Send comments on the Agency's need for this information, the accuracy of the provided burden estimates, and any suggested methods for minimizing respondent burden, including through the use of automated collection techniques to the following addresses. Please refer to EPA ICR No. 1863.01 in any correspondence.

Ms. Sandy Farmer, U.S. Environmental Protection Agency, OP Regulatory Information Division (2137), 401 M Street, SW, Washington, DC 20460; and

Office of Information and Regulatory Affairs, Office of Management and Budget, Attention: Desk Officer for EPA, 725 17th Street, NW, Washington, DC 20503.

Dated: November 30, 1998.

Richard T. Westlund,

Acting Director, Regulatory Information Division.

[FR Doc. 98–32414 Filed 12–4–98; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[FRL–6198–2]

Notice of Public Meetings on Drinking Water Issues

Notice is hereby given that the Environmental Protection Agency (EPA) is holding a public meeting on December 15 and 16, 1998 at the Park Hyatt Hotel, 24th and M Street, NW., Washington DC for the purpose of information exchange with stakeholders on issues related to the development of regulations to control microbial pathogens and disinfection byproducts in drinking water, including a Stage 2 Disinfectants/Disinfection Byproducts Rule and a Long-term 2 Enhanced Surface Water Treatment Rule. The meeting will start at 9:00 AM on December 15 and will adjourn on December 16 at 3:30 PM. The meeting will provide: (1) A review of the Stage 1 Disinfectants/Disinfection Byproducts Rule and the Interim Enhanced Surface Water Treatment Rule; (2) an overview of the Information Collection Rule and research supporting the drinking water rules; (3) an opportunity for stakeholders to discuss the issues and process for completing the Stage 2 deliberations; and (4) an opportunity to discuss schedules for subsequent meetings.

EPA is inviting all interested members of the public to participate in the meeting. As with all previous meetings in this series, to the extent that space is available, EPA is instituting an open door policy to allow any member of the public to attend any of the meetings for any length of time. Approximately 50 seats will be available for the public. Seats will be available on a first-come, first served basis.

For additional information about the meeting, please contact Ephraim King or Mike Cox of EPA's Office of Ground Water and Drinking Water at (202) 260–7575 or by e-mail at cox.michael@epamail.epa.gov.

Dated: December 1, 1998.

Elizabeth Fellows,

Acting Director, Office of Ground Water and Drinking Water.

[FR Doc. 98–32413 Filed 12–4–98; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–848; FRL–6047–2]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticide petitions proposing the establishment of regulations for residues of certain pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–848, must be received on or before January 6, 1999.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), 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
Mary Waller (PM 21)	Rm. 247, CM #2, 703–308–9354, e-mail:waller.mary@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Cynthia Giles-Parker (PM 22).	Rm. 247, 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–848] (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 (insert docket number) 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: November 24, 1998.

James Jones,

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. BASF Corporation, Agricultural Products

PP 7E4874

EPA has received a pesticide petition (PP 7E4874) from BASF Corporation, Agricultural Products, P.O. Box 13528, Research Triangle Park, NC 27709, 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 an import tolerance for residues of the fungicide fenpropimorph, (+)-cis-4-(3-(4-tert-butylphenyl)-2-methylpropyl)-2,6-dimethylmorpholine in or on the raw agricultural commodity bananas at 1.5 parts per million (ppm) of which no more than 0.3 ppm is found in the pulp. 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 and animal metabolism*. BASF Corporation notes that metabolism in plants and animals is understood.

2. *Analytical method*. The method of analysis includes extraction, liquid/liquid partition, column clean-up, and quantitation by gas chromatography/nitrogen-phosphorus detector. The overall fortification recoveries from the unpeeled, whole banana, and the peeled (pulp) samples together averaged 87.1% ± 9.3% (N=76).

3. *Magnitude of residues*. Fifteen crop residue trials were conducted in the banana growing regions of Mexico, South, and Central America including three sites in Colombia, four sites in Costa Rica, four sites in Ecuador, one site in Guatemala, two sites in Honduras, and one site in Mexico. Four sequential applications were made at the target rate of 545 g/ha to both bagged and unbagged bananas at each site. Fruit from both the bagged and unbagged treatments were harvested at 0 days following the last application.

Whole fruit (peel and pulp) samples and pulp only samples were analyzed for all treatments at all sites. Under typical practices (bagged bananas) residue in the whole fruit ranged from < the limit of quantitation (LOQ) (0.050 milligrams/kilogram (mg/kg)) to a maximum of 0.4 mg/kg. Banana pulp residues from bagged bananas ranged from < the LOQ (0.050 mg/kg to 0.20 mg/kg and averaged 0.0518 mg/kg. The average value was calculated by assuming all values below the LOQ were equal to one-half the LOQ or 0.025 mg/kg.

