Summary: EPA expressed environmental concerns regarding potential herbicide contamination of ground waters, and surface waters, including wetlands and indicated that there was insufficient information in the DEIS regarding ground water depths, soil types, wetlands, and the water quality/aquatics monitoring program. The Final EIS should fully assess and mitigate all potential impacts of the management actions.

ERP No. D–BLM–J65267–WY Rating EO2, Gillette South Coal Bed Methane Project, Approval of an Application for a Permit to Drill (APD), Powder River Basin, Buffalo Resource Area, Campbell County, WY.

Summary: EPA expressed environmental objections to the proposed action due to potential air quality, water quality (surface discharge and ground water depletion) and wildlife adverse impacts. EPA requested the above issues be addressed in the Final EIS.

ERP No. D–UAF–G11031–TX Rating EC2, Programmatic EIS—Kelly Air Force Base (AFB), Disposal and Reuse, Implementation, San Antonio County, TX.

Summary: EPA had expressed environmental concerns and has requested additional information including noise impact mitigation.

ERP No. DS–BLM–J65191–00 Rating EC2, Standards for Rangeland Health and Guidelines for Livestock Grazing Management on Bureau of Land Management Administered Lands, Implementation, MT, ND and SD.

Summary: EPA expressed environmental concerns regarding protection of surface water quality and the ability to achieve water quality standards. The Final EIS should fully assess and mitigate all potential impacts of the management actions.

FINAL EISs

ERP No. F–COE–L36104–WA, Howard A. Hanson Dam Continued Operation and Maintenance Plan, Implementation, Green River, King County, WA.

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

ERP No. F–DOE–L09811–00, Wildlife Mitigation Program Standards and Guidelines, Implementation, Columbia River Basin, WA, OR, ID, MT, WY and NV.

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

ERP No. F–FHW–L40198–WA, North Spokane Freeway Project, Improvements Transportation through the City of Spokane and Spokane County between I–90, Spokane County, WA.

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

ERP No. F–NPS–K65187–CA, Santa Rosa Island Resources Management Plan, Improvements of Water Quality and Conservation of Rare Species and their Habitats, Channel Islands National Park, Santa Barbara County, CA.

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

ERP No. F–USA–H11004–MO, U.S. Army Chemical School and U.S. Army Military Police School Relocation to Fort Leonard Wood (FLW) from Fort McClellan, Alabama, Implementation, Cities of St. Robert, Waynesville, Richland, Dixon, Crocker, Rolla, Houston and Lebanon; Pulaski, Texas, Phelps and Laclede Counties, MO.

Summary: EPA expressed environmental objections to unknown human health risks and ecological risks resulting from generated oil fog used during obscurant training. Also EPA objected to initiating any new activities at the installation during the air quality permit renewal process which were not specifically evaluated with the preferred alternative within the EIS. EPA suggested that there be continuing public involvement during implementation of the preferred alternative.

Dated: June 17, 1997.

William D. Dickerson,

Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 97–16234 Filed 6–19–97; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–5844–9]

Clean Air Act Advisory Committee; Mobile Source Technical Advisory Subcommittee Notification of Public Advisory Subcommittee Open Meeting

Pursuant to the Federal Advisory Committee Act, Public Law 92–463, notice is hereby given that the Mobile Source Technical Advisory Subcommittee of the Clean Air Act Advisory Committee will meet on July 16, 1997 at 9:30 am to 4 pm (Eastern Standard Time) at Dupont Plaza Hotel—Embassy Hall, 1500 New Hampshire Avenue, N.W., Washington, DC 20036, Ph: 800/841–0003. This is an open meeting and seating will be on a first-come basis. During this meeting, the subcommittee will hear progress reports from its workgroups, approve its report to the Clean Air Act Advisory Committee, and be briefed on and discuss other current issues in the mobile source program.

Members of the public requesting further technical information should contact Philip A. Lorang, Designated Federal Officer of the Mobile Sources Technical Review Subcommittee of FACA, at the U.S. EPA, 2565 Plymouth Road, Ann Arbor, MI 48105 at 313/668–4374, fax 313/741–7821, or email lorang.phil@epamail.epa.gov. Members requesting further administrative information should contact Jennifer Criss, Mobile Sources Technical Advisory Subcommittee Management Officer at the U.S. EPA, 2565 Plymouth Road, Ann Arbor, MI 48105 313/668–4518 FACA Helpline, fax 313/741–7821, or email criss.jennifer@epamail.epa.gov. Written comments of any length (with at least 20 copies provided) should be sent to the subcommittee no later than July 4, 1997.

The Mobile Source Technical Advisory Subcommittee expects that public statements presented at its meetings will not be repetitive of previously submitted oral or written statements.

Margo T. Oge,

Director, Office of Mobile Sources.

