telephone (202) 260–5886; FAX (202) 260–7118; or via e-mail at: <miller.tom@epa.gov>. Single copies of the guidelines information provided to the Committee can be obtained by contacting Mr. Brett Snyder, Director, Economy and Environment Division (2172), Office of Policy, US Environmental Protection Agency, 401 M Street SW., Washington DC 20460, telephone (202) 260–5610, fax (202) 260-2685; or via email at: <snyder.brett@epa.gov>.

Providing Oral or Written Comments at SAB Meetings

The Science Advisory Board expects that public statements presented at its meetings will not be repetitive of previously submitted oral or written statements. In general, each individual or group making an oral presentation will be limited to a total time of ten minutes. For teleconference meetings, opportunities for oral comment will usually be limited to no more than three minutes per speaker and no more than fifteen minutes total. Written comments (at least 35 copies) received in the SAB Staff Office sufficiently prior to a meeting date (usually one week before the meeting), may be mailed to the relevant SAB committee or subcommittee; comments received too close to the meeting date will normally be provided to the committee at its meeting, or mailed soon after receipt by the Agency.

Additional information concerning the Science Advisory Board, its structure, function, and composition, may be found on the SAB Website (http://www.epa.gov/sab) and in The Annual Report of the Staff Director which is available from the SAB Publications Staff at (202) 260–4126 or via fax at (202) 260–1889.

Meeting Access

Individuals requiring special accommodation at this teleconference meeting, including wheelchair access to the conference room, should contact Mr. Miller at least five business days prior to the meeting so that appropriate arrangements can be made.

Dated: June 16, 1999.

A. Robert Flaak,

Acting Staff Director, Science Advisory Board.

[FR Doc. 99–16234 Filed 6–24–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6367–4]

Science Advisory Board; Public Advisory Committee Meeting

Pursuant to the Federal Advisory Committee Act, Public Law 92–463, notice is hereby given that the Science Advisory Board's (SAB) Executive Committee (EC) will meet on Tuesday, July 13, and Wednesday, July 14, 1999. The meeting will convene each day at 8:30 am, in the Administrator's Conference Room 1103 West Tower of the U.S. Environmental Protection Agency Headquarters Building at 401 M Street, SW, Washington, DC 20460, and adjourn no later than 5:30 pm on each day. All times noted are Eastern Time. The meeting is open to the public, however, seating is limited and available on a first come basis. Documents that are the subject of SAB reviews are normally available from the originating U.S. Environmental Protection Agency (EPA) office and are *not* available from the SAB Office. Public drafts of SAB reports are available to the Agency and the public from the SAB office. Details on availability are noted below.

At this meeting, the Executive Committee will receive updates from its committees and subcommittees concerning their recent and planned activities. As part of these updates, some committees will present draft reports for Executive Committee review and approval. Copies of these drafts will be available on the SAB Website (see below for site address) two weeks prior to the meeting or may be obtained from Ms. Tillery-Gadson (see address below).

In addition, the Board anticipates interacting with various senior Agency officials on issues of general interest, as well as issues currently before or proposed for future Board consideration.

For Further Information—Any member of the public wishing further information concerning the meeting or who wishes to submit comments should contact Dr. John R. Fowle, III, Acting Designated Federal Officer for the Executive Committee, Science Advisory Board (1400), U.S. EPA, Washington, DC 20460, phone (202) 260–8325; fax (202) 260–7118; or via e-mail at: <fowle.jack@epa.gov>. Copies of the draft meeting agenda and the draft reports will be available on the SAB Website (www.epa.gov/sab) approximately two weeks prior to the meeting. Alternatively, these materials can be obtained from Ms. Priscilla Tillery-Gadson at the above address and fax number or via phone (202) 260–4126 or via e-mail: <tillery.priscilla@epa.gov>.

Providing Oral or Written Comments at SAB Meetings

The Science Advisory Board expects that public statements presented at its meetings will not be repetitive of previously submitted oral or written statements. In general, each individual or group making an oral presentation will be limited to a total time of ten minutes. For conference call meetings, opportunities for oral comment will be limited to no more than five minutes per speaker and no more than fifteen minutes total. Written comments (at least 35 copies) received in the SAB Staff Office sufficiently prior to the meeting date, may be mailed to the Committee prior to its meeting; comments received too close to the meeting date will normally be provided to the Committee at its meeting. Written comments may be provided to the Committee up until the time of the meeting.

