(LD_{50}): 1530 milligrams/kilograms (mg/kg); Dermal LD_{50}: 2740 mg/kg.

2. *Genotoxicty*. Nothing in the available literature indicates that phosphoric acid or phosphate ion are considered to be genotoxic or mutagenic.

3. *Reproductive and developmental toxicity*. Nothing in the available literature indicates that phosphoric acid or phosphate ion are developmental or reproductive toxins. They are generally recognized as safe and are normal constituents in the human diet.

4. *Subchronic toxicity*. Nothing in the available literature indicates long-term exposure of phosphoric acid/phosphate ion produces any adverse toxicological effects unless it is ingested at a concentration where it produces corrosive or other effects on the gastric mucosa. There are no studies that indicate that prolonged exposure to low concentrations of phosphoric acid/phosphate ion produce cumulative toxicity since they are normal constituents of cells.

5. *Chronic toxicity*. Chronic exposure would not produce any additional effect over what is noted in subchronic exposure, therefore, no additional concerns are warranted. Nothing in the literature indicates that phosphoric acid may be carcinogenic.

6. *Animal metabolism*. Phosphoric acid is a normal constituent of cells. It is used for many purposes including buffering of the blood, high energy bonds, DNA synthesis, etc. A discussion of the metabolism is not relevant.

7. *Metabolite toxicology*. Phosphoric acid and phosphate are not metabolized by the body, but rather serve as major components in cellular structure and processes. A discussion of metabolite toxicity is not relevant.

8. *Endocrine disruption*. A review of information from the Agency for Toxic Substances and Disease Registry indicates that potential endocrine effects from exposure to phosphoric acid or phosphate ion have not been studied. To the best of our knowledge, nothing in the available literature suggests that phosphoric acid acts as an endocrine disrupter or that it possesses intrinsic hormonal activity.

C. Aggregate Exposure

1. *Dietary exposure*. Acute: There are no acute toxicological concerns for phosphoric acid, therefore, an acute dietary risk assessment is not required. Chronic Indirect: Using a worst-case scenario, the exposure would be 0.0065 mg/kg/day for a 70 kg person (adult) and 0.008 mg/kg/day for a 28 kg person (child).

i. *Food*. Chronic Direct: A typical adult ingests approximately one to two grams of phosphoric acid/phosphate per day as phosphorus via the diet. Following ingestion, it is absorbed by the gastrointestinal tract. In the plasma and in intra and extracellular fluid, the pH is such that the phosphoric acid exists in its ionized form, phosphate. The approximate concentration of phosphate in the plasma is 4 mg/100 milliliters (mls). Phosphate serves many biological purposes including buffering the blood, serving as a constituent of cell membranes, providing high energy phosphate bonds for cellular energy demands, maintaining DNA structure and many other functions. Phosphate is also a major constituent of the skeletal system. It is excreted in the urine and needs to be replenished on an ongoing basis. The normal human diet contains significant quantities of phosphate. Phosphate is also derived from phosphoric acid as a consequence of its direct addition to food, as approved under 21 CFR 582.1073. When used as a food contact surface sanitizer, the residue that would be introduced into food will be insignificant compared to the normal dietary intake of phosphoric acid/phosphate ion. Based on this, there are no toxicological concerns resulting from exposures to residues of phosphoric acid resulting from the use of sanitizing solutions.

ii. *Drinking water*. Acute: Since there are no acute toxicological concerns for phosphoric acid, an acute drinking water risk assessment should not be required. Chronic: There are no toxicological concerns about the exposure of low concentrations of phosphate ion in the drinking water. Although it is possible that trace amounts of phosphates used as a sanitizer may ultimately get into drinking water, no adverse health effects would result. The amount of ''naturally occurring phosphate'' in water will greatly exceed the amount derived from sanitizing solutions.

2. *Non-dietary exposure*. The exposure phosphoric acid/phosphates in non-occupational settings is minimal. Phosphates may be present in some products including general purpose cleaners, soaps, etc. however, dermal absorption would be insignificant. Since phosphate is a relatively significant constituent of the diet, non-occupational exposure will be small by comparison.

D. Cumulative Effects

Over 99% of the exposure to phosphoric acid/phosphates is expected to be via the diet. Small amounts of phosphoric acid/phosphate exposure will be the result of non-food uses. The amount of phosphoric acid/phosphate exposure resulting from indirect exposure to sanitizing solutions will be virtually zero. Since phosphoric acid/phosphate in the diet poses no toxicological risk, the cumulative toxicity resulting from this additional exposure is negligible.

E. Safety Determination

1. *U.S. population*. Since there are not adverse toxicological effects resulting from normal dietary concentrations of phosphoric acid/phosphate ion, there is no need to determine aggregate risks, or to conduct a safety determination. Phosphoric acid is generally recognized as safe and the incremental exposure due to its us as an inert in a food contact surface sanitizer is negligible.

2. *Infants and children*. As in adults, infants and children use phosphoric acid as a basic constituent of cellular metabolism, energy production and cell structure. Children are at no greater ''risk'' from exposure to phosphoric acid. Therefore, as with adults, a safety determination is not appropriate.

F. International Tolerances

No Codex maximum residue levels have been established for phosphoric acid.

[FR Doc. 99–22747 Filed 8–31–99; 8:45 am]
BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–885; FRL–6096–8]

Notice of Filing Pesticide Petitions to Establish a Tolerance for Certain Pesticide Chemicals in or on Food

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 docket control number [PF–885], must be received on or before October 1, 1999.

ADDRESSES: Comments may be submitted by mail, electronically, or in person. Please follow the detailed instructions for each method as provided in Unit I.C. of the **SUPPLEMENTARY INFORMATION** section. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–885 in the subject line on the first page of your response.

FOR FURTHER INFORMATION CONTACT: By mail: Shaja Brothers, Registration Support Branch, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460; telephone number: (703) 308–3194; and e-mail address: brothers.shaja@epa.gov.