[FR Doc. 97–16211 Filed 6–19–97; 8:45 am]

BILLING CODE 6560–50–M

ENVIRONMENTAL PROTECTION AGENCY

[PF–743; FRL–5723–7]

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–743, must be received on or before July 21, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7506C), 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. 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/Regulatory Leader listed in the table below:

Product Manager/Regulatory Leader	Office location/telephone number	Address
Marion Johnson (PM 10)	Rm. 210, CM #2, 703–305–6788, e-mail:johnson.marion@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Indira Gairola (Reg. Leader).	4th floor, CS #1, 703–308–8371, e-mail: gairola.indira@epamail.epa.gov.	2800 Crystal Drive, Arlington, VA

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

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–743] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket number [PF–743] 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: June 12,1997

James Jones,

Acting Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim without editing them in any way. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Rhone-Poulenc Ag Company

PP-7F4832

EPA has received pesticide petition PP-7F4832 from Rhone-Poulenc Ag Company, P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709. This petition proposes, pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C.346a, to amend 40 CFR part 180 by establishing a tolerance for the combined residues of the insecticide fipronil (5-amino-1-[2,6-dichloro-4-(trifluoro-methyl)phenyl]-4-[1*R, S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile; and 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile; and 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(1*RS*)-(trifluoromethyl)]-1*H*-pyrazole-3-carbonitrile on or in the following raw agricultural commodities: potatoes at 0.02 parts per million (ppm), sweet potatoes at 0.02 ppm, rice grain at 0.02 ppm, rice straw at 0.10 ppm, cottonseed at 0.05 ppm, and cotton gin trash at 3.0 ppm. The proposed analytical method is by gas chromatography using a Ni63 electron capture or mass selective detector. 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 this petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Metabolism*. The metabolism of fipronil is adequately understood. Adequate data on the nature of the residues in both plant and animals, including identification of major metabolites and degradates of fipronil, are available. In plants and animal the metabolism of fipronil proceeds via oxidation of the sulfoxide to yield sulfone MB 46136 and hydrolysis of nitrile to yield amide RPA 200766. A limited amount of reduction of sulfoxide to yield sulfide MB 45950 occurs in some cases. In cases where

fipronil is exposed to light for extended periods of time (i.e., foliar applications), photo products MB 46513 and RAP 104615 are often observed. Further transformation of the primary metabolites affords minor amounts of carboxylic acid RPA 200761, amide RPA 105320 and 4-protiopyrazole MB 45897.

2. *Practical analytical method*. Validated analytical methods are available for detecting and measuring levels of fipronil and its metabolites in field corn, cotton, potato and rice raw agricultural commodities and their respective processing fractions and animal tissues. Residues are extracted from corn grain, fodder and forage with 75:25 acetonitrile: water and from the remaining corn substrates with acetonitrile. Acetonitrile: water is also used to extract residues from cottonseed, cotton gin by-products (gin trash), hulls and meal and rice grain and straw. An aliquot of the extract is partioned against hexane to remove lipids. After the addition of water and the removal of acetonitrile, fipronil and its metabolites are then partitioned into dichloromethane. Column chromatography is utilized for clean up / removal of coextractive unknowns. For potato tubers, wet peel, dry peel, flakes and chips and animal tissues, the extraction solvent is a mixture of acetonitrile:acetone (70:30). Samples clean up is effected by column chromatography. Quantification of fipronil and its metabolites is accomplished by gas chromatography using a Ni63 electron capture or mass selective detector.

B. Toxicology Profile

1. *Acute toxicity*. The acute oral LD_{50} in rats is 97 mg/kg. The dermal LD_{50} values in rats and rabbits are greater than 2,000 mg/kg and 354 mg/kg, respectively. The inhalation LC_{50} for a 4-hour exposure (nose only) is 0.39 mg/L. Slight skin and moderate eye irritation are observed in rabbits with complete clearing within 7 days for skin and 14 days for eye. Fipronil is not a dermal sensitizer in guinea pigs (Buehler method).

2. *Genotoxicity*. Fipronil was negative in both *in vitro* and *in vivo* assays conducted to investigate gene mutations, DNA damage, and chromosomal aberrations.

3. *Developmental/reproductive effects*. Rat and rabbit developmental toxicity studies were negative at doses up to 20 mg/kg/day and 1 mg/kg/day, respectively. In a two-generation rat study, the NOEL for reproductive toxicity was 30 ppm (2.64 mg/kg/day for both sexes combined).

4. *Subchronic effects*. The NOELs in rats and dogs were 5 ppm (0.35 mg/kg/day for both sexes combined) and 2 mg/kg/day, respectively.