Information concerning the Science Advisory Board, its structure, function, and composition, may be found in The FY1998 Annual Report of the Staff Director which is available from the SAB Committee Evaluation and Support Staff (CESS) by contacting US EPA, Science Advisory Board (1400), Attention: CESS, 401 M Street, SW, Washington, DC 20460 or via fax (202) 260–1889. Additional information concerning the SAB can be found on the SAB Website at: http://www.epa.gov/sab

Individuals requiring special accommodation at this meeting, including wheelchair access, should contact Dr. Fowle at least five business days prior to the meeting so that appropriate arrangements can be made.

Dated: June 17, 1999.

A. Robert Flaak,

Acting Staff Director, Science Advisory Board.

[FR Doc. 99–16235 Filed 6–24–99 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–788A and PF–848A; FRL–6076–9]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the amendment of pesticide petitions 1F3989, and 7F4900, 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–788A, and PF–848A, must be received on or before July 26, 1999.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information and Services Divison (7502C), Office of Pesticides Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically 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. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: Cynthia Giles-Parker, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 247, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–7740; e-mail: giles-parker.cynthia@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemical 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 this petition contains 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–788A], and [PF–848A] (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number (PF–788A), and (PF–848A) 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 9, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner summary of the pesticide petition is printed below as required by section 408(d)(3) of the FFDCA. The summary of the petition was prepared by the petitioner and represents the views of the petitioner. 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.

Rohm and Haas Company

PP 1F3989 and 7F4900

Amended Petitions

In the **Federal Registers** of January 30, 1998 (63 FR 4631) (FRL–5766–2), and December 7, 1998 (63 FR 67476) (FRL–6047–2), EPA issued a notice of filing announcing that it had received pesticide petitions (PP) 1F3989, and 7F4900 from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA 19106–2399, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d) proposing to amend 40 CFR part 180. In petition 1F3989, Rohm and Haas Company proposed among other things, to establish a time-limited tolerance for residues of fenbuconazole (α-(2-[4-chlorophenyl]-ethyl)-α-phenyl-3-(1*H*-1,2,4-triazole)-1-propanenitrile] in or on stone fruits (except plums and prunes) at 2.0 ppm. In petition 7F4900, Rohm and Haas Company proposed, among other things, to establish permanent tolerances for fenbuconazole in or on grapefruit at 1.0 ppm, citrus oil (grapefruit) at 35.0 ppm, and grapefruit pulp, dried at 4.0 ppm.

Today's notice of filing announces the receipt of pesticide petitions from Rohm and Haas Company proposing to amend PP 1F3989 and 7F4900 by establishing tolerances for residues of fenbuconazole (α-(2-[4-chlorophenyl]-ethyl)-α-phenyl-3-(1*H*-1,2,4-triazole)-1-propanenitrile] plus RH-9129 and RH-9130, the diastereomeric lactone metabolites of fenbuconazole [5-(4-chlorophenyl)-dihydro-3-phenyl-3-(methyl-1*H*-1,2,4-triazole-1-yl)-2-3*H*-furanone) in or on the raw agricultural commodities plums at 2.0 parts per million (ppm), plums, dried (prunes) at 7.0 ppm (PP 1F3989), and for oranges at 1.0 ppm, orange, dry pulp at 4.0 ppm, and orange, citrus oil at 16 ppm (7F4900). 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 support granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of fenbuconazole in plants (wheat, peaches, and sugar beets) is adequately understood for the purpose of these tolerances. The metabolism of fenbuconazole in all crops was similar and involves oxidation of the benzylic position alpha to the chlorophenyl ring. The metabolites which result from this

path are the benzylic alcohols and their conjugates, including sulfates and glucuronides, the iminolactones, the lactones, and the ketoacid, all resulting from intramolecular cyclization. A second pathway is oxidation of the unchlorinated ring to produce the 3- and 4-phenols and their conjugates. Combinations of the above two pathways produce phenol-lactones and their conjugates. A third pathway is cleavage of the triazole moiety, which produces free triazole and its conjugates.

