Summary: EPA continues to have environmental concerns with the stability of the tailings impoundment, the potential for Acid mine drainage, adequacy of bonding and effectiveness of the reclamation plan.

Dated: June 29, 1999.

William D. Dickerson,

Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 99–16936 Filed 7–1–99; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[ER–FRL–6244–1]

Environmental Impact Statements; Notice of Availability

Responsible Agency: Office of Federal Activities, General Information (202) 564–7167 OR (202) 564–7153.

Weekly receipt of Environmental Impact Statements

Filed June 21, 1999 Through June 25, 1999

Pursuant to 40 CFR 1506.9.

EIS No. 990210, FINAL EIS, AFS, MT, Pinkham Timber Sales and Associated Activities, Implementation, Kootenai National Forest, Rexford Ranger District, Lincoln County, MT, Due: August 02, 1999, Contact: Terry Chute (406) 296–2536.

EIS No. 990211, DRAFT EIS, IBR, CA, Programmatic—CALFED Bay-Delta Program, Develop and Implement Long-Term Comprehensive Plan to Restore Ecological Health and Improve Water Management, San Francisco Bay—Sacramento/San Joaquin River Bay-Delta, CA, Due: September 23, 1999, Contact: Rick Breitenbach (916) 657–2666.

EIS No. 990212, DRAFT SUPPLEMENT, COE, VA, Southeastern Public Service Authority of Virginia Regional Landfill Expansion Project, Revised Wetland Mitigation Plan and New Information on Waste Projections, COE Section 404 Permit Issuance, Cities of Chesapeake, Norfolk, Portsmouth, Suffolk, and Virginia Beach, Isle of Wight and Southampton Counties, VA, Due: August 16, 1999, Contact: Pamela K. Painter (757) 441–7654.

EIS No. 990213, FINAL SUPPLEMENT, AFS, CO, Telluride Ski Area Expansion Project, Implementation, New/Additional Information, Special-Use-Permit and COE Section 404 Permit, Grand Mesa Uncompahgre and Gunnison National Forests, Norwood Ranger District, San Miguel County, CO, Due: August 02, 1999, Contact: Arthur Bauer (970) 327–4261.

EIS No. 990214, FINAL EIS, FHW, CT, I–95 at New Haven Harbor Crossing (Quinnipiac River Bridge) Improvement, from Interchange 43 southwest to Interchange 53 northeast, Funding, COE Section 10 and 404 Permits, U.S. Coast Guard Bridge Permit, New Haven, East and West Haven, CT, Due: August 02, 1999, Contact: Donald West (860) 659–6703.

EIS No. 990215, DRAFT EIS, FRC, WA, Warm Creek (No. 10865) and Clearwater Creek (No. 11485) Hydroelectric Project, Issuance of License for the Construction and Operation, Located in the Middle Fork Nooksack river (MFNR) Basin, WA, Due: August 16, 1999, Contact: Timothy Looney (202) 219–2852.

EIS No. 990216, DRAFT EIS, USN, ME, South Weymouth Naval Air Station, Disposal and Reuse, Norfolk and Plymouth Counties, ME, Due: August 16, 1999, Contact: Robert K. Ostermueller (610) 595–0759.

EIS No. 990217, DRAFT EIS, BLM, ID, Dry Valley Mine—South Extension Project, Construction of two New Open Pit Mine, Special-Use-Permit, COE Section 404 Permit, Public and Private Land Used, Caribou County, ID, Due: August 31, 1999, Contact: Jeff Cundick (208) 478–6354. The US Department of Agriculture's, Forest Service and the US Department of Interior's Bureau of Land management are Joint Lead Agencies for this Project.

EIS No. 990218, FINAL EIS, BLM, ID, Owyhee Resource Management Plan, Implementation, Lower Snake River District, Owyhee County, ID, Due: August 02, 1999, Contact: Wallace Evans (208) 373–3803.

