review and approval process conducted by the New Mexico Environment Department.

Dated: January 27, 1998.

William D. Dickerson,

Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 98–2376 Filed 1–29–98; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[ER–FRL–5488–4]

Environmental Impact Statements; Notice of Availability

Responsible Agency: Office of Federal Activities, General Information (202) 564–7167 OR (202) 564–7153.

Weekly receipt of Environmental Impact Statements Filed January 19, 1998 Through January 23, 1998 Pursuant to 40 CFR 1506.9.

EIS No. 980012, DRAFT EIS, COE, CA, Santa Clara River and Major Tributaries Project, Approval of 404 Permit and 1603 Streambed Alteration Agreement, City Santa Clarita, Los Angeles County, CA, Due: March 25, 1998, Contact: Bruce Henderson (805) 641–1128.

EIS No. 980013, DRAFT EIS, FHW, NM, Paseo del Volcon Corridor, Acquisition of Right-of-Way and Construction of Roadway, from the Intersection of I–40 to Intersection of NM–44 near the Town of Bernalillo, Bernalillo and Sandoval Counties, NM, Due: March 16, 1998, Contact: Gregory D. Rawlings (505) 820–2027.

EIS No. 980014, DRAFT EIS, AFS, OR, Nicore Mining Project, Implementation, Plan-of-Operations, Mining of Four Sites, Road Construction, Reconstruction, Hauling and Stockpiling of Ore, Rough and Ready Creek Watershed, Illinois Valley Ranger District, Siskiyou National Forest, Medford District, Josephine County, OR, Due: March 16, 1998, Contact: Rochelle Desser (541) 592–2166.

EIS No. 980015, FINAL SUPPLEMENT, COE, PA, Lower Monogahela River Navigation System, Locks and Dam Nos 2, 3 and 4 Improvement, Additional Documentation, Disposal and Dredge and Excavated Material, Funding, Allegheny, Washington and Westmoreland Counties, PA, Due: March 02, 1998, Contact: James Purdy (412) 395–7224.

Dated: January 27, 1998.

William D. Dickerson,

Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 98–2377 Filed 1–29–98; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[PF–788; FRL–5766–2]

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–788, must be received on or before March 2, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, 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. 119 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.joannes@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Cynthia Giles-Parker (PM 22).	Rm. 229, CM #2, 703–305–7740, e-mail: giles-parker.cynthia@epamail.epa.gov.	Do.

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–788] (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 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by

the docket control number [PF–788] and appropriate petition number. Electronic comments on this 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: January 22, 1998.

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. FMC Corporation

PP 7F4795

EPA has received a pesticide petition (PP 7F4795) from FMC Corporation, 1735 Market Street, Philadelphia, PA 19103, 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 residues of carfentrazone-ethyl in or on the raw agricultural commodities (RAC) cereal grain at 0.1 parts per million (ppm), 0.3 ppm in or on hay; 0.2 ppm in or on straw; 1.0 ppm in or on forage; 0.15 ppm in or on stover and 0.1 ppm in or on sweet corn, K + CWHR (kernels plus cob with husk removed) and in or on the RACs soybeans and soybean seed at 0.1 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. *Plant metabolism*. The metabolism of carfentrazone-ethyl in plants is adequately understood. Corn, wheat, and soybean metabolism studies with carfentrazone-ethyl have shown uptake of material into plant tissue with no significant movement into grain or seeds. All three plants extensively metabolized carfentrazone-ethyl and exhibited a similar metabolic pathway. The residues of concern are the combined residues of carfentrazone-ethyl and carfentrazone-ethyl-chloropropionic acid.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of carfentrazone and its metabolites in or on food with a limit of quantitation (LOQ) that allows monitoring of food with residues at or above the levels set in the tolerances. The analytical method for carfentrazone-ethyl involves separate analyses for parent and its metabolites. The parent is analyzed by GC/ECD. The metabolites are derivatized with boron trifluoride and acetic anhydride for analysis by GC/MSD using selective ion monitoring.

3. *Magnitude of residues*. Carfentrazone-ethyl 50DF was applied postemergent to 28 wheat trials, 24 corn trials, and 22 soybean trials in the appropriate EPA regions. The RACs were harvested at the appropriate growth stages and subsequent analyses determined that the residues of carfentrazone-ethyl and its metabolites will not exceed the proposed tolerances of 1.0, 0.3, 0.2, and 0.1 ppm for wheat forage, hay, straw, and grain, respectively; 0.1 ppm each for corn forage, fodder, and grain; and 0.1 ppm for soybean seed. Residue data from a cow feeding study demonstrated that no accumulation of carfentrazone-ethyl or its metabolites occurred in milk or tissues.

B. Toxicological Profile

1. *Acute toxicity*. Carfentrazone-ethyl demonstrates low oral, dermal and inhalation toxicity. The acute oral LD_{50} value in the rat was greater than 5,000 milligram/kilograms (mg/kg), the acute dermal LD_{50} value in the rat was greater than 4,000 mg/kg and the acute inhalation LC_{50} value in the rat was greater than 5.09 mg/L/4h. Carfentrazone-ethyl is non-irritating to rabbit skin and minimally irritating to rabbit eyes. It did not cause skin sensitization in guinea pigs. An acute neurotoxicity study in the rat had a systemic No observed adverse effect level (NOAEL) of 500 mg/kg based on clinical signs and decreased motor activity levels; the NOAEL for neurotoxicity was greater than 2,000 mg/kg (highest dose tested); (HDT) based on the lack of neurotoxic clinical signs or effects on neuropathology.

2. *Genotoxicity*. Carfentrazone-ethyl did not cause mutations in the Ames assay with or without metabolic activation. There was a positive response in the Chromosome Aberration assay without activation but a negative response with activation. The Mouse Micronucleus assay (an *in vivo* test which also measures chromosome damage), the CHO/HGPRT forward mutation assay and the Unscheduled DNA Synthesis assay were negative. The overwhelming weight of the evidence supports the conclusion that Carfentrazone-ethyl is not genotoxic.

3. *Reproductive and developmental toxicity*. Carfentrazone-ethyl is not considered to be a reproductive or a developmental toxin. In the 2-generation reproduction study, the No observed effect level (NOEL) for reproductive toxicity was greater than 4,000 ppm (greater than 323 to greater than 409 mg/kg/day). In the developmental toxicity studies, the rat and rabbit maternal NOELs were 100 mg/kg/day and 150 mg/kg/day, respectively. The developmental NOEL for the rabbit was greater than 300 mg/kg/day which was the highest dose tested and for the rat the NOEL was 600 mg/kg/day based on increased litter incidences of thickened and wavy ribs at 1,250 mg/kg/day. These two findings (thickened and wavy ribs) are not considered adverse effects of treatment but related delays in rib development which are generally believed to be reversible.

4. *Subchronic toxicity*. Ninety-day feeding studies were conducted in mice, rats and dogs with Carfentrazone-ethyl. The NOEL for the mouse study was 4,000 ppm (571 mg/kg/day), for the rat study was 1,000 ppm (57.9 mg/kg/day for males; 72.4 mg/kg/day for females) and for dogs was 150 mg/kg/day. A 90-day subchronic neurotoxicity study in the rat had a systemic NOEL of 1,000 ppm (59.0 mg/kg/day for males; 70.7 mg/kg/day for females) based on decreases in body weights, body weight gains and food consumption at 10,000 ppm; the neurotoxicity NOEL was greater than 20,000 ppm (1,178.3 mg/kg/day for males; 1,433.5 mg/kg/day for females) which was the highest dose tested.