5. *Chronic effects*. The NOELs in 1-year dietary dog and 2-year dietary rat studies were 0.3 mg/kg/day and 0.5 ppm, respectively, based on clinical signs. The chronic Reference Dose (RfD) of 0.0002 mg/kg/day established by EPA is based on the NOEL from the chronic rat study (equivalent to 0.02 mg/kg/day in male rats and 0.03 mg/kg/day in female rats) divided by an uncertainty factor of 100 to account for inter- and intra-species variation.

6. *Carcinogenicity*. Fipronil was not carcinogenic when administered to mice at any dose level tested. In rats, thyroid tumors were observed only at 300 ppm (highest dose tested) (HDT). Mechanistic data indicate that these tumors are related to an imbalance of thyroid hormones and are specific to the rat. EPA's Health Effects Division Carcinogenicity Peer Review Committee classified fipronil in Group C and recommended that RfD methodology, i.e. non-linear or threshold, be used for the estimation of human risk.

7. *Endocrine effects*. No evidence of estrogenic or androgenic effects were noted in any study with fipronil. No adverse effects on mating or fertility indices and gestation, live birth, or weaning indices were noted in a two-generation rat reproduction study. In a developmental neurotoxicity study, devlopment of pups was delayed only at a dose producing maternal toxicity which resulted in smaller, less developed pups. However, even in the presence of maternal toxicity, the pups developed fully and were comparable to controls by study termination.

C. Aggregate exposure/cumulative effects

1. *Dietary exposure*. A chronic dietary assessment for fipronil use in/on corn demonstrates that the most realistic scenario, i.e. anticipated residues with estimated market share, results in exposures of less than 32% of the RfD for all subgroups including the most sensitive subgroup, children 1 to 6 years of age. Therefore, chronic dietary exposure to fipronil residues from both primary and secondary sources, as a result of its use on field corn, potatoes, rice, and cotton does not represent a significant risk to any segment of the population.

An acute dietary analysis using tolerances, 100% market share, and a NOAEL of 5.0 mg/kg from the acute neurotoxicity study results in Margins of Exposure (MOEs) for all segments of the population of over 2,000 for the 95th percentile and over 1,000 for both the 99th and 99.9th percentile. A more realistic assessment using anticipated residues would result in considerably higher MOEs. However, even with extremely conservative assumptions, sufficient MOEs exist for acute dietary exposure to fipronil residues from both primary and secondary sources. Therefore, fipronil use on field corn, potatoes, rice, and cotton does not represent a significant acute dietary risk to any segment of the population.

2. *Drinking water exposure*. The combined factors of low mobility, moderate persistence, and low application rates result in fipronil and its metabolites having little potential to reach groundwater as a result of movement through the soil profile or of surface run-off. Thus, the potential for ground water and/or surface water contamination by fipronil and its degradates is expected to be very low.

3. *Non-occupational exposure* . Fipronil is currently registered for use on golf and commercial turfgrass under the brand name CHIPCO CHOICETM and for treatment of cats and dogs for fleas and ticks under the brand name FRONTLINE. These uses are not expected to contribute significantly to overall exposure. Fipronil has an extremely low vapor pressure and low dermal penetration. These properties minimize the amount of actual exposure that might occur. The application of fipronil on golf and commercial turf using a slit applicator which places the granule well into or below the thatch reduces the likelihood of post application exposure. Further, as these areas have only limited human activity involving minimal dermal contact with treated turf, potential exposure is expected to be negligible. Exposure due to the application of FRONTLINE is also expected to be low. The particle size characteristics of the spray product result in negligible inhalation exposure while the use of gloves, as required on the label in conjunction with the low dermal penetration rate of fipronil, result in minimal exposure via the dermal route. The affinity of fipronil for the sebum and hair of animals and its one to three month efficacy indicate that the material remains on the pet and is not bioavailable to those coming in contact with the pet. Pending uses which include use of fipronil as a termiticide and use in ant/roach baits are also anticipated to present negligible exposure.

4. *Cumulative risk*. Fipronil belongs to a novel chemical class of insecticides known as phenylpyrazoles. It is the only compound from this class of chemistry registered for use as an insecticide.

Fipronil exhibits a mode of action different from traditional organophosphate, carbamate, or pyrethroid insecticides. Fipronil acts by binding within the chloride channel of the GABA receptor. There is no indication that effects from fipronil would be cumulative with any other pesticide.