EIS No. 990219, FINAL EIS, SFW, WI, Karner Blue Butterfly Habitat Conservation Plan State-wide, Application for an Incidental Take Permit, several counties, WI, Due: August 02, 1999, Contact: Lisa Mandel (612) 713–5343.

Dated: June 29, 1999.

William D. Dickerson,

Director, NEPA Compliance Division, Office of Federal Activities.

[FR Doc. 99–16937 Filed 7–1–99; 8:45 am]

BILLING CODE 6560–50–U

ENVIRONMENTAL PROTECTION AGENCY

[PF–878; FRL–6085–6]

Notice of Filing; Pesticide Petition

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–878, must be received on or before August 2, 1999.

ADDRESSES: By mail submit written comments to: Information and Records Integrity Branch, Public Information 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 (CBI).'' No confidential business information (CBI) 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 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: Joseph Tavano, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: Rm. 214, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 305–6411; e-mail: tavano.joseph@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 food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number [PF–878] (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@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 control number (PF-878) and appropriate petition number. Electronic comments on this proposed rule may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: June 23, 1999.

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. Rohm and Haas Company

PP 7F4824

EPA has received a pesticide petition (PP 7F4824) 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 establishing a tolerances for indirect or inadvertent residues of tebufenozide (benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl)hydrazide) and its metabolite RH-111,788 in or on the raw agricultural commodity (RAC) foliage of legume vegetables at 0.1 parts per million (ppm) and forage, fodder hay, and straw of cereal grains at 0.5 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. Tebufenozide, the metabolite, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-[4-(1-hydroxyethyl) benzoyl, and sugar conjugates of the metabolite were detected in a confined rotation crop study.

2. *Analytical method*. Validated high performance liquid chromatographic (HPLC) analytical methods using ultraviolet (UV) or mass selective (MS) detection are employed for measuring residues of tebufenozide and its metabolite in grains, forage, fodder, stover, hay, and straw. 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 (LOQ) of the method for all matrices is 0.02 ppm for tebufenozide and its metabolite.

3. *Magnitude of residues*. Field rotation crop residue trials were conducted and residues of tebufenozide and its metabolite were measured. Results of analyses showed that residues of tebufenozide and its metabolite will not exceed 0.1 ppm in forage of legumes and 0.5 ppm in forage, hay, or straw of cereal grains.

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 (Ms/Fs) - Toxicity Category IV; dermal LD_{50} in the rat is = 5,000 milligrams/kilograms (mg/kg) for Ms/Fs - Toxicity Category III; inhalation LD_{50} in the rat is > 4.5 milligrams/per liter (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 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*—i. 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 milliliters/kilograms (ml/kg). There was no evidence of maternal or developmental toxicity; the maternal and developmental toxicity NOAEL was 1,000 mg/kg/day.

ii. 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 no-observed adverse effect level (NOAEL) was 1,000 mg/kg/day.

iii. 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 Ms/Fs, respectively) and the lowest-observed adverse effect level (LOAEL) was 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively) based on decreased body weight (bwt) 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 Ms/Fs, respectively) and the LOAEL was 2,000 ppm (154.8/171.1 mg/kg/day for Ms/Fs, 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 Ms/Fs, respectively) with a NOAEL of 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively).

iv. 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 Ms/Fs, respectively), and the was 200 ppm (12.6/14.6 mg/kg/day in Ms/Fs), based on histopathological findings (congestion and extramedullary hematopoiesis) in the spleen. Additionally, at 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs), treatment-related findings included reduced parental bwt 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 Ms/Fs), and the LOAEL was 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs) based on decreased bwt on postnatal days 14 and 21.

4. *Subchronic toxicity*. In a 21–day dermal toxicity study, Crl: CD rats (6/sex/dose) received repeated dermal administration of either the technical 96.1% product RH-75,992 at 1,000 mg/kg/day limit dose (LTD) or the formulation 23.1% a.i. product RH-755,992 2F at 0, 62.5, 250, or 1,000 mg/kg/day, 6 hours/day, 5 days/week for 21 days. Under conditions of this study, RH-75,992 Technical or RH-75,992 2F demonstrated no systemic toxicity or dermal irritation at the highest dose tested (HDT) 1,000 mg/kg/ during the 21 day study. Based on these results, the NOAEL for systemic toxicity and dermal irritation in both sexes is 1,000 mg/kg/day HDT. A LOAEL for systemic toxicity and dermal irritation was not established.