5. *Chronic toxicity*. Carfentrazone-ethyl is not carcinogenic to rats or mice. A 2-Year Combined Chronic Toxicity/Oncogenicity study in the rat was negative for carcinogenicity and had a chronic toxicity NOEL of 200 ppm (9 mg/kg/day) for males and 50 ppm (3 mg/kg/day) for females based on red fluorescent granules consistent with porphyrin deposits in the liver at the 500 and 200 ppm levels, respectively.

An 18 Month Oncogenicity study in the mouse had a carcinogenic NOEL that was greater than 7,000 ppm (>1,090 mg/kg/day for males; >1,296 mg/kg/day for females) based on no evidence of carcinogenicity at the highest dose tested. A 1-Year Oral Toxicity study in the dog had a NOEL of 50 mg/kg/day based on isolated increases in urine porphyrins in the 150 mg/kg/day group (this finding was not considered adverse).

Using the Guidelines for Carcinogen Risk Assessment, carfentrazone-ethyl should be classified as Group ''E'' for carcinogenicity -- no evidence of carcinogenicity -- based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18-month feeding study in mice and a 2-year feeding study in rats at the dosage levels tested. The doses tested are adequate for identifying a cancer risk. Thus, a cancer risk assessment is not necessary.

6. *Animal metabolism*. The metabolism of carfentrazone-ethyl in animals is adequately understood. Carfentrazone-ethyl was extensively metabolized and readily eliminated following oral administration to rats, goats, and poultry via excreta. All three animals exhibited a similar metabolic pathway. As in plants, the parent chemical was metabolized by hydrolytic mechanisms to predominantly form carfentrazone-ethyl-chloropropionic acid which was readily excreted.

7. *Endocrine disruption*. An evaluation of the potential effects on the endocrine systems of mammals has not been determined; however, no evidence of such effects were reported in the chronic or reproductive toxicology studies described above. There was no observed pathology of the endocrine organs in these studies. There is no evidence at this time that carfentrazone-ethyl causes endocrine effects.

C. Aggregate Exposure

1. *Dietary exposure*— i. *Acute dietary*. The Agency has determine that there is no concern for an acute dietary risk assessment since the available data do not indicate any evidence of significant toxicity from a 1-day or single event exposure by the oral route (**Federal Register**: September 30, 1997, 62 FR 51032-51038). Thus an acute dietary risk assessment is not necessary.

ii. *Chronic dietary*. Based on the available toxicity data, the EPA has established a provisional Reference Dose (RfD) for carfentrazone-ethyl of 0.06 mg/kg/day. The RfD for carfentrazone-ethyl is based on a 90-day feeding study in rats with a threshold NOEL of 57.9 mg/kg/day and an uncertainty factor of 100, with an additional modifying factor of 10 to account for the fact that the chronic studies have not yet been reviewed by the EPA. For purposes of assessing the potential chronic dietary exposure, a Tier 1 dietary risk assessment was conducted based on the Theoretical Maximum Residue Contribution (TMRC) from the proposed tolerances for carfentrazone-ethyl on soybeans at 0.1 ppm, wheat at 0.2 ppm and corn (field) at 0.15 ppm. (The TMRC is a ''worse case'' estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are established are treated and that pesticide residues are present at the tolerance levels.) At this time the dietary exposure to residues of carfentrazone-ethyl in or on food will be limited to residues on soybeans, wheat and corn. There are no other established U.S. tolerances for carfentrazone-ethyl, and there are no registered uses for carfentrazone-ethyl on food or feed crops in the U.S. In conducting this exposure assessment, the following very conservative assumptions were made--100% of soybeans, wheat and corn will contain carfentrazone-ethyl residues and those residues would be at the level of the tolerance which result in an overestimate of human exposure.

2. *Food*. Dietary exposure from the proposed uses would account for 1.3% or less of the RfD in subpopulations (including infants and children).

3. *Drinking water*. Studies have indicated that carfentrazone-ethyl will not move into groundwater, therefore water has not been included in the dietary risk assessment.

4. *Non-dietary exposure*. No specific worker exposure tests have been conducted with carfentrazone-ethyl. The potential for non-occupational exposure to the general population has not been fully assessed. No specific worker exposure tests have been conducted with carfentrazone-ethyl.

D. Cumulative Effects

EPA is also required to consider the potential for cumulative effects of carfentrazone-ethyl and other substances that have a common mechanism of toxicity. EPA consideration of a common mechanism of toxicity is not appropriate at this time since EPA does not have information to indicate that toxic effects produced by carfentrazone-ethyl would be cumulative with those of any other chemical compounds; thus only the potential risks of carfentrazone-ethyl are considered in this exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described and based on the completeness and reliability of the toxicity data, the aggregate exposure to carfentrazone-ethyl will utilize 0.61% of the RfD for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD. Therefore, 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 from aggregate exposure to residues of carfentrazone-ethyl, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of carfentrazone-ethyl, EPA considers data from developmental toxicity studies in the rat and rabbit and the 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to effects on the reproductive capacity of males and females exposed to the pesticide. Developmental toxicity was not observed in developmental toxicity studies using rats and rabbits. In these studies, the rat and rabbit maternal NOELs were 100 mg/kg/day and 150 mg/kg/day, respectively. The developmental NOEL for the rabbit was greater than 300 mg/kg/day which was the highest dose tested and for the rat was 600 mg/kg/day based on increased litter incidences of thickened and wavy ribs. These two findings are not considered adverse effects of treatment but related delays in rib development which are generally believed to be reversible.

In a 2-generation reproduction study in rats, no reproductive toxicity was observed under the conditions of the study at 4,000 ppm which was the highest dose tested.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database relative to pre- and post-natal effects for children is complete and an additional uncertainty factor is not warranted. Therefore at this time, the provisional RfD of 0.06 mg/kg/day is

appropriate for assessing aggregate risk to infants and children.

3. *Reference dose (RfD).* Using the conservative exposure assumptions described above, the percent of the RfD that will be utilized by aggregate exposure to residues of carfentrazone-ethyl for non-nursing infants (<1 year old) would be 0.28% and for children 1-6 years of age would be 1.37% (the most highly exposed.

F. International Tolerances

There are no Codex Alimentarius Commission (Codex) Maximum Residue Levels (MRLs) for carfentrazone-ethyl on any crops at this time. However, MRLs for small grains in Europe have been proposed which consist of carfentrazone-ethyl and carfentrazone-ethyl-chloropropionic acid. (PM 23)

2. Rohm and Haas Company

PP 2F4127 2F4135, 3F4194, 3H5663, 7F4887, and 7F4900

EPA has received six pesticide petitions (PP 2F4127, 2F4135, 3F4194, 3H5663, 7F4887, and 7F4900) from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA 19106-2399, proposing pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing permanent tolerances for almond, apple, and grapefruit and time-limited tolerances for wheat and animal commodities for residues of [alpha-(2-(4-chlorophenyl)-ethyl)-alpha-phenyl-3-(1*H*-1,2,4-triazole)-1-propanenitrile (fenbuconazole) in or on the raw agricultural commodities (RAC) almond nuts at 0.05 parts per million (ppm); almond hulls at 3.0 ppm; apples at 0.4 ppm; apple pomace, wet at 1.0 ppm; grapefruit at 1.0 ppm; citrus oil (grapefruit) at 35.0 ppm; grapefruit pulp, dried at 4.0 ppm; sugar beet root at 0.2 ppm; sugar beet top at 9.0 ppm; sugar beet pulp, dried at 1.0 ppm; sugar beet molasses at 0.4 ppm; wheat grain at 0.05 ppm; wheat straw at 10.0 ppm; fat of cattle, hogs, horses, goats, and sheep at 0.05 ppm; and liver of cattle, hogs, horses, goats, and sheep at 0.3 ppm. The analytical method involves soxhlet extraction, partitioning, redissolving, clean-up, and analysis by gas-liquid chromatography using nitrogen specific thermionic detection. EPA has determined that the petitions contain 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 petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