Under worst-case practices (unbagged bananas) residue in the whole fruit ranged from < the LOQ (0.050 mg/kg to a maximum of 1.4 mg/kg. Banana pulp residues from unbagged bananas ranged from < the LOQ (0.050 mg/kg to 0.43 mg/kg and averaged 0.1149 mg/kg. The average value was calculated by assuming all values below the LOQ were equal to one-half the LOQ or 0.025 mg/kg.

B. Toxicological Profile

1. *Acute toxicity*. Based on available acute toxicity data fenpropimorph does not pose any acute toxicity risks. These studies are not required for an import tolerance, but we have provided the following paragraph to demonstrate that fenpropimorph is not an acute toxicant. The acute toxicity studies place technical fenpropimorph in acute toxicity category III for acute oral, dermal, inhalation, and skin irritation; and in acute toxicity category IV for eye irritation and the technical material is not a skin sensitizer. Additionally, results of an acute oral neurotoxicity and a subchronic oral feeding neurotoxicity study demonstrated that fenpropimorph was not a neurotoxic compound.

2. *Genotoxicity*. A Modified Ames Test (1 Study; point mutation): Negative; *In Vitro* Cytogenetics-Human lymphocytes (1 Study; Chromosome Aberrations): Negative; Mouse Micronucleus Assay (1 Study; Chromosome Aberrations): Negative; *In Vitro* UDS Test Using Rat Hepatocytes (1 Study; DNA damage and repair): Negative; fenpropimorph has been tested in a total of 4 genetic toxicology assays. These assays were performed both *in vitro* and *in vivo* and multiple assays were conducted for each of the three EPA Guideline requirement categories. Based on the data presented in this petition, fenpropimorph does not induce gene mutations and does not induce other effects indicative of genotoxicity. Fenpropimorph does not pose a mutagenic hazard to humans.

3. *Reproductive and developmental toxicity*. A 2-generation reproduction study with rats fed dosages of 0, 0.625, 1.25, and 2.5 milligrams/kilogram/day (mg/kg/day) (average mg/kg/day dose levels for both male and female rats) with a reproductive no observed adverse effect level (NOAEL) of 2.5 mg/kg/day and with a parental NOAEL of 2.5 mg/kg/day based on; (i) no treatment-related clinical signs, significant body weight changes, parameters of fertility and gestation, or macro- or histopathological changes were observed for the parental F0 and F1 at all dose levels tested; (ii) in the F1 litters, a slight increased incidence of stillborn pups, unfolding of the ear, and slight reduced body weight development during lactation were observed in the 2.5 mg/kg/day dose level group; and (iii) in the F2 litters, no treatment-related effects were observed at all dose levels tested.

A developmental prenatal study was conducted via oral gavage in rats resulted in dosages of 0,2.5, 10, 40, and 160 highest dose tested (HDT) mg/kg/day from day 6 to 15 of gestation with a development toxicity NOAEL of 40 mg/kg/day and a maternal toxicity of 10 mg/kg/day based on the following: (i) signs of maternal toxicity, in the form of

decreased body weights and/or clinical signs observed at dose levels > 40 mg/kg/day; (ii) maternal animals in the 160 mg/kg/day dose group showed an increased incidence of vaginal bleeding from day 10 to 19 of gestation and increased placental weight; (iii) maternal animals in the 160 mg/kg/day dose group showed an increase in the number of resorptions as compared to controls; (iv) decreases in fetal body weights and size and number of viable fetus were observed at the HDT; (v) a significant number of fetuses had a finding of cleft palate in the high dose group tested were observed; and (vi) litters from animals treated at the lower doses remained entirely unaffected.

A second developmental perinatal study was conducted via oral gavage in rats resulted in dosages of 0, 2.5, 10, 40, and 160 HDT mg/kg/day from day 15 to 21 of gestation with a development toxicity NOAEL of 40 mg/kg/day and a maternal toxicity of 40 mg/kg/day based on the following: (i) four animals died on days 1 to 6 after delivery; (ii) signs of maternal toxicity, in the form of decreased body weights and/or clinical signs observed at the top dose level; (iii) at birth, body weight was significantly reduced in the pups of the top dose group; (iv) the brood care at the top dose group animals was generally unsatisfactory and led to a high perinatal mortality of the fetuses with only 30 viable fetuses left on day 1 post partum, the dead fetuses showed no increased incidence of malformations; (v) the few surviving pups of the dams at the 160 mg/kg/day dose group showed decreases in fetal body weights and size was retarded, no disturbances were found in the functional and behavioral tests that were conducted on the surviving pups; (vi) at necropsy, all dams showed comparable number of implantations and the animals scarified as scheduled revealed no treatment-related changes and also the mean organ weights were similar in treated and untreated groups; and (vii) litters from animals treated at the lower doses remained entirely unaffected and no pathological findings were also noted in these pups.

