motor vehicle recycling program are derived from Section 114 of the Act. An agency may not conduct or sponsor, and a person is not required to respond to, a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations are listed in 40 CFR Part 9 and 48 CFR Chapter 15. The **Federal Register** Notice required under 5 CFR 1320.8(d), soliciting comments on this collection of information was published on 9/4/98 (63 FR 47284); no comments were received.

Burden Statement: The annual public reporting and recordkeeping burden for this collection of information is estimated to average .13 hours per response. Burden means the total time, effort, or financial resources expended by persons to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This reduction is due primarily to revisions in the estimates of the number of service facilities that must complete certifications for the equipment they have purchased. The Agency estimates that no more than 10,000 existing facilities, plus 4,000 new facilities, will need to complete the certification forms in any year. In addition, the reduction in burden hours from the original ICR is due in part to a revision in the estimate of the time it takes for a service facility manager to fill out the certification form. Compiling certification information and submitting it to EPA is estimated to be one half hour based on the limited nature of the information requested, and ease of obtaining the information. Compiling information from training programs and submitting it to EPA is estimated at two hours because of the brief nature of the document. The information can easily be incorporated into an establishment's mailing system. Compiling information on the independent laboratory equipment testing programs, requires independent laboratories to assemble test methodology, list equipment requirements, and review the SAE standards. EPA estimated one hour to compile the information. Substantially identical equipment submission of information is estimated at an hour to obtain information from a standard equipment owners manual. Regarding small containers purchased for resale only, EPA estimated one hour of industry time for recordkeeping requirements. To record names and addresses of off-site reclamation or recycling, EPA estimated five minutes based on the limited nature of the information requested and ease of obtaining the information. These estimates include the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

Respondents/Affected Entities: Automotive Technicians.

Estimated Number of Respondents: 56,037.

Number of Responses: 70,037.

Estimated Total Annual Hour Burden: 8,882 hours.

Estimated Total Annualized Cost Burden: $0.

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. 1617.03 and OMB Control No. 2060–0247 in any correspondence.

Ms. Sandy Farmer, U.S. Environmental Protection Agency, Office of Policy 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: February 10, 1999.

Richard T. Westlund,

Acting Director, Regulatory Information Division.

[FR Doc. 99–3836 Filed 2–16–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[PF–860; FRL–6060–1]

Rohm and Haas Company; 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–860, must be received on or before March 19, 1999.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch, Information Resources 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, Crystal Mall 2 (CM #2), 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. 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: By mail: Joseph Tavano, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location/telephone and e-mail address: Rm. 214, 1921 Jefferson Davis Hwy, Arlington, VA, Crystal Mall 2 (CM #2), 703–305–6411, e-mail: tavano.joseph@epamail.epa.gov.

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 raw 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 grantinig of the

petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–860 (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 (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: February 4, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Below summaries of the pesticide petitions are printed. The summaries of the petitions were prepared by the petitioner. 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

1. 7F4815

EPA has received a revised pesticide petition (7F4815) from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of tebufenozide [benzoic acid, 3,5-dimethyl-, 1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide] in or on the raw agricultural commodity crop grouping, pome fruit at 1.25 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of tebufenozide in plants (grapes, apples, rice and sugar beets) is adequately understood for the purpose of this tolerance. The metabolism of tebufenozide in all crops was similar and involves oxidation of the alkyl substituents of the aromatic rings primarily at the benzylic positions. The extent of metabolism and degree of oxidation are a function of time from application to harvest. In all crops, parent compound comprised the majority of the total dosage. None of the metabolites were in excess of 10% of the total dosage.

2. *Analytical method*. A validated high performance liquid chromatographic (HPLC) analytical method using ultraviolet (UV) detection is employed for measuring residues of tebufenozide in pome fruit. The method involves extraction by blending with solvents, purification of the extracts by liquid-liquid partitions and final purification of the residues using solid phase extraction column chromatography. The limit of quantitation of the method in pome fruit is 0.02 ppm.

3. *Magnitude of residues*. Magnitude of the residue studies were conducted in apples and pears using the maximum application rate of 0.308 pounds active ingredient per acre applied 6 times during the growing season. Fruit were collected 14 days after the last application and were analyzed for residues of tebufenozide. The average residue in apples from 12 trials was 0.52 ppm and the average residue detected in pears from 6 trials was 0.27 ppm. A tolerance of 1.25 ppm is proposed for residues of tebufenozide in or on pome fruit.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies with technical grade: Oral LD_{50} in the rat is > 5 grams for males and females - Toxicity Category IV; dermal LD_{50} in the rat is = 5,000 milligram/kilogram (mg/kg) for males and females - Toxicity Category III; inhalation LD_{50} in the rat is > 4.5 mg/l - Toxicity Category III; primary eye irritation study in the rabbit is a non-irritant; primary skin irritation in the rabbit > 5 mg - Toxicity Category IV. Tebufenozide is not a sensitizer.