The tolerance expression for fenbuconazole residues in or on almond nuts or hulls, apples or apple process fractions, grapefruit and all related commodities, sugar beets, and wheat grain or straw is α-(2-(4-chlorophenyl)-ethyl)-α-phenyl-(1*H*-1,2,4-triazole-1-propanenitrile, plus cis-5-(4-chlorophenyl) dihydro-3-phenyl-3-(1*H*-1,2,4-triazole-1-ylmethyl-)-2(3*H*)-furanone, plus trans-5-(4-chlorophenyl) dihydro-3-phenyl-3-(1*H*-1,2,4-triazole-1-ylmethyl-)-2(3*H*)-furanone. Residues of these compounds are combined and expressed as parent compound to determine the total residue in or on almond nuts or hulls, apples or apple process fractions, grapefruit and all related commodities, sugar beets and all related commodities, and wheat grain or straw.

The tolerance expression for fenbuconazole residues in or on animal fat is α-(2-(4-chlorophenyl)-ethyl)-α-phenyl-(1*H*-1,2,4-triazole-1-propanenitrile, plus 4-chloro-α-(hydroxymethyl)-α-phenyl-benzenebutanenitrile. Residues of these compounds are combined and expressed as parent compound to determine the total residue.

The tolerance expression for fenbuconazole residues in or on animal liver is α-(2-(4-chlorophenyl)-ethyl)-α-phenyl-(1*H*-1,2,4-triazole-1-propanenitrile, plus cis-5-(4-chlorophenyl) dihydro-3-phenyl-3-(1*H*-1,2,4-triazole-1-ylmethyl-)-2(3*H*)-furanone, plus trans-5-(4-chlorophenyl) dihydro-3-phenyl-3-(1*H*-1,2,4-triazole-1-ylmethyl-)-2(3*H*)-furanone, plus 4-chloro-α-(hydroxymethyl)-α-phenyl-benzenebutanenitrile. Residues of these compounds are combined and expressed as parent compound to determine the total residue.

Analytical methods to measure the components of the residue in or on almond nuts and almond hulls, apples, apple process fractions, grapefruit, sugar beets, wheat grain and wheat straw, and animal commodities have been validated and accurately quantify residues of fenbuconazole. The residues of fenbuconazole will not exceed the proposed Permanent Tolerances in/on apples or apple process fractions, in/on almonds or related commodities, in/on grapefruit or related commodities following foliar treatment, on sugar beets or related commodities, or in/on wheat or related commodities following foliar or seed treatment.

1. *Analytical method.* Fenbuconazole residues (parent plus lactones) are measured at an analytical sensitivity of 0.01 mg/kg in apples, and wheat grain and straw by soxhlet extraction of samples in methanol, partitioning into methylene chloride, redissolving in toluene, clean-up on silica gel, and gas-liquid chromatography (GLC) analysis using nitrogen specific thermionic detection. Fenbuconazole residues are measured at an analytical sensitivity of 0.01 mg/kg in fat and liver in essentially the same manner except that one of the analytes in these matrices, 4-chloro-α-(hydroxymethyl)-α-phenyl-benzenebutanenitrile, is measured at a sensitivity of 0.05 ppm.

2. *Magnitude of residues*— i. *Wheat.* Residue studies have been conducted in accordance with the geographic distribution mandated by the EPA for wheat. In the wheat grain, the raw agricultural commodity, the fenbuconazole residues ranged from no detectable residue (NDR < LOQ = 0.01 mg/kg) to approximately 0.01 ppm. In wheat straw the fenbuconazole residues ranged from approximately 0.05 ppm to approximately 4.5 ppm. Residues were measured in processed fractions of wheat including cleaned grain, bread, patent flour, flour, red dog, bran, shorts/germ, and middlings. The EPA concluded that no concentration above the residue levels in the RAC occurred so no tolerances for any of these commodities were required. Tolerances of 0.05 ppm in wheat grain and 10 ppm in wheat straw are proposed based on these data.

Feeding studies in the cow, goat, and hen indicated that the only animal commodities which require tolerances are fat and liver. There were no significant residues in eggs or milk at any dose level. In cows there were residues in fat only at the 10x level in one animal at 0.06 mg/kg. Liver contained quantifiable residues in all dose groups and the magnitude of the residue correlated closely with the dose level. At study day 28 the 1 x livers averaged 0.08 mg/kg. Residues declined significantly during the depuration period. In the fat and liver one of the components of the fenbuconazole tolerance expression has a LOQ = 0.05 mg/kg. Because there were detectable residues only in liver, not fat, at the 1x level, the LOQ of the least sensitive component drives the fat tolerance. Tolerances of 0.05 ppm in fat and 0.3 ppm in liver are proposed based on the animal data.

Tolerances for wheat process fractions and wheat rotation crops are not required because no concentration of residues occurs in process fractions of wheat and no residues occur in rotation crops.

ii. *Apples.* Residue studies have been conducted in accordance with the geographic distribution mandated by the EPA for apples. In the apples, the raw agricultural commodity (RAC), the fenbuconazole residues ranged from approximately 0.1 mg/kg to approximately 0.3 mg/kg. Residues were measured in process fractions of apples, apple juice, and apple pomace. Concentration above the residue levels in the RAC occurred only in the pomace at approximately two-fold. Thus, no tolerance for juice is required, but a tolerance for pomace is required.

Seven field trials on apples were carried out in 1990 in six states: Pennsylvania, Washington, North Carolina, Michigan, Virginia, and West Virginia. Two application rates were used in each of the studies, the anticipated maximum application rate of 0.14 kg ai/ha and a 2x exaggerated rate of 0.28 kg ai/ha. A total of eight to ten applications were made at the normal timing in each trial, and the fruit was harvested at 0, 7, and 13 or 14 days after the final application. All samples were frozen immediately after they were harvested and were kept frozen until analysis, or shipped fresh immediately after harvest and processed and frozen immediately upon receipt and kept frozen until analysis. Samples were analyzed using the residue analytical method for RH-7592 parent and metabolites in stone fruit, and residues were corrected for average fortification recoveries. As would be expected, the residue levels were seen to increase with decreased PHI and increased application rate. The average half-life of residue decline for six studies was 11.9 days. The average parent residue at 13-14 PHI at the 0.14 kg ai/ha rate was 0.086 mg/kg.

Formulation bridging studies were conducted on apples in 1993. Apples grown in Washington and Pennsylvania were treated, in separate plots, with the 2F and 75 WP formulations of fenbuconazole at a rate of 0.14 kg ai/ha/application. A total of ten or twelve applications were made using an airblast sprayer at the normal timing of each trial, and the fruit was harvested at 14 days after the final application (14 day Pre-Harvest Interval or PHI). Samples were shipped fresh immediately after harvest and frozen immediately upon receipt and kept frozen until processing and subsequent analysis. Samples were analyzed using the residue analytical method for RH-7592 parent and metabolites in stone fruit, but residues were not corrected for average fortification recoveries. Total residues from the two trials were 0.226 and 0.135 mg/kg in the 2F formulation, and 0.184 and 0.162 mg/kg in the 75WP formulation. There were no significant differences in apparent residues found from the use of the two formulations, and residues due to parent compound constituted greater than 85% of the total residues found on the fruit.

