residues in drinking water and exposure from non-occupational sources.

Based on the available acute toxicity data, Monsanto believes that glyphosate does not pose any acute dietary risks.

2. *Drinking water*. A Maximum Concentration Level (MCL) has been established for residues of glyphosate in drinking water at 0.7 mg/l since glyphosate is approved for direct application to water. The MCL represents the level at which no known or anticipated adverse health effects occur, allowing for an adequate margin of safety (MOE), and is based on the RfD.

Monsanto reports that glyphosate adsorbs strongly to soil and is not expected to move vertically below the 6-inch soil layer; residues are expected to be immobile in soil. Glyphosate is readily degraded by soil microbes to AMPA, which is degraded to carbon dioxide. Monsanto believes that glyphosate and AMPA are not likely to move to ground water due to their strong adsorptive characteristics. However, due to its aquatic use patterns and through erosion, glyphosate does have the potential to enter surface waters, where, according to Monsanto, it will adsorb to sediment and undergo microbial degradation.

3. *Non-dietary exposure*. Exposure (non-occupational) of the general population to glyphosate is expected based on the currently-registered uses; however, due to the low acute toxicity and lack of other toxicological concerns, Monsanto believes that the risk posed by non-occupational exposure (NOE) to glyphosate is minimal.

D. Cumulative Effects

Because the existing data base is insufficient to fully assess cumulative toxic effects that may be caused by glyphosate along with other chemical compound(s) that may share a common mechanism of toxicity, Monsanto believes that any consideration of such an analysis of toxicity is inappropriate at this time.

E. Safety Determination

1. *U.S. population*. The TMRC for existing, published tolerances for glyphosate is 0.021460 mg/kg/bwt/day or 1.0% of the RfD for the overall U.S. population. Even using conservative exposure assumptions and substituting the more widely consumed jackfruit, sugar apple and lychee, there is not enough exposure to calculate a significant contribution to the TMRC. As the exposure from durian, mangosteen and rambutan would be even less, the aggregate exposure of these three fruits will not add to the RfD for the overall U.S. population. EPA generally has no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Monsanto concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of glyphosate, including all anticipated dietary exposure and all other non-occupational exposures.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of glyphosate, data were considered from developmental toxicity studies in the rat and rabbit and multi-generation reproduction studies in rats.

No birth defects were observed in the offspring of rats given glyphosate by gavage at dose levels of 0, 300, 1,000, and 3,500 mg/kg/day on days 6 through 19 of gestation. The NOEL for this study was 1,000 mg/kg/day based on maternal and developmental toxicity observed at the HDT, 3,500 mg/kg/day. The high-dose in this study was 3.5 times higher than the limit dose that is currently required by the guidelines.

No birth defects were observed in the offspring of rabbits given glyphosate by gavage at dose levels of 0, 75, 175, and 350 mg/kg/day on days 6 through 27 of gestation. The NOEL for this study is considered to be 175 mg/kg/day based on maternal toxicity at the high-dose of 350 mg/kg/day. Because no developmental toxicity was observed at any dose level, the developmental NOEL is considered to be 350 mg/kg/day.

Male and female rats were fed glyphosate at dose levels of 0, 3, 10, and 30 mg/kg/day every day throughout the production of three successive generations. No adverse treatment-related effects on reproduction were observed. Because no toxicity was noted even at the HDT, a second reproduction study at higher dose levels (HDLs) was performed and is described below.

Male and female rats were fed glyphosate at dose levels of 0, 100, 500, and 1,500 mg/kg/day every day throughout the production of two successive generations. Reduced body weights and soft stools occurred at 1,500 mg/kg/day (3% of the diet); therefore, the systemic NOEL is considered to be 500 mg/kg/day. Glyphosate did not affect the ability of rats to mate, conceive, carry or deliver normal offspring at any dose level.

3. *Reference dose*. The TMRC for existing, published and pending tolerances (including durian, mangosteen, and rambutan) for glyphosate range from 0.015 for nursing infants to 0.049 for non-nursing infants (0.8 to 2.5% of the RfD). EPA generally has no concern for exposures below 100% of the RfD. Therefore, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, Monsanto concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of glyphosate, including all anticipated dietary exposure and all other non-occupational exposures.

4. *Endocrine effects*. No known factors were identified in sub-chronic, chronic or developmental toxicity studies to indicate any endocrine-modulating activity by glyphosate.

