 2) or a more realistic (Tier 3) methodology. In the Tier 2 analysis, MOEs were well over 1,000 up to the 95th percentile of exposure for all population groups. In the Tier 3 analysis and assuming 100% of the crop treated, MOEs up to the 99th percentile of exposure were greater than 1,000. Assuming 25% of the crop treated, MOEs were greater than 1,000 up to the 99.9th percentile of exposure. Results of both the chronic and acute dietary exposure analyses clearly demonstrate a reasonable certainty that no harm will result from the use of emamectin benzoate.

3. *Infants and children.* It is Merck's position that the administration of emamectin benzoate has not been shown to cause developmental or reproductive effects at dose levels below those that are maternally toxic. Even if it were decided to use the 0.6 mg/kg NOEL from the rat developmental neurotoxicity study as an endpoint from which to calculate an RfD, the resulting RfD would not yield a different regulatory outcome unless a very high additional uncertainty factor were also employed. Use of such an extra uncertainty factor is not justified for several reasons. Emamectin benzoate is not a teratogen. In developmental toxicity testing, the compound caused no developmental effects in rabbits; in rats, it caused no malformations, and caused skeletal effects typical of developmental delay only at severely maternally toxic doses. Likewise, no reproductive toxicity or toxicity to pups was seen in the two-generation reproductive toxicity study except at parentally toxic doses. In the developmental neurotoxicity study, tremors, hind-leg splay, and behavioral effects were seen in pups at a dose level (3.6/2.5 mg/kg/day) at which no maternal clinical signs were noted. However, the dams in the study were discarded after the lactation period without gross necropsy or microscopic examination. In studies in which rats dosed at similar levels were examined microscopically, effects (central and peripheral neural lesions) were seen.

The clinical signs of avermectin-family neurotoxicity seen in neonatal rats are unlikely to be useful predictors of human risk. Young rats are considerably more sensitive to avermectin-type compounds than either adult rats or humans and other primates. (In neonatal rats, unlike humans, the P-glycoprotein levels are only a small fraction of the levels seen in adult rats.) Moreover, data from clinical experience with ivermectin, a related human drug, and studies on ivermectin and abamectin, a related pesticide, demonstrate that both the neonatal rat and the CF-1 mouse overpredict the toxicity of the avermectin-type compounds to humans and to non-human primates.

*F. International Tolerances*

No Codex maximum residue levels (MRLs) have been established for residues of emamectin benzoate. (PM 13)

**3. Novartis Crop Protection Inc.**

*PP 0E3875*

EPA has received a pesticide petition (0E3875) from Novartis Crop Protection Inc., PO Box 18300, Greensboro, NC 27419. The petition proposes, to amend 40 CFR part 180, by establishing a permanent import tolerance for the residues of the fungicide cyproconazole, (2*RS*,3*RS*)-2-(4-chlorophenyl)-3-cyclopropyl-1-1(1*H*-1,2,4-triazole-1-yl)butan-2-ol, (CAS #94361–06–5; PC Code 128993) in or on the raw agricultural commodity coffee beans at 0.1 part per million (ppm). The time-limited tolerance of 0.1 ppm in or on coffee beans established in the **Federal Register** of September 27, 1995 (60 FR 49795) will expire July 1, 1997.

*A. Chemical Uses*

Cyproconazole, (2*RS*,3*RS*)-2-(4-chlorophenyl)-3-cyclopropyl-1-(1*H*-1, 2, 4-triazole-1-yl)butan-2-ol, is a broad spectrum fungicide that has been classified as an ergosterol-biosynthesis inhibitor. It is used to control a variety of fungi, including coffee rust, in several coffee producing countries. Rates range from a preventative treatment of 20 g ai/ha to a maximum curative treatment of 50 g ai/ha with a 30 day pre-harvest interval (PHI) and annual maximum of 100 g ai/ha.