Seven field residue trials were conducted on apples in 1995, in California, Colorado, Michigan, New York, Ohio, Oregon, and Washington. Apples were treated with dilute (0.014 kg ai/hl) and concentrate (0.035 kg ai/hl) sprays of the 2F formulation of fenbuconazole at a 0.14 kg ai/ha. A total of eight to ten applications were made using airblast sprayers, with first application at early bud break and subsequent applications on a 10-14 day schedule through bloom and a 14 to 21 day schedule in the cover sprays until harvest. The apples were harvested by hand at a PHI of 14 days. Residue samples were analyzed using the residue analytical method for RH-7592 parent and metabolites in stone fruit, but residues were not corrected for average fortification recoveries. Samples from three sites were also analyzed using the residue analytical method for metabolite RH-7905. Metabolite RH-7905 was not detected in any of the samples. The total residues from the concentrate sprays ranged from 0.015 to 0.274 mg/kg and averaged 0.137 mg/kg. The total residues from the dilute sprays ranged from 0.019 to 0.295 mg/kg and averaged 0.139 mg/kg. There is not a significant difference in the magnitude of the residues between dilute and concentrate spray volumes of the 2F formulation of fenbuconazole.

An additional residue study was conducted on apples grown in Pennsylvania in 1994 and the fruit was used for a processing study. The apples received nine foliar applications of the 2F formulation of fenbuconazole at the normal timing at a rate of 0.14 kg ai/ha/application. The fruit was harvested 14 days after the last treatment. The RAC samples were shipped fresh and either immediately processed or frozen for storage. All RAC and processed samples were analyzed within a less than 30 day period, eliminating the need for generation of storage stability data. The apples were processed at the Food Research Laboratory of Cornell University using methodology simulating commercial apple processing. Briefly, the processing consisted of washing the apples in water, grinding in a hammer mill to apple mash, and pressing of the mash to form both fresh apple juice and wet pomace. The juice was either canned (sampled as unpasteurized juice) or canned and pasteurized (sampled as pasteurized juice). The wet pomace (moisture content 69%) was also sampled. All samples were frozen on generation and stored frozen until analysis. Samples were analyzed using the residue analytical method for RH-7592 and metabolites in stone fruit, and residues were not corrected for average fortification recovery. The average total residues for each component, and its concentration factor, were as follows: unwashed fruit 0.065 mg/kg NA, washed fruit 0.070 mg/kg NA, wet pomace 0.159 mg/kg 2.46, unpasteurized juice 0.004 mg/kg 0.06, pasteurized juice 0 mg/kg 0.00. No concentration of residues was seen in the human diet component, i.e. apple juice. Concentration of residues of approximately 2-fold was seen in wet pomace, which is not a component of the human diet.

Feeding studies in the cow, goat, and hen indicated that the only animal commodities which require tolerances are fat and liver. There were no significant residues in eggs or milk at any dose level. Residues in animals declined significantly during the depuration period. In the fat and liver one of the components of the fenbuconazole tolerance expression has a LOQ = 0.05 mg/kg. Because there were detectable residues only in liver, not fat, the LOQ of the least sensitive component drives the fat tolerance. Tolerances of 0.05 ppm in fat and 0.3 ppm in liver were proposed based on the animal data.

Tolerances for other apple process fractions and for rotation crops are not required because no concentration of residues occurs in other process fractions of apples and rotation crops are not a concern for perennial crops.

iii. *Almonds.* Residue studies have been conducted in accordance with the geographic distribution mandated by the EPA for almonds. There are no process fractions of almonds. Six field trials in almonds were carried out at five sites in California in 1987. In all of the studies, the anticipated maximum application rate of 0.11 kg ai/ha and a 2X exaggerated rate of 0.22 kg ai/ha. A total of three applications were made at the normal timing in all trials, and the almonds were harvested at maturity, 127-200 days after the final application. Samples were shipped fresh or frozen. Hulls were separated from the nuts and processed in a Hobart food processor with dry ice or in a Wiley Mill without dry ice. Nuts were shelled and the nutmeat homogenized in a Waring food processor with dry ice. The processed samples were stored frozen until analysis. Samples were analyzed using the residue analytical method for RH-

7592 and metabolites. No residue in any nutmeat sample at the 1x application rate reached 0.01 mg/kg. Residues in the hull at the 1x rate ranged from 0.1 to 1.5 mg/kg. One nutmeat sample treated at the 2x rate had a quantifiable residue of 0.027 mg/kg. The remainder had no detectable residue. Hull sample residues from the 2x rate ranged from 0.5 to 6.6 mg/kg.

Feeding studies in the cow, goat, and hen indicated that the only animal commodities which require tolerances are fat and liver. There were no significant residues in eggs or milk at any dose level. Residues in animals declined significantly during the depuration period. In the fat and liver one of the components of the fenbuconazole tolerance expression has a LOQ = 0.05 mg/kg. Because there were detectable residues only in liver, not fat, the LOQ of the least sensitive component drives the fat tolerance. Tolerances of 0.05 ppm in fat and 0.3 ppm in liver were proposed based on the animal data.

Tolerances for almond process fractions and rotational crops are not required because there are no process fractions of almonds and rotational crops are not a concern for perennial crops.

iv. *Grapefruit.* Trials included both grapefruit and orange, so the following text covers the residue results for both. Six residue trials were conducted in 1993 on grapefruit and oranges grown in Texas, Florida and California (one grapefruit and one orange trial at each site). Three airblast sprayer applications of the 2F formulation of fenbuconazole at the rate of 0.28 kg ai/ha/application were made at the normal timing, and the fruit was harvested by hand at Pre-Harvest Intervals (PHIs) of 0 days (all trials), and approximately 15, 30 and 60 days (three trials). The whole fruit was analyzed using the residue analytical method for RH-7592 parent and metabolites in stone fruit and residues were not corrected for average fortification recoveries. The average total residue in whole grapefruit at 0 day PHI was 0.344 mg/kg, with a range of 0.190 - 0.499 mg/kg. The average total residue in whole oranges at 0 day PHI was 0.438 mg/kg, with a range of 0.339 - 0.528 mg/kg. For both fruits, the 0 day PHI residues were >97% parent. In the three trials which measured residue decline, the average total residue value had decreased to about 40% of the original value by 60 PHI.

Residue trials were conducted in 1993 and 1994 on grapefruit and oranges grown in seven different locations. Sites with both grapefruit and orange trials were in Texas (2) and Florida (3), and in California there was one site for oranges and another for grapefruit. Three airblast sprayer applications of the 2F formulation of fenbuconazole at the rate of 0.28 kg ai/ha/application were made at the normal timing, and the fruit was harvested by hand on the day of the final application (for a 0 day Pre-Harvest Interval). The fruit was processed in two different ways: as whole fruit, or as pulp only with the peel discarded. Samples were analyzed using the residue analytical method for RH-7592 parent and metabolites in stone fruit, and residues were not corrected for average fortification recoveries. Six of the RAC samples were also analyzed using the residue analytical method for metabolite RH-7905 (the glucoside conjugate). No detectable residues of RH-7905 were found in any sample. Average total residue for whole oranges was 0.238 mg/kg, and 0.0082 mg/kg for orange pulp. Average total residue for whole grapefruit was 0.141 mg/kg, and 0.0078 mg/kg for grapefruit pulp. Nearly all of the fenbuconazole residues lie on the peel, and [NDR] no detectable residue to LOQ levels are seen in the edible portion of the fruit, i.e. the pulp.