2. *Analytical method*. An adequate enforcement method is available to enforce the established and proposed tolerances. Quantitation of fenbuconazole residues (parent plus lactones) at an analytical sensitivity of 0.01 milligrams/kilogram (mg/kg) is accomplished by soxhlet extraction of samples in methanol, partitioning into methylene chloride, redissolving in toluene, clean up on silica gel, and gas liquid chromatography using nitrogen specific thermionic detection.

3. *Magnitude of residues*. Field residue trials were conducted with an aqueous flowable formulation of fenbuconazole in geographically representative regions of the United States. The results from these studies support the proposed tolerances, and clearly indicate that the lactone metabolites (RH–9129 and RH–9130) are minor contributors to the total residue.

i. *Oranges*. A total of 16 field residue trials were conducted in oranges. Three applications were made at 0.25 pounds active ingredient/acre (lb ai/A), twice the maximum use rate of 0.125 lb ai/A, and whole fruit was harvested on the same day as the last application. The highest field residue value in whole fruit was 0.752 ppm. The average field residue value in whole fruit was 0.276 ppm. The highest field residue value in the edible pulp from five field trials was 0.0104 ppm. The average field residue value in pulp was 0.005 ppm. Residues were measured in orange process fractions including, juice, dried pulp, and cold press (citrus) oil. In the processing study, three applications were made at 0.25 lb ai/A, twice the maximum use rate of 0.125 lb ai/A, and the fruit were harvested seven days after the last application. Fruit was processed into multiple components. No residues (<0.01 ppm) were detected in juice, thus were was no concentration of residues in fresh juice. The average residues in dried pulp (cattle feed) and citrus oil (defined as a non-ready-to-eat processed commodity) were 4.1- and 32.1-times the amount of residues in fresh oranges, respectively.

ii. *Plums*. A total of 10 field residue trials were conducted in plums. Six to nine applications were made at the maximum use rate of 0.1 lb ai/A, and whole fruit was harvested on the same day as the last application. The highest field residue value in whole fruit was 0.315 ppm; the next highest field residue value was 0.071 ppm. The average field residue value in whole fruit was 0.062 ppm. Residues were measured in dried plums (prunes) in three residue trials. Six applications were made at the maximum use rate of 0.1 lb ai/A, and whole fruit was harvested on the same day as the last application. Dried plums contained residues of 0.0244, 0.04, and 0.139 ppm.

B. Toxicological Profile

1. *Acute toxicity*. Fenbuconazole is practically non-toxic after administration by the oral and dermal routes, and was not significantly toxic to rats after a 4–hour inhalation exposure. Fenbuconazole is classified as not irritating to skin and inconsequentially irritating to the eyes. It is not a skin sensitizer.

2. *Genotoxicity*. Fenbuconazole was negative (non-mutagenic) in an Ames assay with and without hepatic enzyme activation. Fenbuconazole was negative in a hypoxanthine guanine phosphoribosyl transferase (HGPRT) gene mutation assay using Chinese hamster ovary (CHO) cells in culture when tested with and without hepatic enzyme activation. In isolated rat hepatocytes, fenbuconazole did not induce unscheduled DNA synthesis (UDS) or repair. Fenbuconazole did not produce chromosome effects in rats *in vivo*. On the basis of the results from this battery of tests, it is concluded that fenbuconazole is not mutagenic or genotoxic.

3. *Reproductive and developmental toxicity*—i. *Rat developmental toxicity*. In the developmental study in rats, the maternal (systemic) no-observed adverse effect level (NOAEL) was 30 mg/kg/day based on decreases in body weight (bwt) and body weight gain at the lowest-observed adverse effect level (LOAEL) of 75 mg/kg/day. The developmental (fetal) NOAEL was 30 mg/kg/day based on an increase in post implantation loss and a significant decrease in the number of live fetuses per dam at the LOAEL of 75 mg/kg/day.

ii. *Rabbit developmental toxicity*. In the developmental study in rabbits, the maternal (systemic) NOAEL was 10 mg/kg/day based on decreased bwt gain at the LOAEL of 30 mg/kg/day. The developmental (fetal) NOAEL was 30 mg/kg/day based on increased resorptions at the LOAEL of 60 mg/kg/day.

iii. *Rat reproduction*. In the 2-generation reproduction toxicity study in rats, the maternal (systemic) NOAEL was 4 mg/kg/day based on decreased bwt and food consumption, increased number of dams delivering nonviable offspring, and increases in adrenal and thyroid weights at the LOAEL of 40 mg/kg/day. The reproductive (pup) NOAEL was 40 mg/kg/day, the highest dose tested (HDT).