A series of two developmental study, Study A dose levels were 0, 2.4, 12, 36, and 60 mg/kg/day and, Study B dose levels were 0, 7.5, 15, and 30 mg/kg/day were conducted via oral gavage in rabbits resulted in dosages of 0, 2.4, 7.5, 12, 15, 30, 36, and 60 HDT mg/kg/day with a development toxicity NOAEL of 15 mg/kg/day and a maternal toxicity of 15 mg/kg/day based on the following: (i) Severe clinical signs and/or mortality were observed at dose levels > 30 mg/kg/day; (ii) decreased body weight, food consumption, and absorption/premature delivery in the 36 and 60 mg/kg/day dose groups which survived to the end of the studies; (iii) fetal effects consisted of high number of dead fetuses and several gross malformations (pseudo ancylosis, syndactylia, micromelia, aplasia of the twelveth rib) at the HDT; and (iv) pseudo ancylosis was also seen in 1 fetus from the 12 mg/kg/day dose group and in 6 fetuses in the 36 mg/kg/day dose level, but this finding is known to occur spontaneously in rabbits of this strain used and the contractures usually normalize during early stages of life. Due to the severe effect at the high dose level (HDL), these effects may be considered to represent a specific teratogenic effect of the treatment.

4. *Chronic toxicity*. Based on review of the available data, BASF believes the Reference Dose (RfD) for fenpropimorph will be based on a 2-year feeding study in rats with a threshold NOAEL of 0.3 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.003 mg/kg/day. The following are summaries of the pertinent toxicity data supporting fenpropimorph tolerances. Additionally, these are summaries of EPA reviewed Phase III Toxicology Summaries prepared by BASF Corporation for EPA.

A 1 year feeding study in dogs fed dosages of 0, 0.8, 3.2, or 12.7 mg/kg/day with a NOAEL of 3.2 mg/kg/day based on the following effects: (i) no changes in body weights nor food consumption for both the high dose male and female dogs were observed at all tested dose levels as compared to controls; (ii) blood biochemistry values were slightly increased in high dose males (alkaline phosphatase) and females (alanine aminotransferase); (iii) the cholininesterase from plasma, red blood cells, and brain showed comparable activities in treated and untreated dogs; and (iv) neither organ weight analyses nor macro- and histopathological examinations demonstrated any treatment related effects as compared to controls.

A combined chronic feeding/oncogenicity study was performed in rats being fed dosages of 0, 0.2, 0.3, 1.7 and 8.8 mg/kg/day (males) and 0, 0.2, 0.4, 2.1, and 11.2 mg/kg/day (females) with a NOAEL of 0.3 mg/kg/day (males) and 0.4 mg/kg/day (females) based on the following effects: (i) decreased in body weights were observed in both males and female rat at dose levels > 1.7 mg/kg/day with a very slight progression of severity to the upper level; (ii) decreased food consumption in female rats at the HDT; (iii) significantly lower activities of plasma cholinesterase were noted in male and female rats in the HDT where as no effect was found for red blood cell cholinesterase values; (iv) at terminal sacrifice, reduced activities of brain cholinesterase were detected in males, only, at the 1.7 and 8.8 mg/kg/day dose levels groups tested; (v) increased liver weights for females at dose levels > 2.1 mg/kg/day and in males of the top dose group; (vi) microscopic findings were observed in the liver of male and female rats in the HDLs, only; and (vii) no increased incidence of neoplasms occurred at any dose levels tested in this study.

A carcinogenicity study in mice fed dosages of 0, 0.5, 3.0, 16, and 106 HDT mg/kg/day (males) and 0, 0.5, 3.5, 17, and 118 HDT mg/kg/day (females) with a NOAEL of 3.0 and 3.5 mg/kg/day for male and female mice, respectively, based on the following effects: (i) decreased body weights and slight inferior food conversion ratio were observed in both male and female mice at the HDT; (ii) decreased cholinesterase activities were observed in red blood cells for female mice in the 17 and 118 mg/kg/day dose level tested at terminal sacrifice; (iii) at the HDT increased liver weights were observed for female mice at terminal sacrifice and in males at interim sacrifice after 52 weeks; and (iv) no increased incidence of neoplasms occurred at any dose levels tested in this study.