5. *Chronic toxicity*—i. In a 1–year dog feeding study with a LOAEL of 250 ppm, 9 mg/kg/day for Ms/Fs dogs based on decreases in red blood cells (RBC), HCT, and HGB, increases in Heinz bodies, methemoglobin, MCV, MCH, reticulocytes, platelets, plasma total bilirubin, spleen weight, and spleen/bwt ratio, and liver/bwt 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).

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

iii. 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 Ms/Fs, respectively).

6. *Animal metabolism*. The pharmacokinetics and metabolism of tebufenozide were studied in female Sprague-Dawley rats (3-6/sex/group) receiving a single oral dose of 3 or 250 mg/kg of RH-5992 ^{14}C labeled in one of three positions (A-ring, B-ring or N-butylcarbon). The extent of absorption was not established. The majority of the radiolabeled material was eliminated or excreted in the feces within 48 hours within 48 hours; small amounts (1 to 7% of the administered dose) were excreted in the urine and only traces were excreted in expired air or remained in the tissues. There was no tendency for bioaccumulation. Absorption and excretion were rapid. A total of 11 metabolites, in addition to the parent compound, were identified in the feces; the parent compound accounted for 96 to 99% of the administered radioactivity in the high dose group and 35 to 43% in the low dose group. No parent compound was found in the urine; urinary metabolites were not characterized. The identity of several fecal metabolites was confirmed by mass spectral analysis and other fecal metabolites were tentatively identified by cochromatography with synthetic standards. A pathway of metabolism was proposed based on these data. Metabolism proceeded primarily by oxidation of the three benzyl carbons, two methyl groups on the B-ring and an ethyl group on the A-ring to alcohols, aldehydes or acids. The type of metabolite produced varies depending on the position oxidized and extent of oxidation. The butyl group on the quaternary nitrogen also can be cleaved (minor), but there was no fragmentation of the molecule between the benzyl rings.

No qualitative differences in metabolism were observed between sexes, when high or low dose groups were compared or when different labeled versions of the molecule were compared.

7. *Metabolite toxicology*. The absorption and metabolism of tebufenozide were studied in a group of M/F bile-duct cannulated rats. Over a 72–hour period, biliary excretion accounted for 30% M to 34% F of the administered dose while urinary excretion accounted for about 5% of the administered dose and the carcass accounted for < 0.5% of the administered dose for both Ms/Fs. Thus systemic absorption (percent of dose recovered in the bile, urine and carcass) was 35% M to 39% F. The majority of the radioactivity in the bile (20% M to 24% F of the administered dose) was excreted within the first 6 hours post-dosing indicating rapid absorption. Furthermore, urinary excretion of the metabolites was essentially complete within 24 hours post-dosing. A large amount 67% F to 70% M of the administered dose was unabsorbed and excreted in the feces by 72 hours. Total recovery of radioactivity was 105% of the administered dose.

A total of 13 metabolites were identified in the bile; the parent compound was not identified, i.e. unabsorbed compound, nor were the primary oxidation products seen in the feces in the pharmacokinetics study. The proposed metabolic pathway proceeded primarily by oxidation of the benzylic carbons to alcohols, aldehydes, or acids. Bile contained most of the other highly oxidized products found in the feces. The most significant individual bile metabolites accounted for 5% to 18% of the total radioactivity (F and/or M). Bile also contained the previously undetected (in the pharmacokinetics study) ''A'' ring ketone and the ''B'' ring diol. The other major components were characterized as high molecular weight conjugates. No individual bile metabolite for > 5% of the total administered dose. Total bile

radioactivity accounted for about 17% of the total administered dose.