2. *Genotoxicty*. Several mutagenicity tests which were all negative. These include an Ames assay with and without metabolic activation, an *in vivo* cytogenetic assay in rat bone marrow cells, and *in vitro* chromosome aberration assay in Chinese hampster ovary (CHO) cells, a CHO/Hypoxanthine guanine phophoribosyl transferase (HGPRT) assay, a reverse mutation assay with *E. Coli*, and an unscheduled DNA synthesis (UDS) assay in rat hepatocytes.

3. *Reproductive and developmental toxicity*. In a prenatal developmental toxicity study in Sprague-Dawley rats 25/group Tebufenozide was administered on gestation days 6–15 by gavage in aqueous methyl cellulose at dose levels of 50, 250, or 1,000 mg/kg/day and a dose volume of 10 ml/kg. There was no evidence of maternal or developmental toxicity; the maternal and developmental toxicity no observed adverse effect level (NOAEL) was 1,000 mg/kg/day.

In a prenatal developmental toxicity study conducted in New Zealand white rabbits 20/group Tebufenozide was administered in 5 ml/kg of aqueous methyl cellulose at gavage doses of 50, 250, or 1,000 mg/kg/day on gestation days 7–19. No evidence of maternal or developmental toxicity was observed; the maternal and developmental toxicity NOAEL was 1,000 mg/kg/day.

In a 1993 2-generation reproduction study in Sprague-Dawley rats tebufenozide was administered at dietary concentrations of 0, 10, 150, or 1,000 ppm (0, 0.8, 11.5, or 154.8 mg/kg/day for males and 0, 0.9, 12.8, or 171.1 mg/kg/day for females). The parental systemic NOAEL was 10 ppm (0.8/0.9 mg/kg/day for males and females, respectively) and the lowest observed effect level (LOEL) was 150 ppm (11.5/12.8 mg/kg/day for males and females, respectively) based on decreased body weight, body weight gain, and food consumption in males, and increased incidence and/or severity of splenic pigmentation. In addition, there was an increased incidence and severity of extramedullary hematopoiesis at 2,000 ppm. The reproductive NOAEL was 150 ppm. (11.5/12.8 mg/kg/day for males and females, respectively) and the LOEL

was 2,000 ppm (154.8/171.1 mg/kg/day for males and females, respectively) based on an increase in the number of pregnant females with increased gestation duration and dystocia. Effects in the offspring consisted of decreased number of pups per litter on postnatal days 0 and/or 4 at 2,000 ppm (154.8/171.1 mg/kg/day for males and females, respectively) with a NOAEL of 150 ppm (11.5/12.8 mg/kg/day for males and females, respectively).

In a 1995 2-generation reproduction study in rats Tebufenozide was administered at dietary concentrations of 0, 25, 200, or 2,000 ppm (0, 1.6, 12.6, or 126.0 mg/kg/day for males and 0, 1.8, 14.6, or 143.2 mg/kg/day for females). For parental systemic toxicity, the NOAEL was 25 ppm (1.6/1.8 mg/kg/day in males and females, respectively), and the LOEL was 200 ppm (12.6/14.6 mg/kg/day in males and females), based on histopathological findings (congestion and extramedullary hematopoiesis) in the spleen. Additionally, at 2,000 ppm (126.0/143.2 mg/kg/day in M/F), treatment-related findings included reduced parental body weight gain and increased incidence of hemosiderin-laden cells in the spleen. Columnar changes in the vaginal squamous epithelium and reduced uterine and ovarian weights were also observed at 2,000 ppm, but the toxicological significance was unknown. For offspring, the systemic NOAEL was 200 ppm. (12.6/14.6 mg/kg/day in males and females), and the LOEL was 2,000 ppm (126.0/143.2 mg/kg/day in M/F) based on decreased body weight on postnatal days 14 and 21.

4. *Subchronic toxicity*. In a prenatal developmental toxicity study in Sprague-Dawley rats 25/group Tebufenozide was administered on gestation days 6–15 by gavage in aqueous methyl cellulose at dose levels of 50, 250, or 1,000 mg/kg/day and a dose volume of 10 ml/kg. There was no evidence of maternal or developmental toxicity; the maternal and developmental toxicity NOAEL was 1,000 mg/kg/day.

5. *Chronic toxicity*. A 1-year dog feeding study with a LOEL of 250 ppm, 9 mg/kg/day for male and female dogs based on decreases in RBC, HCT, and HGB, increases in Heinz bodies, methemoglobin, MCV, MCH, reticulocytes, platelets, plasma total bilirubin, spleen weight, and spleen/body weight ratio, and liver/body weight ratio. Hematopoiesis and sinusoidal engorgement occurred in the spleen, and hyperplasia occurred in the marrow of the femur and sternum. The liver showed an increased pigment in the Kupffer cells. The NOAEL for systemic toxicity in both sexes is 50 ppm (1.9 mg/kg/day).

An 18-month mouse carcinogenicity study with no carcinogenicity observed at dosage levels up to and including 1,000 ppm.