F. International Tolerances

Codex maximum residue levels (MRLs) have not been established for residues of glyphosate on durian, mangosteen and rambutan. (Sidney Jackson).

[FR Doc. 98–22430 Filed 8–25–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–825; FRL–6023–4]

Notice of Filing of Pesticide Tolerance Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

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

DATES: Comments, identified by the docket control number PF–825, must be received on or before September 25, 1998.

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

Comments and data may also be submitted electronically by following the instructions under ''SUPPLEMENTARY INFORMATION.'' No Confidential Business Information (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. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Mark Dow	Rm. 214, CM #2, 703–305–5533; e-mail: Dow.mark@epamail.epa.gov.	1921 Jefferson Davis Hwy, Arlington, VA
Mary L. Waller	Rm. 247, CM #2, 703 308–9354; e-mail: waller.mary@epamail.epa.gov.	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment of regulations for residues of certain pesticide chemicals in or on various raw food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports grantinig of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice, as well as the public version, has been established for this notice of filing under docket control number PF–825 (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''.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comment and data will also be accepted on disks in Wordperfect 5.1/6.1 file format or ASCII file format. All comments and data in electronic form must be identified by the docket control number (PF–825) and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

Authority: 21 U.S.C. 346a.

List of Subjects

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

Dated: August 10, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Below summaries of the pesticide petitions are printed. The summaries of the petitions were prepared by the petitioners. 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. Novartis Crop Protection, Inc.

PP 7E4919 and 8F4978

EPA has received two pesticide petitions (7E4919 and 8F4978 from Novartis Crop Protection, Inc., 410 Swing Road, Greensboro, NC 27419 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 tolerances for residues of fludioxonil (4–(2,2-difluoro-1,3-benzodioxol-4-yl)-1*H*-pyrrole-3-carbonitrile) in or on the raw agricultural commodities: grapes at 1.00 parts per million (ppm) (7E4919); canola, peanuts, sunflowers, leafy vegetables except brassica (Crop Group 4); brassica leafy vegetables (Crop Group 5); legume vegetables (Crop Group 6); foliage of legume vegetables (Crop Group 7); fruiting vegetables (Crop Group 8); cucurbit vegetables (Crop Group 9); forage, fodder, and straw of cereal grains (Crop Group 16); grass , forage, fodder, and hay (Crop Group 17); and non-grass animal feeds (Crop Group 18) at 0.01 ppm; root and tuber vegetables (Crop Group 1); leaves of root and tuber vegetables (Crop Group 2); bulb vegetables (Crop Group 3); cereal grains (Crop Group 15); and herbs and spices (Crop Group 19) at 0.02 ppm; and cotton at 0.05 ppm (8F4978). 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 fludioxonil is adequately understood for the purpose of the proposed tolerances. The residues of regulatory concern is the parent compound only. Metabolism in grapes involves oxidation of the pyrrole ring, primarily at the 2 and 5 positions. Subsequent opening of the oxidized pyrrole ring yields a metabolite with an amide plus a carboxylic acid group. This open-ring metabolite undergoes further oxidation at the bridgehead carbon followed by decarboxylation.

2. *Analytical method*. Novartis has developed and validated analytical methodology for enforcement purposes as part of the original corn, sorghum, and potato registrations. This method (Novartis Crop Protection Method AG-597B) has passed an Agency petition method validation (PMV) and is currently the enforcement method for potatoes. As part of this petition, Novartis has validated the method on the crops, fractions, and crop representatives of each crop grouping associated with this submittal. The method validation study (ABR-97060) contains recovery data on over eighty individual substrates. In most cases, a limit of quantitation of 0.01 ppm of fludioxonil was achieved. For several very difficult substrates, a limit of quantitation of 0.02 ppm and for cotton substrates a limit of 0.05 ppm were achieved.

For the analysis of grapes, grape juice, and wine the analytical Method AG-579B14 is proposed as the regulatory enforcement method. It has been validated by the Agency as an enforcement method for fludioxonil as AG-57912. In Method AG-579B14, whole fruit or wine samples are extracted with acetonitrile/water (90/10). Red and white grapes, as well as red and white wine samples were analyzed by this method. Recoveries (from 0.02 ppm to 1.0 ppm) ranged from 73% to 114% with a mean of 92% (n=15).