1. *Cyproconazole safety.* A battery of acute toxicity studies was conducted placing technical cyproconazole in Toxicity Category III and IV.

i. *90-day rat study.* A NOEL for this study was not attained, but the NOEL is estimated to be less than 1.0 mg/kg.

ii. *13-week feeding study in dogs.* NOEL of 20 ppm (0.8 mg/kg/day) and an LEL of 100 ppm (4 mg/kg/day) based on included slack muscle tone, depressed body weight gain, and decreases in bilirubin, total cholesterol, HDL-cholesterol, triglycerides, total protein, and albumin. There were increases in platelet counts, alkaline phosphatase, gamma glutamyl transferase, absolute

and relative liver weights, relative kidney weights, and relative brain weights. Liver toxicity was indicated by hepatomegaly.

iii. *21-day dermal study.* NOEL was 250 mg/kg and the LEL was 1,250 mg/kg. Effects included depressed body weight gain and food consumption and increased levels of AST, creatinine, and cholesterol.

iv. *1-year dog study.* NOEL of 30 ppm (1.0 mg/kg/day) and an LEL of 100 ppm (3.2 mg/kg/day) based on laminal eosinophilic intrahepatocytic bodies observed in all males and two females at the high dose, and in one male at the mid-level dose.

v. *A mouse carcinogenicity study.* NOEL for systemic toxicity of 15 ppm (1.8 mg/kg for males and 2.6 mg/kg for females). The LEL was 100 ppm (13.2 mg/kg for males and 17.7 mg/kg for females) based on a significantly increased incidence of hepatic single cell necrosis and diffuse hepatocytic hypertrophy at the two highest levels.

vi. *A rat chronic/carcinogenicity study.* The NOEL for systemic toxicity was 50 ppm. The LEL was 350 ppm based on slightly decreased body weights in the high-dose females and increased incidence of fatty infiltration of the liver in the high-dose males.

vii. *A rat developmental toxicity study.* NOEL for maternal toxicity was 6 mg/kg, and the LEL was 12 mg/kg based on decreased body weight gain during dosing. The NOEL for developmental toxicity was 6 mg/kg. The LEL was 12 mg/kg based on the increased incidence of supernumerary ribs.

viii. *A chinchilla rabbit developmental toxicity study.* NOEL for maternal toxicity was 10 mg/kg (equivocal). The LEL was 50 mg/kg based on decreased body weight gain during dosing. Developmental effects were also evaluated. Hydrocephalus internus was observed in 1 fetus at each treatment level. Therefore, the NOEL for developmental toxicity was set at less than 2 mg/kg, and the LEL was 2 mg/kg.

ix. *A New Zealand white rabbit developmental toxicity study.* NOEL for maternal toxicity was 10 mg/kg, and the LEL was 50 mg/kg based on decreased body weight gain. There was also evidence of developmental toxicity. The NOEL for developmental toxicity was 2 mg/kg, and the LEL was 10 mg/kg based on the increased incidence of malformed fetuses and litters with malformed fetuses.

x. *A rat two-generation reproduction study.* systemic NOEL for parental toxicity was set at 20 ppm (1.7 mg/kg) based on liver effects at 10.6 mg/kg/day. For reproductive toxicity, the NOEL was set at 4 ppm (0.4 mg/kg) and the LEL at 20 ppm (1.7 mg/kg) based on increased gestation length in the F0 dams and decreased F1 litter sizes.

xi. *Several mutagenicity studies.* Mutagenicity potential of cyproconazole was tested in several studies considered acceptable by the Agency. Since the results of two chromosomal aberration assays indicated the cyproconazole is clastogenic, additional mutagenicity data were requested to address an identified heritable risk concern. For the potential to induce chromosome aberrations in CHO cells, cyproconazole was positive under non-activated and activated conditions, thus supporting the evidence that cyproconazole is clastogenic in this test system. However, cyproconazole was negative in Salmonella, mouse micronucleus, and SHE/cell transformation assays. A dominant-lethal assay in rats was submitted and was negative. Based on this evidence, the concern for a possible heritable effect was not pursued.

xii. *Metabolism/pharmacokinetics studies.* Cyproconazole was shown to be extensively metabolized in the rat. Unchanged cyproconazole and 13 metabolites were isolated and identified, and 35 metabolites were detected in the excreta. Excretion was relatively rapid with the majority of the radioactivity appearing in the feces as a result of biliary elimination. Residues were found in renal fat, adrenals, kidney and liver, although no significant tissue radioactivity was observed at 168 hours post-dose.