Feeding studies in the cow, goat, and hen indicated that the only animal commodities which require tolerances are fat and liver. There were no significant residues in eggs or milk at any dose level. Residues in animals declined significantly during the depuration period. In the fat and liver one of the components of the fenbuconazole tolerance expression has a LOQ = 0.05 mg/kg. Because there were detectable residues only in liver, not fat, the LOQ of the least sensitive component drives the fat tolerance. Tolerances of 0.05 ppm in fat and 0.3 ppm in liver were proposed based on the animal data. Tolerances for rotational crops are not required for tree fruits.

v. *Sugar beets.* Residue studies have been conducted in accordance with the geographic distribution mandated by the EPA for sugar beets. Following full season foliar treatment, the residues of fenbuconazole were higher in the sugar beet tops than in the root. Combined residues in root averaged 0.415 mg/kg. Residues in tops were more variable, and ranged from 0.56-8.89 mg/kg. In a formulation bridging study the residues were higher in the sugar beet tops compared to the root. Total root residues in the 75WP formulation ranged from 0.0061 to 0.268 mg/kg and averaged 0.0616 mg/kg. Total root residues in the 2F formulation ranged from 0.0223 to 0.0523 mg/kg and averaged 0.0328 mg/kg. Total top residues averaged 2.15 mg/kg in the 75WP formulation, and 2.69 mg/kg in the 2F formulation. There was no significant difference in residues between formulations of fenbuconazole. In a processing study the concentration factor for each component was: root - 1.0X, dry pulp - 5.39X, molasses - 1.82X, and refined sugar - 0.1X. Compared to raw roots, a reduction of residues was seen in the human diet component, sugar. Concentration of residues was seen in molasses and dry pulp, neither of which is a component of the human diet.

Tolerances for rotational crops are not required because EPA determined under the wheat petition that rotational crops are not a concern for fenbuconazole.

B. Toxicological Profile

The toxicology of fenbuconazole is summarized in the following sections. There is no evidence to suggest that human infants and children will be more sensitive than adults, that fenbuconazole will modulate human endocrine systems at anticipated dietary exposures, or cause cancer in humans at the dietary exposures anticipated for this fungicide. While the biochemical target for the fungicidal activity of members of the DMI class is shared, it cannot be concluded that the mode of action of fenbuconazole which produces phytotoxic effects in plants or toxic effects in animals is also common to a single class of chemicals.

1. *Acute toxicity*. Fenbuconazole is practically nontoxic after administration by the oral, dermal and respiratory routes. The acute oral LD_{50} in mice and rats is >2,000 mg/kg. The acute dermal LD_{50} in rats is >5,000 mg/kg. Fenbuconazole was not significantly toxic to rats after a 4-hour inhalation exposure, with an LD_{50} value of >2.1 mg/L. Fenbuconazole is classified as not irritating to skin (Draize score = 0), inconsequentially irritating to the eyes (mean irritation score = 0), and it is not a sensitizer. No evidence exists regarding differential sensitivity of children and adults to acute exposure.

2. *Mutagenicity*. Fenbuconazole has been adequately tested in a variety of *in vitro* and *in vivo* mutagenicity tests. It is negative in the Ames test, negative in *in vitro* and *in vivo* somatic and germ cell tests, and did not induce unscheduled DNA synthesis (UDS). Fenbuconazole is not genotoxic.

3. *Reproductive and developmental toxicity*. These conclusions were extracted from the **Federal Register** of May 24, 1995 (60 FR 27419). Fenbuconazole is not teratogenic. The maternal no observable effect level (NOEL) in rabbits was 10 mg/kg/day and

30 mg/kg/day in rats. The fetal NOEL was 30 mg/kg/day in both species. The parental NOEL was 4.0 mg/kg/day (80 ppm) in a 2-generation reproduction study in rats. The reproductive NOEL in this study was greater than 40.0 mg/kg/day (800 ppm; highest dose tested). Fenbuconazole had no effect on male reproductive organs or reproductive performance at any dose. The adult lowest observed effect level (LOEL) was 40.0 mg/kg/day (800 ppm; highest dose tested). Systemic effects of decreased body weight gain; maternal deaths; and hepatocellular, adrenal, and thyroid follicular cell hypertrophy were observed. No effects on neonatal survival or growth occurred below the adult toxic levels. Fenbuconazole does not produce birth defects and is not toxic to the developing fetus at doses below those which are toxic to the mother.

4. *Subchronic toxicity*. In a 21-day dermal toxicity study in the rat, the NOEL was greater than 1,000 mg/kg/day, with no effects seen at this limit dose.

5. *Chronic toxicity*. In 2-year combined chronic toxicity/oncogenicity studies in rats, the NOEL was 80 ppm (3.03 mg/kg/day for males and 4.02 mg/kg/day for females) based on decreased body weight, and liver and thyroid hypertrophy. In a 1-year chronic toxicity study in dogs, the NOEL was 150 ppm (3.75 mg/kg/day) based on decreased body weight, and increased liver weight. The LOEL was 1,200 ppm (30 mg/kg/day). In a 78-week oncogenicity study in mice, the NOEL was 10 ppm (1.43 mg/kg/day). The LOEL was 200 ppm (26.3 mg/kg/day, males) and 650 ppm (104.6 mg/kg/day, females) based on increased liver weights and histopathological effects on the liver. These effects were consistent with chronic enzyme induction from high dose dietary exposure.

A Reference Dose (RfD) for systemic effects at 0.03 mg/kg/day was established by EPA in 1995 based on the NOEL of 3.0 mg/kg/day from the rat chronic study. This RfD adequately protects both adults and children.

6. *Carcinogenicity*. Twenty-four-month rat chronic feeding/carcinogenicity studies with fenbuconazole showed effects at 800 and 1,600 ppm. Fenbuconazole produced a minimal, but statistically significant increase in the incidence of combined thyroid follicular cell benign and malignant tumors. These findings occurred only in male rats following life-time ingestion of very high levels (800 and 1,600 ppm in the diet) fenbuconazole. Ancillary mode-of-action studies demonstrated that the increased incidence of thyroid tumors was secondary to increased liver metabolism and biliary excretion of thyroid hormone in the rat. This mode of action is a nonlinear phenomenon in that thyroid tumors occur only at high doses where there is an increase in liver mass and metabolic capacity of the liver. At lower doses of fenbuconazole in rats, the liver is unaffected and there is no occurrence of the secondary thyroid tumors. Worst-case estimates of dietary intake of fenbuconazole in human adults and children indicate effects on the liver or thyroid, including thyroid tumors, will not occur, and there is a reasonable certainty of no harm.

In support of the findings above, EPA's Science Advisory Board has approved a final thyroid tumor policy, confirming that it is reasonable to regulate chemicals on the basis that there exists a threshold level for thyroid tumor formation, conditional upon providing plausible evidence that a secondary mode of action is operative. This decision supports a widely-held and internationally respected scientific position.

In a 78-week oncogenicity study in mice there was no statistically significant increase of any tumor type in males. There were no liver tumors in the control females and liver tumor incidences in treated females just exceeded the historical control range. However, there was a statistically significant increase in combined liver adenomas and carcinomas in females at the high dose only (1,300 ppm; 208.8 mg/kg/day). In ancillary mode-of-action studies in female mice, the increased tumor incidence was associated with changes in several parameters in mouse liver following high doses of fenbuconazole including: an increase in P450 enzymes (predominately of the CYP 2B type), an increase in cell proliferation, an increase in hepatocyte hypertrophy, and an increase in liver mass (or weight). Changes in these liver parameters as well as the occurrence of the low incidence of liver tumors were nonlinear with respect to dose (i.e., were observed only at high dietary doses of fenbuconazole). Similar findings have been shown with several pharmaceuticals, including phenobarbital, which is not carcinogenic in man. The nonlinear relationship observed with respect to liver changes (including the low incidence of tumors) and dose in the mouse indicates that these findings should be carefully considered in deciding the relevance of high-dose animal tumors to human dietary exposure.