3. *Magnitude of residues*. Residue trials were conducted on cotton, wheat, radishes, lettuce, cucumbers and peas in the major crop growing areas of the U.S. in addition to residue trials previously on corn, sorghum, potatoes and grapes. Several trials were conducted on each crop. Rates were 0.5×, 1.0×, 2.5× and 5.0× of the proposed use rate on all crops except cotton where 1.0× and 3.0× were used.

From 6 cotton trials, field trash, gin trash, un-delinted seed and cottonseed fractions (hulls, meal, refined oil) were analyzed for fludioxonil at a method limit of determination of 0.05 ppm. At this level, no quantifiable residues of fludioxonil were found in any RAC or fraction at the proposed or the exaggerated (3×) rate.

Seven trials were completed on wheat. At a method limit of quantification of 0.02 ppm, no quantifiable residues of fludioxonil were observed in any RAC at the proposed treatment rate or at rates up to 5× the proposed treatment rate.

Five trials were completed on radishes which represents the absolute worst case for potential uptake of residues because of its very rapid growth and short growing season (27–55 days in these studies). Both root and top samples from all rates in all 5 trials were analyzed at a method limit of determination of 0.01 ppm. No fludioxonil residue (<0.01 ppm) was found in any root or top sample at the proposed use rate or at rates up to 5× the proposed use rate.

Mature lettuce leaves from all treatment rates of the 6 trials were analyzed for fludioxonil at a method limit of determination of 0.01 ppm. No fludioxonil residue was found in any lettuce sample at the proposed use rate or up to 5× the proposed use rate.

Cucumbers from all treatment rates in all 6 trials were analyzed at a method limit of determination of 0.01 ppm. No fludioxonil residue was found in any cucumber sample at the proposed use rate or up to 5× the proposed use rate.

Peas with pods from 5 trials were analyzed at a method limit of determination of 0.01 ppm. No fludioxonil residue was found in any pea sample at the proposed use rate or at rates up to 5× the proposed use rate.

Thirty (30) field trials were conducted under maximum label rates on ten varieties of grapes in the major grape-growing regions of France, Switzerland, and Chile. Grape residue data were generated from fifty (50) samples treated at the maximum use rate. Data on transfer to grape juice were generated from sixteen (16) samples and data concerning transfer to wine were based on twenty-six (26) samples. Raisin data were produced from sixteen (16) samples.

Supplemental data (including fifteen (15) decline curves) were generated using exaggerated rates (due to multiple applications) on 89 additional samples of whole fruit. Supplemental data were also provided on eight additional juice samples and on twelve additional wine samples. Raisin data on eight samples were also provided. These data demonstrate dose response, provide additional decline information, provide additional information on transfer to juice and wine, and show that residue data obtained from other grape-growing countries (Italy and South Africa) fully support the results obtained from France, Switzerland, and Chile. The raisin data also demonstrate no significant concentration of residues.

Analysis of mature grapes at harvest following a single foliar application of fludioxonil at 500 grams a.i./ha at flowering, up to the beginning of bunch closing, resulted in maximum whole fruit residues of 0.77 ppm. Similarly, analysis of grapes at harvest following two foliar applications of fludioxonil at 250 grams a.i./ha/application at flowering and again at stages up to fruit softening resulted in maximum whole fruit residues of 0.33 ppm. These results suggest that the application at flowering does not contribute to the residue in fruit. The data fully support an import tolerance of 1 ppm on grapes imported into the U.S..

The data support a 60–day pre-harvest interval (PHI) as listed on the GEOXE label for France. The data also support Chilean, Slovenian, and Bosnian PHIs of 15–days, 7–days (berries) to 21–days (applications to the vine), and 21–days, respectively for the combination product, Switch . There is no PHI on the Swiss Switch label, but the second application is limited by the label to mid-August, which results in a PHI greater than 21 days.

No significant concentration of residues was observed in grape juice, wine, or raisins. Thus, tolerances are not required for these processing fractions.

B. Toxicological Profile

1. *Acute toxicity*. Fludioxonil and end use formulations have very low toxicity to the mammalian species by the oral, dermal, or inhalation route. The dose needed to kill 50% of animals was calculated to be greater than 5,000 mg/kg (oral), 2,000 mg/kg (dermal), and 2.6 mg/L (inhalation) in these studies. The eye and skin irritations seen in animals upon acute exposure indicate that no more than transient and slight irritation. No sensitizing potential was noted with either the technical material or the formulated product.