No major qualitative differences in biliary metabolites were observed between sexes. The metabolic profile in the bile was similar to the metabolic profile in the feces and urine.

C. Aggregate Exposure

1. *Dietary exposure—From food and feed uses.* 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 0.5 ppm in or on kiwifruit to 10 ppm in leafy and cole crop vegetables. The current petition requests establishment of tolerances due to indirect or inadvertent residues of tebufenozide and its metabolite in or on foliage of legume vegetables and forage, straw, and hay of cereal grains. 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 and are presented in the following discussion.

2. *Food*—i. *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 1–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 LTD during gestation to pregnant rats or rabbits. This risk is considered to be negligible.

ii. *Chronic exposure and risk.* The RfD used for the chronic dietary analysis is 0.018 mg/kg/day. In conducting this chronic dietary (food) exposure assessment, Rohm and Haas used tolerance level residues for pecans, walnuts, wine, and sherry, imported apples and all other commodities with established or pending tebufenozide tolerances; and percent crop-treated (%CT) information on some of these crops. Further refinement using anticipated residue values and additional %CT information would result in a lower estimate of chronic dietary exposure. The Novigen DEEM system was used for this chronic dietary exposure analysis. The subgroups listed below are the U.S. population (48 contiguous States); those for infants and children; and the other subgroups (adult) for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48 contiguous States). The results are summarized below:

Groups	RfD (Percentage)
U.S. Population	10.0
All Infants (<1 year)	12.2
Nursing Infants (<1 year old)	5.7
Non-Nursing Infants (<1 year old)	15.0
Children (1-6 years old)	22.5
Children (7-12 years old)	14.1
Females (13 + years old, nursing)	10.1
U.S. Population (autumn season)	10.3
U.S. Population (winter season)	10.1
Non-Hispanic Blacks	10.4
Non-Hispanic Other than Black or White	11.0
Northeast Region	10.3
Southern Region	10.1
Western Region	10.5
Pacific Region	10.7

3. *Drinking water*—i. *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.

ii. *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 previously by EPA using GENEEC (surface water) and 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 appropriate chronic drinking water level of concern (DWLOC). In each case, the calculated exposures based on model data were below the DWLOC.

4. *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*—i *Acute risk*. Since no acute toxicological endpoints were established, no acute aggregate risk exists.

ii. *Chronic risk*. 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 10.0% 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 drinking water level of concern (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.

iii. *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.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide, EPA previously considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 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 postnatal toxicity and the completeness of the data base 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 MOE analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty 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 MOE/safety factor.

3. *Developmental toxicity studies*—i. *Rats*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 250 mg/kg/day. The LOAEL was 1,000 mg/kg/day, based on decreased bwt and food consumption. The developmental (pup) NOAEL was 1,000 mg/kg/day (HGT)

ii. *Rabbits*. In a developmental toxicity study in rabbits, the maternal and developmental NOAELs were 1,000 mg/kg/day highest dose tested (HDT).

iii. *Reproductive toxicity study*. 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 Ms and 0, 0.9, 12.8, or 171.1 mg/kg/day for Fs).The parental systemic NOAEL was 10 ppm (0.8/0.9 mg/kg/day for Ms/Fs, respectively) and the LOAEL was 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively) based on decreased bwt 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 Ms/Fs, respectively) and the LOAEL was 2,000 ppm (154.8/171.1 mg/kg/day for Ms/Fs, 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 Ms/Fs, respectively) with a NOAEL of 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, 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 Ms/Fs, respectively), and the LOAEL 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 Ms/Fs), 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 Ms/Fs), and the LOAEL was 2,000 ppm (126.0/143.2 mg/kg/day in M/F) based on decreased bwt on postnatal days 14 and 21.

iv. *Pre- and postnatal sensitivity*. The toxicology data base for tebufenozide is complete and includes acceptable developmental toxicity studies in both rats and rabbits as well as a 2-generation reproductive toxicity studies in rats. EPA determined that the data provided no indication of increased sensitivity of rats or rabbits to *in utero* and/or postnatal exposure to tebufenozide. No maternal or developmental findings were observed in the prenatal developmental toxicity studies at doses up to 1,000 mg/kg/day in rats and rabbits. In the 2-generation reproduction studies in rats, effects occurred at the same or lower treatment levels in the adults as in the offspring.