The Carcinogenicity Peer Review Committee (PRC) of the Health Effects Division (HED) classified fenbuconazole as a Group C tumorigen (possible human carcinogen with limited evidence of carcinogenicity in animals). The PRC used a low-dose extrapolation model. The $Q1^*$ risk factor applied (1.06×10^{-2} $(mg/kg/day)^{-1}$) was based on the rat oncogenicity study and surface area was estimated by $(body\ weight)^{3/4}$.

Since the PRC published the above estimate they have agreed that low-dose extrapolation for fenbuconazole, based on rat thyroid tumors, is inappropriate given the EPA's policy regarding thyroid tumors and the data which exist for fenbuconazole. The PRC agrees that the more appropriate dataset for the low-dose extrapolation and risk factor estimate is the mouse. From these data a $Q1^*$ of (0.36×10^{-2} $(mg/kg/day)^{-1}$) is calculated when surface area is estimated by $(body\ weight)^{3/4}$. All estimates of dietary oncogenic risk are based on this risk factor.

Since fenbuconazole will not leach into groundwater (see below) there is no increased cancer risk from this source. Neither is fenbuconazole registered for residential use, so there is no risk from non-occupational residential exposure either. All estimates of excess risk to cancer are from dietary sources.

7. *Endocrine effects.* The mammalian endocrine system includes estrogen and androgens as well as several other hormone systems. Fenbuconazole does not interfere with the reproductive hormones. Thus, fenbuconazole is not estrogenic or androgenic.

While fenbuconazole interferes with thyroid hormones in rats by increasing thyroid hormone excretion, it does so only secondarily and only above those dietary levels which induce metabolism in the liver. These effects are reversible in rats, and humans are far less sensitive to these effects than rats. The RfD protects against liver induction because it is substantially below the animal NOEL. As noted previously, maximal human exposures are far below the RfD level, and effects on human thyroid will not occur at anticipated dietary levels.

We know of no instances of proven or alleged adverse reproductive or developmental effects to domestic animals or wildlife as a result of exposure to fenbuconazole or its residues. In fact, no effects should be seen because fenbuconazole has low octanol/water partition coefficients and is known not to bioaccumulate. Fenbuconazole is excreted within 48 hours after dosing in mammalian studies.

C. Aggregate Exposure

1. *Dietary exposure— Food*. i. *Wheat*. For wheat, children 1 to 6 years old, not infants, are the highest consumers (g/kg bw/d basis). For children 1-6 the dietary TMRC for existing tolerances utilizes only 5% of the RfD. The dietary TMRC for wheat in this group is estimated to be 0.00016 mg/kg/day and uses 0.52% of the RfD. Additional dietary exposure (TMRC) to fenbuconazole from residues which might be transferred to animal fat and liver from treated wheat is estimated to be 0.00006 mg/kg/day and uses 0.22% of the RfD. No residues occur in animal meats, milk, or eggs. Thus, the TMRC, the worst-case exposure, in the two most sensitive subpopulations of consumers, non-nursing infants less than one year old and children 1 to 6 years old, still utilizes less than 18% and less than 6%, respectively, of the fenbuconazole RfD. The dietary TMRCs for other children and for adults utilize less than this.

The calculated additional cancer risk for wheat ($Q1^* = 0.36 \times 10^{-2}$ (mg/kg/day)$^{-1}$) has an upper-bound of 0.2×10^{-6}. The calculated additional cancer risk for animal fat and liver has an upper-bound of 0.1×10^{-6}. The upper bound estimate on excess cancer risk for all uses including wheat is 0.7×10^{-6}. The estimate shows that the TMRC, the worst-case exposure, for consumers to fenbuconazole presents a reasonable certainty of no harm. The actual residue contribution is anticipated to be significantly less than this estimate.

ii. *Apples*. The EPA used the DRES model to estimate consumer dietary exposure to fenbuconazole residues for the most recently approved tolerance in bananas (memorandum of E.A. Doyle, February 8, 1995). (memorandum of E.A. Doyle, 8 February 1995). The EPA used the Theoretical Maximum Residue Contribution (TMRC) for pecans and bananas, and adjusted the TMRC for the stone fruit crop group by excluding plums/prunes and limiting sales volume to 12.8% of the available stone fruit market. From this EPA calculated an upper-bound risk of 0.9 x 10-6 for additional cancer risk ($Q1^* = 1.06 \times 10^{-2}$ (mg/kg/day)-1). (**Federal Register** of May 24, 1995 (60 FR 27419)). This estimate does not reflect the change in $Q1^*$, the use of the DEEM database, the percent crop treated for all crops, or average residues. When these factors are included the aggregate lifetime exposure for consumers to fenbuconazole has an upper bound risk estimate of 0.18×10^{-6} for apples and 0.28×10^{-6} for all pending and approved uses combined. The theoretical maximum estimated exposure to the most sensitive subpopulation, non-nursing infants less than one year old, for this same scenario utilizes no more than 0.89% of the RfD. Thus, the addition of fenbuconazole use on apples meets the EPA criterion of reasonable certainty of no harm.

iii. *Almonds*. The consumer dietary exposure to fenbuconazole residues was estimated for the most recently approved tolerance in bananas (memorandum of E.A. Doyle, 8 February 1995). The EPA used the Theoretical Maximum Residue Contribution (TMRC) for pecans and bananas, and adjusted the TMRC for the stone fruit crop group by excluding plums/prunes and limiting sales volume to 12.8% of the available stone fruit market. From this EPA calculated an upper-bound risk of 0.9×10^{-6} for additional cancer risk ($Q1^* = 1.06 \times 10^{-2}$ (mg/kg/day)$^{-1}$). (**Federal Register** of May 24, 1995 (60 FR 27419)). This estimate does not reflect the change in $Q1^*$, the use of the DEEM database, the percent crop treated for all crops, or average residues. When these factors are included the aggregate lifetime exposure for consumers to fenbuconazole has an upper bound cancer risk estimate of 7.5×10^{-11} for almonds and 0.28×10^{-6} for all pending and approved uses combined. The theoretical maximum estimated exposure to the most sensitive subpopulation, non-nursing infants less than one year old, for this same scenario utilizes no more than 0.89% of the RfD. Thus, the addition of fenbuconazole use on almonds meets the EPA criterion of reasonable certainty of no harm.

This estimate shows that the estimated exposure for consumers to fenbuconazole presents a reasonable certainty of no harm. The actual dietary residue contribution will likely be less than this estimate.

iv. *Grapefruit*. The consumer dietary exposure to fenbuconazole residues was estimated for the most recently approved tolerance in bananas (memorandum of E.A. Doyle, 8 February 1995). The EPA used the Theoretical Maximum Residue Contribution (TMRC) for pecans and bananas, and adjusted the TMRC for the stone fruit crop group by excluding plums/prunes and limiting sales volume to 12.8% of the available stone fruit market. From this EPA calculated an upper-bound risk of 0.9×10^{-6} for additional cancer risk ($Q1^* = 1.06 \times 10^{-2}$ (mg/kg/day)$^{-1}$). (**Federal Register** of May 24, 1995 (60 FR 27419)). This estimate does not reflect the change in $Q1^*$, the use of the DEEM database, the percent crop treated for all crops, or average residues. When the new $Q1^*$ of (0.36×10^{-2} (mg/kg/day)$^{-1}$) and surface area estimated by (body weight)$^{3/4}$ plus the other factors are included, the aggregate lifetime exposure to consumers to fenbuconazole has an upper bound risk estimate of 7.0 x 10^{-8} for grapefruit and 0.17 x 10^{-6} for all pending and approved uses combined. The theoretical maximum estimated exposure to the most sensitive subpopulation, non-nursing infants less than one year old, for this same scenario utilizes no more than 0.39% of the RfD. Thus, the addition of fenbuconazole use on grapefruit meets the EPA criterion of reasonable certainty of no harm.