A 2-year rat carcinogenicity with no carcinogenicity observed at dosage levels up to and including 2,000 ppm (97 mg/kg/day and 125 mg/kg/day for males and females, respectively).

6. *Animal metabolism*. The adsorption, distribution, excretion and metabolism of tebufenozide in rats was investigated. Tebufenozide is partially absorbed, is rapidly excreted and does not accumulate in tissues. Although tebufenozide is mainly excreted unchanged, a number of polar metabolites were identified. These metabolites are products of oxidation of the benzylic ethyl or methyl side chains of the molecule. These metabolites were detected in plant and other animal (rat, goat, hen) metabolism studies.

7. *Metabolite toxicology*. Common metabolic pathways for tebufenozide have been identified in both plants (grape, apple, rice and sugar beet) and animals (rat, goat, hen). The metabolic pathway common to both plants and animals involves oxidation of the alkyl substituents (ethyl and methyl groups) of the aromatic rings primarily at the benzylic positions. Extensive degradation and elimination of polar metabolites occurs in animals such that residue are unlikely to accumulate in humans or animals exposed to these residues through the diet.

8. *Endocrine disruption*. The toxicology profile of tebufenozide shows no evidence of physiological effects characteristic of the disruption of the hormone estrogen. Based on structure-activity information, tebufenozide is unlikely to exhibit estrogenic activity. Tebufenozide was not active in a direct *in vitro* estrogen binding assay. No indicators of estrogenic or other endocrine effects were observed in mammalian chronic studies or in mammalian and avian reproduction studies. Ecdysone has no known effects in vertebrates. Overall, the weight of evidence provides no indication that tebufenozide has endocrine activity in vertebrates.

C. Aggregate Exposure

1. *Dietary exposure* — i. *Food*. Tolerances have been established (40 CFR 180.482) for the residues of tebufenozide, in or on walnuts at 0.1 ppm, apples at 1.0 ppm, pecans at 0.01 ppm and wine grapes at 0.5 ppm. Numerous section 18 tolerances have been established at levels ranging from 0.3 ppm in sugar beet roots to 5.0 ppm in turnip tops. Other tolerance petitions are pending at EPA with proposed tolerances ranging from 0.3 ppm in or on sugarcane to 10 ppm in cole crop vegetables. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide as follows:

ii. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a one day or single exposure. Toxicity observed in oral toxicity studies were not attributable to a single dose (exposure). No neuro or systemic toxicity was observed in rats given a single oral administration of tebufenozide at 0, 500, 1,000 or 2,000 mg/kg. No maternal or developmental toxicity was observed following oral administration of tebufenozide at 1,000 mg/kg/day (Limit-Dose) during gestation to pregnant rats or rabbits. This risk is considered to be negligible.

2. *Chronic exposure and risk* — i. The reference dose (RfD) used for the chronic dietary analysis is 0.018 mg/kg/day. In conducting this exposure assessment, Rohm and Haas has made very conservative assumptions 100% of pecans, walnuts, wine and sherry, pome fruit and all other commodities having tebufenozide tolerances or pending tolerances will contain tebufenozide residues, and those residues would be at the level of the tolerance which result in an overestimate of human dietary exposure. Thus, in making a safety determination for this tolerance, Rohm and Haas is taking into account this conservative exposure assessment. The existing tebufenozide tolerances published, pending, and including the necessary section 18 tolerance(s) resulted in a Theoretical Maximum Residue Contribution (TMRC) that is equivalent to the following percentages of the RfD:

U.S. Population (35.6% of RfD);
All Infants (<1 year) (63.8%);
Nursing Infants (<1 year old) (41.0% of RfD);
Non-Nursing Infants (<1 year old) (73.3% of RfD);
Children (1-6 years old) (81.8% of RfD);
Children (7-12 years old) (50.0% of RfD);
Females (13 + years old, nursing) (40.0% of RfD);
Non-Hispanic Whites (35.8%);
Non-Hispanic Other than Black or White (40.8% of RfD);
Northeast Region (38.2% of RfD);
Western Region (37.6%);

Pacific Region (37.6%).

The subgroups listed above are subgroups for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48 States).

ii. *Drinking water — Acute exposure and risk*. Because no acute dietary endpoint was determined, Rohm and Haas concludes that there is a reasonable certainty of no harm from acute exposure from drinking water.

iii. *Chronic exposure and risk*. Submitted environmental fate studies suggest that tebufenozide is moderately persistent to persistent and mobile. Under certain conditions tebufenozide appears to have the potential to contaminate ground and surface water through runoff and leaching; subsequently potentially contaminating drinking water. There are no established Maximum Contaminant Levels (MCL) for residues of tebufenozide in drinking water and no Health Advisories (HA) have been issued for tebufenozide therefore these could not be used as comparative values for risk assessment. Therefore, potential residue levels for drinking water exposure were calculated using Generic expected environmental concentration (GENEEC (surface water)) and screening concentration in ground water (SCIGROW (ground water)) for human health risk assessment. Because of the wide range of half-life values (66–729 days) reported for the aerobic soil metabolism input parameter a range of potential exposure values were calculated. In each case the worst case upper bound exposure limits were then compared to appropriate chronic drinking water level of concern (DWLOC). In each case the calculated exposures based on model data were below the DWLOC.