4. *Acute risk*. Since no acute toxicological endpoints were established, no acute aggregate risk exists.

5. *Chronic risk*. Using the conservative exposure assumptions described above, Rohm and Haas has concluded that aggregate exposure to tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide from food will utilize from 10.0% of the RfD for the U.S. population to 22.5% of the RfD for children 1-6 years old. The potential for exposure to tebufenozide in drinking water does not exceed EPA's level of concern. There are currently no tebufenozide residential or non-dietary exposure scenarios. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas does not expect the aggregate exposure to exceed 100% of the RfD. Rohm and Haas concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide residues.

6. *Short- or intermediate-term risk*. Since no short- and intermediate-term toxicological endpoints were established by EPA, no acute aggregate risk exists.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican maximum residue levels (MRLs) established for tebufenozide in rotation crops so no harmonization issues are required for this action.

2. Rohm and Haas Company

9F5077

EPA has received a pesticide petition (9F5077) from Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA 19106-2399 proposing, pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of tebufenozide (benzoic acid, 3,5-dimethyul-,1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl)hydrazide) in or on the RAC crop grouping, tree nuts, at 0.1 ppm and in or almond hulls at 25 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*. Validated high performance liquid chromatographic (HPLC) analytical methods using ultraviolet (UV) or mass selective (MS) detection are employed for measuring residues of tebufenozide and its metabolite in nut meat and almond hulls. 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 (LOQ) of the method for all matrices is 0.01 ppm for tebufenozide.

3. *Magnitude of residues*. Field residue trials were conducted in the representative nut crops pecans and almonds and residues of tebufenozide were measured in nut meat and almond hulls. Results of analyses showed that residues of tebufenozide will not exceed 0.1 ppm in nut meat and 25 ppm in almond hulls.

B. Toxicological Profile

1. *Acute toxicity*. Acute toxicity studies with technical grade. Oral LD_{50} in the rat is > 5 grams for Ms/Fs - Toxicity Category IV; dermal LD_{50} in the rat is = 5,000 mg/kg for Ms/Fs - 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 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*—i. 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.

ii. 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.

iii. 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 M/F, respectively) and the LOAEL was 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively) based on decreased bwt 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 M/F, respectively) and the LOAEL was 2,000 ppm (154.8/171.1 mg/kg/day for M/F, 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 Ms/Fs, respectively) with a NOAEL of 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively).

iv. 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 Ms/Fs, respectively), and the LOAEL was 200 ppm (12.6/14.6 mg/kg/day in Ms/Fs), based on histopathological findings (congestion and extramedullary hematopoiesis) in the spleen. Additionally, at 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs), treatment-related findings included reduced parental bwt 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 Ms/Fs), and the LOAEL was 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs) based on decreased bwt on postnatal days 14 and 21.

4. *Subchronic toxicity*. In a 21-day dermal toxicity study, Crl: CD rats (6/sex/dose) received repeated dermal administration of either the technical 96.1% product RH-75,992 at 1,000 mg/kg/day limit dose (LTD) or the formulation 23.1% a.i. product RH-755,992 2F at 0, 62.5, 250, or 1,000 mg/kg/day, 6 hours/day, 5 days/week for 21–days. Under conditions of this study, RH-75,992 Technical or RH-75,992 2F demonstrated no systemic toxicity or dermal irritation at the highest dose tested (HDT) 1,000 mg/kg during the 21–day study. Based on these results, the NOAEL for systemic toxicity and dermal irritation in both sexes is 1,000 mg/kg/day HDT. A LOAEL for systemic toxicity and dermal irritation was not established.