This estimate shows that the estimated exposure for consumers to fenbuconazole presents a reasonable certainty of no harm. The actual dietary residue contribution will likely be less than this estimate.

v. *Sugar beets*. The consumer dietary exposure to fenbuconazole residues was estimated for the most recently approved tolerance in bananas (memorandum of E.A. Doyle, 8 February 1995). The EPA used the TMRC for pecans and bananas, and adjusted the TMRC for the stone fruit crop group by excluding plums/prunes and limiting sales volume to 12.8% of the available stone fruit market. From this EPA calculated an upper-bound risk of 0.9 x 10^{-6} for additional cancer risk ($Q1^* = 1.06 \times 10^{-2}$ (mg/kg/day)$^{-1}$). (**Federal Register** of May 24, 1995 (60 FR 27419)). This estimate does not reflect the change in $Q1^*$, the use of the DEEM database, the percent crop treated for all crops, or average residues. When the new $Q1^*$ of (0.36×10^{-2} (mg/kg/day)$^{-1}$)) and surface area estimated by (body weight)$^{3/4}$ plus the other factors are included the aggregate lifetime exposure for consumers to fenbuconazole has an upper bound cancer risk estimate of 1.0 x 10^{-8} for sugar beets and 0.17 x 10^{-6} for all pending and approved uses combined. The theoretical maximum estimated exposure to the most sensitive subpopulation, non-nursing infants less than one year old, for this same scenario utilizes no more than 0.01% of the RfD for sugar beets and 0.39% of the RfD for all crops combined. Thus, the addition of fenbuconazole use on sugar beets meets the EPA criterion of reasonable certainty of no harm.

2. *Drinking water*. Fenbuconazole has minimal tendency to contaminate groundwater or drinking water because of its adsorptive properties on soil, solubility in water, and degradation rate. Data from laboratory studies and field dissipation studies have been used in the USDA PRZM/GLEAMS computer model to predict the movement of fenbuconazole. The model predicts that fenbuconazole will not leach into groundwater, even if heavy rainfall is simulated. The modeling predictions are

consistent with the data from environmental studies in the laboratory and the results of actual field dissipation studies. There are no data on passage of fenbuconazole through water treatment facilities and there are no State water monitoring programs which target fenbuconazole.

3. *Non-dietary exposure.* Fenbuconazole has no veterinary applications and is not approved for use in swimming pools. It is not labeled for application to residential lawns or for use on ornamentals, nor is fenbuconazole applied to golf courses or other recreational areas. Therefore, there are no data to suggest that these exposures could occur. Any acute exposures to children would come from dietary exposure or inadvertent dermal contact. As previously discussed, fenbuconazole is neither orally or dermally acutely toxic. Thus, there is a reasonable certainty that no exposure would occur to adults, infants or children from these sources.

D. Cumulative Effects

The toxicological effects of fenbuconazole are related to the effects on rodent liver. These are manifest in rats and mice differently. Fenbuconazole causes liver toxicity in rats and mice in the form of hepatocyte enlargement and enzyme induction. In rats the liver enzyme induction causes increased biliary removal of thyroxin and the hepatotoxicity leads to elevated thyroid stimulating hormone levels with subsequent development of thyroid gland hyperplasia and tumors. This process is reversible and demonstrates a dose level below which no thyroid gland stimulation can be demonstrated in rats. Liver toxicity in the mouse is manifest by hepatocyte enlargement, enzyme induction, and hepatocellular hyperplasia (cell proliferation). These processes are associated with the appearance of a small number of liver tumors. In both cases, rats and mice, the initiating event(s) do not occur below a given dose, i.e., the effects are nonlinear, and the processes are reversible. Therefore, since the tumors do not occur at doses below which hepatocyte enlargement and enzyme induction occur, the RfD protects against tumors because it is substantially below the NOEL for liver effects and maximal human exposures are below the RfD. Effects on human thyroid will not occur at anticipated dietary levels. The mode of action data should be carefully considered in deciding the relevance of these high-dose animal tumors to human dietary exposure.

Extensive data are available on the biochemical mode of action by which fenbuconazole produces animal tumors in both rats and mice. However, there are no data which suggest that the mode of action by which fenbuconazole produces these animal tumors or any other toxicological effect is common to all fungicides of this class. In fact, the closest structural analog to fenbuconazole among registered fungicides of this class is not tumorigenic in animals even at maximally tolerated doses and has a different spectrum of toxicological effects.

E. Safety Determination

1. *U.S. population*— i. *Wheat.* The Rohm and Haas Company estimates the risk to the U.S. adult population from use of fenbuconazole on wheat as utilizing approximately 0.36% of the RfD. Using the EPA low dose extrapolation model and the risk factor based on the mouse data (0.36 x 10-6 $(mg/kg/day)^{-1}$) the excess cancer risk from dietary sources for fenbuconazole use on wheat and the associated animal commodities is estimated at 0.3×10^{-6}. The upper bound estimate on excess cancer risk for all uses including wheat is 0.7×10^{-6}.

This assumes that all of the wheat consumed in the U.S. will contain residues of fenbuconazole (in actuality a small fraction of the total crop is likely to be treated). The combined risk for wheat plus registered uses will not exceed either the dietary risk standard established by the Food Quality Protection Act (FQPA) for the US population, (one x 10^{-6}), or the RfD.

The sole acute risk would be for women of childbearing age. The EPA/OREB calculated that the worst-case Margin of Exposure (MOE) for fenbuconazole measured against the developmental LOEL would be greater than 30,000. This is clearly adequate. The MOE would be even higher for consumer dietary exposure from any source. Thus, there is adequate safety for this group and there is a reasonable certainty that no harm will result from fenbuconazole use on wheat.

ii. *Apples.* When the DEEM database is used and the assumptions in the above calculations the Rohm and Haas Company estimates the risk to the U.S. adult population from use of fenbuconazole on apples as utilizing approximately 0.17% of the RfD. The calculated upper bound estimate on excess cancer risk for all uses (apples, apricots, almonds, bananas, cherries, nectarines, peaches, pecans, and wheat, plus the associated processing and animal commodities) is 0.28×10^{-6}.

The combined risk for apples plus registered uses plus almonds and wheat will not exceed the dietary risk standards established by the FQPA for the US population (one x 10^{-6} excess cancer risk, or the RfD).

The sole acute risk would be for women of childbearing age. The EPA/OREB calculated that the worst-case Margin of Exposure (MOE) for fenbuconazole measured against the developmental LOEL would be greater than 30,000. This is clearly adequate. The MOE would be even higher for consumer dietary exposure from any source. Thus, there is adequate safety for this group and there is a reasonable certainty that no harm will result from fenbuconazole use on apples.

iii. *Almonds.* When the DEEM database is used and the assumptions in the above calculations the Rohm and Haas Company estimates the risk to the U.S. adult population from use of fenbuconazole on almonds as utilizing approximately 0.00007% of the RfD. The calculated upper bound estimate on excess cancer risk for all uses (apples, apricots, almonds, bananas, cherries, nectarines, peaches, pecans, and wheat, plus the associated processing and animal commodities) is 0.28×10^{-6}.