2. *Threshold effects.*—i. *Chronic effects.* Based on available chronic toxicity data, EPA has set the reference dose (RfD) used in the dietary exposure analysis at 0.01 mg/kg bwt/day. This RfD is based on a NOEL of 30.0 ppm (1.00 mg/kg bwt/day) from a 1-year dog feeding study and an uncertainty factor of 100 to account for interspecies extrapolation and intraspecies variability.

ii. *Acute effects.* The risk from acute dietary exposure to cyproconazole is considered by Novartis to be very low. The lowest NOEL in a short term exposure scenario, identified as 2 mg/kg in the rabbit teratology study, is 2-fold higher than the chronic NOEL (see above). Since chronic exposure assessment (see below), based on some worst-case assumptions, resulted in margins of exposure in the thousands for even the most impacted population subgroup, Novartis believes that the margin of exposure for acute exposure would be much higher than one hundred for any population groups; margins of exposure of 100 or more are considered satisfactory by the Agency.

3. *Non-threshold effects.* The HED Carcinogenicity Peer Review Committee has classified cyproconazole as a Group ''B2'' carcinogen (probable human carcinogen) based on findings of liver tumors in both sexes of mice administered adequate doses of cyproconazole, its possible clastogenic activity, tumors in rats and mice administered structurally related analogues and the lack of an adequate rat carcinogenicity study. The committee assigned cyproconazole a risk characterization value, Q1*, of 3.0 × 10–1 (mg/kg/day)-1 derived from liver tumor data obtained in male mice.

*B. Aggregate Exposure*

The anticipated residue contributions (ARC) as percentages of the RfD are <0.1% for the general population and all sub-populations and geographic regions. The chronic dietary exposure analysis for cyproconazole is calculated using anticipated residues for coffee and 100% treatment of all crops. This estimate is not a worst-case estimate of dietary exposure but still exaggerates exposure. Based on this calculation, Novartis believes the chronic dietary risk from the recommended use is far below the level which would trigger a concern.

Other potential sources for exposure are drinking water and non-occupational exposure. No cyproconazole-based products are labeled for residential use. Non-occupational exposure for cyproconazole has not been estimated since the current registrations for cyproconazole-based products are limited to commercial and agricultural turf treatment. Field studies have demonstrated that cyproconazole does not leach to groundwater or accumulate in the soil. The average half life of cyproconazole in field dissipation studies was <50 days. The field characteristics of cyproconazole, combined with its use pattern, make surface water contamination unlikely. Thus, Novartis believes the potential for non-occupational and drinking water exposure to the general population is insignificant.

*C. Safety Determination*

1. *U.S. population.* All non-occupational exposure of cyproconazole in the U.S. is due to its use in the production of imported coffee beans. The anticipated residue contribution (ARC) is 0.000001 mg/kg/day for the general population and, 0.000002 mg/kg/day for females, 20 years old and older. Novartis has calculated that the ARC will consume 0.01% and 0.02% of the RfD for the general population and

females 20 years old or older, respectively. Lifetime carcinogenic risk for dietary exposure based on quantitative risk assessment and a $Q_1$* of $3.0 \times 10^{-1}$ (mg/kg/day)-1, is $3.15 \times 10^{-7}$. EPA generally has no concern for exposures below 100 percent of the RfD or lifetime carcinogenic risks less than $1 \times 10^{-6}$. Therefore, Novartis concludes that there is a reasonable certainty that no harm will result from aggregate exposure to cyproconazole residues via the use on coffee beans.

The consideration of a common mechanism of toxicity is not appropriate at this time because Novartis and EPA do not have information to indicate that toxic effects produced by cyproconazole would be cumulative with those of any other chemical compounds.

2. *Infants and children.* For dietary risk assessments, no exposure is apportioned to infants and children because they do not normally consume coffee. There is also no non-occupational exposure to infants and children. Based on the completeness and reliability of the toxicity data and the practical non-exposure to cyproconazole, Novartis concludes that there is a reasonable certainty that no harm will result to infants and children from the aggregate exposure of residues of cyproconazole including all anticipated dietary exposure and all other non-occupational exposures.

*D. Estrogenic Effects*

Cyproconazole does not belong to a class of chemicals known for having adverse effects on the endocrine system. No estrogenic effects have been observed in the various short and long term studies conducted with various mammalian species.