5. *Chronic toxicity*—i. In a 1 year dog feeding study with a LOAEL of 250 ppm, 9 mg/kg/day for Ms/Fs 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/bwt ratio, and liver/bwt 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).

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

iii. 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 Ms/Fs, respectively).

6. *Animal metabolism*. The pharmacokinetics and metabolism of tebufenozide were studied in female Sprague-Dawley rats (3-6/sex/group) receiving a single oral dose of 3 or 250 mg/kg of RH-5992 14C labeled in one of three positions (A-ring, B-ring or N-butylcarbon). The extent of absorption was not established. The majority of the radiolabeled material was eliminated or excreted in the feces within 48 hours; small amounts (1 to 7% of the administered dose) were excreted in the urine and only traces were excreted in expired air or remained in the tissues. There was no tendency for bioaccumulation. Absorption and excretion were rapid. A total of 11 metabolites, in addition to the parent compound, were identified in the feces; the parent compound accounted for 96 to 99% of the administered radioactivity in the high dose group and 35 to 43% in the low dose group. No parent compound was found in the urine; urinary metabolites were not characterized. The identity of several fecal metabolites was confirmed by mass spectral analysis and other fecal metabolites were tentatively identified by cochromatography with synthetic standards. A pathway of metabolism was proposed based on these data. Metabolism proceeded primarily by oxidation of the three benzyl carbons, two methyl groups on the B-ring and an ethyl group on the A-ring to alcohols, aldehydes or acids. The type of metabolite produced varies depending on the position oxidized and extent of oxidation. The butyl group on the quaternary nitrogen also can be cleaved (minor), but there was no fragmentation of the molecule between the benzyl rings.

No qualitative differences in metabolism were observed between sexes, when high or low dose groups were compared or when different labeled versions of the molecule were compared.

7. *Metabolite toxicology*. The absorption and metabolism of tebufenozide were studied in a group of Ms/Fs bile-duct cannulated rats. Over a 72-hour period, biliary excretion accounted for 30% Ms to 34% Fs of the administered dose while urinary excretion accounted for about 5% of the administered dose and the carcass accounted for < 0.5% of the administered dose for both Ms/Fs. Thus systemic absorption (percent of dose recovered in the bile, urine and carcass) was 35% Ms to 39% Fs. The majority of the radioactivity in the bile (20% Ms to 24% Fs of the administered dose) was excreted within the first 6 hours post-dosing indicating rapid absorption. Furthermore, urinary excretion of the metabolites was essentially complete within 24 hours post-dosing. A large amount (67% Fs to 70% Ms) of the administered dose was unabsorbed and excreted in the feces by 72 hours. Total recovery of radioactivity was 105% of the administered dose.

A total of 13 metabolites were identified in the bile; the parent compound was not identified, i.e. unabsorbed compound, nor were the primary oxidation products seen in the feces in the pharmacokinetics study. The proposed metabolic pathway proceeded primarily by oxidation of the benzylic carbons to alcohols, aldehydes or acids. Bile contained most of the other highly oxidized products found in the feces. The most significant individual bile metabolites accounted for 5% to 18% of the total radioactivity (Fs and/or Ms). Bile also contained the previously undetected (in the pharmacokinetics study) ''A'' ring ketone and the ''B'' ring diol. The other major components were characterized as high molecular weight conjugates. No individual bile metabolite accounted for > 5% of the total administered dose. Total bile radioactivity accounted for about 17% of the total administered dose. No major qualitative differences in biliary metabolites were observed between sexes. The metabolic profile in the bile was similar to the metabolic profile in the feces and urine.

C. Aggregate Exposure

1. *Dietary exposure—From food and feed uses.* 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 0.5 ppm in or on kiwifruit to 10 ppm in leafy and cole crop vegetables. The current petition requests establishment of tolerances in or on tree nuts and almond hulls. 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.