2. *Non-dietary exposure*. Tebufenozide is not currently registered for use on any residential non-food sites. Therefore there is no chronic, short- or intermediate-term exposure scenario.

D. Cumulative Effects

Cumulative exposure to substances with common mechanism of toxicity. Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' The Agency believes that ''available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency hassome information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides as to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

EPA does not have, at this time, available data to determine whether tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, Rohm and Haas has not assumed that tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions described above, and taking into account the completeness and reliability of the toxicity data, Rohm and Haas has concluded that dietary (food only) exposure to tebufenozide will utilize 35.6% of the RfD for the U.S. population. Submitted environmental fate studies suggest that tebufenozide is moderately persistent to persistent and mobile; thus, tebufenozide could potentially leach to ground water and runoff to surface water under certain environmental conditions. The modeling data for tebufenozide indicate levels less than OPP's DWLOC. 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. There are no registered residential uses of tebufenozide. Since there is no potential for exposure to tebufenozide from residential uses, Rohm and Haas does not expect the aggregate exposure to exceed 100% of the RfD.

Since, tebufenozide has been classified as a Group E, ''no evidence of carcinogenicity for humans,'' this risk does not exist.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of tebufenozide, data from developmental toxicity studies in the rat and rabbit and two 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 pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. Developmental toxicity was not observed in developmental studies using rats and rabbits. The NOAEL for developmental effects in both rats and rabbits was 1,000 mg/kg/day, which is the limit dose for testing in developmental studies.

In the 2-generation reproductive toxicity study in the rat, the reproductive/ developmental toxicity NOAEL of 12.1 mg/kg/day was 14-fold higher than the parental (systemic) toxicity NOAEL (0.85 mg/kg/day). The reproductive (pup) LOEL of 171.1 mg/

kg/day was based on a slight increase in both generations in the number of pregnant females that either did not deliver or had difficulty and had to be sacrificed. In addition, the length of gestation increased and implantation sites decreased significantly in F_1 dams. These effects were not replicated at the same dose in a second 2-generation rat reproduction study. In this second study, reproductive effects were not observed at 2,000 ppm (the NOAEL equal to 149–195 mg/kg/day) and the NOAEL for systemic toxicity was determined to be 25 ppm (1.9–2.3 mg/kg/day).

Because these reproductive effects occurred in the presence of parental (systemic) toxicity and were not replicated at the same doses in a second study, these data do not indicate an increased pre-natal or post-natal sensitivity to children and infants (that infants and children might be more sensitive than adults) to tebufenozide exposure. FFDCA section 408 provides that EPA shall apply an additional safety factor for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the data base unless EPA concludes that a different margin of safety is appropriate. Based on current toxicological data discussed above, an additional uncertainty factor is not warranted and the RfD at 0.018 mg/kg/day is appropriate for assessing aggregate risk to infants and children. Rohm and Haas concludes that there is a reasonable certainty that no harm will occur to infants and children from aggregate exposure to residues of tebufenozide.

F. International Tolerances

There are no approved CODEX maximum residue levels (MRLs) established for residues of tebufenozide. At the 1996 Joint Meeting for Pesticide Residues, the FAO expert panel considered residue data for pome fruit and proposed an MRL of 1.0 mg/kg. An MRL of 1.0 mg/kg was established for apples in Canada.

2. 7F4863

EPA has received a revised pesticide petition (7F4863) from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of tebufenozide [benzoic acid, 3,5-dimethyl-,1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide] in or on the raw agricultural commodity sugarcane and molasses at 1.0 and 6.0 parts per million (ppm) respectively. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. Plant metabolism. The metabolism of tebufenozide in plants (grapes, apples, rice and sugar beets) is adequately understood for the purpose of this tolerance. The metabolism of tebufenozide in all crops was similar and involves oxidation of the alkyl substituents of the aromatic rings primarily at the benzylic positions. The extent of metabolism and degree of oxidation are a function of time from application to harvest. In all crops, parent compound comprised the majority of the total dosage. None of the metabolites were in excess of 10% of the total dosage.

2. *Analytical method*. A high performance liquid chromatographic (HPLC) analytical method using ultraviolet (UV) detection has been validated for sugarcane, molasses and refined sugar. For all matrices, the methods involve extraction by blending with solvents, purification of the extracts by liquid-liquid partitions and final purification of the residues using solid phase extraction column chromatography. The limit of quantitation of the method is 0.01 ppm.