*E. Chemical Residue*

The nature of the residue in coffee is fully understood. A metabolism study in coffee, using triazole-labeled cyproconazole, was submitted and was acceptable. Cyproconazole per se was the primary component of the residue. A metabolism study in wheat was conducted to determine the fate of the phenyl portion of cyproconazole in plants. Results of the study have been submitted and the Agency found that residues from the wheat metabolism study were not significantly different from the coffee metabolism study.

Adequate enforcement methodology has been submitted to the EPA and has passed a method validation trial by EPA's analytical laboratories. Additional data has been submitted to demonstrate that residues of several other pesticides registered for use on coffee do not interfere with the method. Prior to publication in the Pesticide Analytical Manual, Vol. II, the enforcement methodology is being made available in the interim to anyone who is interested in pesticide enforcement when requested from: Calvin Furlow, Public Response and Program Resource Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 1130A, CM#2, 1921 Jefferson Davis Hwy., Arlington, VA (703) 305–5937.

*F. Environmental Fate*

No domestic use of cyproconazole is associated with the established tolerance in coffee.

*G. International Tolerances*

No international tolerances have been established under CODEX for cyproconazole. (PM 21)

**4. ZENECA Ag Products**

*PP 6F4790*

EPA has received a pesticide petition (PP 6F4790) from ZENECA Ag Products, 1800 Concord Pike, P.O. Box 15458, Wilmington, DE 19850–5458, proposing to amend 40 CFR part 180 by establishing a tolerance for residues of tralkoxydim, 2-cyclohexen-1-one, 2[1-(ethoxyimino) propyl]-3-hydroxy-5-(2,4,6-trimethylphenyl)-(9CI) in or on the food commodities barley grain, barley straw, barley hay, wheat grain, wheat forage, wheat straw, and wheat hay at 0.1 parts per million (ppm). The proposed analytical method is High Pressure Liquid Chromatography with ultra-violet detection (HPLC-UV).

*A. Residue Chemistry*

1. *Plant metabolism.* Wheat Plant metabolism was evaluated in wheat. 14C-Tralkoxydim, labeled in the equivalent C4/C6 positions of the cyclohexenone ring, was applied as a foliar spray to field-grown spring wheat. A single application was made at a rate of 0.31 lb ai/acre at Zadok's growth stage 31. A representative forage sample was harvested 22 days post-application. The remainder of the crop was harvested at maturity, 96 days post-application, then separated into straw and grain prior to analysis.

The total radioactive residues (TRR) in forage, straw and grain were 0.71, 1.29 and 0.013 mg/kg tralkoxydim equivalents, respectively. No residues of parent were detected and at least ten individual components were initially observed, demonstrating extensive metabolism of tralkoxydim. Characterization of the total radioactive residue in grain by extraction indicates that no single component exceeds 0.01 mg/kg. Also, in both forage and straw, the same complex metabolic profile was evident. Characterization showed that none of the metabolites exceeded 3.6% TRR (0.05 mg/kg) in any of the fractions examined.

2. *Analytical method.* The method of analysis uses High Pressure Liquid Chromatography. It is method PRAM 99A and it has been validated using independent laboratory confirmatory trials as described in US EPA PR Notice 88–5. The method is for extraction and quantification of tralkoxydim residues in wheat and barley crops. Grain, straw, or forage is extracted into acetonitrile, filtered, and re-extracted into dichloromethane. The organic layer is used for analysis. The limit of detection of the analytical method is 0.02 ppm, while the limit of quantification is 0.1 ppm.

3. *Magnitude of residues.* ZENECA requests registration of 2 concentrations of tralkoxydim, 80% and 40% for ACHIEVE 80DG and ACHIEVE 40DG, respectively. These products use the same rate of application and demonstrate that there are no detectable residues on wheat and barley crops when either product is used according to the label directions.

*Wheat: ACHIEVE 80DG containing 80% tralkoxydim.* Residue data are available for tralkoxydim applied postemergence on wheat at the maximum label rate of 0.25 lb ai/A. Application was made from full tillering to first detectable node growth stage. In 1995, a total of 20 magnitude of the residue trials were conducted on spring wheat. There were no detectable residues (<0.02 ppm LOD) on wheat grain or straw in any of the trials at the pre-harvest interval of 60 days. There were no detectable residues on hay at the pre-harvest interval of 45 days. There were no detectable residues on immature forage at the pre-harvest interval of 30 days.