For technical questions, contact the appropriate Product Manager: Joseph Tavano, telephone number: (703) 305–6411 and e-mail address: tavano.joseph@epa.gov.; or Cynthia Giles-Parker (PM 22), telephone number: (703) 305–7740 and e-mail address: giles-parker.cynthia@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be affected by this action if you are an agricultural producer, food manufacturer or pesticide manufacturer. Potentially affected categories and entities may include, but are not limited to:

Categories	NAICS	Examples of potentially affected entities
Industry	111 112 311 32532	Crop production Animal production Food manufacturing Pesticide manufacturing

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in the table could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether or not this action might apply to certain entities. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the ''FOR FURTHER INFORMATION CONTACT'' section.

B. How Can I Get Additional Information, Including Copies of this Document and Other Related Documents?

1. *Electronically.* You may obtain electronic copies of this document, and certain other related documents that might be available electronically, from the EPA Internet Home Page at http://www.epa.gov/. To access this document, on the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register**--Environmental Documents.'' You can also go directly to the **Federal Register** listings at http://www.epa.gov/fedrgstr/.

2. In person. The Agency has established an official record for this action under docket control number PF–885. The official record consists of the documents specifically referenced in this action, any public comments received during an applicable comment period, and other information related to this action, including any information claimed as confidential business information (CBI). This official record includes the documents that are physically located in the docket, as well as the documents that are referenced in those documents. The public version of the official record does not include any information claimed as CBI. The public version of the official record, which includes printed, paper versions of any electronic comments submitted during an applicable comment period, is available for inspection in the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

C. How and to Whom Do I Submit Comments?

You may submit comments through the mail, in person, or electronically. To ensure proper receipt by EPA, it is imperative that you identify docket control number PF–885 in the subject line on the first page of your response.

1. *By mail.* Submit your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460.

2. *In person or by courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Information Resources and Services Division (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. The PIRIB is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The PIRIB telephone number is (703) 305–5805.

3. *Electronically.* You may submit your comments electronically by E-mail to: ''*opp-docket@epa.gov* ,'' or you can submit a computer disk as described above. Do not submit any information electronically that you consider to be CBI. Avoid the use of special characters and any form of encryption. Electronic submissions will be accepted in Wordperfect 5.1/6.1 or ASCII file format. All comments in electronic form must be identified by docket control number PF–885. Electronic comments may also be filed online at many Federal Depository Libraries.

D. How Should I Handle CBI That I Want to Submit to the Agency?

Do not submit any information electronically that you consider to be CBI. You may claim information that you submit to EPA in response to this document as CBI by marking any part or all of that information as CBI. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public version of the official record. Information not marked confidential will be included in the public version of the official record without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person identified in the ''FOR FURTHER INFORMATION CONTACT'' section.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket control number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities

under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

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

Dated: August 19, 1999.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required bysection 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. IR-4 Project

PP 6E4603, 6E4787, and 7E4878

EPA has received pesticide petitions [PP 6E4603, 6E4787, and 7E4878] from the Interregional Research Project Number 4 (IR-4), New Jersey Agricultural Experiment Station, P. O. Box 231 Rutgers University, New Brunswick, NJ 08903 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 tolerances for combined residues of the herbicide, pendimethalin [N-(1-ethylpropyl)-3,4-dimethyl-2,6-dinitrobenzenamine, and its 3, 5-dinitrobenzyl alcohol metabolite (CL 202347) in or on the food commodities as follows:

1 *PP 6E4603*. Proposes the establishment of a tolerance for carrots at 0.5 parts per million (ppm).

2 *PP 6E4787*. Proposes the establishment of a tolerance for citrus fruit crop group at 0.1 ppm.

3. *PP 7E4878*. Proposes the establishment of tolerances, with regional registration for peppermint and spearmint tops at 0.2 ppm, and peppermint and spearmint oil at 1.0 ppm. Registration will be limited to Idaho, Oregon, and Washington based on the geographical representation of the residue data submitted to EPA.

EPA has determined that the petitions contain data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petitions. Additional data may be needed before EPA rules on the petitions.

A. Residue Chemistry

1. *Plant metabolism*. The qualitative nature of the residues of pendimethalin in plants is understood based on adequate studies conducted with [^{14} C]-pendimethalin on various crops. Pendimethalin and its 3,5-dinitrobenzyl alcohol metabolite (CL202347) are the only residues of concern.

2. *Analytical method*. Section 408 (b)(3) of the amended FFDCA requires EPA to determine that there is a practical method for detecting and measuring levels of the pesticide chemical residue in or on food and that the tolerance be set at a level at or above limit of detection of the designated method. The Gas Chromatography (GC) of pendimethalin and (CL202347) analytical methods, M691 and M692, are proposed as the enforcement methods for the residues in carrots; M1999 is the proposed method for citrus fruit crop group, and processed citrus commodities; and M1930.01 has been proposed for mint and mint oil. All methods utilize electron capture detectors and have a limit of quantitation (LOQ) of 0.05 ppm for the respective residues of concern.

3. *Magnitude of residues*—i. Residue field trials were conducted in seven major carrot producing states in the United States at both the 1x rate of 2 pounds (lbs) active ingredient/acre (ai/A) and an exaggerated rate of 4 lbs ai/A (2x the typical application rate). Maximum pendimethalin residues recovered from carrot samples treated with these applications were 0.10 ppm from the 1x treatment and 0.16 ppm from the 2x treatment. For the alcohol metabolite, CL202347, the maximum recovered residues ranged from 0.29 ppm from the 1x treatment to 0.44 ppm from the 2x treatment. The registrant believes that the results from these studies support the proposed tolerance of 0.5 ppm pendimethalin in or on carrots.

ii. Residue field trials were conducted on oranges, grapefruits, and lemons in major citrus fruit crop group producing states in the United States at a 1.5x rate of 6 lbs ai/A and an exaggerated 3x rate of 12 lbs ai/A. The plots were treated with pendimethalin at a variety of different intervals prior to harvest. The raw agricultural commodity (RAC) samples were also processed into wet and dried pulp, molasses, oil and juice. RAC samples taken from plots treated one day prior to harvest, a worst case residue situation, resulted in residues of 0.008 ppm (in grapefruit) or less. No residues were recovered from wet pulp and juice samples at the 0.005 ppm level. Residues of pendimethalin were recovered at 0.005 ppm in dried pulp, 0.009 ppm in molasses and 0.026 ppm in orange oil. It should be noted that data for wet pulp and molasses are no longer required as per Table I of the Residue Chemistry Test Guidelines EPA OPPTS 860.1000. The registrant believes that the results from these studies are adequate to support the proposed tolerance of 0.1 ppm pendimethalin in or on citrus fruit crop group, and in processed citrus commodities.

