cereal grains are 0.1 mg/kg and is 0.01mg/kg for processed foods after treatment with PH_3 generated from metal phosphides. This corresponds to the levels set both by Environmental Protection Agency/the NH & MRC of Australia and the Codex Alimentarius Commission of the WHO/FAO.

2. *Analytical methodology.* The maximum residue limit recommended by the Codex Alimentarius Commission of the WHO/FAO for phosphine in raw cereals is 0.1mg/kg and in milled cereals and a range of foodstuffs including nuts it is 0.01mg/kg. An improved method for the determination of phosphine residues in a range of stored foodstuffs with a limit of detection better than 0.0001mg/kg is described by K.A. Scudamore and G.Goodship (Ref: ''Determination of Phosphine Residues in Fumigated Cereals and other Foodstuffs.'' Pestic. Sci. 1986, 37; 385-395). The method has been used to obtain data on the amount of phosphine, which remains in these commodities after treatment at typical dosage levels and on its persistence during storage. Results show that in cereal grains and nuts residues fall quickly to below. Internationally recommended levels although ultra trace amounts (less than 0.001 mg/kg) of phosphine could be detected several months after treatment in all the commodities examined.

3. *Crop residue data.* While phosphine is not applied to growing plants or crops it is a well-established fumigant of cereal grain and stored products.

4. *Fate of residues.* The possible reactions of absorbed phosphine within the commodity matrics to form inorganic phosphorous compounds have been detailed. In warm-blooded animals, phosphorous acid and phosphoric acid are formed or else phosphate. The volatile nature of phosphine (boiling point minus 87°C) and its limited solubility ensures that any phosphine absorbed in a foodstuff during treatment would be negligible and rapidly lost. Residue of phosphine held for any length of time is less than 0.001 mg/kg i.e., 0.001 ppm. Phosphoric acid has many uses including an acidulate and flavor in beverages of the soft drink type.

5. *Maximum residue limits— i. Overseas.* The maximum residue limit recommended by the Codex Alimentarius Commission of the WHO/FAO for phosphine in raw cereals is 0.1 mg/kg and in milled cereals and a range of foodstuffs including nuts is 0.01 mg/kg. (Ref: ''Codex Maximum Limits for Pesticide Residues'' Codex Alimentarius Commission Volume XIII, Rome 1983).

ii. *Australia.* The 100th session of the National Health and Medical Research Council, November 1985 gave the maximum residue limit in cereal grains of 0.1 mg/kg; and in flour and other milled cereal products, breakfast cereals, dried fruit, dried vegetables, all other dried foods, spices, nuts, peanuts, cocoa, beans and honey a limit of 0.01 mg/kg. The maximum residue limit is set at or about the limit of analytical determination. If the substance were to occur at or below this limit it is considered that no hazard to human health would occur. (Ref: ''Standard for Maximum Residue Limits of Pesticides, Agricultural Chemical, Feed Activities, Veterinary Medicines and Noxious Substances in Food'' Commonwealth Dept. of Health, Commonwealth of Australia 1986. ISBN 0644 04688 0).

iii. *U.S.A.* Tolerances have been established for commodities fumigated by the fumigant PH_3 generated from metal phosphides. Maximum residue limits for cereal grains are 0.1 mg/kg and is 0.01mg/kg for processed foods after treatment with PH3 generated from metal phosphides.

E. Residue Detection and Removal

See Section D Above

F. Endocrine Effects

Phosphine degrades to phosphates and phosphoric acid or else phosphates, in warm-blooded animals (Ref: ''The Agrochemicals Handbook'', Royal Society of Chemistry, 1986). It has been shown that there is no overt toxicity associates with the residue low levels (order 0.001 ppm) of phosphine products, in fact, a major buffering system of the body utilizes polybasic phosphates; and phosphoric acid is used as an acidulate and flavor in beverages of soft drink type (Ref: The Merck Index, 9th Edition, 7153).