4. *Subchronic toxicity*—i. *Rat 90–day oral study*. A subchronic feeding study in rats conducted for 13–weeks resulted in a NOAEL of 20 ppm (1.3 and 1.5 mg/kg/day in males and females, respectively). Minimal liver hypertrophy was observed in males at the LOAEL of 80 ppm. Increased liver weight, hepatic hypertrophy, thyroid hypertrophy, and decreased bwt were observed at the higher doses (400 and 1,600 ppm).

ii. *Mouse 90–day oral study*. A subchronic feeding study in mice conducted for 13–weeks resulted in a NOAEL of 60 ppm (11.1 and 17.6 mg/kg/day in males and females, respectively). Increased liver weight, hypertrophy in the liver (males), and increases in clinical chemistry parameters (males) were observed at the LOAEL of 180 ppm. These effects were all observed in females at 540 ppm in addition to males.

iii. *Dog 90–day oral study*. A subchronic feeding study in dogs conducted for 13–weeks resulted in a NOAEL of 100 ppm (3.3 and 3.5 mg/kg/day in males and females, respectively). At the LOAEL of 400 ppm, increased liver weight, clinical chemistry parameters, and liver hypertrophy (males) were observed.

iv. *Rat 4–week dermal study*. In a 21–day dermal toxicity study in the rat, the NOAEL was greater than 1,000 mg/kg/day, with no effects seen at this limit dose.

5. *Chronic toxicity*—i. *Dog*. A 1–year feeding study in dogs resulted in a NOAEL of 15 ppm (0.62 mg/kg/day) for females and 150 ppm (5.2 mg/kg/day) for males. Decreased bwt, increased liver weight, liver hypertrophy, and pigment in the liver were observed at the LOAEL of 150 and 1,200 ppm in females and males, respectively.

ii. *Mouse*. A 78–week chronic/oncogenicity study was conducted in male and female mice at 0, 10, 200 (males only), 650, and 1,300 ppm (females only). The NOAEL was 10 ppm (1.4 mg/kg/day), and the LOAEL was 200 ppm (26.3 mg/kg/day) for males and 650 ppm (104.6 mg/kg/day) for females based on increased liver weight and

histopathological effects on the liver, which were consistent with chronic enzyme induction. There was no statistically significant increase of any tumor type in males, 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). There were no liver tumors in the control females, and liver tumor incidences in treated females just exceeded the historical control range. 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 weight. Changes in these liver parameters as well as the occurrence of the low incidence of liver tumors were non-linear 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 humans. The non-linear dose response relationship observed with respect to liver changes (including the low incidence of tumors) in the mouse indicates that these findings should be carefully considered in deciding the relevance of high-dose animal tumors to human dietary exposure.

iii. *Rat*. A 24-month chronic/oncogenicity study in male and female rats was conducted at 0, 8, 80, and 800 ppm fenbuconazole, and a second 24-month chronic/oncogenicity was conducted in male rats at 0, 800, and 1,600 ppm. The NOAEL was 80 ppm (3 and 4 mg/kg/day in males and females, respectively), and the LOAEL was 800 ppm (31 and 43 mg/kg/day in males and females, respectively) based on decreased bwt, increased liver and thyroid weights, and liver and thyroid hypertrophy. 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) of 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 non-linear phenomenon in that thyroid tumors occur only at high doses where there is an increase in liver weight 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 that 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.

The reference dose (RfD) of 0.03 mg/kg/day was established by the Agency based on the NOAEL of 3.0 mg/kg/day in the chronic rat feeding study and an uncertainty factor of 100.

The Carcinogenicity Peer Review Committee (CPRC) of the Health Effects Division (HED) of EPA has classified fenbuconazole as a Group C tumorigen (possible human carcinogen with limited evidence of carcinogenicity in animals). The Committee has decided that it is appropriate to use a low-dose extrapolation model based on the mouse data with the Q_{1*} of 0.359×10^{-2} $(mg/kg/day)^{-1}$ and surface area estimated by $(bwt)^{3/4}$. All estimates of dietary oncogenic risk are based on this risk factor.