5. *Endocrine disruption*. No specific tests have been performed with fenpropimorph to determine whether the chemical may have an effect in humans that is similar to an effect produced by naturally occurring estrogen or other endocrine effects. However, there are significant findings in other relevant toxicity studies, i.e. teratology and multi-generation reproductive studies, that would suggest fenpropimorph produces endocrine related effects.

C. Aggregate Exposure

Based on the information above it is concluded that the RfD used to assess safety to children should be 0.003 mg/kg/day dose level established in the 2-year rat oral feeding study. Using the assumption stated for the general population, BASF concluded that the most sensitive child population group is that of children > 1 year. Using the same RfD and the same conservative exposure assumptions employed in the dietary risk analysis for the general population. It was calculated that the exposure to this group to be approximately > 11% of the RfD for all uses proposed in this document. Therefore, based on the completeness and reliability of the

toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to residues of fenpropimorph, including all anticipated dietary exposure.

1. *Dietary exposure*. For the purpose of assessing the potential chronic dietary exposure, BASF has estimated aggregate exposure based on Theoretical Maximum Residue Contribution (TMRC) from the tolerance of fenpropimorph on bananas at 0.3 ppm the maximum residue found in bananas. The TMRC is a ''worse case'' estimate of dietary exposure since it is assumed that 100% of all crops for which the tolerances are established are treated and that pesticide residues are always found at tolerance levels. Based on the expected RfD of 0.003 mg/kg/day (from the NOAEL determined in the 2-year feeding study in rats and a 100 fold safety factor) and the tolerance level residue chronic dietary exposure of the general population is less than 2.5% of the RfD. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of fenpropimorph, including all anticipated dietary exposure.

2. *Food*. BASF has reviewed the available toxicology database to determine the endpoints of concern. For Fenpropimorph BASF believes there is no concern regarding an acute dietary risk since the available data do not indicate any evidence of significant toxicity from a 1-day or single, event exposure by the oral route.

3. *Drinking water/Non-dietary exposure*. There are no other potential sources (such as in drinking water and exposure from non-occupational sources) of exposure to fenpropimorph for the general population to residues of fenpropimorph due to the fact the action being requested is to establish an import tolerance, only.

4. *Threshold and non-threshold effects*. The proposed RfD for fenpropimorph is based on a 2-year feeding study in rats with a threshold NOAEL of 0.3 mg/kg/day. Using an uncertainty factor of 100, the RfD is calculated to be 0.003 mg/kg/day. Fenpropimorph is considered not to be a carcinogenic material. Therefore, it should be regulated by the traditional RfD approach to quantify human risk.

D. Cumulative Effects

BASF has considered the potential for cumulative effects of fenpropimorph and other substances that have a common mechanism of toxicity. BASF is not aware of any other active ingredients which is structurally similar to fenpropimorph that are registered on bananas. Therefore, BASF has considered only the potential risks of fenpropimorph in its exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the exposure assumptions described above, based on the completeness and there liability of the toxicity data, BASF has estimated that aggregate exposure to fenpropimorph will utilize > 2.5% of the RfD for the U.S. population. EPA generally has no concern for exposure below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data, and the exposure assessment discussed above, BASF concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of fenpropimorph, including all anticipated dietary exposure.

2. *Infants and children*. The findings in the rat and rabbit are most likely as a result of excessive maternal toxicity, treatment of pregnant rats and rabbits with fenpropimorph induced embryotoxic effects which manifested themselves in the form of early resorptions and structural anomalies in the offspring. In both the rat and rabbit, the dose-effect relationship was rather steep and showed clear threshold levels. At dose levels below the threshold of maternal toxicity, reproductive parameters as well as the offsprings remained entirely unaffected. This data demonstrates that the rat and rabbit are similarly sensitive to fenpropimorph. Additionally, the NOAEL of 0.3 mg/kg/day from the chronic rat study used to set the RfD is 33x to 50x lower than the maternal NOAELs established in the rat and rabbit teratology studies, respectively. The developmental effects observed in either the rat or rabbit occurred only at maternally toxic doses. Therefore, no additional safety factor is needed for children.