G. Exposure to Infants and Children

Summary. Commodities fumigated with PH_3 at the recommended dosage levels leaves very little residue in the order of 0,001ppm (see part D) Long term feeding studies showed that ingestion of PH_3 fumigated dirt by the rat for 2 years does not cause any marked modification of growth, food intake, nitrogen balance, body composition, functional behavior or the incidence of type of tumors. The product should however, at all times be kept out of reach of children or other uncertified applicators due to acute inhalation toxicity.

H. Reasonable Grounds

ECO_2FUMETM is a mixture of two well known fumigants PH_3 and CO_2. Tolerances have already been established for PH_3 generated from Aluminum and Magnesium phosphide. Maximum residue limits for cereal grains are 0.1 mg/kg and is 0.01mg/kg for processed foods after treatment with PH_3 generated from metal phosphides. CO_2 is exempt from tolerance. Use of ECO_2FUMETM results in approximately 75% less PH_3 being used for fumigation as compared to PH_3 from metal phosphides ECO_2FUMETM has recorded residue levels of below 0,001ppm. (PM 14)

[FR Doc. 97–24694 Filed 9–16–97; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[PF–753; FRL–5735–5]

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–753, must be received on or before October 17, 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 listed in the table below:

Product Manager	Office location/telephone number	Address
Joanne Miller (PM 23) ...	Rm. 237, CM #2, 703–305–6224, e-mail:miller.joanne@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
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–753] (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–753] 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: September 5,1997

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. DowElanco

PP 7F4851

EPA has received a pesticide petition (PP 7F4851) from DowElanco, 9330 Zionsville Road, Indianapolis, IN 46268–1054, 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 flumethsulam in or on the raw agricultural commodity dry beans at 0.05 ppm. The proposed analytical method involves homogenization, filtration, partition and cleanup with analysis by high performance liquid chromatography using UV detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism in plants is adequately understood. No metabolites of significance were detected in plant metabolism studies.

2. *Analytical method*. There is a practical analytical method for detecting and measuring levels of flumetsulam in or on food with a limit of quantitation (LOQ) of 0.010 ppm, and a limit of detection of 0.005 ppm that allows monitoring of food with residues at or above the levels set in these tolerances. EPA has provided information on this method to FDA. The method is availabe to anyone who is interested in pesticide residue enforcement.

3. *Magnitude of residues*. No detectable residues of flumetsulam were found in any of the drybean samples obtained from multiple sites and multiple varieties and analyzed using a method with a limit of detection of 0.005 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Flumetsulam has low acute toxicity. The rat oral LD_{50} is >5,000 mg/kg or greater for males and females. The rabbit dermal LD_{50} is >2,000 mg/kg and the rat inhalation LC_{50} is >1.2mg/L air (the highest attainable concentration). In addition, flumetsulam is not a skin sensitizer in guinea pigs, is not a dermal irritant and is not an ocular irritant. Therefore based on the available acute toxicity data, flumetsulam does not pose any acute dietary risks.

2. *Genotoxicty*. Flumetsulam is not genotoxic. The following studies have been conducted and all were negative for genotoxic responses: a dominant lethal assay, an *In vivo* rat cytogenic study, an *In vitro Salmonella* and *Saccharomyces assay*, an *in vivo* mouse host–mediated assay, and an unscheduled DNA synthesis assay in rats.

3. *Reproductive and developmental toxicity*. In a 2–generation reproduction study in rats, there was no compound–related reproductive toxicity. The No-Observed-Effect Level (NOEL) was greater than 1,000 mg/kg/day. Developmental toxicity was studied using rats and rabbits. The developmental study in rats resulted in a developmental NOEL greater than 1.000 mg/kg/day (highest dose tested) and a maternal NOEL of 500 mg/kg/day. A study in rabbits resulted in a

developmental NOEL equal to or greater than 700 mg/kg/day (highest dose tested) with a maternal NOEL of 100 mg/kg/day and a maternal LOEL (lowest observed effect level) of 500 mg/kg/day evidenced by decreased body weight gain. Based on all of the data for flumetsulam, there is no evidence of developmental toxicity at dose levels that do not result in maternal toxicity.