6. *Animal metabolism*. The absorption, distribution, excretion, and metabolism of fenbuconazole in rats, goats, and hens were investigated. Following oral administration, fenbuconazole was completely and rapidly absorbed, extensively metabolized by oxidation/hydroxylation and conjugation, and rapidly and essentially completely excreted predominately in the feces. Fenbuconazole did not accumulate in tissues.

7. *Metabolite toxicology*. Common metabolic pathways for fenbuconazole have been identified in both plants (wheat, peaches, and sugar beets) and animals (rat, goat, and hen). The metabolic pathway common to both plants and animals involves oxidation of the benzylic position alpha to the chlorophenyl ring. The metabolites which result from this path are the benzylic alcohols and their conjugates, including sulfates and glucuronides, the iminolactones, the lactones, and the ketoacid, all resulting from intramolecular cyclization. A second pathway is oxidation of the unchlorinated ring to produce the 3- and 4-phenols and their conjugates. Combinations of the above two pathways produce phenol-lactones and their conjugates. A third pathway is cleavage of the triazole moiety, which produces free triazole and its conjugates. Extensive degradation and elimination of polar metabolites occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet.

8. *Endocrine disruption*. The mammalian endocrine system includes estrogen and androgens as well as other hormonal systems. Fenbuconazole is not known to interfere with reproductive hormones; thus, fenbuconazole should not be considered to be estrogenic or androgenic. There are no known instances of proven or alleged adverse reproductive or developmental effects to people, domestic animals, or wildlife as a result of exposure to fenbuconazole or its residues.

C. Aggregate Exposure

1. *Dietary exposure*—i. *Food*. Permanent tolerances have been established (40 CFR 180.480) or proposed for the residues of fenbuconazole in or on a variety of raw agricultural commodities:

Commodity	Tolerance (ppm)
Almond nutmeat	0.05 (P)[1]
Almond hulls	3.0 (P)
Apples	0.4 (P)
Apple pomace, wet	1.0 (P)
Banana (whole fruit)	4.0
Banana (pulp)	0.05

Commodity	Tolerance (ppm)
Blueberry	0.3 (P)
Cattle, fat	0.05 (P)[3]
Cattle, liver	0.1 (P)[4]
Citrus oil (grapefruit)	35.0 (P)
Grapefruit	1.0 (P)
Grapefruit juice	N/R[2]
Molasses (beet)	0.4[5]
Pecans	0.1
Pulp, dried (beet)	1.0
Pulp, dry (grapefruit)	4.0 (P)
Refined sugar	N/R[2]
Stone Fruit (except plum/prune)	2.0
Sugar beet (root)	0.2 (P)
Sugar beet (top)	9.0 (P)
Wheat (grain)	0.05 (P)
Wheat (straw)	10.0 (P)

[1] (P): Proposed tolerance;
[2] Tolerance not required because concentration factor is < 1 in processing study;
[3] An identical tolerance is pending for fat in poultry, hogs, horses, sheep, and goats;
[4] An identical tolerance is pending for liver in poultry, hogs, horses, sheep, and goats;
[5] For livestock feed; not a human dietary component.

Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from fenbuconazole as follows:

a. *Acute exposure and risk*. No acute endpoint was identified for fenbuconazole, and no acute risk assessment is required.

b. *Chronic exposure and risk*. Risk associated with chronic dietary exposure from fenbuconazole was assessed on four levels. In the first assessment, tolerance level residues and 100% crop treated were assumed. In the second assessment, tolerance level residues and Rohm and Haas Company's conservative estimates of the highest achievable percent crop treated refinements were assumed. Rohm and Haas Company's percent of crop treated estimates used in the assessments are almonds = 50%, blueberry = 30%, grapefruit = 30%, bananas = 20%, apples = 15%, oranges = 15%, pecans = 11%, sugar beets = 3%, and wheat = 0.3%. In the third assessment, average field trial (anticipated) residues and 100% crop treated were assumed. In the fourth assessment, average field trial residues and Rohm and Haas Company's percent of crop treated estimates indicated above were assumed. Rohm and Haas Company's processing factors for apple, orange, and grapefruit juice were assumed in all four assessments. One hundred percent crop treated was assumed when calculating the dietary burden from which secondary residue tolerances in meat and fat were derived. A 12.8% crop treated refinement was used for stone fruit in all four assessments June 10, 1998 (FR 63 31636) (FRL 5791–5). The Anticipated Residue Contribution (ARC) from all proposed and existing food uses of fenbuconazole was assessed.