The combined risk for almonds plus registered uses plus apples and wheat will not exceed the dietary risk standards established by the FQPA for the US population (one x 10^{-6} excess cancer risk, or the RfD).

The sole acute risk would be for women of childbearing age. The EPA/OREB calculated that the worst-case Margin of Exposure (MOE) for fenbuconazole measured against the developmental LOEL would be greater than 30,000. This is clearly adequate. The MOE would be even higher for consumer dietary exposure from any source. Thus, there is adequate safety for this group and there is a reasonable certainty that no harm will result from fenbuconazole use on almonds.

iv. *Grapefruit.* When the DEEM database is used and the assumptions in the above calculations the Rohm and Haas Company estimates the risk to the U.S. adult population from use of fenbuconazole on grapefruit as utilizing approximately 0.06% of the RfD. The calculated upper bound estimate on excess cancer risk for all uses (apples, apricots, almonds, bananas, cherries, grapefruit, nectarines, peaches, pecans, sugar beets, and wheat, plus the associated processing and animal commodities) is 0.17×10^{-6}.

The combined risk for grapefruit plus registered and pending uses will not exceed the dietary risk standards established by the FQPA for the U.S.

population (one x 10-6 excess cancer risk, or the RfD).

The sole acute risk would be for women of childbearing age. The EPA/OREB calculated that the worst-case Margin of Exposure (MOE) for fenbuconazole measured against the developmental LOEL would be greater than 30,000. This is clearly adequate. The MOE would be even higher for consumer dietary exposure from any source. Thus, there is adequate safety for this group and there is a reasonable certainty that no harm will result from fenbuconazole use on grapefruit.

v. *Sugar beets.* When the DEEM database is used and the assumptions in the above calculations the Rohm and Haas Company estimates the risk to the U.S. adult population from use of fenbuconazole on sugar beets as utilizing approximately 0.009% of the RfD. The calculated upper bound estimate on excess cancer risk for all uses (apples, apricots, almonds, bananas, cherries, grapefruit, nectarines, peaches, pecans, sugar beets, and wheat, plus the associated processing and animal commodities) is 0.17×10^{-6}. Therefore, the combined risk for sugar beets plus registered and pending uses will not exceed the dietary risk standards established by the FQPA for the U.S. population (one $\times 10^{-6}$ excess cancer risk, or the RfD).

The sole acute risk would be for women of childbearing age. The EPA/OREB calculated that the worst-case Margin of Exposure (MOE) for fenbuconazole measured against the developmental LOEL would be greater than 30,000. This is clearly adequate. The MOE would be even higher for consumer dietary exposure from any source. Thus, there is adequate safety for this group and there is a reasonable certainty that no harm will result from fenbuconazole use on sugar beets.

2. *Infants and children*— i. *Wheat.* The reproductive and developmental toxicity data base for fenbuconazole is complete. There is no selective increase in toxicity to developing animals. Thus, there is no evidence that prenatal and postnatal exposure would present unusual or disproportionate hazard to infants or children. Therefore, there is no need to impose an additional uncertainty factor to protect infants and children.

The EPA calculated the dietary risk to infants and children for existing tolerances. The estimated dietary exposure (TMRC) for this subpopulation is 0.00522 mg/kg/day which represents only 17% of the RfD; no other subgroup used in excess of 17% of the RfD. The EPA estimated lifetime oncogenic risk in the range of one in a million at 0.9×10^{-6}, using ($Q1^* = 1.06 \times 10^{-2}$ (mg/kg/day)$^{-1}$). (**Federal Register** of May 24, 1995 (60 FR 27419)).

For the wheat use the most sensitive subgroup is children 1 to 6 years old and the estimated risk to this subgroup is less than 18% of the RfD. Utilizing the risk factor ($Q1^* = 0.36 \times 10^{-2}$ (mg/kg/day)$^{-1}$), the estimated excess cancer risk for the U.S. population is less than 1×10^{-6}. Therefore the wheat use is safe within the meaning of the FQPA and there is a reasonable certainty that no harm will result to infants or children from the approval of fenbuconazole use on wheat.

ii. *Apples and almonds.* The reproductive and developmental toxicity data base for fenbuconazole is complete. There is no selective increase in toxicity to developing animals. Thus, there is no evidence that prenatal and postnatal exposure would present unusual or disproportionate hazard to infants or children. Therefore, there is no need to impose an additional uncertainty factor to protect infants and children. The dietary exposure estimate for children utilizes only 0.89% of the RfD.

iii. *Grapefruit and sugar beets.* The reproductive and developmental toxicity data base for fenbuconazole is complete. There is no selective increase in toxicity to developing animals. Thus, there is no evidence that prenatal and postnatal exposure would present unusual or disproportionate hazard to infants or children. Therefore, there is no need to impose an additional uncertainty factor to protect infants and children. The dietary exposure estimate for children utilizes only 0.39% of the RfD.

F. Environmental Fate

Fenbuconazole has little to no mobility in soil (Koc = 4425). It is stable to hydrolysis and aqueous photolysis in buffered solutions, but does degrade photolytically in natural waters and soil (half-life 87 and 79 days, respectively). Laboratory soil metabolism half-lives or DT50 values for fenbuconazole range from 29 to 532 days under terrestrial conditions and from 442 to 906 days in soil exposed to aquatic conditions. Field-trial soil dissipation studies had half-lives ranging from 157 to 407 days and indicated no significant downward movement of residues. These field trials show fenbuconazole degrades more rapidly outdoors than in laboratory metabolism studies. When material was applied in a single application, fenbuconazole degraded to about 50% of the applied material in less than 60 days. In wheat the DT50 in green heads was measured as 18 days and in green wheat stalks the DT50 was 84.4 days. These results only reflect foliar dissipation in wheat at the particular growth stage(s) during the study and not at all stages of wheat. The results of residue decline analyses in a number of environmental media support the EPA conclusion that there is no environmental hazard associated with the proposed agricultural use of this chemical.

G. International Tolerances

There are no Codex Maximum Residue Levels (MRLs) for fenbuconazole, but the fenbuconazole database will be evaluated by the WHO and the FAO Expert Panels at the Joint Meeting on Pesticide Residues (JMPR) in September 1997. An Allowable Daily Intake (ADI (RfD)) of 0.03 mg/kg/day is proposed and a total of 36 Codex MRLs are proposed in the data submission. (PM 22)

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FEDERAL COMMUNICATIONS COMMISSION

[Report No. 2250]

Petitions For Reconsideration and Clarification of Action in Rulemaking Proceedings

January 27, 1998.

Petitions for reconsideration and clarification have been filed in the Commission's rulemaking proceedings listed in this Public Notice and published pursuant to 47 CFR Section 1.429(e). The full text of these documents are available for viewing and copying in Room 239, 1919 M Street, N.W., Washington, D.C. or may be purchased from the Commission's copy contractor, ITS, Inc. (202) 857–3800. Oppositions to these petitions must be filed February 17, 1998. See Section 1.4(b)(1) of the Commission's rule (47 CFR 1.4(b)(1)). Replies to an opposition must be filed by February 24, 1998.

Subject: Amendment of the Commission's Regulatory Policies to Allow Non-U.S.-Licensed Space Stations to Provide Domestic and International Satellite Service in the United States (IB Docket No. 96–111).

Amendment of Section 25.131 of the Commission's Rules and Regulations to Eliminate the Licensing Requirement for Certain International Receive-Only Earth Stations (CC Docket No. 93–23, RM–7931).