2. *Genotoxicity*. Mutagenicity potential of fludioxonil was tested in several studies. In the Chinese hamster ovary cell assay, some clastogenic and polyploidogenic effects were seen at or near the precipitating concentration of the test substance. However, results were negative in the Ames assay, Chinese hamster V79 cell assay, hepatocyte DNA repair assay, rat hepatocyte micronucleus test, mouse bone marrow test, and Chinese hamster bone marrow test. A dominant lethal test conducted in the mouse was also negative.

3. *Reproductive and developmental toxicity*. Fludioxonil is not a teratogen and does not affect reproduction or fertility. No fetal toxicity was observed even at the highest dose tested in both the rabbit (300 mg/kg) and the rat (1,000 mg/kg) teratogenicity studies. In a two–generation rat reproduction study, a reduction of pup body weight was seen at the highest feeding level of 3,000 ppm in the presence of maternal toxicity. The NOEL was 300 ppm for both maternal and fetal toxicity in this study.

4. *Subchronic toxicity*. In a 90–day dietary toxicity study the kidney and liver have been identified as target organs. In a subchronic study in rats, the NOEL was 10 ppm based on liver toxicity. In a subchronic study in mice, the NOEL was 100 ppm based on blue urine (a metabolite); the maximum tolerated dose was 7,000 ppm. In a subchronic study in dogs, the NOEL was 200 ppm based on clinical observations; the maximum tolerated dose was 8,000 ppm.

5. *Chronic toxicity*. In an 1–year chronic toxicity study in dogs, the NOEL was 100 ppm based on body weight effects; the maximum tolerated dose was 8,000 ppm.

Two 18-month dietary oncogenicity studies were performed in mice. While a NOEL of 1,000 ppm was clearly established in the first study, its highest feeding level (3,000 ppm) did not meet the criteria for a maximum tolerated dose. In the second 18-month study, the

maximum tolerated dose was determined to be 5,000 ppm based on kidney effects. There were no treatment-related increases in neoplasia at any dose level tested in either study.In a combined chronic toxicity/oncogenicity study in rats, the incidence of liver tumors in top-dose females (3,000 ppm) was marginally higher than the concurrent controls but within historical control range. The NOEL for chronic toxicity was 1,000 ppm in both sexes.

6. *Animal metabolism*. The metabolism of fludioxonil in rats is adequately understood. The compound is rapidly absorbed and excreted. In rats, excretion in the feces is greater than excretion via the urine. Metabolism involves primarily oxidation at the 2 position of the pyrrole ring, with minor amounts of oxidation at the 5 position of the pyrrole ring and the 4 position of the phenyl ring. All of these oxidized metabolites are conjugated with glucuronic acid and sulfuric acid and then rapidly eliminated.

7. *Metabolite toxicology*. The residues of concern for tolerance setting purposes is the parent compound. Consequently, there is no additional concern for toxicity of metabolites. In grapes, fludioxonil is metabolized only to a limited extent. The metabolites thus formed have also been found in the rat. The major metabolites are those that result from the oxidation of the pyrrole ring and they are rapidly excreted upon conjugation. Consequently, there is no additional concern for toxicity of any metabolites in grapes.

8. *Endocrine disruption*. Fludioxonil does not belong to a class of chemicals known for having adverse effects on the endocrine system. No estrogenic effects have been observed in the various short and long term studies conducted with various mammalian species.

C. Aggregate Exposure

1. *Dietary exposure* —i. *Food*. For purposes of assessing the potential dietary exposure under the proposed tolerance, Novartis has estimated aggregate exposure based on the theoretical maximum residue concentration (TMRC) from the tolerance level of 1.0 ppm in or on grapes and from the established or proposed tolerance levels. The TMRC is a worse case estimate of dietary exposure since it is assumed that 100% of all crops for which tolerances are proposed or established are treated and that pesticide residues are present at the tolerance levels.