The RfD used for the chronic dietary analysis is 0.03 mg/kg/day. Potential chronic exposures were estimated using NOVIGEN'S Dietary Exposure Evaluation Model (DEEM™, Version 5.31), which uses USDA food consumption data from the 1989–1992 survey. The existing and proposed fenbuconazole tolerances, and average fenbuconazole residues result in ARCs that are equivalent to the following percentages of the RfD:

Population Subgroup	DEEM[1] %RfD	DEEM[2] %RfD	DEEM[3] %RfD	DEEM[4] %RfD
U.S. Population (48 States)	2.7	0.9	0.4	0.1
Non-Hispanic Other than Black or White	3.5	1.0	0.5	0.2
All Infants (< 1-year old)	6.1	3.5	1.0	0.4
Nursing Infants (< 1-year old)	2.2	0.8	0.5	0.1
Non-Nursing Infants (< 1-year old)	7.7	4.7	1.3	0.5
Children (1-6 years old)	6.4	1.8	1.1	0.3
Children (7-12 years old)	4.2	1.2	0.7	0.2
Females (13+ / Nursing)	3.2	0.8	0.5	0.1

[1] Assumes residues are present at tolerance levels and 100% crop treated (12.8% stone fruit);
[2] Assumes residues are present at tolerance levels and includes percent crop treated refinements;
[3] Assumes residues are present at their average field trial residue levels and 100% crop treated (12.8% stone fruit); and
[4] Assumes residues are present at their average field trial residue levels, and includes percent crop treated refinements.

c. *Aggregate cancer risk for U.S. population*. Fenbuconazole has been classified as a Group C Carcinogen with a Q^{1*} value of 0.00359 mg/kg/day^{-1}. Cancer risk assessments for all existing and proposed food uses for the U.S. population are as follows:

Assumptions/Refinements	All Crops	Orange & Proc. Frac.	Plums/Prunes
Tolerance residue levels and 100% crop treated (12.8% stone fruit) assumed:	2.90E-06	1.05E-06	1.46E-07
Tolerance residue levels and percent crop treated refinements assumed:	9.24E-07	1.57E-07	1.46E-07
Anticipated residue levels and 100% crop treated (12.8% stone fruit) assumed:	4.65E-07	1.6E-08	3E-09
Anticipated residue levels and percent crop treated refinements assumed:	1.44E-07	2E-09	3E-09

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. Computer modeling of laboratory and field dissipation data using EPA's Pesticide Root Zone Model (PRZM) and USDA's Groundwater Loading Effects of Agricultural Management Systems (GLEAMS) models predict 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 is no established Maximum Concentration Level (MCL) for residues of fenbuconazole in drinking water. No drinking water health advisory levels have been established for fenbuconazole. There is no entry for fenbuconazole in the ''Pesticides in Groundwater Database'' (EPA 734–12–92–001; September, 1992).

3. *Non-dietary exposure*. Fenbuconazole is not currently registered for any indoor or outdoor residential uses; therefore, no non-dietary residential exposure is anticipated.

D. Cumulative Effects

The potential for cumulative effects of fenbuconazole with other substances that have a common mechanism of toxicity was considered. Fenbuconazole belongs to the class of fungicide chemicals known as triazoles, which have demethylase inhibition capability. The toxicological effects of fenbuconazole are related to its effects on rodent thyroid and liver. Extensive data are available on the biochemical mode of action by which fenbuconazole produces animal tumors in rats and mice. These data indicate that the initiating events do not occur below a given dose, and that the processes are reversible. There are no data which suggest that the mode of action by which fenbuconazole produces these animal tumors or any other toxicological effect is common to all fungicides of this class. In fact, the closest structural analog to fenbuconazole among registered fungicides of this class is not tumorigenic in animals even at maximally tolerated doses and has a different spectrum of toxicological effects.