3. *Magnitude of residues*. Magnitude of the residue and processing studies were conducted in sugarcane using the maximum proposed label rate. Samples were collected 14 days after the last application and were analyzed for residues of tebufenozide. The residue data support a tolerance of 1.0 ppm for sugarcane and 6.0 ppm for molasses. Residues were not found in refined sugar and no tolerance is needed for this commodity.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies with technical grade: Oral LD_{50} in the rat is > 5 grams for males and females - Toxicity Category IV; dermal LD_{50} in the rat is = 5,000 milligram/kilogram (mg/kg) for males and females - Toxicity Category III; inhalation LD_{50} in the rat is > 4.5 mg/l - Toxicity Category III; primary eye irritation study in the rabbit is a non-irritant; primary skin irritation in the rabbit > 5 mg - Toxicity Category IV. Tebufenozide is not a sensitizer.

2. *Genotoxicty*. Several mutagenicity tests which were all negative. These include an Ames assay with and without metabolic activation, an *in vivo* cytogenetic assay in rat bone marrow cells, and *in vitro* chromosome aberration assay in CHO cells, a CHO/HGPRT assay, a reverse mutation assay with *E. Coli*, and an unscheduled DNA synthesis assay (UDS) in rat hepatocytes.

3. *Reproductive and developmental toxicity*. In a prenatal developmental toxicity study in Sprague-Dawley rats 25/group Tebufenozide was administered on gestation days 6–15 by gavage in aqueous methyl cellulose at dose levels of 50, 250, or 1,000 mg/kg/day and a dose volume of 10 ml/kg. There was no evidence of maternal or developmental toxicity; the maternal and developmental toxicity no observed adverse effect level (NOAEL) was 1,000 mg/kg/day.

In a prenatal developmental toxicity study conducted in New Zealand white rabbits 20/group Tebufenozide was administered in 5 ml/kg of aqueous methyl cellulose at gavage doses of 50, 250, or 1,000 mg/kg/day on gestation days 7-19. No evidence of maternal or developmental toxicity was observed; the maternal and developmental toxicity NOAEL was 1,000 mg/kg/day.

In a 1993 2-generation reproduction study in Sprague-Dawley rats tebufenozide was administered at dietary concentrations of 0, 10, 150, or 1,000 ppm (0, 0.8, 11.5, or 154.8 mg/kg/day for males and 0, 0.9, 12.8, or 171.1 mg/kg/day for females). The parental systemic NOAEL was 10 ppm (0.8/0.9 mg/kg/day for males and females, respectively) and the lowest observed effect level (LOEL) was 150 ppm (11.5/12.8 mg/kg/day for males and females, respectively) based on decreased body weight, body weight gain, and food consumption in males, and increased incidence and/or severity of splenic pigmentation. In addition, there was an increased incidence and severity of extramedullary hematopoiesis at 2,000 ppm. The reproductive NOAEL was 150 ppm. (11.5/12.8 mg/kg/day for males and females, respectively) and the LOEL was 2,000 ppm (154.8/171.1 mg/kg/day for males and females, respectively) based on an increase in the number of pregnant females with increased gestation duration and dystocia. Effects in the offspring consisted of decreased number of pups per litter on postnatal days 0 and/or 4 at 2,000 ppm (154.8/171.1 mg/kg/day for males and females, respectively) with a NOAEL of 150 ppm (11.5/12.8 mg/kg/day for males and females, respectively).

In a 1995 2-generation reproduction study in rats Tebufenozide was administered at dietary concentrations of 0, 25, 200, or 2,000 ppm (0, 1.6, 12.6, or 126.0 mg/kg/day for males and 0, 1.8, 14.6, or 143.2 mg/kg/day for females). For parental systemic toxicity, the NOAEL was 25 ppm (1.6/1.8 mg/kg/day in males and females, respectively), and the LOEL was 200 ppm (12.6/14.6 mg/kg/day in males and females), based on histopathological findings (congestion and extramedullary hematopoiesis) in the spleen. Additionally, at 2,000 ppm (126.0/143.2 mg/kg/day in M/F), treatment-related findings included reduced parental body weight gain and increased incidence of hemosiderin-laden cells in the spleen. Columnar changes in the vaginal squamous epithelium and reduced uterine and ovarian weights were also observed at 2,000 ppm, but the toxicological significance was unknown. For offspring, the systemic NOAEL was 200 ppm. (12.6/14.6 mg/kg/day in males and females), and the LOEL was 2,000 ppm (126.0/143.2 mg/kg/day in M/F) based on decreased body weight on postnatal days 14 and 21.

4. *Subchronic toxicity*. In a prenatal developmental toxicity study in Sprague-Dawley rats 25/group Tebufenozide was administered on gestation days 6–15 by gavage in aqueous methyl cellulose at dose levels of 50, 250, or 1,000 mg/kg/day and a dose volume of 10 ml/kg. There was no evidence of maternal or developmental toxicity; the maternal and developmental toxicity NOAEL was 1,000 mg/kg/day.

5. *Chronic toxicity*. A 1-year dog feeding study with a LOEL of 250 ppm, 9 mg/kg/day for male and female dogs based on decreases in RBC, HCT, and HGB, increases in Heinz bodies, methemoglobin, MCV, MCH, reticulocytes, platelets, plasma total bilirubin, spleen weight, and spleen/body weight ratio, and liver/body weight ratio. Hematopoiesis and sinusoidal engorgement occurred in the spleen, and hyperplasia occurred in the marrow of the femur and sternum. The liver showed an increased pigment in the Kupffer cells. The NOAEL for systemic toxicity in both sexes is 50 ppm (1.9 mg/kg/day).