Fludioxonil's current registered use for seed treatment on corn and sorghum seeds does not contribute to dietary exposure because there are no detectable residues. EPA has ruled that these uses are food uses not requiring tolerances. For potato seed treatment, a tolerance of 0.02 ppm has been set. In conducting this exposure assessment, very conservative assumptions have been used (i.e., 100% of potatoes and grapes will contain fludioxonil residues at tolerance levels), resulting in an overestimate of human exposure.

ii. *Drinking water*. Exposure of the general population to residues of fludioxonil from drinking water is considered unlikely for two reasons: (1) the import tolerance for grapes would not lead to the exposure of the general population to residues of pesticides in drinking water; and (2) the movement of fludioxonil into groundwater is highly unlikely due to its chemistry. In addition, the EPA has not established a Maximum Contaminant Level for residues of fludioxonil in drinking water.

2. *Non-dietary exposure*. Non-occupational exposure for fludioxonil has not been calculated since the current registration for fludioxonil is limited to commercial crop production. Since the chemical is not used in or around the home, Novartis considers the potential for non-occupational exposure to the general population to be non-existent.

D. Cumulative Effects

Consideration of a common mechanism of toxicity is not appropriate at this time since Novartis is unaware of any reliable information that indicates that toxic effects produced by fludioxonil would be cumulative with those of any other chemical compounds. Consequently, Novartis is considering the potential risks of only fludioxonil in its aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Based on the available chronic toxicity data, EPA has set the Reference Dose (RfD) for fludioxonil at 0.03 mg/kg/day. This RfD is based on a 1-year feeding study in dogs with a No Observed Effect-Level (NOEL) of 3.3 mg/kg/day (100 ppm) and an uncertainty factor of 100. No additional uncertainty factor was judged to be necessary as body weight was the most sensitive indicator of toxicity in that study.

2. *Infants and children*. Using GENEEC water and aggregate exposures (water plus diet) 5.65% and 5.75% of the RfD were obtained for the most sensitive sub-populations, non-nursing infants and children (1–6 years), respectively. Aggregate exposure (water plus diet) utilizing the summed SCI-GROW estimated water concentrations (turf and seed treatment uses) resulted in an overall exposure of 1.72% of the RfD for the U.S. population. Aggregated exposure (water plus dietary) to non-nursing infants and children (1–6 years) was 3.49% and 4.69% of the RfD, respectively, using the combined turf and seed treatment water estimates. It should be noted that the aggregate exposure assessment greatly overestimates exposure since both GENEEC and SCI-GROW models generate extremely conservative and unrealistic water concentrations. In addition, all non-detected residues were assumed to be at the limit of quantitation and no market share adjustment was made. Therefore, a more than reasonable certainty exists that no harm will result from exposure to fludioxonil residues through food and water consumption if the proposed uses are registered.

F. International Tolerances

There are no Codex maximum residue levels established for residues of fludioxonil. (Mary L. Waller)

2. Rohm and Haas Company

PP 8F4994

EPA has received a pesticide petition (PP 8F4994) 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 tolerance for residues of Triazamate (Acetic acid, [[1-[(dimethylamino)carbonyl]-3–(1,1-dimethylethyl)-1*H*–1,2,4-triazol-5-yl]-,ethyl ester) in or on the raw agricultural commodity leafy green vegetables (crop subgroup 4A) at 2.5 parts per million (ppm); leaf petioles (crop subgroup 4B) at 0.6 ppm; head and stem Brassica (crop subgroup 5A) at 12.5 ppm and leafy Brassica (crop subgrop 5B) at 5.75 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 triazamate in plants (apples, potatoes, sugar beets) is adequately understood for the purpose of these tolerances. None of these crops are fed to animals and livestock metabolism studies are

not required. The metabolism of triazamate involves oxidative demethylation of the carbamoyl group. Parent compound is rapidly metabolized and is either not found or found at trace levels in plants. The majority of the total dosage is present as other non-cholinesterase inhibiting metabolites whose structures do not contain the dimethylcarbamoyl moiety. Tolerances for residues of triazamate should be expressed as the total residue from triazamate and its only cholinesterase-inhibiting metabolite RH-0422.

2. *Analytical method*. An analytical method employing liquid chromatography followed by two-stage mass spectroscopy detection has been developed and validated for residues of triazamate and RH-0422 in leafy and cole crop vegetables. The method involves extraction by blending with solvents and purification of the extracts by solid phase extraction chromatography. The limit of quantitation of the method is 0.01 ppm for both analytes.