iii. Residue field trials were conducted in two major mint producing states in the United States at both the 1x rate of 2 lbs ai/A and an exaggerated rate of 10 lbs ai/A (5x the typical application rate). Fresh mint foliage samples were either harvested and directly analyzed or processed into mint oil before analyses. The registrant believes that the results from these studies support the proposed tolerances of 0.2 ppm pendimethalin in mint foliage (leaves and stems) and 1.0 ppm pendimethalin in mint oil.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral lethal dose (LD_{50}) values for pendimethalin technical in the Wistar rat are 1,250 milligrams/kilograms/body weight (mg/kg/bwt) (males) and 1,050 mg/kg/bwt (females). The acute dermal LD_{50} was greater than 5,000 mg/kg in New Zealand white rabbits. The 4–hour rat inhalation lethal concentration (LC_{50}) was > 320 milligram per liter (mg/L) (nominal concentration). Pendimethalin was shown to be slightly irritating to rabbit eyes and non-irritating to rabbit skin. Pendimethalin did not cause skin sensitization in guinea pigs.

2. *Genotoxicity*. Extensive mutagenicity studies conducted to investigate point and gene mutations, DNA damage and chromosomal aberration, using *in vitro* and *in vivo* test systems show pendimethalin to be non-genotoxic.

3. *Reproductive and developmental toxicity*. Results from a 2-generation rat reproduction study showed the no-observed adverse effect level (NOAEL) for parental and reproductive toxicity to be 2,500 ppm (172 mg/kg bwt/day) and the lowest-observed adverse effect level (LOAEL) to be 5,000 ppm (346 mg/kg/bwt/day). No developmental toxicity was observed in either the rat or rabbit developmental toxicity studies, nor was there any evidence in the 2-generation rat reproduction study that there was developmental or reproductive toxicity at dose levels below those in which parental toxicity was observed. For rabbits, the developmental toxicity NOAEL was > 60 mg/kg/day, the highest dose tested (HDT). The maternal NOAEL was > 60 mg/kg/day, based on mortality observed at 125 mg/kg/day in a pilot study. For rats, there were no maternal or developmental effects at any dose level and the NOAELs for both maternal and developmental effects were ≧ 500 mg/kg/day, the HDT.

4. *Subchronic toxicity*. A 90–day feeding study was conducted in rats and dogs. The NOAELs for these studies were 500 ppm (50 mg/kg/bwt/day) and 2,500 ppm (62.5 mg/kg/bwt/day) for the rat and dog studies, respectively.

5. *Chronic toxicity*. The chronic toxicity of pendimethalin has been extensively investigated in three species (i. e., the rat, mouse, and dog). The results are as follows:

i. *Rats*. In an initial 2–year feeding study in Sprague-Dawley rats, conducted at dose levels of 0, 100, 500, and 5,000 ppm (corresponding to dietary intakes of 0, 5, 25, and 250 mg/kg/bwt/day, respectively), a clear NOAEL was established at 500 ppm (25 mg/kg/bwt/day). The LOAEL was set at 5,000 ppm (250 mg/kg/bwt/day) based on decreased survival, body weight gain and food consumption, increased gamma glutamyl transferase and cholesterol, an increase in absolute and/or relative liver weight, generalized icterus, dark adipose tissue in females, diffusely dark thyroids and follicular cell hyperplasia of the thyroid. In a second 2–year feeding study in rats, conducted at dose levels of 0, 1,250, 2,500, 3,750, and 5,000 ppm (corresponding to dietary intakes of 0, 51, 103, 154, and 213 mg/kg/bwt/day, respectively), a NOAEL was not determined. The LOAEL of less than or equal to 1,250 ppm (≧ 51 mg/kg/bwt/day) was based on non-neoplastic thyroid follicular cell changes and increased liver weight.

ii. *Mouse*. Pendimethalin technical was administered at dietary concentrations of 100, 500, and 5,000 ppm (corresponding to dose levels of 12.3, 62.3 and 622.1 mg/kg/bwt/day in males and 15.6, 78.3, and 806.9 mg/kg/bwt/day in females) to CD-1 mice for 18-months. In this study, the NOAEL was 500 ppm (62.3 mg/kg/bwt/day) and the LOAEL, based on mortality, body weight decrease, organ weight changes and amyloidosis, was 5,000 ppm (622.1 mg/kg/ bwt/day).

iii. *Dog*. In a 2–year oral (capsule) study, conducted at dose levels of 0, 12.5, 50 and 200 mg/kg/bwt/day, the NOAEL was equal to or greater than the maximum dose tested ≧ 200 mg/kg/bwt/day with no LOAEL established.

Pendimethalin has been classified as a Group C, ''possible human carcinogen,'' chemical by EPA based on a statistically significant increased trend and pairwise comparison between the high dose group and controls for thyroid follicular cell adenomas in male and female rats. EPA recommended using the chronic population adjusted dose (cPAD) approach for quantification of human risk. Therefore, the cPAD is deemed protective of all chronic human health effects, including cancer.

6. *Animal metabolism*. Adequate goat and poultry metabolism studies are available for pendimethalin. As no poultry feed items are associated with carrots, citrus fruit crop group processed citrus commodities, or mint, poultry metabolism studies are not relevant to this petition. In addition, the registrant has determined that there is no reasonable expectation of finite pendimethalin residues of concern in animal commodities as a result of use on multiple crops and no tolerances for pendimethalin residues of concern in livestock commodities are needed.