D. Safety Determinations

1. *U.S General population*. Both aggregate and dietary exposure assessments demonstrate that all current and pending uses of fipronil do not pose any significant risk to the general population. Therefore, based on a very complete database, there is reasonable certainty that no harm will result from aggregate exposure to the chemical residue including all anticipated dietary exposures and all other exposures for which there is reliable information.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fipronil, the available developmental and reproductive toxicity studies were considered. Developmental toxicity studies in two species indicate that fipronil has no teratogenic potential at any dose level. Further, no adverse effects on fetal development were observed in rats or rabbits even in the presence of maternal toxicity. In a two-generation rat reproduction study, effects on pups were seen only at the highest dose tested in the presence of parental toxicity. In a developmental neurotoxicity study, development of pups was delayed only at a dose producing maternal toxicity which resulted in smaller, less developed pups. However, even in the presence of maternal toxicity, the pups developed fully and were comparable to controls by study termination. Thus, maternal and developmental NOELs and LELs were comparable in all studies indicating no increase susceptibility of developing organisms. Further, the NOEL of 0.02 mg/kg/day from the 2-year rat study, which was used to calculate the RfD for fipronil, is already lower than the NOELs from developmental studies by a factor of 45 to 1,000 times. As a hundredfold uncertainty factor is already used to calculate the RfD which is based on a NOEL significantly lower than NOELs from all developmental and reproductive studies, an additional uncertainty factor is not warranted and the RfD of 0.0002 mg/kg/day is appropriate for assessing risk to infants and children.

E. International Tolerances

There are no Codex maximum residue levels established for fipronil. (Marion Johnson)

2. Rhone-Poulenc Ag Company

PP-5F4426

EPA has received pesticide petition (PP) 5F4426 from Rhone-Poulenc Ag Company, P.O. Box 12014, 2 T.W. Alexander Drive, Research Triangle Park, NC 27709. This petition proposes, pursuant to section 408(d) of the Federal Food, Drug and Cosmetic Act (FFDCA), 21 U.S.C. 346a, to amend 40 CFR part 180 by establishing a tolerance for the combined residues of the insecticide fipronil (5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[1*R, S*)-(trifluoromethyl)sulfinyl]-1*H*-pyrazole-3-carbonitrile) and its metabolites 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluor omethyl) sulfonyl]-1*H*-pyrazole-3-carbonitrile; and 5-amino-1-[2,6-dichloro-4-(trifluoromethyl)phenyl]-4-[(trifluoromethyl)thio]-1*H*-pyrazole-3-carbonitrile on or in the following raw agricultural commodities: corn grain at 0.02 parts per million (ppm), corn forage at 0.15 ppm and corn stover at 0.15 ppm; in the animal product commodities of cattle, goats, horses and sheep: fat at 0.40 ppm, liver at 0.10 ppm, meat at 0.04 ppm, meat by-products (except liver) at 0.04 ppm, beef kidney at 0.03 ppm, and milk fat at 0.70 ppm; in the animal product commodities of hogs: fat at 0.04 ppm, liver at 0.02 ppm, meat at 0.01 ppm and meat by-products (except liver) at 0.01 ppm; in the animal product commodities of poultry: eggs at 0.03 ppm, fat at 0.05 ppm and meat at 0.02 ppm. The proposed analytical method is by gas chromatography using a Ni63 electron capture or mass selective detector. 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 this petition. Additional data may be needed before EPA rules on the petition.

As required by section 408(d) of the FFDCA, as recently amended by the Food Quality protection Act (FQPA), Rhone-Poulenc Ag Company included in the petition a summary of the petition and authorization for the summary to be published in the Federal Register in a notice of receipt of the petition. The summary represents the views of Rhone-Poulenc Ag Company; EPA is in the process of evaluating the petition. As required by section 408 (d)(3), EPA is including the summary as a part of this notice of filing. EPA may have made minor edits to the summary for the purpose of clarity.

A. Residue Chemistry

1. *Metabolism*. The metabolism of fipronil is adequately understood. Adequate data on the nature of the residues in both plant and animals, including identification of major metabolites and degradates of fipronil, are available. In plants and animal the metabolism of fipronil proceeds via oxidation of the sulfoxide to yield sulfone and hydrolysis of nitrile to yield the amide. Fipronil and its sulfone and amide constitute greater than 75% of the identified residues in all studies. A limited amount of reduction of sulfoxide to yield the sulfide occurs in some cases. Further transformation of the primary metabolites affords minor amounts of the carboxylic acid, the amide and the 4-protiopyrazole.

2. *Practical analytical method*. A validated analytical method is available for detecting and measuring levels of fipronil and its metabolites in field corn raw agricultural commodities (grain, forage and fodder) and its processing fractions (oil and starch). Residues are extracted from corn grain, fodder and forage with 75:25 acetonitrile:water and from the remaining corn substrates with acetonitrile. An aliquot of the extract is partitioned against hexane to remove lipids. After the addition of water and the removal of acetonitrile, fipronil and its metabolites are partitioned into dichloromethane. Column chromatography is utilized for clean up / removal of coextractive unknowns. Quantification of fipronil and its metabolites is accomplished by gas chromatography using a Ni63 electron capture or mass selective detector.