An 18-month mouse carcinogenicity study with no carcinogenicity observed at dosage levels up to and including 1,000 ppm.

A 2-year rat carcinogenicity with no carcinogenicity observed at dosage levels up to and including 2,000 ppm (97 mg/kg/day and 125 mg/kg/day for males and females, respectively).

6. *Animal metabolism*. The adsorption, distribution, excretion and metabolism of tebufenozide in rats was investigated. Tebufenozide is partially absorbed, is rapidly excreted and does not accumulate in tissues. Although tebufenozide is mainly excreted unchanged, a number of polar metabolites were identified. These metabolites are products of oxidation of the benzylic ethyl or methyl side chains of the molecule. These metabolites were detected in plant and other animal (rat, goat, hen) metabolism studies.

7. *Metabolite toxicology*. Common metabolic pathways for tebufenozide have been identified in both plants (grape, apple, rice and sugar beet) and animals (rat, goat, hen). The metabolic pathway common to both plants and animals involves oxidation of the alkyl substituents (ethyl and methyl groups) of the aromatic rings primarily at the benzylic positions. Extensive degradation and elimination of polar metabolites occurs in animals such that residue are unlikely to accumulate in humans or animals exposed to these residues through the diet.

8. *Endocrine disruption*. The toxicology profile of tebufenozide shows no evidence of physiological effects characteristic of the disruption of the hormone estrogen. Based on structure-activity information, tebufenozide is unlikely to exhibit estrogenic activity. Tebufenozide was not active in a direct *in vitro* estrogen binding assay. No indicators of estrogenic or other endocrine effects were observed in mammalian chronic studies or in mammalian and avian reproduction studies. Ecdysone has no known effects in vertebrates. Overall, the weight of evidence provides no indication that tebufenozide has endocrine activity in vertebrates.

C. Aggregate Exposure

1. *Dietary exposure* —i. *Food*. Tolerances have been established (40 CFR 180.482) for the residues of tebufenozide, in or on walnuts at 0.1 ppm, apples at 1.0 ppm, pecans at 0.01 ppm and wine grapes at 0.5 ppm. Numerous section 18 tolerances have also been established. Other tolerance petitions are pending at EPA with proposed tolerances. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide as follows:

a. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a one day or single exposure. Toxicity observed in oral toxicity studies were not attributable to a single dose (exposure). No neuro or systemic toxicity was observed in rats given a single oral administration of tebufenozide at 0, 500, 1,000 or 2,000 mg/kg. No maternal or developmental toxicity was observed following oral administration of tebufenozide at 1,000 mg/kg/day (Limit-Dose) during gestation to pregnant rats or rabbits. This risk is considered to be negligible.

b. *Chronic exposure and risk*. The RfD used for the chronic dietary analysis is 0.018 mg/kg/day. In conducting this exposure assessment, Rohm and Haas has made very conservative assumptions 100% of pecans, walnuts, wine and sherry, pome fruit and all other commodities having tebufenozide tolerances or pending tolerances will contain tebufenozide residues, and those residues would be at the level of the tolerance which result in an overestimate of human dietary exposure. Thus, in making a safety determination for this tolerance, Rohm and Haas is taking into account this conservative exposure assessment. Using the Dietary Exposure Evaluation Model (Version 5.03b, licensed by Novigen Sciences Inc.) which uses USDA food consumption data from the 1989–1992 survey and the appropriate concentration or reduction factors, the existing tebufenozide tolerances published, pending, and including the necessary section 18 tolerance(s) resulted in a Theoretical Maximum Residue Contribution (TMRC) that is equivalent to the following percentages of the RfD:

U.S. Population (35.8% of RfD);
Northeast Region (37.5% of RfD);
Western Region (39.8%);
Pacific Region (40.9%)All Infants (<1 year) (36.3%);
Nursing Infants (<1 year old) (16.8% of RfD);
Non-Nursing Infants (<1 year old) (44.5% of RfD);
Children (1-6 years old) (61.9% of RfD);
Children (7-12 years old) (45.6% of RfD);
Females (13 + years old, nursing) (30.6% of RfD);
Non-Hispanic Whites (36.0%);
Non-Hispanic Other than Black or White (43.1% of RfD).