7. *Endocrine disruption*. Collective results from several mechanistic studies provide support that pendimethalin disrupts thyroid-pituitary hormonal balance. An analysis of the data obtained from these studies supports fluctuations in thyroid hormones (T3 and/or T4) at dietary concentrations of 500 ppm (31 mg/kg/bwt/day) and greater. However, no fluctuations in thyroid hormones were observed at 100 ppm (10 mg/kg/bwt/day) in either of the 14–day special feeding studies, supporting a NOAEL for thyroid effects of 100 ppm or 10 mg/kg/bwt/day. As the cPAD is based on the NOAEL of 10 mg/kg/bwt/day obtained from these studies, thyroid hormonal changes are already accounted for in the characterization of the potential risks to humans. Moreover, because of species differences in thyroid gland physiology, slight fluctuations in thyroid hormone levels noted in rats may not be applicable to humans. In addition, collective organ weights and histopathological findings from the 2-generation rat reproduction study, as well as from the subchronic and chronic toxicity studies in 3 different animal species demonstrate no apparent estrogenic effects or treatment-related effects on any other component of the endocrine system.

C. Aggregate Exposure

Pendimethalin is widely used as a pre-emergent herbicide to control broad-leaf weeds in both food and non-food crops, as well as non-agricultural use sites including residential lawns. In examining aggregate exposure, FQPA directs EPA to consider available information concerning exposures from the pesticide residue in food and water (dietary) and all other non-occupational exposures. The primary non-food sources of exposure the Agency evaluates include drinking water (whether from groundwater or surface water), and exposure through pesticide use in gardens, lawns, or buildings (residential and other indoor uses). The potential for aggregate exposure from all registered and proposed uses is discussed below:

1. *Dietary (food) exposure*. Tolerances have been established (40 CFR 180.361) for the combined residues of pendimethalin and its 3,5-dinitrobenzyl alcohol metabolite (CL 202347) in or on a variety of food commodities at levels ranging from 0.05 ppm in rice grain to 0.1 ppm in corn, peanuts, soybeans and other commodities. Based on conservative assumptions of tolerance level residues and 100% crop treatment with pendimethalin, the EPA's Dietary Exposure Evaluation Model (DEEM) estimates chronic dietary exposure to pendimethalin from all currently registered uses to be only 0.00042 mg/kg/day (< 1% cPAD) for the overall U. S. population. The estimated most highly exposed DEEM subgroup for pendimethalin is non-nursing infants at a level of 0.00140 mg/kg/day (< 2%).

Additional maximum dietary contributions, (of up to 0.000498 mg/kg/bwt/day and 0.001294 mg/kg/bwt/day for the general U.S. population and for non-nursing infants less than 1–year old, respectively) anticipated from use on carrots and citrus fruit crop group will still utilize < 1% (actual 0.5%) and < 2% (actual 1.3%) of the cPAD for the respective population subgroups. The additional dietary burden that will result from the pendimethalin tolerances in mint and mint oil will also be insignificant. Thus, the American Cyanamid Company believes that there should be no reason for concern from the additional dietary burden that will result from the proposed tolerances of pendimethalin in carrots, citrus fruit

crop group, and mint because the contribution to the cPAD will be insignificant.

i. *Drinking water.* Pendimethalin has low water solubility and a strong absorption to soil, which makes it essentially immobile in all soil types. Therefore, American Cyanamid Company concludes that there is no concern for the potential for pendimethalin to runoff to surface water or leach to ground water. No Maximum Concentration Level and no Health Advisory Level has been established for residues of pendimethalin in drinking water. A pendimethalin drinking water exposure analysis for a 10 kg child shows that a chronic exposure from a worst case dietary intake (drinking water only) of 0.0018 mg/kg/day would utilize < 2% of the cPAD. Thus, the American Cyanamid Company believes that contributions to the dietary burden from residues of pendimethalin in water, alone, would be inconsequential.

2. *Non-dietary exposure.* Pendimethalin is currently registered for use on the following residential and non-food sites: ornamental lawns, grasses, ground covers, turf, and ornamental plantings, which are short- and intermediate-term non-occupational exposure scenarios. Thus, the American Cyanamid Company believes that the estimates margins of exposure (MOEs) for residential applicators (MOE = 833) and residential post-application exposures to children (MOE = 111) are more than adequate.

D. Cumulative Effects

The Agency has not yet published guidelines to determine whether pendimethalin 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, pendimethalin does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, the American Cyanamid Company assumes that pendimethalin does not have a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population.* Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data, the American Cyanamid Company concludes that the total aggregate exposure to pendimethalin from food will utilizes less than 1% of the cPAD for the overall U.S. population. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to pendimethalin in drinking water and from non-dietary non-occupational exposures, the American Cyanamid Company does not expect the aggregate exposure to exceed 100% of the cPAD. The registrant concludes that the aggregate risks estimated from the following three scenarios: (i) < 4% of the cPAD for chronic dietary exposures (food plus water), (ii) MOE = 680 for chronic dietary exposures (food plus water) plus residential applicator exposures, and (iii) MOE = 107 for chronic dietary exposures (food plus water) plus residential post-application exposures to children, do not exceed the Agency's levels of concern. Thus, the American Cyanamid Company concludes that there is a reasonable certainty that no harm will result from aggregate exposure to pendimethalin residues as a result of the establishment of the proposed tolerance in carrots, citrus fruit crop group, and processed citrus commodities, mint and mint oil.

2. *Infants and children.* The major identifiable subgroup with the highest aggregate exposure is non-nursing infants less than 1–year old. In assessing the potential for additional sensitivity of infants and children to residues of pendimethalin, the data from developmental toxicity studies in the rat and rabbit, and a 2-generation reproduction study in the rat has been considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during prenatal development. Reproduction studies provide information relating to effects on the reproductive capabilities of parental animals from exposure to the pesticide as well as additional data on systemic toxicity.

The prenatal and postnatal toxicology data base for pendimethalin is complete with respect to current toxicological data requirements. The data base does not indicate a potential for increased sensitivity from prenatal or postnatal exposure. As mentioned in item B.3. above, no developmental toxicity was observed in either the rat or rabbit developmental toxicity studies, nor was there any evidence in the 2-generation rat reproduction study that there was developmental or reproductive toxicity at dose levels below those in which parental toxicity was observed. For rabbits, the developmental toxicity NOAEL was > 60 mg/kg/day, the HDT. The maternal NOAEL was > 60 mg/kg/day, based mortality observed at 125 mg/kg/day in a pilot study. For rats, there were no maternal or developmental effects at any dose level and the NOAELs for both maternal and developmental effects were ≧ 500 mg/kg/day, the HDT. In the 2-generation reproductive toxicity study in rats, the parental and reproductive NOAELs were 172 mg/kg/day. The reproductive LOAEL of 346 mg/kg/day was based on decreased pup weight, which occurred in the presence of parental (systemic) toxicity at 346 mg/kg/day.