B. Toxicology Profile

1. *Acute toxicity*. The acute oral LD_{50} in rats is 97 mg/kg. The dermal LD_{50} values in rats and rabbits are greater than 2,000 mg/kg and 354 mg/kg, respectively. The inhalation LC_{50} for a 2-hour exposure (nose only) is 0.39 mg/L. Slight skin and moderate eye irritation are observed in rabbits with complete clearing within 7 days for skin and 14 days for eye. Fipronil is not a dermal sensitizer in guinea pigs (Buehler method).

2. *Genotoxicity*. Fipronil was negative in both *in vitro* and *in vivo* assays conducted to investigate gene mutations, DNA damage, and chromosomal aberrations.

3. *Developmental/reproductive effects*. Rat and rabbit developmental

toxicity studies were negative at doses up to 20 mg/kg/day and 1 mg/kg/day, respectively. In a 2–generation rat study, the NOEL for reproductive toxicity was 30 ppm (2.64 mg/kg/day for both sexes combined).

4. *Subchronic effects*. The NOELs in rats and dogs were 5 ppm (0.35 mg/kg/day for both sexes combined) and 2 mg/kg/day, respectively.

5. *Chronic effects*. The NOELs in 1-year dietary dog and 2-year dietary rat studies were 0.3 mg/kg/day and 0.5 ppm, respectively, based on clinical signs. The chronic Reference Dose (RfD) of 0.0002 mg/kg/day established by EPA is based on the NOEL from the chronic rat study (equivalent to 0.02 mg/kg/day in male rats and 0.03 mg/kg/day in female rats) divided by an uncertainty factor of 100 to account for inter- and intra-species variation.

6. *Carcinogenicity*. Fipronil was not carcinogenic when administered to mice at any dose level tested. In rats, thyroid tumors were observed only at 300 ppm (HDT). Mechanistic data indicate that these tumors are related to an imbalance of thyroid hormones and are specific to the rat. EPA's Health Effects Division Carcinogenicity Peer Review Committee classified fipronil in Group C and recommended that RfD methodology, i.e. non-linear or threshold, be used for the estimation of human risk.

7. *Endocrine effects*. No evidence of estrogenic or androgenic effects were noted in any study with fipronil. No adverse effects on mating or fertility indices and gestation, live birth, or weaning indices were noted in a two-generation rat reproduction study. In a developmental neurotoxicity study, development of pups was delayed only at a dose producing maternal toxicity which resulted in smaller, less developed pups. However, even in the presence of maternal toxicity, the pups developed fully and were comparable to controls by study termination.

C. Aggregate Exposure/Cumulative Effects

1. *Dietary exposure*. A chronic dietary assessment for fipronil use in/on corn demonstrates that the most realistic scenario, i.e. anticipated residues with estimated market share, results in exposures of less than 3% of the RfD for all subgroups including the most sensitive subgroup, children 1 to 6 years of age. Scenarios using tolerances and estimated market share, as well as anticipated residues and 100% crop treated, demonstrated exposures of less than 40% of the RfD for the most sensitive subgroup (children 1 to 6 years of age) and less than 15% of the RfD for the US population in general. Therefore, chronic dietary exposure to fipronil residues from both primary and secondary sources, as a result of its use on field corn, does not represent a significant risk to any segment of the population.

An acute dietary analysis using tolerances, assuming fipronil in milk fat only with a tolerance of 0.7 ppm, 1989-92 consumption data, and a NOAEL of 5.0 mg/kg from the acute neurotoxicity study results in Margins of Exposure (MOEs) for all segments of the population of over 2,000 for the 95th percentile and over 1,000 for both the 99th and 99.9th percentile. A more realistic assessment using anticipated residues would result in considerably higher MOEs. However, even with extremely conservative assumptions, sufficient MOEs exist for acute dietary exposure to fipronil residues from both primary and secondary sources. Therefore, fipronil use on field corn does not represent a significant acute dietary risk to any segment of the population.

2. *Drinking water exposure*. The combined factors of low mobility, moderate persistence, low application rates, and in-furrow application result in fipronil and its metabolites having little potential to reach groundwater as a result of movement through the soil profile or of surface run-off. Thus, the potential for ground water and/or surface water contamination by fipronil and its degradates is expected to be very low.

3. *Non-occupational exposure*. Fipronil is currently registered for use on golf and commercial turfgrass under the brand name CHIPCO CHOICETM and for treatment of cats and dogs for fleas and ticks under the brand name FRONTLINE . These uses are not expected to contribute significantly to overall exposure. Fipronil has an extremely low vapor pressure and low dermal penetration. These properties minimize the amount of actual exposure that might occur. The application of fipronil on golf and commercial turf using a slit applicator which places the granule well into or below the thatch reduces the likelihood of post application exposure. Further, as these areas have only limited human activity involving minimal dermal contact with treated turf, potential exposure is expected to be negligible. Exposure due to the application of FRONTLINE is also expected to be low. The particle sizecharacteristics of the spray product result in negligible inhalation exposure while the use of gloves, as required on the label in conjunction with the low dermal penetration rate of fipronil, result in minimal exposure via the dermal route. The affinity of fipronil for the sebum and hair of animals and its one to three month efficacy indicate that the material remains on the pet and is not bioavailable to those coming in contact with the pet. Pending uses which include use of fipronil as a termiticide and use in ant/roach baits are also anticipated to present negligible exposure.