A 2-generation reproduction study with rats fed dosages of 0, 0.625, 1.25, and 2.5 mg/kg/day (average mg/kg/day dose levels for both male and female rats) with a reproductive NOAEL of 2.5 mg/kg/day and with a parental NOAEL of 2.5 mg/kg/day based on: (i) no treatment-related clinical signs, significant body weight changes, parameters of fertility and gestation, or macro-or histopathological changes were observed for the parental F0 and F1 at all dose levels tested; and (ii) in the F1 litters, a slight increased incidence of stillborn pups, unfolding of the ear, and slight reduced body weight development during lactation were observed in the 2.5 mg/kg/day doselevel group; (iii) in the F2 litters, no treatment-related effects were observed at all dose levels tested. As stated above, the NOAEL of 0.3 mg/kg/day from the chronic rat study used to set the RfD is approximately 8x lower than the maternal NOAEL established in the rat reproduction study. Therefore, no additional safety factor is needed for children.

F. International Tolerances

A maximum residue level has not been established under Codex Alimentarius Commission for fenpropimorph in any of the crops petitioned: bananas.

2. Rohm and Haas Company

PP 1F3995, 1F3989 and 2F4154

EPA has received data intended to satisfy the conditions which caused time-limits to be placed on the tolerances proposed by the three pesticide petitions PP 1F3995, 1F3989, and 2F4154 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, 21 U.S.C. 346a(d), to amend 40 CFR part 180 by extending until December 31, 2001 the time-limited tolerances for residues of fenbuconazole (alpha-(2-(4-chlorophenyl)-ethyl)-alpha-phenyl-3-(1H-1,2,4-triazole)-1-propanenitrile) in or on the raw agricultural commodities bananas at 0.3 parts per million (ppm), banana pulp at 0.05 ppm, stone fruits (except plums and prunes) at 2.0 ppm, and pecans at 0.1 ppm. EPA has determined that the submissions concern the additional data requirements as elements set forth in section 408(f)(1) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time.

A summary of the data that support the tolerances, and of exposure to and risks from the use of fenbuconazole, is printed below. This summary of the petitions was prepared by the registrant and represents the views of the registrant. EPA is publishing the petition summary with only minor editing changes. The petition summary includes an announcement of the availability of the analytical methods available to EPA for the detection and

measurement of the pesticide chemical residues.

A. Residue Chemistry

The tolerance expression for fenbuconazole residues in or on bananas, banana pulp, pecans, and stone fruit (except plums and prunes) is the combined residues of fenbuconazole (alpha-(2-(4-chlorophenyl)-ethyl)-alpha-phenyl-3-(1H-1,2,4-triazole)-1-propanenitrile) and its metabolites cis-5-(4-chlorophenyl)-dihydro-3-phenyl-3-(1H-1,2,4-triazole-1-ylmethyl)-2-3H-furanone and trans-5-(4-chlorophenyl)-dihydro-3-phenyl-3-(1H-1,2,4-triazole-1-ylmethyl)-2-3H-furanone. Residues of these compounds are combined and expressed as parent compound to determine the total residue in or on bananas, banana pulp, pecans, and stone fruit (except plums and prunes). No changes in the tolerances of fenbuconazole or in the tolerance expression (parent plus lactone metabolites) for pecans, bananas, or stone fruit from that indicated in 40 CFR 180.480 will be necessary for the tolerance extensions. Current tolerances for fenbuconazole are 0.3 ppm for banana whole fruit, 0.05 ppm for banana pulp, 0.1 ppm for pecans, and 2.0 ppm for the stone fruit crop group (except plums and prunes). There is also a current time-limited (Section 18) tolerance for fenbuconazole on blueberries of 1.0 ppm.

1. *Analytical method*. Fenbuconazole residues (parent plus lactones) are measured in pecans, stone fruit, and bananas at an analytical sensitivity of 0.01 milligrams/kilogram (mg/kg) 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.

2. *Magnitude of residues*—i. *Pecans*. Four field trials were conducted in pecans. Eight to ten applications were made at the maximum use rate of 0.125 lb a.i./A, and nuts were harvested 28 days after the last application. Field residue values in nutmeat for the four trials were 0.004, 0.004, <0.01, and <0.01 ppm.

ii. *Bananas*. Fourteen field trials were conducted on pulp from bagged bananas, and nine field trials were conducted on whole fruit from bagged bananas. Bagged bananas are typically used in commerce. Eight applications (5 and 7 applications in two trials) were made at the maximum use rate of 0.09 lb a.i./A and bananas were harvested on the last day of application. The highest field residue values were 0.019 ppm in pulp and 0.0589 ppm in whole fruit. The average field residue values were 0.004 ppm in pulp and 0.010 ppm in whole fruit.