Two (2) winter wheat trials were conducted in 1995 to determine forage residues of tralkoxydim in winter wheat, using ACHIEVE DG, 80% concentration (ACHIEVE 80DG). The product was applied at the maximum label rate at growth stages from advanced tillering to full tillering. The winter wheat forage data showed no detectable residues at either 16 or 18 days after treatment. These results fall well within the proposed forage pre-harvest interval of 30 days.

*ACHIEVE 40DG containing 40% tralkoxydim.* There were 3 magnitude of the residue trials conducted on spring wheat in 1994 and one trial was

conducted in 1993. In addition, 6 trials were conducted in Canada during 1986 and 1987. (Note: The Canadian trials were conducted using a 50% concentration of tralkoxydim at a higher use rate of 0.3 - 0.6 lb ai/A). There were no detectable residues (<0.02 ppm LOD) on wheat grain or straw in any of the trials at the pre-harvest interval of 60 days. There were no detectable residues on hay at the pre-harvest interval of 45 days. There were no detectable residues on immature forage at the pre-harvest interval of 30 days. Despite having no detectable residues of tralkoxydim at 0.02 ppm, it is proposed that the tolerance level be based on the limit of quantification (LOQ) of the tolerance enforcement method, which has been validated to 0.1 ppm for tralkoxydim. The proposed tolerance of 0.1 ppm for wheat grain, forage, straw and hay is five (5) times greater than any residue that would result from the application of ACHIEVE DG arising from the proposed use pattern.

*Wheat Products (processing).* The wheat processing study demonstrated that there are no detectable residues (<0.02 ppm) in the bran, flour, middlings, shorts, and germ. Therefore, no food or feed additive tolerances are required for processed wheat commodities.

*Barley: ACHIEVE 80DG containing 80% tralkoxydim.* A total of 12 magnitude of the residue trials were conducted in 1995 on barley crops for tralkoxydim applied postemergence at the maximum label rate of 0.25 lb ai/A. The product was applied at full tillering to first detectable node growth stage. There were no detectable residues (<0.02 ppm) on barley grain or straw at the pre-harvest interval of 60 days. There were no detectable residues in hay at the pre-harvest interval of 45 days.

*ACHIEVE 40DG containing 40% tralkoxydim.* In 1994, 3 magnitude of the residue trials were conducted on barley using ACHIEVE DG, 40% concentration (ACHIEVE 40DG). In addition, 6 magnitude of the residue trails that were conducted in Canada during 1986 and 1987. (Note: The Canadian trials were conducted using a 50% concentration of tralkoxydim at a higher use rate of 0.3 - 0.6 lb ai/A). There were no detectable residues (<0.02 ppm) on barley grain or straw at the pre-harvest interval of 60 days. There were no detectable residues in hay at the pre-harvest interval of 45 days.

Despite having no detectable residues of tralkoxydim at 0.02 ppm, it is proposed that the tolerance level be based on the limit of quantification (LOQ) of the tolerance enforcement method, which has been validated to 0.1 ppm for tralkoxydim. The proposed tolerance of 0.1 ppm for barley grain, hay and straw is five (5) times greater than any residue that would result from the application of ACHIEVE DG arising from the proposed use pattern.

*Barley Products (processing).* The barley processing study demonstrated that there are no detectable residues (<0.02 ppm) in the pearled barley, flour and bran. Therefore, no food or feed additive tolerances are required.

*Animal Products.* Based on the results of the poultry and ruminant metabolism studies, the extensive metabolism and rapid excretion of either tralkoxydim or any of its metabolites, and the poultry and ruminant consumption of commodities used in animal feed, there are no expected residues of tralkoxydim in meat, milk, or eggs.

*B. Toxicological Profile*

1. *Acute toxicity.* Tralkoxydim technical results of the acute toxicity testing: acute oral in the rat LD50 > 934 mg/kg, acute dermal in the rat LD50 > 2,000 mg/kg, acute inhalation in the rat LD50 > 3.5 mg/L, eye irritation in the rabbit showed mild irritancy, skin irritation in the rabbit showed a slight irritancy. Tralkoxydim is not a skin sensitizer.