4. *Cumulative risk*. Fipronil belongs to a novel chemical class of insecticides known as phenylpyrazoles. It is the only compound from this class of chemistry registered for use as an insecticide. Fipronil exhibits a mode of action different from traditional organophosphate, carbamate, or pyrethroid insecticides. Fipronil acts by binding within the chloride channel of the GABA receptor. There is no indication that effects from fipronil would be cumulative with any other pesticide.

D. Safety Determinations

5. *U.S. general population*. Both aggregate and dietary exposure assessments demonstrate that all current and pending uses of fipronil do not pose any significant risk to the general population. Therefore, based on a very complete database, there is reasonable certainty that no harm will result from aggregate exposure to the chemical residue including all anticipated dietary exposures and all other exposures for which there is reliable information.

6. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of fipronil, the available developmental and reproductive toxicity studies were considered. Developmental toxicity studies in two species indicate that fipronil has no teratogenic potential at any dose level. Further, no adverse effects on fetal development were observed in rats or rabbits even in the presence of maternal toxicity. In a two-generation rat reproduction study, effects on pups were seen only at the highest dose tested in the presence of parental toxicity. In a developmental neurotoxicity study, development of pups was delayed only at a dose producing maternal toxicity which resulted in smaller, less developed pups. However, even in the presence of maternal toxicity, the pups developed fully and were comparable to controls by study termination. Thus, maternal and developmental NOELs and LELs were comparable in all studies indicating no increase susceptibility of developing organisms. Further, the NOEL of 0.02 mg/kg/day from the 2-year rat study, which was used to calculate the RfD for fipronil, is already lower

than the NOELs from developmental studies by a factor of 45 to 1,000 times. As a hundredfold uncertainty factor is already used to calculate the RfD which is based on a NOEL significantly lower than NOELs from all developmental and reproductive studies, an additional uncertainty factor is not warranted and the RfD of 0.0002 mg/kg/day is appropriate for assessing risk to infants and children.

E. International Tolerances

There are no Codex maximum residue levels established for fipronil. (Marion Johnson)

3. Zeneca Ag Products

PP-6E4675

EPA has received a pesticide petition (PP 6E4675) from Zeneca Ag Products, 1800 Concord Pike, P.O. Box 15458, Wilmington, Delaware 19850–5458, proposing pursuant to 408(e) of the Federal Food, Drug, and Cosmetic Act, 21 U.S.C. 346a(e), to amend 40 CFR 180.1001(d) by establishing an exemption from the requirement for a tolerance for residues of the inert ingredient titantium dioxide when used in pesticide formulations used on growing crops.

Pursuant to section 408 (d)(2)(A)(i) of the FFDCA, as amended, Zeneca Ag Products has submitted the following summary of information, data and arguments in support of their pesticide petition. This summary was prepared by Zeneca and EPA has not fully evaluated the merits of the petition. EPA edited the summary to clarify that the conclusions and arguments were the petitioner's and not necessarily EPA's.

A. Residue Chemistry

Titanium(Ti) is the eighth most abundant element in the earth's crust and consequently spontaneously enters the food chain to some degree. Humans are estimated to consume approximately 300 µg Ti/day in food. Since the various forms of titanium, including titanium dioxide, are so abundant as a background element, estimations of residues resulting from use as an inert ingredient in a pesticide formulation would not be of value in determining the overall impact of this particular use.

Analytical method. There are two approved AIHA methods for analysis of titanium residues: (1) Hydrogen peroxide colormetric method with a sensitivity of 2 µg Ti; and (2) Atomic absorption with a sensitivity of 1.9 µg/ml.

B. Toxicological Profile

Titanium dioxide (TiO_2) is the most commercially important of all the titanium compounds. TiO_2 is an opaque powder that is approved for use as a colorant in cosmetics (21 CFR 73.2575 and 21 CFR 73.3126), pharmaceuticals (21 CFR 73.575) and foods, as well as in an extensive range of industrial uses (e.g. paper, paints, enamels and plastics) throughout the world. Titanium dioxide is exempt from the requirement for a tolerance when used as a colorant in pesticide formulations (40 CFR 180.1001). In the **Federal Register** of June 20, 1988, EPA announced that it was deleting titanium dioxide from the list of toxic chemicals under section 313 of Title III of the Superfund Amendments. This rule concluded that titanium dioxide will not cause significant adverse effects to humans or to the environment.