iii. *Stone fruit*—a. *Peaches*. Ten field trials were conducted on peaches. Seven to ten applications were made at the maximum use rate of 0.1 lb a.i./A and fruit were harvested on the last day of application. The highest field residue value was 0.5096 ppm, and the average field residue value was 0.351 ppm.

b. *Cherries*. Eleven field trials were conducted on cherries. Five to six applications were made at the maximum use rate of 0.1 lb a.i./A and fruit were harvested on the last day of application. The highest field residue value was 0.641 ppm, and the average field residue value was 0.434 ppm.

c. *Apricots*. Two field trials were conducted on apricots. Six applications were made at the maximum use rate of 0.125 lb a.i./A and fruit was harvested on the last day of application. The field residue values in four samples measured were 0.168, 0.226, 0.268, and 0.279 ppm.

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 andrespiratory 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), in consequentially 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, negative in *in vitro* and *in vivo* somatic and germcell tests, and did not induce unscheduled in DNA synthesis (UDS). Fenbuconazole is not genotoxic.

3. *Reproductive and developmental toxicity*. These conclusions were extracted from 60 FR 27419, May 24, 1995. Fenbuconazole is not teratogenic. The maternal no observed adverse effect level (NOAEL) in rabbits was 10 mg/kg/day and 30 mg/kg/day in rats. The fetal NOAEL was 30 mg/kg/day in both species. The parental NOAEL was 4.0 mg/kg/day (80 ppm) in a 2-generation reproduction study in rats. The reproductive NOAEL in this study was greater than 40.0 mg/kg/day (800 ppm; highest dose tested (HDT)). Fenbuconazole had no effect on male reproductive organs or reproductive performance at any dose. The adult lowest observed adverse effect level (LOAEL) was 40.0 mg/kg/day (800 ppm; HDT). Systemic effects of decreased body weight gain; maternal deaths; and hepatocellular, adrenal, and thyroid follicular cell hypertrophy were observed. No effects on neonatal survival or growth occurred below the adult toxic levels. Fenbuconazole does not produce birth defects and is not toxic to the developing fetus at doses below those which are toxic to the mother.

4. *Subchronic toxicity*. In a 21 day dermal toxicity study in the rat, the NOAEL 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 NOAEL 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 NOAEL was 150 ppm (3.75 mg/kg/day) based on decreased body weight, and increased liver weight. The LOAEL was 1,200 ppm (30 mg/kg/day). In a 78 week oncogenicity study in mice, the NOAEL was 10 ppm (1.43 mg/kg/day). The LOAEL was 200 ppm (26.3 mg/kg/day, males) and 650 ppm (104.6mg/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 NOAEL of 3.0 mg/kg/day from the rat chronic study. This RfD adequately protects both adults and children.

Twenty-four month rat chronic feeding/carcinogenicity studies with fenbuconazole showed effects at 800 and 1,600 ppm. Fenbuconazole produced a minimal, but statistically

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

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

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

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

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

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

6. *Endocrine disruption*. 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 NOAEL. 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 and Risk

1. *Dietary exposure—Chronic exposure and risk*. Risk associated with chronic dietary exposure from fenbuconazole was assessed on two level using two dietary exposure models. In the first assessment, tolerance level residues were assumed and in the second assessment average field trial residues were used. Both assessments assumed 100% of crop treated, except for stone fruit in which 12.8% of crop treated was assumed 63 FR 31636, June 10, 1998, (FRL 5791-9). Residues in pulp from bagged bananas were used in the assessments, since only bagged bananas are used in commerce. The Anticipated Residue Contribution (ARC) from all existing food uses of fenbuconazole was assessed; these foods included stone fruit (except plums, and prunes), bananas, pecans, and blueberries).

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, and the EPA's Dietary Risk Evaluation System (DRES), which uses USDA food consumption data from 1977-1978. The existing 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	DRES[1] %RfD	DRES[2] %RfD
U. S. Population (48 States)	0.2%	<0.01%	0.31%	0.06%
Nursing Infants (<1 year old)	0.4%	0.1%	1.47%	0.27%
Non-Nursing Infants (<1year old)	1.3%	0.2%	2.46%	0.45%

Population Subgroup	DEEM[1] %RfD	DEEM[2] %RfD	DRES[1] %RfD	DRES[2] %RfD
Children (1-6 years old)	0.5%	0.1%	0.74%	0.14%
Children (7-12 years old)	0.3%	<0.01%	0.44%	0.08%
Females (13+/nursing)	0.3%	<0.01%	0.28%	0.05%

[1] Assumes residues are present at tolerance levels and 100% of crop treated except stone fruit (12.8% of crop treated).
[2] Assumes residues are present at their average field residue levels and 100% of crop treated except stone fruit (12.8% of crop treated).