3. *Magnitude of residues*. A total of 58 field residue trials in geographically representative regions of the U.S. was conducted with a 50% wettable powder formulation in the representative crops for the leafy and cole crop vegetable crop groups. Three or four applications were made at 0.25 lb. a.i./acre. Samples were harvested at 7 days after the last application. The highest detected value (sum of the residues of triazamate and RH-0422) in an individual sample was 0.63 ppm in head lettuce, 1.62 ppm in leaf lettuce, 2.23 ppm in spinach, 0.54 ppm in celery, 11.5 ppm in broccoli, 4.86 in cabbage and 5.54 ppm in mustard greens.

B. Toxicological Profile

1. *Acute toxicity*. Triazamate is a moderately toxic cholinesterase inhibitor belonging to the carbamate class. Triazamate Technical was moderately toxic to rats following a single oral dose (LD_{50} = 50–200 mg/kg), and after a 4–hr inhalation exposure (LC_{50} value of > 0.47 mg/L); and was minimally to slightly toxic to rats following a single dermal dose (LD_{50} >5,000 mg/kg). In a guideline acute neurotoxicity study with triazamate in the rat, the NOEL for clinical signs was 5 mg/kg based on the observation of cholinergic signs in 1 of 10 male rats at 25 mg/kg. Triazamate was practically non-irritating to the skin, moderately irritating to eyes in rabbits and did not produce delayed contact hypersensitivity in the guinea pig.

2. *Genotoxicty*. Triazamate is not mutagenic or genotoxic. Triazamate Technical was negative (non-mutagenic) in an Ames assay with and without hepatic enzyme activation. Triazamate Technical was negative in a hypoxanthine guanine phophoribosyl transferase (HGPRT) gene mutation assay using Chinese hamster ovary (CHO) cells in culture when tested with and without hepatic enzyme activation. In isolated rat hepatocytes, triazamate did not induce unscheduled DNA synthesis (UDS) or repair when tested up to the maximum soluble concentration in culture medium. Triazamate did not produce chromosome aberrations in an in vitro assay using Chinese hamster ovary cells (CHO) or an in vivo mouse micronucleus assay.

3. *Reproductive and developmental toxicity*. In a developmental toxicity study in rats with Triazamate Technical, the no-observed-effect-level (NOEL) for developmental toxicity was 64 mg/kg (highest dose tested) (HDT). The NOEL for maternal toxicity was 16 mg/kg based on clinical signs of cholinergic toxicity at 64 mg/kg.

In a developmental toxicity study in rabbits with Triazamate Technical, the NOEL for developmental toxicity was 10 mg/kg (HDT). The NOEL for maternal toxicity was 0.5 mg/kg based on clinical signs and decreased body weight at 10 mg/kg.

In a two-generation reproduction study in rats with Triazamate Technical, the NOEL for reproductive effects was 1,500 ppm (101 and 132 milligrams/kilograms/day (mg/kg/day) for males and females, respectively; HDT). The NOEL for parental toxicity was 10 ppm (0.7 and 0.9 mg/kg/day for males and females, respectively) based on decreased plasma and RBC cholinesterase activities at 250 ppm (17 and 21 mg/kg/day for males and females, respectively).

The acceptable developmental studies (prenatal developmental toxicity studies in rats and rabbits and two-generation reproduction study in rats) provided no indication of increased sensitivity of rats or rabbits to in utero and or post-natal exposure to triazamate. Triazamate Technical is not a developmental or reproductive toxicant.

4. *Subchronic toxicity*. In subacute and subchronic dietary toxicity studies, Triazamate Technical produced no evidence of adverse effects other than those associated with cholinesterase inhibition:

i. In a 90–day dietary toxicity study with Triazamate Technical in the rat, the NOEL for blood cholinesterase inhibition was 50 ppm (3.2 and 3.9 mg/kg/day for males and females, respectively), based on decreases in plasma and RBC cholinesterase activities at 500 ppm (32 and 39 mg/kg/day for males and females, respectively). The NOEL for brain cholinesterase inhibition and/or clinical signs was 500 ppm (32 and 39 mg/kg/day for males and females respectively) based on decreased brain cholinesterase activity and decreased body weight gain and feed consumption at 1,500 ppm (93 and 117 mg/kg/day for males and females, respectively).