2. *Genotoxicity.*

| Assay | Type | Result |
|---|---|---|
| *In vitro* | Ames | negative |
| | Mouse lymphoma | negative |
| | Human lymphocyte cytogenetics | negative |
| *In vivo* | Mouse micronucleus | negative |
| | UDS | negative |

3. *Reproductive and developmental toxicity.* (Reproductive toxicity) Tralkoxydim showed no evidence of reproductive toxicity to rats. Tralkoxydim was dosed to rats at levels of 2.5 mg/kg/day (50 ppm), 10 mg/kg/day (200 ppm) and 50 mg/kg/day (1,000 ppm) in a 3 generation reproductive toxicity study.

| Study Type Reproductive Toxicity | NOEL | Effect Description |
|---|---|---|
| Rat (diet) 3 generation. | NOEL = 10 mg/kg/day (200 ppm). | LEL is 1,000 ppm based on reduced litter weights and weight gain in pups and bodyweight gain effects, food consumption and reduced liver weights in adults |

(Developmental toxicity) Tralkoxydim caused no clear dose related developmental effects in the rabbit. At a dose of 30 mg/kg/day, tralkoxydim caused some developmental effects in the rat manifested by skeletal defects including single misshapen centra. The NOEL for developmental toxicity was established at 3 mg/kg/day.

| Study Type Developmental Toxicity | NOEL/LEL | Effect Description |
|---|---|---|
| Rabbit (by gavage). | NOEL = 2.5 mg/kg/day fetotoxicity LEL = 20 mg/kg/day NOEL = 20 mg/kg/day maternal. | No clear dose-related developmental effects. LEL effect, increased partially ossified 2nd lumbar transverse process. |
| Rat (by gavage) | NOEL = 3 mg/kg/day fetotoxicity and developmental LEL = 30 mg/kg/day NOEL = 30 mg/kg/day maternal. | LEL for maternal toxicity is 300 mg/kg/day maternal death and overt toxicity. Developmental LEL is 30 mg/kg/day, skeletal defects includes single misshapen centra. |

| Study Type Developmental Toxicity | NOEL/LEL | Effect Description |
|---|---|---|
| Rat (by gavage) | NOEL = 3 mg/kg/day LEL = 200 mg/kg/day maternal, fetotoxicity and developmental. | LEL for fetotoxicity effect, increased post-implantation loss. Developmental effect fused or misshapen centra. Maternal LEL is based on moralities & overt signs of toxicity. |

4. *Subchronic toxicity*. Tralkoxydim is of low subchronic toxicity in 21-day dermal testing.

5. *Chronic toxicity*. Tralkoxydim is not a carcinogen in the rat. The dose levels used in the 2 year combined chronic/oncogenicity study on rats were as follows.

| Tralkoxydim in Diet (ppm) | Male rat (mg/kg/day) | Female rat (mg/kg/day) |
|---|---|---|
| 50 | 2.3 | 3.0 |
| 500 | 23.1 | 30.1 |
| 2,500 | 117.9 | 162.8 |

Tralkoxydim administration was associated with an increase in the incidence of benign Leydig cell tumors in the male rat at the top-dose of 2,500 ppm, only. This increase represented an exacerbation of a naturally occurring tumor type in the male rat and was considered to be the result of a physiological response to tralkoxydim administration. There was no evidence of a treatment-related effect or incidence of any other tumor type (malignant or benign) in male or female rats at any dose.

*Oncogenicity - Hamster*. Tralkoxydim is not an oncogen in the hamster. The dose levels used in the combined chronic toxicity/oncogenicity study on hamsters were as shown in the table below.

| Tralkoxydim in Diet (ppm) | Male hamster (mg/kg/day) | Female hamster (mg/kg/day) |
|---|---|---|
| 250 | 14.9 | 14.8 |
| 2,500 | 153.0 | 148.3 |
| 7,500 | 438.6 | 427.9 |

There was no increased tumor incidence or early onset of tumors in hamsters receiving up to 7,500 ppm tralkoxydim in the diet. The NOEL was established at 250 ppm, equivalent to 15 mg/kg bodyweight/day.