D. Aggregate Cancer Risk for U.S. Population

Fenbuconazole has been classified as a Group C Carcinogen with a Q,* value of 0.00359 mg/kg/day^{-1}. Assuming fenbuconazole residues are present at tolerance levels and assuming 100% crop treated, except stone fruit (12.8% of crop treated assumed), give a cancer risk assessment for existing food uses for the U.S. population of 3.31 x 10^{-7} for the DRES and DEEM analyses, respectively. Assuming fenbuconazole residues are present at average field residue levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed), gives a cancer risk assessment for existing food uses for the U.S. population of 6.34 x 10^{-8} and 4.94 x 10^{-8} for the DRES and DEEM analyses, respectively.

The individual crop cancer risk assessments for bananas, stone fruit, pecans, and blueberries were 4.11 x 10^{-8}, 2.78 x 10^{-7}, 1.73 x 10^{-9}, and 9.74 x 10^{-9}, respectively (DRES analysis), and were 5.11 x 10^{-8}, 1.67 x 10^{-7}, 7.37 x 10^{-10}, and 1.38 x 10^{-8}, respectively (DEEM analysis).

1. *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.

2. *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.

E. Cumulative Effects

The toxicological effects of fenbuconazole are related to its effects on rodent liver. These are manifested 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 NOAEL 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.

F. Safety Determination.

1. *All crops (current food uses).* The exposure to fenbuconazole from all current food uses will utilize 1.3% (non-nursing infants < 1 year old) and 0.4% (nursing infants < 1 year old) of the RfD (DEEM analysis), and will utilize 2.46% (non-nursing infants < 1 year old) and 1.47% (nursing infants < 1 year old) of the RfD (DRES analysis), assuming residues are present at tolerance levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed). The percent of the RfD that will be utilized by children 1-6 years old and 7-12 years old is 0.5 and 0.3%, respectively (DEEM analysis), and 0.74 and 0.44%, respectively (DRES analysis), assuming residues are present at tolerance levels and assuming 100% crop treated, except stone fruit.

2. *Stone Fruit (except plums and prunes).* The exposure to fenbuconazole from stone fruit (excluding plums and prunes) will utilize 1.1% of the RfD for non-nursing infants < 1 year old, 0.3% of the RfD for nursing infants < 1 year old, 0.4% of the RfD for children 1-6 years old, and 0.2% of the RfD for children 7-12 years old (DEEM analysis) assuming residues are present at tolerance levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed).

3. *Bananas.* The exposure to fenbuconazole from bananas will utilize 0.2% of the RfD for non-nursing infants < 1 year old, 0.1% of the RfD for nursing infants < 1 year old, 0.1% of the RfD for children 1-6 years old, and 0.1% of the RfD for children 7-12 years old (DEEM analysis) assuming residues are present at tolerance levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed).

4. *Pecans.* The exposure to fenbuconazole from pecans will utilize

<0.01% of the RfD for each of the population subgroups: non-nursing infants < 1 year old, nursing infants < 1 year old, children 1-6 years old, and children 7-12 years old (DEEM analysis) assuming residues are present at tolerance levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed).

5. *Blueberries*. The exposure to fenbuconazole from blueberries, will utilize < 0.01% of the RfD for each of the population subgroups, non-nursing infants < 1 year old, nursing infants < 1 year old, children 1-6 years old, and children 7-12 years old (DEEM analysis) assuming residues are present at tolerance levels and assuming 100% of crop treated, except stone fruit (12.8% of crop treated assumed).

Section 408 of the FFDCA provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a margin of exposure (MOE) analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. In either case, EPA generally defines the level of appreciable risk as exposure that is greater than 1/100 of the NOAEL in the animal study appropriate to the particular risk assessment. This hundredfold uncertainty (safety) factor/MOE exposure (safety) is designed to account for combined inter- and intra-species variability. EPA believes that reliable data support using the standard hundredfold margin/factor but not the additional tenfold margin/factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard margin/factor.

The Agency FQPA Safety Factor Committee removed the additional 10x safety factor to account for sensitivity of infants and children. Rohm and Haas Company concludes that there is a reasonable certainty that no harm will result from exposure to fenbuconazole residues to the U.S. population or to infants and children.