The subgroups listed above are subgroups for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48 States).

ii. *Drinking water*. Acute exposure and risk. Because no acute dietary endpoint was determined, Rohm and

Haas concludes that there is a reasonable certainty of no harm from acute exposure from drinking water.

iii. *Chronic exposure and risk.* Submitted environmental fate studies suggest that tebufenozide is moderately persistent to persistent and mobile. Under certain conditions tebufenozide appears to have the potential to contaminate ground and surface water through runoff and leaching; subsequently potentially contaminating drinking water. There are no established Maximum Contaminant Levels (MCL) for residues of tebufenozide in drinking water and no Health Advisories (HA) have been issued for tebufenozide therefore these could not be used as comparative values for risk assessment. Therefore, potential residue levels for drinking water exposure were calculated using Generic expected environmental concentration (GENEEC (surface water)) and screening concentration in ground water (SCIGROW (ground water)) for human health risk assessment. Because of the wide range of half-life values (66–729 days) reported for the aerobic soil metabolism input parameter a range of potential exposure values were calculated. In each case the worst case upper bound exposure limits were then compared to appropriate chronic drinking water level of concern (DWLOC). In each case the calculated exposures based on model data were below the DWLOC.

2. *Non-dietary exposure.* Tebufenozide is not currently registered for use on any residential non-food sites. Therefore there is no chronic, short- or intermediate-term exposure scenario.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' The Agency believes that ''available information'' in this context might include not only toxicity, chemistry, and exposure data, but also scientific policies and methodologies for understanding common mechanisms of toxicity and conducting cumulative risk assessments. For most pesticides, although the Agency has some information in its files that may turn out to be helpful in eventually determining whether a pesticide shares a common mechanism of toxicity with any other substances, EPA does not at this time have the methodologies to resolve the complex scientific issues concerning common mechanism of toxicity in a meaningful way. EPA has begun a pilot process to study this issue further through the examination of particular classes of pesticides. The Agency hopes that the results of this pilot process will increase the Agency's scientific understanding of this question such that EPA will be able to develop and apply scientific principles for better determining which chemicals have a common mechanism of toxicity and evaluating the cumulative effects of such chemicals. The Agency anticipates, however, that even as its understanding of the science of common mechanisms increases, decisions on specific classes of chemicals will be heavily dependent on chemical specific data, much of which may not be presently available.

Although at present the Agency does not know how to apply the information in its files concerning common mechanism issues to most risk assessments, there are pesticides as to which the common mechanism issues can be resolved. These pesticides include pesticides that are toxicologically dissimilar to existing chemical substances (in which case the Agency can conclude that it is unlikely that a pesticide shares a common mechanism of activity with other substances) and pesticides that produce a common toxic metabolite (in which case common mechanism of activity will be assumed).

EPA does not have, at this time, available data to determine whether tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, Rohm and Haas has not assumed that tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide has a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions described above, and taking into account the completeness and reliability of the toxicity data, Rohm and Haas has concluded that dietary (food only) exposure to tebufenozide will utilize 35.8% of the RfD for the U.S. population. Submitted environmental fate studies suggest that tebufenozide is moderately persistent to persistent and mobile; thus, tebufenozide could potentially leach to ground water and runoff to surface water under certain environmental conditions. The modeling data for tebufenozide indicate levels less than OPP's DWLOC. 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. There are no registered residential uses of tebufenozide. Since there is no potential for exposure to tebufenozide from residential uses, Rohm and Haas does not expect the aggregate exposure to exceed 100% of the RfD.

Short- and intermediate-term risk. Short- and intermediate-term aggregate exposure takes into account chronic dietary food and water (considered to be a background exposure level) plus indoor and outdoor residential exposure. Since there are currently no registered indoor or outdoor residential non-dietary uses of tebufenozide and no short- or intermediate-term toxic endpoints, short- or intermediate-term aggregate risk does not exist.

Since, tebufenozide has been classified as a Group E, ''no evidence of carcinogenicity for humans,'' this risk does not exist.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of tebufenozide, data from developmental toxicity studies in the rat and rabbit and two 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 pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. Developmental toxicity was not observed in developmental studies using rats and rabbits. The NOAEL for developmental effects in both rats and rabbits was 1,000 mg/kg/day, which is the limit dose for testing in developmental studies.

In the 2-generation reproductive toxicity study in the rat, the reproductive/ developmental toxicity NOAEL of 12.1 mg/kg/day was 14-fold higher than the parental (systemic) toxicity NOAEL (0.85 mg/kg/day). The

reproductive (pup) LOEL of 171.1 mg/kg/day was based on a slight increase in both generations in the number of pregnant females that either did not deliver or had difficulty and had to be sacrificed. In addition, the length of gestation increased and implantation sites decreased significantly in F_1 dams. These effects were not replicated at the same dose in a second 2-generation rat reproduction study. In this second study, reproductive effects were not observed at 2,000 ppm (the NOAEL equal to 149–195 mg/kg/day) and the NOAEL for systemic toxicity was determined to be 25 ppm (1.9–2.3 mg/kg/day).

Because these reproductive effects occurred in the presence of parental (systemic) toxicity and were not replicated at the same doses in a second study, these data do not indicate an increased pre-natal or post-natal sensitivity to children and infants (that infants and children might be more sensitive than adults) to tebufenozide exposure. FFDCA section 408 provides that EPA shall apply an additional safety factor for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the data base unless EPA concludes that a different margin of safety is appropriate. Based on current toxicological data discussed above, an additional uncertainty factor is not warranted and the RfD at 0.018 mg/kg/day is appropriate for assessing aggregate risk to infants and children. Rohm and Haas concludes that there is a reasonable certainty that no harm will occur to infants and children from aggregate exposure to residues of tebufenozide.