| Study Type Oncogenicity | NOEL/LEL | Effect Description |
|---|---|---|
| Hamster (diet) | NOEL = 250 ppm (15 mg/kg/day) LEL = 2,500 ppm | LEL effect: decreased lymphocyte numbers (in males only) and increased liver lipofuscin pigment at 2,500 and 7,500 ppm. |

The hamster instead of the mouse was selected as the second test species for oncogenicity testing because laboratory mice developed hepatic porphyria at low doses of tralkoxydim. Extensive mechanism data in support of the mouse specific porphyria has been provided. The results of these studies led ZENECA to the conclusion that the mouse was not an appropriate second test species for chronic toxicity/oncogenicity testing of tralkoxydim since the level of sensitivity in the mouse precluded the administration of a dose sufficient to determine chronic/oncogenicity effects in a lifetime feeding study.

*One-Year Feeding Study - Dog*. Tralkoxydim was administered to groups of 4 beagle dogs at dose levels of 0, 0.5, 5.0, and 50 mg/kg/day, as a daily oral dose in the food. At 50 mg/kg/day there was hepatotoxicity (marked increase in liver weight) and an effect on the adrenal gland (increase in weight and cortical vacuolation). At a dose of 5 mg/kg/day, the following changes were not considered toxicologically significant: a slight increase in adrenal weight relative to body weight in males, and a slight adaptive effect in the liver of one male dog considered to be abnormally susceptible. These changes are of no toxicological significance.

The resulting NOEL from this study is 0.5 mg/kg/day. Based on the EPA review of tralkoxydim toxicity data, the NOEL from this study was recommended for use in establishing a provisional RfD.

The resulting RfD, with an uncertainty factor of 100 is 0.005 mg/kg/day.

6. *Animal metabolism*. Tralkoxydim is well absorbed and completely metabolized in the rat. Excretion is rapid and there is no accumulation of tralkoxydim or metabolites. There are no significant plant metabolites that are not animal metabolites.

7. *Metabolite toxicology*. Toxicity testing results for the tralkoxydim parent compound is indicative of any metabolites, either in the plant or animal.

*C. Aggregate Exposure*

1. *Dietary exposure (Food)*. Tralkoxydim is to be used on wheat and barley crops, only. For the purposes of assessing the potential dietary exposure, ZENECA estimated aggregate exposure based on the Theoretical Maximum Residue Contribution (TMRC) from the tolerances of tralkoxydim on wheat at 0.1 ppm and barley at 0.1 ppm. This is a worst case estimate of aggregate exposure and assumes 100% of the wheat and barley crops in the United States will have residues of tralkoxydim at the 0.1 ppm. Dietary exposure to residues of tralkoxydim in or on food

will be limited to residues on wheat and barley, and food derived from wheat and barley. Based on animal metabolism data and because there are no residues on the crops at time of harvest or at grazing intervals, we have concluded that there is reasonable expectation that no measurable residues of tralkoxydim will occur in meat, milk, poultry, or eggs from this use. Since tralkoxydim is a new herbicide, there are no other established U.S. tolerances for tralkoxydim.

Due to no detectable residues in grain at harvest, even after processing, the dietary risk assessment has been conducted on the basis of the limit of quantification of 0.1 mg/kg. This is significantly above (5×) the limit of detection of tralkoxydim residues of 0.02 mg/kg determined by ZENECA's analytical methods used in the magnitude of residue studies. However, even using a tolerance level of 0.1 mg/kg (limit of quantification) the chronic assessment for tralkoxydim indicates less than 10% of the RfD is consumed, for any given subpopulation, even assuming 100% market share. Based on a review of available toxicity data for tralkoxydim, there are no toxicological endpoints of concern for acute dietary risk.

Agricultural use of tralkoxydim on wheat and barley, therefore, does not represent an acute or chronic risk to the U.S. population, infants, children, or any other of the 23 subpopulations evaluated in this assessment.