G. International Tolerances

There are no Codex maximum residue limits (MRLs) for fenbuconazole, but the fenbuconazole database was evaluated by the WHO and FAO Expert Panels at the Joint Meeting on Pesticide Residues (JMPR) in September, 1997. An ADI (RfD) of 0.03 mg/kg/day was proposed and accepted (Pesticide Residues in Food—WHO/FAO Report 1997; No. 145), and a total of 36 Codex MRLs, including MRLs for pecans, stone fruit, and bananas, have been submitted for review.

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FEDERAL COMMUNICATIONS COMMISSION

[CC Docket No. 95–155]

Toll Free Service Access Codes

AGENCY: Federal Communications Commission.

ACTION: Notice; letter.

SUMMARY: The Network Services Division, Common Carrier Bureau, has issued a letter stating that 145 RespOrgs failed to report to Database Service Management, Inc., as required, that they gave notice to all of their subscribers having right of first refusal for set-aside 888 numbers. By December 11, 1998, these RespOrgs must explain why they failed to comply with this requirement and must describe their actions to remedy their non-compliance. RespOrgs that fail to submit explanations or that fail to provide satisfactory explanations will be subject to possible forfeiture penalties, decertification as RespOrgs, or fines, imprisonment, or both.

FOR FURTHER INFORMATION CONTACT: Marty Schwimmer 202–418–2334.

SUPPLEMENTARY INFORMATION: The Bureau's letter is attached.

Federal Communications Commission.

Anna M. Gomez,

Chief, Network Services Division, Common Carrier Bureau.

Attachment

November 24, 1998.

Mr. Michael Wade
President, Database Service Management, Inc.
6 Corporate Place
Room PYA—1F286
Piscataway, NJ 08854–4157

Re: RespOrg non-compliance with the set-aside 888 number right-of-first-refusal process—Requirement for specified RespOrgs to submit letters of explanation by December 11, 1998

Dear Mr. Wade: The Bureau's letter to you dated April 15, 1998, initiated the process for subscribers to exercise their right of first refusal to request 888 numbers that had been set aside for them. It required RespOrgs to give notice of this right to their subscribers. Further, among other things, it required RespOrgs, for each set-aside 888 number, to submit to DSMI either the subscriber's request to accept or reject an 888 set-aside number, with documentation, or certification that the subscriber did not respond to the notice.

The Bureau's letter to you dated May 15, 1998, extended to August 21, 1998, the time for RespOrgs to give the required notice to their subscribers, although it provided that requests received from subscribers after that date must still be processed. It also explained that the certification that RespOrgs were required to provide for subscribers who did not respond must include contact information containing the subscriber's name, address, and phone number, as well as the date and means by which the RespOrg notified the subscriber.

The attachment to this letter summarizes the RespOrgs' compliance with this process, using information provided by your staff in response to the Bureau's request. For each of 179 RespOrgs, the attachment shows the total percentage of requests and certifications of no response reported to DSMI as of October 5, 1998, based on the RespOrg's initial count of set-aside 888 numbers as of July 1998. It indicates that only 34 RespOrgs reported subscriber notification results for all of their set-aside 888 numbers (100%). Of the remaining 145 RespOrgs, 93 reported results for some but not all of their set-aside 888 numbers (0.1% to 99.7%), and 52 did not report any results for their set-aside 888 numbers (0%).

The Commission is concerned that a RespOrg's failure to report that it gave notice to each of its set-aside 888 number subscribers may indicate that the RespOrg is operating in defiance of Commission orders, that it is warehousing set-aside 888 numbers or the corresponding 800 numbers, or that it has falsely indicated that it has identified subscribers for those numbers. The Commission stated last year that it may penalize RespOrgs that warehouse toll free numbers, by imposing forfeiture penalties on them or referring them to the Department of Justice to determine whether a fine, imprisonment, or both are warranted, or may decertify them as RespOrgs. It also stated that RespOrgs that falsely indicate they have identified subscribers for particular numbers may be criminally liable for false statements under Title 18 of the United States Code. The Commission stated as follows:

''We conclude that the Commission's exclusive jurisdiction over the portions of the North American Numbering Plan that pertain to the United States, found at section 251(e)(1) of the Communications Act, as amended, authorizes the Commission to penalize RespOrgs that warehouse toll free numbers. We may impose a forfeiture penalty under section 503(b). In addition, if a person violates a provision of the Communications Act or a rule or regulation issued by the Commission under authority of the Communications Act, the Commission can refer the matter to the Department of Justice to determine whether a fine, imprisonment, or both are warranted under section 501 or section 502 of the Communications Act. We also may limit any RespOrg's allocation of toll free numbers or possibly decertify it as