FFDCA section 408 provides that EPA may apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base. Based on current toxicological data requirements, the toxicology data base for pendimethalin is complete. Furthermore, the reproductive NOAEL of 172 mg/kg/day is seventeen-fold higher than the NOAEL of 10 mg/kg/day used for the cPAD. Additionally, the reproductive LOAEL occurred in the presence of parental (systemic) toxicity, and there was no evidence of developmental toxicity in either the rat or the rabbit studies. Therefore, the American Cyanamid Company believes that these proposed tolerances do not represent any unacceptable prenatal or postnatal risk to infants and children.

Using the conservative exposure assumptions described above, and based on previous EPA reports, the American Cyanamid Company has concluded that aggregate exposure to pendimethalin from food will utilize less than 2% of the cPAD for infants and children. EPA generally has no concern for exposures below 100% of the cPAD because the cPAD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Despite the potential for exposure to pendimethalin in drinking water and from non-dietary, non-occupational exposure, the American Cyanamid Company does not expect the aggregate exposure to exceed 100% of the cPAD. Thus, the registrant concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to pendimethalin residues.

F. International Tolerances

There are no Codex, Canadian or Mexican International Maximum Residue Levels established for residues of pendimethalin in carrots, citrus fruit

crop group and processed citrus commodities, or mint at this time.

2. Rohm and Haas Company

PP 7F4824

EPA has received a pesticide petition (PP 7F4824) from Rohm and Haas Company, 100 Independence Mall West, Phila., 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 indirect or inadvertent residues of tebufenozide [benzoic acid, 3,5-dimethyl-, 1-(1,1-dimethylethyl)-2-(4-ethylbenzoyl) hydrazide] and its metabolite [Benzoic acid, 3,5-dimethyl-1-(1,1-dimethylethyl)-2-[4-(1-hydroxyethyl) benzoyl] hydrazide] in or on the RAC grass forage, fodder and hay at 0.5 parts per million (ppm) and forage, fodder, straw and hay of nongrass animal feeds 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 - Toxicity Category IV; dermal LD_{50} in the rat is = 5,000 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 (UDS) assay 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 males and females, respectively) and the lowest observed adverse effect level (LOAEL) 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 LOAEL 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 NOEL 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 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 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 LOAEL 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 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 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 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. A 1–year dog feeding study with a (LOAEL) of 250 ppm, 9 mg/kg/day for male and female 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/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).

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 males and females, 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 male and female bile-duct cannulated rats. Over a 72 hour period, biliary excretion accounted for 30% male to 34% female 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 males and females. Thus systemic absorption (percent of dose recovered in the bile, urine and carcass) was 35% male to 39% female. The majority of the radioactivity in the bile (20% male to 24% female 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% (female) to 70% (male) 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 oxidizedproducts found in the feces. The most significant individual bile metabolites accounted for 5% to 18% of the total radioactivity (female and/or male). 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, pome fruit at 1.5 ppm, pecans at 0.01, kiwifruit at 0.5ppm, leafy and cole crop vegetables at 10 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. The current petition requests establishment of tolerances due to indirect or inadvertent residues of tebufenozide and its metabolite in or on grass forage, fodder and hay and forage, fodder, straw and hay of nongrass animal feeds 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 followingdiscussion:

i. *Food—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.

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 (a) tolerance level residues for pecans, walnuts, wine and sherry, imported apples and all other commodities with established or pending tebufenozide tolerances; and (b) 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 (c) the U.S. Population (48

States); (d) those for infants and children; and (e) the other subgroups (adult) for which the percentage of the reference dose (RfD) occupied is greater than that occupied by the subgroup U.S. population (48 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%

iii. *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.

iv. *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.

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*— 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 groundwater and runoff to surface water under certain environmental conditions. The modeling data for tebufenozide indicate levels less than OPP's drinking water levels 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*— i. *In general*. 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 prenatal 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.

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.

The 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.

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.

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.

3. Rohm and Haas Company

PP 9F5058

EPA has received a pesticide petition (PP 9F5058) 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 combined residues of RH-117281 Technical Benzamide-3,5-dichloro-N-(3-chloro-1-ethyl-1-methyl-2oxopropyl)-4-methyl and metabolites 3,5-dichloro-4-hydroxy methyl-benzoic acid and 3,5-dichloro-1,4-benzene dicarboxylic (RH-141452 and RH-141455) in or on the raw agricultural commodity (RAC) potatoes at 0.1 parts per million (ppm), grapes at 5 ppm, and raisins at 15 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 RH-117281 Technical in plants (grapes and potatoes) is adequately understood for the purposes of these tolerances. There were no significant metabolites other than the parent compound in grapes. Residues in grapes were surface residues of parent RH-117281 and minor amounts of hydrolysis and photolysis degradates. In potatoes, two minor rat metabolites, RH-141452 and RH-141455, comprised the majority of the residue. No other metabolites were present in excess of 10% of the total dosage. It is most likely that the source of these residues is extremely low level uptake of highly degraded metabolites from the soil, rather than metabolism within the plant, since these compounds are highly metabolized, but there are no intermediate products found in the potato.

2. *Animal metabolism*. The metabolism of RH-117281 Technical in food-producing animals (dairy goats) is adequately understood. Hen metabolism is not required for the current submission because no components of grape or potato are fed to poultry. Metabolism in laboratory and food-producing animals was similar and extensive, occurring through multiple pathways involving primary hydrolysis, glutathione-mediated reactions, and reductive dehalogenation; secondary oxidation; and terminal glucuronic and amino acid conjugation. RH-117281 Technical and its residues are rapidly excreted in animals. No significant residues in these food commodities.

3. *Analytical method*. Tolerance enforcement methods using gas chromatography/electron capture detection (GC/ECD) or gas chromatography/mass selective detection (GC/MSD), have been developed for RH-117281 in grapes, grape juice and raisins. The limit of quantification (LOQ) is 0.01 ppm for all matrices. Average recoveries are 95.8-106% for grapes, 84.2-101% for juice, and 85.9-108% for raisins, over the range of fortifications.