E. Safety Determination

1. *U.S. population*—i. *Acute exposure and risk*. Since no acute endpoint was identified for fenbuconazole, no acute risk assessment is required.

ii. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of fenbuconazole from existing, pending, and proposed tolerances is 2.7% for the U.S. population, assuming residues are present at their tolerance levels and 100% crop treated (12.8% for stone fruit). Aggregate exposure is not expected to exceed 100%. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas concludes that there is a reasonable certainty that no harm will result from aggregate exposure to fenbuconazole residues to the U.S. population.

2. *Infants and children—Safety factor for Infants and children*—i. *General*. In assessing the potential for additional sensitivity of infants and children to residues of fenbuconazole, data from developmental toxicity studies in the rat and rabbit, and 2-generation reproduction studies in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

ii. *Developmental toxicity studies*—a. *Rat*. In the developmental study in rats, the maternal (systemic) NOAEL was 30 mg/kg/day based on decreases in bwt and bwt gain at the LOAEL of 75 mg/kg/day. The developmental (fetal) NOAEL was 30 mg/kg/day based on an increase in post implantation loss and a significant decrease in the number of live fetuses per dam at the LOAEL of 75 mg/kg/day.

b. *Rabbit*. In the developmental study in rabbits, the maternal (systemic) NOAEL was 10 mg/kg/day based on decreased bwt gain at the LOAEL of 30 mg/kg/day. The developmental (fetal) NOAEL was 30 mg/kg/day based on increased resorptions at the LOAEL of 60 mg/kg/day.

iii. *Reproductive toxicity study*. In the 2–generation reproduction toxicity study in rats, the maternal (systemic) NOAEL was 4 mg/kg/day based on decreased bwt and food consumption, increased number of dams delivering nonviable offspring, and increases in adrenal and thyroid weights at the LOAEL of 40 mg/kg/day. The reproductive (pup) NOAEL was 40 mg/kg/day, the highest dose tested (HDT).

iv. *Pre- and Post-Natal sensitivity*. The pre- and post-natal toxicology database for fenbuconazole is complete with respect to current toxicological data requirements. There is a 10-fold difference between the developmental NOAEL of 30 mg/kg/day from the rat and rabbit developmental toxicity studies and the NOAEL of 3 mg/kg/day from the chronic rat feeding study which is the basis of the RfD. It is further noted that in the rabbit and rat developmental toxicity studies, the developmental NOAELs are similar to or greater than the respective maternal NOAELs. In the rat reproduction study, the maternal NOAEL (4 mg/kg/day) was ten times lower than the developmental (pup) and reproductive NOAEL (40 mg/kg/day, the HDT). These studies indicate that there is no additional sensitivity for infants and children in the absence of maternal toxicity for fenbuconazole.

v. *Acute risk*. No acute dietary risk has been identified for fenbuconazole.

vi. *Chronic risk*. Using the exposure assumptions described above, the exposure to fenbuconazole from food will utilize 7.7% (non-nursing infants <

1–year old) and 2.2% (nursing infants < 1–year old) of the RfD assuming residues are present at tolerance levels and 100% crop treated (12.8% for stone fruit), and will utilize 1.3% (non-nursing infants < 1–year old) and 0.5% (nursing infants < 1–year old) of the RfD assuming residues are present at their average field residue levels and 100% crop treated (12.8% for stone fruit). The percent of the RfD that will be used by the food exposure for children 1–6 years old is 6.4 and 1.1% assuming residues are present at tolerance and average field residue levels, respectively, and 100% crop treated (12.8% for stone fruit). The percent of the RfD that will be used by the food exposure for children 7–12 years old is 4.2 and 0.7% assuming residues are present at tolerance and average field residue levels, respectively, and 100% crop treated (12.8% for stone fruit). EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health.

vii. *Conclusion*. It is concluded that reliable and complete data support the use of the 100–fold uncertainty factor, and that an additional 10–fold factor is not needed to ensure the safety of infants and children from dietary exposure.