F. International Tolerances

There are no approved CODEX maximum residue levels (MRLs) established for residues oftebufenozide.

[FR Doc. 99–3662 Filed 2–16–99; 8:45 am]
BILLING CODE 6560–50–F

FEDERAL DEPOSIT INSURANCE CORPORATION

Sunshine Act Meeting

Pursuant to the provisions of the ''Government in the Sunshine Act'' (5 U.S.C. 552b), notice is hereby given that at 10:00 a.m. on Tuesday, February 16, 1999, the Federal Deposit Insurance Corporation's Board of Directors will meet in closed session, pursuant to sections 552b(c) (2), (c)(4), (c)(6), (c)(8), (c)(9)(A)(ii), and (c)(9)(B) of Title 5, United States Code, to consider (1) matters relating to the Corporation's corporate and supervisory activities, and (2) reports from the Office of Inspector General.

The meeting will be held in the Board Room on the sixth floor of the FDIC Building located at 550—17th Street, NW., Washington, DC.

Requests for further information concerning the meeting may be directed to Mr. Robert E. Feldman, Executive Secretary of the Corporation, at (202) 898–6757.

Dated: February 11, 1999.

Federal Deposit Insurance Corporation.

Robert E. Feldman,

Executive Secretary.

[FR Doc. 99–3906 Filed 2–11–99; 5:10 pm]
BILLING CODE 6714–01–M

FEDERAL EMERGENCY MANAGEMENT AGENCY

Open Meeting, Technical Mapping Advisory Council

AGENCY: Federal Emergency Management Agency (FEMA).

ACTION: Notice of meeting.

SUMMARY: In accordance with § 10(a)(2) of the Federal Advisory Committee Act, 5 U.S.C. App. 1, the Federal Emergency Management Agency gives notice that the following meeting will be held:

NAME: Technical Mapping Advisory Council.

DATE OF MEETING: March 1–2, 1999.

PLACE: ASCE Office, 1015 Fifteenth Street, NW., Washington, DC.

TIME: 8:30 a.m. to 5:00 p.m., both days.

PROPOSED AGENDA:

1. Call to order and announcements.
2. Action on minutes of previous two meetings.
3. Plan of action for 1999: Unnumbered A-Zones, Alluvial Fans, Migrating streambeds.
4. Progress Report on the Map Modernization Plan and FY99 study projections.
5. Adjournment.

STATUS: This meeting is open to the public.

FOR FURTHER INFORMATION CONTACT: Michael K. Buckley, P.E., Federal Emergency Management Agency, 500 C Street SW., room 421, Washington, DC 20472, telephone (202) 646–2756 or by facsimile at (202) 646–4596.

SUPPLEMENTARY INFORMATION: This meeting is open to the public with limited seating available on a first-come, first-served basis. Members of the general public who plan to attend the meeting should contact Sally Magee, Federal Emergency Management Agency, 500 C Street SW., room 444, Washington, DC 20472, telephone (202) 646–8242 or by facsimile at (202) 646–4596 on or before December 2, 1998.

Minutes of the meeting will be prepared and will be available upon request 30 days after they have been approved by the next Technical Mapping Advisory Council meeting.

Dated: February 9, 1999.

Michael J. Armstrong,

Associate Director for Mitigation.

[FR Doc. 99–3880 Filed 2–16–99; 8:45 am]
BILLING CODE 6718–04–P

FEDERAL RESERVE SYSTEM

Agency Information Collection Activities: Submission for OMB Review; Comment Request

AGENCY: Board of Governors of the Federal Reserve System (Board).

ACTION: Notice of information collections submitted to OMB for review and approval under Paperwork Reduction Act of 1995.

SUMMARY: In accordance with the requirements of the Paperwork Reduction Act of 1995 (44 U.S.C. chapter 35), the Board, the Federal Deposit Insurance Corporation (FDIC), and the Office of the Comptroller of the Currency (OCC) (the ''agencies'') may not conduct or sponsor, and the respondent is not required to respond to, an information collection that has been extended, revised, or implemented on or after October 1, 1995, unless it displays a currently valid Office of Management and Budget (OMB) control number.

On November 19, 1998 the agencies requested public comments for 60 days on proposed revisions to the Report of Assets and Liabilities of U.S. Branches and Agencies of Foreign Banks (FFIEC 002) and the extension, without revision, of the Report of Assets and Liabilities of Non-U.S. Branches that are Managed or Controlled by a U.S. Branch or Agency of a Foreign Bank (FFIEC 002s). Both reports are currently approved collections of information. The Federal Financial (Institutions Examination Council (FFIEC), of which the agencies are members, has given final approval to the proposed revisions. The Board is publishing the proposed revisions and extension on behalf of the agencies.

DATES: Comments must be submitted on or before March 19, 1999.

ADDRESSES: Interested parties are invited to submit written comments to