2. *Drinking water*. Based on the available studies, exposures are not anticipated to residues of tralkoxydim in drinking water. Tralkoxydim does not leach. It is unlikely that tralkoxydim would be in drinking water. Tralkoxydim is unlikely to enter surface water bodies to any significant degree except by direct accidental over-spray. Should this arise, tralkoxydim will be readily degraded by one or more of a number of contributory processes; studies have shown that degradation in flooded anaerobic soil occurs with a half-life of approximately 25 days, aqueous hydrolysis (pH 5) with a half-life of less than 7 days and aqueous photolysis also with a half-life of less than 7 days. All these processes will ensure that any tralkoxydim entering surface water bodies will be short-lived and tralkoxydim will not result in any significant contamination of potential drinking water sources. Therefore, it is not appropriate to assess aggregate exposure from drinking water.

3. *Non-dietary exposure*. Since tralkoxydim is not registered for residential or turf uses, and does not represent a groundwater contamination concern, exposures from other than dietary or occupational sources are extremely unlikely.

*D. Cumulative Effects*

Tralkoxydim is a new class of chemistry for herbicides used on wheat and barley. Although tralkoxydim is in the chemical class of compounds called cyclohexanediones, it is the only herbicide in this class to be used on wheat and barley crops. No evidence or information exists to suggest that the toxic effects produced by tralkoxydim would be cumulative with those of any other chemical compound.

*E. Safety Determination*

1. *U.S. population*. Using the conservative assumptions described above, based on the completeness and reliability of the toxicity data, the aggregate exposure to tralkoxydim will utilize less than 4% of the RfD for the U.S. Population. EPA generally has no concern for exposures below 100 percent of the RfD. There is reasonable certainty that no harm will result from aggregate exposure to residues of tralkoxydim, including all anticipated dietary exposure.

2. *Infants and children*. In assessing the potential for additional sensitivity for infants and children to residues of tralkoxydim, the three-generation reproductive study in rats and the developmental toxicity studies in the rat and rabbit were considered. Tralkoxydim showed no evidence of reproductive toxicity. Tralkoxydim caused no developmental toxicity in the rabbit. At a dose of 30 mg/kg/day, tralkoxydim caused some developmental effects in the rat manifested by skeletal defects including single fused or misshapen centra. The NOEL for developmental toxicity was established at 3 mg/kg/day.

Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete. Further, for the chemical tralkoxydim, the NOEL at 0.5 mg/kg/day from the dog feeding study which was used to calculate the RfD, is already lower than the NOEL from the developmental study in rats by a factor of approximately 10-fold. In addition, residue field trials have shown that there are no detectable residues of tralkoxydim on wheat and barley, indicating negligible exposure potential. Therefore, an additional uncertainty factor is not warranted and the RfD at 0.005 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

The percentage of the RfD that will be utilized by aggregate exposure to tolerance level residues of tralkoxydim are: 2% for nursing infants, 6% for children 1–6 years, and 5% for children 7–12 years. Therefore, there is reasonable certainty that there will be no harm to these sensitive subgroups of the U.S. population. The agricultural use of tralkoxydim on wheat and barley does not represent an acute or chronic risk to the U.S. population, infants, children or any of the 23 subgroups that were evaluated.

*F. International Tolerances*

There are no Codex Maximum Residue Levels established for tralkoxydim. (PM 25)

[FR Doc. 97–17176 Filed 7–1–97; 8:45 am]
**BILLING CODE 6560–50–F**

## ENVIRONMENTAL PROTECTION AGENCY

**[OPP–64033; FRL 5724–7]**

## Notice of Receipt of Requests for Amendments to Terminate the Use of Methamidophos on All Crops Except Cotton and Potatoes, and to Cancel All Methamidophos 24(c) Food-Use Registrations Not Labeled for Use on Tomatoes Only

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Notice.

**SUMMARY:** In accordance with section 6(f)(1) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended, EPA is issuing a notice of receipt of requests for amendment by Bayer Corporation and Valent USA, the sole U.S. registrants of the insecticide methamidophos, to terminate the use of methamidophos on all agricultural crops except cotton and potatoes by deleting uses from all methamidophos FIFRA section 3 registrations, and to cancel all section 24(c) food-use registrations not labeled for use on tomatoes only.

**DATES:** Public comment on this notice, in order to be considered, must be received by August 1, 1997. Unless EPA publishes a notice in the **Federal Register** modifying this notice, EPA will approve these use terminations and product cancellations and make them effective on December 29, 1997, subject to the existing stocks provision specified herein.

**ADDRESSES:** By mail, submit comments to the Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW.,