2. *Food*—i. *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 1 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 (LTD) during gestation to pregnant rats or rabbits. This risk is considered to be negligible.

ii. *Chronic exposure and risk.* The reference dose (RfD) used for the chronic dietary analysis is 0.018 mg/kg/day. In conducting this chronic dietary (food) exposure assessment, Rohm and Haas used tolerance level residues for nut crops, wine, and sherry, imported apples and all other commodities with established or pending tebufenozide tolerances; and percent crop-treated (%CT) information for some of these crops. Further refinement using anticipated residue values and additional %CT information would result in a lower estimate of chronic dietary exposure. The Novigen DEEM system was used for this chronic dietary exposure analysis. The subgroups listed below are (i) the U.S. population (48 contiguous States); (ii) those for infants and children; and (iii) the other subgroups (adult) for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48 contiguous States). The results are summarized below:

Groups	RfD (percentage)
U.S. Population	10.0
All Infants (< 1 year)	12.2
Nursing Infants (< 1 year old)	5.7
Non-Nursing Infants (< 1 year old)	15.0
Children (1-6 years old)	22.5
Children (7-12 years old)	14.1
Females (13 + years old, nursing)	10.1
U.S. Population (autumn season)	10.3
U.S. Population (winter season)	10.1
Non-Hispanic Blacks	10.4
Non-Hispanic Other than Black or White	11.0
Northeast Region	10.3
Southern Region	10.1
Western Region	10.5
Pacific Region	10.7

3. *Drinking water*—i. *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.

ii. *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 previously by EPA using GENEEC (surface water) and 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.

4. *Non-dietary exposure.* From 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 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*—i. *Acute risk*. Since no acute toxicological endpoints were established, no acute aggregate risk exists.

ii. *Chronic risk*. 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 10.0% 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.

iii. *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.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide, EPA previously considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity. FFDCA section 408 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 postnatal toxicity and the completeness of the data base 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. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability) and not the additional tenfold MOE/uncertainty 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 MOE safety factor.

3. *Developmental toxicity studies*—i. *Rats*. In a developmental toxicity study in rats, the maternal (systemic) NOAEL was 250 mg/kg/day. The LOAEL was 1,000 mg/kg/day, based on decreased bwt and food consumption. The developmental (pup) NOAEL was 1,000 mg/kg/day (HGT).

ii. *Rabbits*. In a developmental toxicity study in rabbits, the maternal and developmental NOAELs were 1,000 mg/kg/day (HDT).

iii. *Reproductive toxicity study*. 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 Ms/Fs, respectively) and the LOAEL was 150 ppm (11.5/12.8 mg/kg/day for Ms/Fs, respectively) based on decreased bwt, bwt 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 Ms/Fs, respectively) and the LOAEL was 2,000 ppm (154.8/171.1 mg/kg/day for Ms/Fs, 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 Ms/Fs, 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 Ms/Fs, respectively), and the LOAEL was 200 ppm (12.6/14.6 mg/kg/day in Ms/Fs), based on histopathological findings (congestion and extramedullary hematopoiesis) in the spleen. Additionally, at 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs), treatment-related findings included reduced parental bwt

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 Ms/Fs), and the LOAEL was 2,000 ppm (126.0/143.2 mg/kg/day in Ms/Fs) based on decreased bwt on postnatal days 14 and 21.

iv. *Pre- and postnatal sensitivity*. The toxicology data base for tebufenozide is complete and includes acceptable developmental toxicity studies in both rats and rabbits as well as a 2-generation reproductive toxicity studies in rats.

EPA determined that the data provided no indication of increased sensitivity of rats or rabbits to *in utero* and/or postnatal exposure to tebufenozide. No maternal or developmental findings were observed in the prenatal developmental toxicity studies at doses up to 1,000 mg/kg/day in rats and rabbits. In the 2-generation reproduction studies in rats, effects occurred at the same or lower treatment levels in the adults as in the offspring.

v. *Acute risk*. Since no acute toxicological endpoints were established, no acute aggregate risk exists.