The wide range of relatively unrestricted uses of titanium dioxide reflects the fact that the compound is held to be toxicologically inert, belonging to that group of materials classified as ≥Generally Accepted as Safe≥ (GRAS). The scientific committee on food coloring materials determined that no ADI need be set for the use of titanium dioxide, as its use does not present any health concerns (1983). Indeed, titanium dioxide is frequently used as a negative control material *in vivo* chronic dust exposure studies and *in vivo* assessments of fibrogenic potential of dusts.

1. *Acute toxicity.* Titanium dioxide (TiO_2) has very low acute toxicity with no deaths in rats administered as much as 24 grams/Kg. No overt signs of toxicity occurred in a person that ingested approximately 1 pound of TiO_2. Skin and eye contact to the dry powder produced no irritation to the skin and very slight irritation to the eyes. An acute 4-hour inhalation exposure at concentrations of 6.82 mg/L produced no mortalities. Intratracheal administration also indicated a low level of acute toxicity. In a 2-week inhalation study, rats exposed to 1.92 mg/L showed a typical dust-cell reaction. Additionally, only a typical dust-cell reaction was noted in rats exposed to 1 mg/L from 4-weeks up to 1-year.

2. *Genotoxicity.* Titanium dioxide has no genotoxic potential as judged from unequivocal negatives in a range of studies *in vitro* and *in vivo*.

3. *Reproductive and developmental toxicity.* No relevant data are available for this material. However, the OECD Screening Information Data Set (SIDS) Manual for 1996, which contains chemical data and regulatory decisions agreed by scientists within the European Community, stated that due to a lack of toxicity resulting from subchronic and chronic exposure to titanium dioxide, specific testing for reproductive and developmental toxicity were not required for TiO_2.

4. *Subchronic toxicity.* Repeated doses ranging from 800 to 1,500 mg/kg of Titanium dioxide for 2-13 months did not produce adverse effects in all species tested. Some of these studies were limited in terms of the number of animals used (group sizes were 1 to 4).

In a comprehensive study reported as part of the NCI program, groups of 50 male and 50 female F344 rats or B6C3F1 mice were fed diets containing 25,000 or 50,000 ppm titanium dioxide for 103 weeks. Even though these doses (equivalent to 1.25 g/kg or 2.5 g/kg in rats and 3.75 g/kg or 7.5 g/kg in mice) were very high (well in excess of the modern guideline limit dose of 20,000 ppm in rat or 7,000 ppm in mouse), there was no significant evidence of chronic toxicity.

5. *Chronic toxicity* —a. *Carcinogenicity*. In an NCI study groups of 50 male and 50 female F344 rats or B6C3F1 mice were fed diets containing 25,000 ppm or 50,000 ppm titanium dioxide for 103 weeks. There were no compound-related increases in tumors. There was a non-statistically significant increase in C-cell adenoma and of thyroid carcinoma in female rats which, it was concluded, was unrelated to titanium dioxide.

In a study in which F344 rats were fed diets containing up to 5% mica coated with titanium dioxide there was no increase in tumors. In addition, there were no tumors in rats or mice injected intraperitoneally (single or multiple doses) or subcutaneously and observed for periods of 18 months or longer.

There are no epidemiological studies following purely oral exposure to titanium dioxide. However, in studies of factory workers exposed to titanium dioxide dust (primarily via inhalation) there was no evidence of increased cancers.

b. *Pulmonary effects of eitanium dioxide.* TiO_2 is considered generally to be inert and this is confirmed by the very low acute inhalation toxicity (LC_{50} 6.82 mg/L). Single administration of TiO_2 by intratracheal instillation may produce changes in the alveolar cell population, lung lining fluid components and lung tissues. Such changes, the majority of which reversed rapidly even with very high lung loading, were consistent with administration of a relatively high dose of an inert, insoluble dust into the lung. The acceptance that TiO_2 is relatively inert in the lung has led to the use of this as a negative control in many studies investigating the pulmonary

effects of particles. Results in the majority of these studies are again consistent with the inert nature of this material.

A number of repeat exposure inhalation studies have been conducted to investigate either the inherent toxicity of TiO_2 or again to investigate the response of the lung to exposure to inert particles. The majority of studies demonstrate that sub-chronic and chronic exposure to realistic concentrations of TiO_2 result in minimal changes consistent with a steady accumulation of inert particles in the lung.

In a 2-year inhalation study, groups of 200 rats were exposed 6-hours a day, 5-days a week to 10, 50 or 250 mg/m of TiO_2. Survival of the exposed animals was comparable to that of the control group, and there were no compound-related clinical signs of toxicity at any dose level.