F. International Tolerances

There are no Codex Maximum Residue Levels (MRLs) for fenbuconazole, but the fenbuconazole database was evaluated by the World Health Organization (WHO) and the Food and Agriculture Organization (FAO) Expert Panels at the Joint Meeting on Pesticide Residues (JMPR) in September 1997. An Allowable Daily Intake (ADI (same as the RfD) of 0.03 mg/kg/day and a total of 32 Codex MRLs were proposed in the JMPR report.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6365–5]

Final NPDES General Permit for Discharges From Petroleum Bulk Stations and Terminals in Texas (TXG340000)

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final Issuance of NPDES general permit.

SUMMARY: EPA Region 6 today issues a National Pollutant Discharge Elimination System (NPDES) general permit authorizing discharges of facility waste water and contact storm water from petroleum bulk stations and terminals in Texas. This permit covers facilities having Standard Industrial Classification (SIC) Code 5171.

The permit has limits on Total Petroleum Hydrocarbons, benzene, Total BTEX (sum of benzene, toluene, ethyl benzene and xylene), Total Lead and pH. There is also a requirement of no acute toxicity as determined by requiring greater than 50% survival in 100% effluent using a 24 hour acute test. In addition, the permit has limits on arsenic, barium, cadmium, chromium, copper, manganese, mercury, nickel, selenium, silver and zinc as contained in Texas Natural Resource Conservation Commission (TNRCC) Regulations for Hazardous Metals (30 TAC 319, Subchapter B), as well as requirements for no discharge of floating solids or visible foam in other than trace amounts, and no discharge of visible oil. There is also the requirement to develop and implement a pollution prevention plan for the storm water discharges authorized by this permit.

DATES: The limits and monitoring requirements in this permit shall become effective on July 26, 1999.

FOR FURTHER INFORMATION CONTACT: Ms. Wilma Turner, EPA Region 6, 1445 Ross Avenue, Dallas, Texas 75202–2733, telephone (214) 665–7516. Copies of the complete response to comments may be obtained from Ms. Turner. The complete response to comments and final permit can also be found on the Internet at http://www.epa.gov/earth1r6/6wq/6wq.htm.

SUPPLEMENTARY INFORMATION: Regulated categories and entities include:

Category	Examples of regulated entities
Industry	Operators of petroleum bulk stations and terminals.

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be regulated by this action. This table lists the types of entities that EPA is now aware could potentially be regulated by this action. Other types of entities not listed in the table could also be regulated. To determine whether your (facility, company, business, organization, etc.) is regulated by this action, you should carefully examine the applicability criteria in Part I, Section A.1 of this permit. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding **FOR FURTHER INFORMATION CONTACT** section.

Pursuant to section 402 of the Clean Water Act (CWA), 33 U.S.C. section 1342, EPA proposed and solicited public comment on NPDES General Permit TXG340000 at 63 FR 41848 (August 5, 1998). The comment period closed on October 5, 1998. Region 6 received written comments from Texas Natural Resources Conservation Commission, Texas Oil and Gas Association, Chevron Products Company, and DynMcDermott Petroleum Operations Company.

EPA Region 6 has considered all comments received. In response to the comments, EPA agrees to extend the time for existing dischargers from no later than 30 days to no later than 90 days from the permit effective date to submit Notices of Intent to be covered by the permit. In addition, EPA agrees to reduce the monitoring frequency for the 24 hour acute toxicity requirement from twice per year to once per year, and to allow a facility with multiple storm water outfalls discharging substantially identical storm water effluents to collect and analyze an effluent sample for one of those outfalls and report that the data also applies to the other substantially identical outfalls.

Other Legal Requirements

A. State Certification

Under section 401(a)(1) of the Act, EPA may not issue an NPDES permit until the State in which the discharge will originate grants or waives certification to ensure compliance with appropriate requirements of the Act and State law. The Region has received certification, dated August 14, 1998, from the Texas Natural Resources Conservation Commission for NPDES General Permit TXG340000.

B. Endangered Species Act

EPA has determined that issuance of this general permit is unlikely to adversely affect any threatened or endangered species or its critical habitat. EPA sought written concurrence from the United States Fish and Wildlife Service on this determination. In a letter dated September 2, 1998, the United States Fish and Wildlife Service concurred with EPA's finding that issuance of this general permit is not likely to adversely affect any federally listed species, provided that two general concerns were addressed in the permit. The first concern was in regard to the 24-hour acute testing requirement. The Service was concerned that the permit language does not specify as to how test