vi. *Chronic risk*. Using the conservative exposure assumptions described above, Rohm and Haas has concluded that aggregate exposure to tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide from food will utilize from 10.0% of the reference dose RfD for the U.S. population to 22.5% of the RfD for children 1-6 years old. The potential for exposure to tebufenozide in drinking water does not exceed EPA's level of concern. There are currently no tebufenozide residential or non-dietary exposure scenarios. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas does not expect the aggregate exposure to exceed 100% of the RfD. Rohm and Haas concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to tebufenozide, benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide residues.

vii. *Short- or intermediate-term risk*. Since no short- and intermediate-term toxicological endpoints were established by EPA, no acute aggregate risk exists.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican maximum residue levels (MRLs) established for tebufenozide in nut crops so no harmonization issues are required for this action.

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FEDERAL COMMUNICATIONS COMMISSION

Notice of Public Information Collection(s) Submitted to OMB for Review and Approval

June 24, 1999

SUMMARY: The Federal Communications Commission, as part of its continuing effort to reduce paperwork burden invites the general public and other Federal agencies to take this opportunity to comment on the following information collection(s), as required by the Paperwork Reduction Act of 1995, Pub. L. 104–13. An agency may not conduct or sponsor a collection of information unless it displays a currently valid control number. No person shall be subject to any penalty for failing to comply with a collection of information subject to the Paperwork Reduction Act (PRA) that does not display a valid control number. Comments are requested concerning (a) whether the proposed collection of information is necessary for the proper performance of the functions of the Commission, including whether the information shall have practical utility; (b) the accuracy of the Commission's burden estimate; (c) ways to enhance the quality, utility, and clarity of the information collected; and (d) ways to minimize the burden of the collection of information on the respondents, including the use of automated collection techniques or other forms of information technology.

DATES: Written comments should be submitted on or before August 2, 1999. If you anticipate that you will be submitting comments, but find it difficult to do so within the period of time allowed by this notice, you should advise the contact listed below as soon as possible.

ADDRESSES: Direct all comments to Judy Boley, Federal Communications Commission, Room 1–C804, 445 12th Street, SW, Washington, DC 20554 or via the Internet to jboley@fcc.gov.

FOR FURTHER INFORMATION CONTACT: For additional information or copies of the information collection(s), contact Judy Boley at 202–418–0214 or via the Internet at jboley@fcc.gov.

SUPPLEMENTARY INFORMATION:

OMB Control Number: 3060–0686.

Title: Streamlining the International Section 214 Authorization Process and Tariff Requirements.

Form Number: N/A.

Type of Review: Revision of a currently approved collection.

Respondents: Business or other for-profit.

Number of Respondents: 1,650.

Estimated Time Per Response: 2 to 20 hours.

Frequency of Response: On occasion reporting requirement, third party disclosure requirement, quarterly, semi-annual and annual reporting requirements.

Total Annual Burden: 73,975 hours.

Total Annual Cost: $12,465,000.

Needs and Uses: The Commission amended Part 63 of its rules in the Report and Order and Order referenced in IB Docket 95–118. When the Commission sought OMB approval of the information collections contained in the *Streamlining Order*, it inadvertently omitted the information collections associated with Sections 63.19 and 63.53(c). Before revising the rules, the information requested under Section 63.19 was authorized pursuant to Section 63.15(c) and 63.17 (approved by OMB under OMB Control Number 3060–0149). The information will be used by the Commission staff in carrying out its duties under the Communications Act. In the *Streamlining Order*, the Commission clarified its notification requirements for carriers that discontinue, reduce or impair service. The Commission will require non-dominant international carriers that seek to discontinue, reduce, or impair service to a community to: (1) Notify their customers in writing sixty days in advance; (2) send a copy of this notification at least sixty days in advance of their action to the Commission. The information collection is necessary for the Commission to maintain effective oversight of U.S. carrier operations. The information will serve the public interest by providing customers with sufficient time to find another international carrier if service is discontinued by their current carrier. In addition, the *Streamlining Order* requires that applicants submitting information or documents in Section 214 proceedings by accompanied by a certified translation in English. English translations of relevant documents that