4. *Subchronic toxicity* . In a 13–week oral feeding study in mice at 5,000 mg/kg/day, slight effects on the liver, kidney, and cecum appeared to represent adaptive responses to treatment and have questionable toxicological significance. The NOEL was 1,000 mg/kg/day (limit dose). In a 13–week oral feeding study in dogs, the lowest–observed–effect level (LOEL) for both male and female dogs was 500 mg/kg/day. A NOEL was not established for males or females. In a 13–week dietary study in rats, the NOEL was 250 mg/kg/day and the LOEL was 1,000 mg/kg/day.

5. *Chronic toxicity*. In a 1–year dietary study in dogs, the NOEL was 100 mg/kg/day and the LOEL was 500 mg/kg/day. The animals were administered feed containing 0, 20, 100, and 500 mg/kg/day. Reduced body weights and inflammatory and atrophic changes in the kidneys occurred in the 500 mg/kg/day dose groups. In a combined feeding carcinogenicity/chronic study in mice there were no treatment–related effects and there was no evidence of a carcinogenic response. Systemic NOEL was greater than or equal to 1,000 mg/kg/day (limit dose); a LOEL was not established. In a combined feeding carcinogenicity/chronic study in rats, renal pathological alterations were seen in males. No treatment-related effects were seen in females at the highest dose (1,000 mg/kg/day) which is the limit dose. There was no carcinogenic response. The NOELs were 500 mg/kg/day in males and 1,000mg/kg/day in females. The LOEL was 1,000 mg/kg/day in males; a LOEL was not established in females. Based on the chronic toxicity data, EPA has established the RfD for flumetsulam at 1.0 milligram (mg)/kilogram (kg)/day. The RfD for flumetsulam is based on the 1–year chronic study in dogs with a NOEL of 100 mg/kg/day and an uncertainty (or safety) factor of 100. Thus, it would not be necessary to require the application of an additional uncertainty factor above the 100–fold factor already applied to the NOEL.

6. *Animal metabolism*. Disposition and metabolism of flumetsulam were tested in male and female rats and male mice at an oral dose of 5 and 1,000 mg/kg for rats and 1,000 mg/kg for mice Flumetsulam was rapidly excreted. The majority of a radioactive dose was excreted in 48 hours of all dose groups. The principle route for elimination was the urine and to a lessor extent by fecal elimination. Detectable levels of residual radioactivity were observed in the carcass and stomach at 72 hours post–dose. HPLC and TLC analysis of urine and fecal extracts showed no apparent metabolism of flumetsulam.

7. *Metabolite toxicology*. There are no flumetsulam metabolites of toxicological significance.

8. *Endocrine effects*. There is no evidence to suggest that flumetsulam has an effectt on any endocrine system.

C. Aggregate Exposure

1. *Food*. For purposes of assessing the potential dietary exposure under these tolerances, exposure is estimated based on the Theoretical Maximum Residue Contribution (TMRC) from the existing and pending tolerances for flumetsulam on food crops. The TMRC is obtained by multiplying the tolerance level residues by the consumption data which estimates the amount of those food products eaten by various population subgroups. Exposure of humans to residues could also result if such residues are transferred to meat, milk, poultry or eggs. The following assumptions were used in conducting this exposure assessment: 100% of the crops were treated, the RAC residues would be at the level of the tolerance, certain processed food residues would be at anticipated (average) levels based on processing studies and all current and pending tolerances were included. This results in an overestimate of human exposure and a conservative assessment of risk. Based on a NOEL of 100 mg/kg/day in a 1–year chronic feeding study in the dog and a hundredfold safety factor the reference dose (RfK) would be 1.0 mg/kg/day. The TMRC for the general population would be 4.1 X 10^{-5} mg/kg/day or 0.0041% of the RfD. For non–nursing infants, the TMRC wold be 1.37 X 10^{-5} mg/kg/day or 0.014% of the RfD.