A tolerance enforcement method using GCECD or GC/MSD detection has also been developed for RH-117281 in potatoes and for the metabolites RH-141452 and RH-141455 in potatoes, potato chips and potato flakes. The LOQ for all analytes is 0.02 ppm for all matrices.

The methods involve extraction with solvent, filtration, liquid-liquid partition, and final purification of the residues using solid phase column chromatography. An independent validation of the methods has been completed.

4. *Magnitude of residues*—i. *Grape*. Twelve field residue trials were conducted over two seasons in four States at either 1.25 lb active ingredient (a.i)/acre and 2.50 lb a.i./acre (1.40 kiligram/hectare Kg/ha and 2.81 Kg/ha) or 2.0 lbs a.i/acre and 4.0 lbs a.i acre (2.25 Kg/ha and 4.49 Kg/ha). Ten applications were made in each trial. In two of the trials, fruit was harvested at 0, 7, 14, and 21 days after the final application. In the remaining trials, samples were taken at 13 or 14 days after the final application. The proposed seasonal use rate is 1.6 lb a.i/acre (1.8 Kg/ha) with a 14– day pre-harvest interval (PHI).

Samples were analyzed for residues of RH-117281. Residue levels in the 34 samples from the 2.0 or 2.5 lb/acre (2.25 and 2.81 kg/ha) rates and 13 or 14 day PHI ranged from 0.218 to 4.52 ppm. The average residue was 0.88 ppm.

These data support a permanent tolerance of 5.0 ppm on grapes. Grape juice (clarified and unclarified) and raisins were generated from two RAC samples from one residue trial. Residues in grape juice were much lower than in the whole fruit, roughly 10% of the levels in the RAC. Residues concentrated in the raisins. The data support a permanent tolerance of 15 ppm on raisins.

ii. *Potatoes*. Sixteen field residue trials were conducted over two seasons in 10 States at either 1.25 lb a.i./acre and 2.50 lb a.i/a (1.40 kg/ha and 2.81 kg/ha) or 2.0 lbs a.i./acre and 4.0 lbs a.i./acre (2.25 kg/ha and 4.49 kg/ha). Ten applications were made in each trial. In two of the trials, tubers were harvested at 0, 3, 7, and 14 days after the final application. In the remaining trials, samples were taken at 3 days after the final application. The proposed maximum seasonal use rate is 1.6 lb a.i./acre (1.8 kg/ha) with a 3–day PHI. Samples were analyzed for parent RH-117281 and the two metabolites RH-141452 and RH-141455.

Samples were below the LOQ in nearly all cases. These residues support the establishment of a permanent tolerance of 0.1 ppm on potatoes.

Twelve residue trials were conducted in 7 regions in Canada during 1998 at 2.0 kg/ha and a PHI of 3–days. There were no residues of any analyte above the LOQ of 0.02 ppm in any sample.

A potato process study was conducted. Residues of two metabolites concentrated in flakes, consistent with loss of water from the potato.

B. Toxicological Profile

1. *Acute toxicity*. RH-117281 Technical was practically non-toxic by ingestion of a singe oral dose in rats and mice lethal dose (LD_{50}) > 5,000 milligram/kilogram (mg/kg), practically non-toxic by dermal application to rats (LD_{50} > 2,000 mg/kg), and practically non-toxic to rats after a 4–hour inhalation exposure with an LC_{50} value of > 5.3 milligrams per liter (mg/L) (highest attainable concentration), is not considered to be a primary eye irritant or a skin irritant and is not a dermal sensitizer. The technical material was non irritating to skin after single applications and moderately irritating to eyes. RH-117281 Technical produced delayed contact hypersensitivity in the guinea pig at concentrations of 2,500 ppm and higher. An acute neurotoxicity study in rats did not produce any neurotoxic or neuropathologic effects with a NOAEL > 2,000 mg/kg.

2. *Genotoxicity*. RH-117281 was nonmutagenic in a standard battery of tests. In *in vitro* assays, RH-117281 showed no evidence of mutagenic activity in an Ames and CHO/HGPRT assays for gene mutation, and no evidence of structural chromosomal aberrations in the CHO *in vitro* cytogenetic study. As predicted by its antitubulin mode of action, mitotic accumulation and polyploidy were noted at cytotoxic doses in the *in vitro* chromosomal assay. However, there was no evidence of structural or numerical chromosomal aberrations when RH-117281 Technical was tested *in vivo* in the mouse micronucleus test.

3. *Reproductive and developmental toxicity*. NOAELs for developmental and maternal toxicity to RH-117281 Technical were established at 1,000 mg/kg/day, highest dose tested (HDT) in both the rat and rabbit. No signs of developmental toxicity were exhibited.

In a 2-generation reproduction study in the rat, RH-117281 Technical had no adverse effects on reproductive performance or pup development at doses up to and exceeding 1474 mg/kg/day, the limit dose tested (LDT). This NOAEL was 20-fold higher than the NOAEL for adult toxicity of 71 mg/kg/day. A delay in periweaning weight gain and associated spleen effects in the F1 and F2a litters were shown in the F2b litters to be a secondary effect related to feed refusal due to palatability of the treated diets, and not to a systemic toxic effect. The consequences of feed refusal due to palatability do not constitute an adverse effect relevant to human health risk assessment.

4. *Subchronic toxicity*. The NOAEL in a 90–day rat subchronic feeding study was 1,509 mg/kg/day in males and 1,622 mg/kg/day in females (HDT). RH-117281 Technical did not produce neurotoxic or neuropathologic effects.

In a 90–day feeding study with mice, the NOAEL was 436 mg/kg/day in males and 574 mg/kg/day in females based on a slight decrease in weight gain among the females only at the LOAEL of 1,666 mg/kg/day.

A 90–day dog feeding study gave a NOAEL of 55 mg/kg/day in males and 62 mg/kg/day in females based on increased liver weights without a corresponding clinical or histopathologic change in females only at 322 mg/kg/day.