In rats, white foci of accumulated material were apparent on the visceral surface and throughout the lung parenchyma at gross necropsy. At 10mg/m this was minimal but marked increases were noted at 50 mg/m and particularly at 250 mg/m. Microscopically, these foci represented not only aggregates of dust or dust containing cells but in most instances the additional biological response of the lung (e.g. pleurisy, collagenized fibrosis associated with cholesterol granulomas, alveoli bronchiolarization, pneumonia, and alveolar cell hyperplasia) to the persistent presence of inert particles. At 250 mg/m in this study, and at 10 mg/m in a subsequent study using a different type (ultrafine) of TiO_2, resulted in an increased incidence of lung tumors at termination. These tumors were either broncho-alveolar or epidermoid/squamous. Such tumors are now known to be a common response of the lung to excessive lung burdens of insoluble dusts, are seen only in the rat and are of questionable relevance to man.

A case-control epidemiology study of male employees exposed to titanium dioxide did not demonstrate an increased risk for lung cancer. In addition, there was no dose-response relationship between titanium dioxide exposure and chronic respiratory disease, pleural thickening, pleural plaques, or pleural nodules.

6. *Animal metabolism*. Data on the absorption of titanium compounds is limited. When male and female rats were fed diet containing 100 g titanium dioxide per kg of diet, for about 32 days, no retention of titanium occurred in the liver, spleen, kidney, bone, plasma or erythrocytes. However, there were measurable amounts (0.06 and 0.11 mg/kg wet weight) in the muscles. Following intravenous injection of 250mg titanium dioxide/kg to rats, there was an exponential disappearance rate from the blood with only about 30% remaining after 10 minutes. Seventy percent of the injected dose was detected in the liver after 5 minutes, rising to almost 80% by 15 minutes. The organ with the next highest concentration was the spleen, after 6 hours. By 24 hours, the highest concentration was in the celiac lymph nodes that drain the lymph from the liver. 1-year after the single injection, the highest tissue concentrations (178.9 mg/gm) were still in these lymph nodes.

7. *Human data*. In a study involving five adult males, each of whom consumed 5g on three consecutive days, there was no significant increase in urinary content of titanium indicating there had been no significant absorption/excretion of the compound. However, titanium dioxide has been found in the lymphatic systems of three workers employed in processing titanium dioxide pigments, indicating the compound can access the tissues, following inhalation exposure. Titanium dioxide is also known to have a long residence time (in the order of a year) in the lung.

C. Aggregate Exposure

Titanium dioxide is currently approved for use in a significant number of pharmaceutical, cosmetic, industrial and food products. Therefore, the potential for aggregate exposure from dietary and non-dietary routes does exist for titanium dioxide. However, the use of titanium dioxide as an inert in a pesticide formulation would not be expected to significantly raise the background levels found naturally in the food chain or general environment. Also, since the acute, subchronic and chronic toxicity testing has shown titanium dioxide to be physiologically inert, there is no concern for adverse health effects resulting from potential aggregate exposures.

D. Cumulative effects

Because of the low toxicity of titanium dioxide and because its presence in the environment is primarily naturally-occurring, Zeneca does not believe that there is any reason to be concerned about the potential for cumulative effects of titanium dioxide and other substances that may have a common mechanism of toxicity.

E. Safety Determination

Titanium dioxide has been shown to be physiologically inert by most routes of exposure, and is classified as ≥Generally Accepted as Safe≥ (GRAS). Based on this information, Zeneca believes that is a reasonable certainty that no harm will result to infants, children, or the general population from aggregate exposure to titanium dioxide residues.

F. Existing Tolerances or Tolerance Exemptions

Titanium dioxide is currently approved by FDA for use in foods, cosmetics and pharmaceuticals. Titanium dioxide also is exempt from the requirement for a tolerance by EPA for use as a colorant in pesticide formulations (40 CFR 180.1001). (Indira Gairola)

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COUNCIL ON ENVIRONMENTAL QUALITY

American Heritage Rivers Initiative

AGENCY: Council on Environmental Quality.

ACTION: Proposal With Request for Comments—Re-Issue of May 19, 1997 Notice With Clarification Section and Revised Schedule.

SUMMARY: In the State of the Union Address, President Clinton announced that he had directed his Cabinet to design an initiative to support communities in their efforts to restore and protect America's rivers. The White House subsequently convened an interagency task force to develop what has come to be known as the American Heritage Rivers initiative. The charter of the interagency task force is to integrate the environmental, historic and economic programs and services of federal agencies to benefit communities. The agencies designing this initiative include the Departments of Agriculture, Commerce, Defense, Energy, Interior, Justice, and Housing and Urban Development, the Environmental Protection agency, Advisory Council on Historic Preservation, Army Corps of Engineers and the National Endowment for the Humanities.

There are many citizens, nongovernmental organizations and local, state and tribal governments working to restore and revitalize their river communities. The Administration is creating the American Heritage Rivers initiative to help these communities restore and protect their river resources in a way that integrates natural resource protection, economic development, and the preservation of historical and