2. *Drinking water*. Another potential source of dietary exposure to residues of pesticides are residues in drinking water. There is no established Maximum Concentration Level for residues of flumetsulam in drinking water. Although there has been limited detections at ppb levels in some of the specially designed studies under highly vulnerable test conditions and at elevated non–labeled application rates, no ongoing monitoring studies, have reported residues of flumetsulam in ground or surface waters.

Based on the physical and chemical characteristics of flumetsulam, such as water solubility and its stability under hydrolysis and photolysis, it has potential for downward movement through the soil profile. Degradation based on over 20 laboratory studies indicated a half–life range of 2 weeks to 4 months with 80% less than 2 months. Degradation is driven primarily by microbial processes. However based on the low application rate and detection in groundwater samples only under extremely vulnerable soil conditions at elevated non–labeled application rates with detections in single digit ppb levels, flumetsulam is not anticipated to be a groundwater contaminant.

In summary, these data on potential water exposure indicate insignificant additional dietary intake of flumetsulam and any exposure is more than compensated for in the conservative dietary risk evaluation. Therefore, it is concluded that there is a reasonable certainty of no harm even at potential upper limit exposures to flumetsulam from drinking water.

3. *Non–dietary exposure*. There are no non–dietary uses for flumetsulam registered under the Federal Insecticide, Fungicide and Rodenticide Act. Potential exposures for children is therefore limited to dietary exposure.

D. Cumulative Effects

The potential for cumulative effects of flumetsulam and other substances that have a common mechanism of toxicity was considered. The mammalian toxicity of flumetsulam is well defined. However, no reliable information exists to indicate that toxic effects produced by flumetsulam would be cumulative with those of any other chemical compound. Additionally, flumetsulam does not appear to produce a toxic metabolite produced by other substances. Therefore, consideration of a common mechanism of toxicity with other compounds is not appropriate at this time. Thus only the potential exposures to flumetsulam were considered in the aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Based on a NOEL of 100 mg/kg/bwt/day from a one-year dog feeding study with a reduced weight and inflammatory and atrophic kidney effect, and using an uncertainty factor of 100 to account for the interspecies extrapolation and intraspecies variability, a Reference Dose (RfD) of 1.0 mg/kg bwt/day was used for this assessment of chronic risk. As indicated, there is no endpoint of concern identified with acute and short–or intermediate–term exposures. The existing and proposed tolerances

would utilize 0.000041 mg/kg bwt/day or less than 0.01% of the RfD for the U.S. population. And, as indicated previously, whatever upper limit might be used for drinking water exposure, the exposure estimate for flumetsulam would not exceed the RfD. Generally, exposures below 100 percent of the RfD are of no concern because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risk to human health. Thus, there is a reasonable certainty that no harm will result from aggregate exposure to flumetsulam residues.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of flumetsulam, data from developmental toxicity studies in the rat and rabbit and a 2–generation reproduction study in the rat were considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism during prenatal development resulting from pesticide exposure to one or both parents. Reproduction studies provide (1) information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and (2) data on systemic toxicity.

As indicated previously, reproductive and developmental toxicity was studied using rats and rabbits. The data base is complete and based on all of the data for flumetsulam, there is no evidence of reproductive or developmental toxicity at dose levels that do not result in maternal toxicity.

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. These data suggest minimal concern for developmental or reproductive toxicity and do not indicate any increased pre- or post–natal sensitivity. Therefore, an additional uncertainty factor is not necessary to protect the safety of infants and children and that the RfD at 1.0 mg/kg/day is appropriate for assessing aggregate risk to infants and children.