No signs of systemic toxicity were observed when RH-117281 Technical was administered dermally to rats for 28 days at a limit dose of 1,000 mg/kg/day. This occurred despite skin irritation at all doses tested (150, 400, and 1,000 mg/kg/day). Similarly, *in vivo* dermal absorption was shown to be low regardless of concentration or formulation type (i.e. < 1-6% of theadministered dose was systemically absorbed after 24 hours).

5. *Chronic toxicity*. In a combined rat chronic/oncogenicity study, the NOAEL for chronic toxicity was 51 mg/kg/day in males and 65 mg/kg/day based on an equivocal increase in relative liver weight at a LOAEL of 328 mg/kg/day in females at the interim sacrifice only. The NOAEL was considered to be 1,058 mg/kg/day in males and 1,331 mg/kg/day in females (HDT, limit dose). No carcinogenicity was observed.

An 18–month mouse carcinogenicity study showed no signs of carcinogenicity or of any other compound-related effect at dosage levels up to 1,021 mg/kg/day in males and 1,289 mg/kg/day in females HDT, limit dose).

The NOAEL in a 1–year feeding study in dogs was 255 mg/kg/day in males and 48 mg/kg/day in females based on minimal effects on body weight (bwt) and body weight gain and increased liver weights in females only at a LOAEL of 278 mg/kg/day.

6. *Animal metabolism*. In pharmacokinetic and metabolism studies in the rat, RH-117281 Technical

was rapidly and extensively absorbed, metabolized and excreted following oral exposure. A total of approximately 60% of the administered dose was systemically absorbed. Plasma levels peaked within 8 hours of dosing, and declined with a half-life of 12-14 hours, consistent with the nearly complete excretion within 48 hours. No evidence of accumulation of the parent compound or its metabolites was observed. The predominant route of excretion was hepatobiliary. Metabolism was found to occur through multiple pathways involving primary hydrolysis, glutathione-mediated reactions, and reductive dehalogenation; secondary oxidation on both the aromatic methyl and the aliphatic side-chain; and terminal glucuronic acid and amino acid conjugation. Altogether, 32 separate metabolites were identified; no single metabolite other than parent RH-117281 accounted for more than 10% of the administered dose. The rapid metabolism and excretion of RH-117281 Technical was a major factor explaining the compound's overall remarkably low toxicity profile in animals.

7. *Metabolite toxicology*. Of these multiple pathways, all three are common to both laboratory (rat) and food-producing animals (goat). Extensive degradation and elimination occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet. There were no significant metabolites other than the parent RH-117281 in grapes. Two minor metabolites in the rat constituted a major portion of the residue in potato tubers in the ^{14}C-metabolism study. RH-141452 and RH-141455 are not considered toxicologically significant as they were practically non-toxic after acute oral administration in mice, non mutagenic in the Ames test, and rapidly excreted essentially unchanged in rats. Actual residues in field trials never exceeded trace levels approximating the LOQ.

8. *Endocrine disruption*. Based on structure-activity and mode of action information as well as the lack of developmental and reproductive toxicity, RH-117281 Technical is unlikely to exhibit endocrine activity. There was no evidence of a functional or histopathologic change in the male or female reproductive tract, and no indicators of an endocrine effect of any kind below limit doses in mammalian subchronic or chronic studies or in mammalian and avian reproduction studies. A slight thyroid effect at the limit dose (994-1139 mg/kg/day) in the subchronic dog studies was secondary to liver hypertrophy and enlargement at that dose. Collectively, the weight of evidence provides no indication of an endocrine effect of RH-117281 Technical.

9. *Toxicological endpoints*— i. *Acute and short term dietary*. No endpoint of concern was identified for acute or short term (1–7 day) dietary exposure to RH-117281 Technical, and no acute or short term risk assessment is required.

ii. *Chronic dietary*. The proposed RfD for RH-117281 Technical is 0.5 mg/kg/day, based on application of a 100-fold uncertainty factor to the chronic NOAELs in the rat and dog of 51 and 48 mg/kg/day, respectively.

iii. *Carcinogen classification*. There was no evidence of oncogenic potential in two well-conducted lifetime feeding studies in rats and mice, at doses up to and including the limit dose. Thus, RH-117281 Technical should be classified as ''unlikely'' to have carcinogenic potential.

C. Aggregate Exposure

1. *Dietary (food) exposure*. Tolerances are proposed for the residues of RH-117281 Technical in or on potatoes (0.1 ppm), grapes (5 ppm), and raisins (15 ppm). The goat metabolism study demonstrated that there is no reasonable expectation of transfer of residues of RH-117281 Technical into meat or milk from potatoes. There are no grape feed commodities fed to livestock, and no potato or grape feed commodities fed to poultry. There are no other established or proposed United States tolerances for RH-117281 Technical, and no currently registered uses in the United States. Risk assessments were conducted by Rohm and Haas to assess dietary exposures and risks from RH-117281 Technical as follows:

i. *Acute exposure and risk*. No acute endpoint was identified for RH-117281 Technical and no acute risk assessment is required.

ii. *Chronic exposure and risk*. For chronic dietary risk assessment, the proposed tolerance values, as well as anticipated (average) residues and processing factors, were used and the assumption that 100% of all potatoes and grapes will contain residues of RH-117281 Technical at the tolerance or anticipated residue levels. Potential chronic exposures were estimated using USDA food consumption data from the 1989-1992 survey. With the proposed tolerances and anticipated residue levels for RH-117281 Technical, the percentage of the 0.5 mg/kg/day reference dose (RfD) utilized as follows:

Group	AnticipatedResidues Total % RfD	Tolerance Levels Total % RfD
U.S. Population 48 States	0.5	0.1
Nursing Infants < 1 year old	1.0	0.2
Non-Nursing Infants < 1–year old	1.2	< 0.1
Children 1-6 years old	1.7	.1
Children 7-12 years old	0.5	0.1

The chronic dietary risks from these uses do not exceed EPA's level of concern.

2. *Drinking water*. No direct information is available on potential for exposure to RH-117281 Technical from drinking water. However, exposure from drinking water is unlikely to occur as a result of the uses on potatoes or grapes. Submitted environmental fate studies indicat0e that Rh-117281 Technical dissipates rapidly from the environment under all conditions tested, and that is not mobile and poses no threat to groundwater. Furthermore, its environmental metabolites are very snort-lived and also have no potential to leach.

There is no established Maximum Concentration Level (MCL) for residues of RH-117281 Technical in drinking water, and no drinking water health advisory levels have been established. There is no entry for RH-117281 Technical in the ''Pesticides in Groundwater Database'' (EPA 734-12-001, September 1992).

i. *Chronic exposure and risk*. Nevertheless, to assess an upper bound on the potential for exposure from drinking water, chronic exposure to RH-117281 Technical in drinking water was estimated using the generic expected environmental concentration (GENEEC) V1.2 and SCI-GROW models, as directed in the Office of Pesticide Program's Interim Approach for Addressing Drinking Water Exposure. GENEEC is a highly conservative model used to estimate residue concentrations in surface water. SCI=GROW is an equally

conservative model used to estimate residue concentrations in shallow, highly vulnerable groundwater (i.e., sites with sandy soils and depth to groundwater of 10 to 20 feet). As indicated in EPA's drinking water exposure guidance, a very small percentage of people in the United States would derive their drinking water from such sources. GENEEC (56–Day average) and SCI-GROW water exposure values utilizes substantially less than 1% of the RfD for adults and children.

3. *Non-dietary exposure*. RH-117281 Technical is not currently registered for any indoor or outdoor residential or structural uses, and no application is pending; therefore, no non-dietary non-occupational exposure is anticipated.

4. *Aggregate exposure and risk*. The anticipated exposure from food and drinking water combined is < 2% of the RfD, and there is no expectation of other non-occupational exposure. Thus, aggregate exposure of RH-117281 Technical does not exceed EPA's level of concern, and is essentially negligible.

D. Cumulative Effects

At this time, no data are available to determine whether RH-117281 Technical has a common mechanism of toxicity with other substances. Thus, it is not appropriate to include this fungicide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, RH-117281 Technical does not appear to produce a toxic metabolite produced by other substances. In addition, the toxicity studies submitted to support this petition indicate that RH-117281 has only limited toxic potential. No toxic endpoints of potential concern were identified. For the purposes of this tolerance action, therefore, RH-117281 Technical [Benzamide-3,5-dichloro-N-(3-Clair-1-ethyl-1-methyl-2-oxopropyl)-4- methyl] is assumed not to have a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*— i. *Acute exposure and risk*. Since no acute endpoint was identified for RH-117281 Technical, no acute risk assessment is required.

ii. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by the dietary (food only) exposure to residues of RH-117281 Technical from the proposed tolerances is 0.5% (tolerance levels) and 0.1% (anticipated residues) for the U.S. population. Aggregate exposure (food and water) are expected to be < 1% RfD. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Rohm and Haas concludes there is a reasonable certainty that no harm will result from aggregate exposure to RH-117281 Technical residues to the U.S. population.

2. *Infants and children*—i. *General*. The potential for additional sensitivity of infants and children to residues of RH-117281 Technical is assessed using data from developmental toxicity studies in the rat and rabbit and 2-generation reproduction studies in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

ii. *Developmental toxicity studies—Rats*. In a developmental toxicity study in rats, the maternal NOAEL was 1,000 mg/kg/day, HDT, and the developmental (pup) NOAEL was 1,000 mg/kg/day HDT.

Rabbits. In a developmental toxicity study in rats, the maternal NOAEL was 1,000 mg/kg/day HDT, and the developmental (pup) NOAEL was 1,000 mg/kg/day HDT.

iii. *Reproductive toxicity study—Rats*. In a multigeneration reproductive toxicity study in rats, theparental (systemic) NOAEL was 71 mg/kg/day, based on an equivocal liver effect at the lowest observed adverse effect levels (LOAEL) of 360 mg/kg/day. The NOAEL for reproductive and developmental effects was 1,471 mg/kg/day HDT. No adverse reproductive or developmental effects were observed.

iv. *Prenatal and postnatal sensitivity*. No developmental or reproductive effects were demonstrated for RH-117281 Technical as a result of systemic exposure at up to limit doses of 1,000 and 1,471 mg/kg/day. Additionally, these NOAELs are greater than 20-fold higher than the NOAELs of 48-51 mg/kg/day from the dog and rat chronic studies which are the basis of the RfD. These developmental and reproductive studies indicate that developing and maturing animals are not more sensitive either pre or postnatally than other age groups to RH-117281 Technical; i.e., RH-117281 Technical does not exhibit additional pre or postnatal sensitivity. Thus, reliable data indicate that an additional FQPA uncertainty factor is not necessary to insure an adequate margin of safety for protection of infants and children.

a. *Acute exposure and risk*. No acute endpoint was identified for RH-117281 Technical, and therefore no acute risk assessment is required.

b. *Chronic exposure and risk*. Using the conservative exposure assumptions described above and taking into account the completeness and reliability of the toxicity data, the percentage of the RfD that will be utilized by dietary (food only) exposure to residues of RH-117281 Technical from the proposed tolerances is 1.0% (tolerance levels) and 0.2% (anticipated residues) for children, 1-infants (< 1–year) and 1.7% (tolerance levels) and 0.1% (anticipated residues) for children, 1-6 years old, the most highly exposed subgroups. Aggregate exposure (food and water) are expected to be < 2% RfD. 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.

F. International Tolerances

There are currently no CODEX, Canadian or Mexican maximum residue levels (MRLs) established for RH-117281 Technical in potatoes, potato chips or flakes, grapes or raisins. Thus, no harmonization issues are required to be resolved for this action.

G. Rotation Crop Restrictions

An outdoor C rotation crop study was conducted, in which leafy, root, and grain crops and soybeans were planted back 30, 137, 210, and 365 days following four applications. No individual metabolite comprised greater than or equal to 0.01 ppm in any matrix.

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6431–4]

Proposed CERCLA Prospective Purchaser Agreement; Canton Industrial Corporation Site; City of Canton, Fulton County, Illinois

AGENCY: Environmental Protection Agency.

ACTION: Notice; request for public comment.

SUMMARY: In accordance with the Comprehensive Environmental Reponse, Compensation, and Liability Act, as amended (''CERCLA''), 42 U.S.C.