The percent of the RfD that will be utilized by the aggregate exposure from all tolerances to flumetsulamill be less than 0.1% for non–nursing infants and for children (1–6 years of age). Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to flumetsulam residues.

F. International Tolerances

There are no Codex maximum residue levels established for flumetsulam. (Joanne Miller)

2. Rohm and Haas Company

PP 2F4127

EPA has received a pesticide petition (PP 2F4127) 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 a permanent tolerance for residues of [alpha-(2-(4-chlorophenyl)-ethyl)-alpha-phenyl-3-(1H-1,2,4-triazole)-1-propanenitrile (fenbuconazole)] in or on the raw agricultural commodities wheat grain; wheat straw; milk; eggs; and meat, fat, and meat by-products of cattle, goats, horses, hogs, poultry, and sheep. The analytical method involves soxhlet extraction, partitioning, redissolving, cleanup, and analysis by gas-liquid chromatography using nitrogen specific thermionic detection. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

The tolerance expression for fenbuconazole residues in or on 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 are combined and expressed as parent compound to determine the total RAC residue in or on wheat grain and wheat 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 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 are combined and expressed as parent compound to determine the total residue.

Analytical methods to measure the components of the residue in or on wheat grain and wheat straw, and in or on animal commodities have been validated and accurately quantify residues of fenbuconazole. The residues of fenbuconazole will not exceed the proposed Permanent Tolerances on wheat or related commodities following foliar or seed treatment of wheat.

1. *Analytical method*. Fenbuconazole residues (parent plus lactones) are measured at an analytical sensitivity of 0.01 mg/kg in 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) 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*. 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.

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

3. *Reproductive and developmental toxicity*. These data cited at 60–FR–27419, May 24,1995. 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 no observable effect level (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, 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 $(\text{body weight})_{3/4}$.

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 data set for the low-

dose extrapolation and risk factor estimate is the mouse. From these data a Q1* of (0.36 x 10^{-2}(mg/kg/day)–1) is calculated when surface area is estimated by (body weight)$_{3/4}$. All estimates of dietary risk must be adjusted to reflect this change.

Since fenbuconazole is unlikely to 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 additional risk from this source 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. *Food.* 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 x 10^{-6} for additional cancer risk (Q1* = 1.06 x 10^{-2} (mg/kg/day)-1). (60 FR 27419; 24 May 1995). This estimate does not reflect the change in Q1*. Using the EPA model and the new risk factor based on the mouse data (Q1* = 0.36 x 10^{-2} (mg/kg/day)-1) the dietary risk for currently registered uses is 0.3 x 10^{-6}. The TMRC for existing tolerances utilizes 17% of the RfD for the most sensitive subpopulation, non-nursing infants less than 1–year old. This is unaffected by the change in Q1*.

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 1– 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 x 10^{-2} (mg/kg/day)–1) has an upper–bound of 0.2 x 10^{-6}. The calculated additional cancer risk for animal fat and liver has an upper–bound of 0.1 x 10^{-6}. The upper bound estimate on excess cancer risk for all uses including wheat is 0.7 x 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.

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. *US population*. 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 (0.36×10^{-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 $\times 10^{-6}$), 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.

2. *Infants and children*. 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). (60 FR 27420; May 24,1995).

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.

F. 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.

G. Environmental Fate Summary

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 DT_{50} values for fenbuconazole range from 29 to 532 days under terrestrial conditions and from 442 to 906 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 DT_{50} in green heads was measured as 18 days and in green wheat stalks the DT_{50} 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.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–754; FRL–5735–8]

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 and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–754, must be received on or before October 17, 1997.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7506C), 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: By mail: Sidney Jackson, Product Manager (PM) 43, Minor Use, Inerts, Emergency Response Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone number: Rm. 274, CM#2, 1921 Jefferson Davis Highway, Arlington, VA., (703) 305–7610. e-mail: jackson.sidney@epamail.epa.gov.