ii. In a guideline subchronic neurotoxicity study (90–day dietary feeding) with Triazamate Technical in the rat, the NOEL for blood cholinesterase inhibition was 10 ppm (0.6 and 0.7 mg/kg/day for males and females, respectively), based on reductions in plasma and RBC cholinesterase activities at 250 ppm (14.3 and 17.1 mg/kg/day for males and females, respectively). The NOEL for brain cholinesterase inhibition and/or clinical signs was 250 ppm (14.3 and 17.1 mg/kg/day for males and females respectively) based on decreases in brain cholinesterase activity and cholinergic signs at 1,500 ppm (87 and 104 mg/kg/day for males and females, respectively).

iii. In a 90–day dietary toxicity study with Triazamate Technical in the mouse, the NOEL for blood cholinesterase inhibition was 2 ppm (0.4 and 0.5 mg/kg/day for males and females, respectively) based on decreases in plasma cholinesterase activity at 25 ppm (4 and 6 mg/kg/day for males and females, respectively). The NOEL for brain cholinesterase and/or clinical signs was 250 ppm (46 and 67 mg/kg/day for males and females, respectively) based on decreases in brain cholinesterase and decreases in body weight and feed consumption at 1,000 ppm (164 and 222 mg/kg/day for males and females, respectively).

iv. In a 90–day dietary toxicity study with Triazamate Technical in the dog, the NOEL for blood cholinesterase inhibition was 1 ppm for males only (0.03 mg/kg/day) based on decreases in plasma cholinesterase at 10 ppm (0.3 mg/kg/day). The dose of 1 ppm was a lowest-observed-effect level (LOEL) for females based on the presence of decreased plasma cholinesterase activity (24%). The NOEL for clinical signs was 10 ppm (0.3 mg/kg/day for males and females) based on a few clinical signs at 100 ppm (3.1 mg/kg/day for males and females).

v. In a 21–day dermal toxicity study with Triazamate Technical, the NOEL blood and brain cholinesterase inhibition was 10 mg/kg based on decreases in plasma, RBC and brain cholinesterase activities at 100 mg/kg.

5. *Chronic toxicity* — i. *Rat, mouse and dog studies.* In chronic dietary toxicity studies, Triazamate Technical produced no evidence of adverse effects other than those associated with cholinesterase inhibition and was not oncogenic in the rat and mouse.

In a combined chronic dietary toxicity/oncogenicity study (24 months) in rats with Triazamate Technical, no evidence of oncogenicity was observed at doses up to 1,250 ppm (62.5 mg/kg/day for males and females; HDT). The NOEL for blood cholinesterase inhibition was 10 ppm (0.5 and 0.6 mg/kg/day for males and females respectively) based on decreases in plasma and RBC cholinesterase activity at 250 ppm (11.5 and 14.5 mg/kg/day in males and females, respectively). The NOEL for brain cholinesterase inhibition and/or clinical signs was 250 ppm (11.5 and 14.5 mg/kg/day in males and females, respectively) based on clinical signs and decreases in brain cholinesterase inhibition at 1,250 ppm (62.5 mg/kg/day for males and females).

In a combined chronic dietary toxicity study (18 months) in mice with Triazamate Technical, no evidence of oncogenicity was observed at doses up to 1,000–1,500 ppm (130–195 mg/kg/day for males and females; HDT). The NOEL for blood cholinesterase inhibition was 1 ppm (0.1 and 0.2 mg/kg/day for males and females, respectively) based on decreased plasma cholinesterase activity at 50 ppm (6.7 and 8.4 mg/kg/day for males and females, respectively). The NOEL for brain cholinesterase inhibition and/or clinical signs was 50 ppm (6.7 and 8.4 mg/kg/day for males and females, respectively) based on decreased brain cholinesterase activity and other evidence of systemic toxicity at 1,000–1,500 ppm (130–195 mg/kg/day for males and females).

In a chronic dietary toxicity study (12 months) in dogs with Triazamate Technical, the NOEL for blood cholinesterase inhibition was 0.9 ppm (0.023 and 0.025 mg/kg/day for males and females, respectively) based on decreased plasma cholinesterase activity at 15.0 ppm (0.42 mg/kg/day for both males and females). The NOEL for brain cholinesterase inhibition was 15.0 ppm (0.42 mg/kg/day for both males and females) based on decreased brain cholinesterase activity at 150 ppm (4.4 and 4.7 mg/kg/day for males and females, respectively).

ii. *Human Studies.* A randomized double-blind, ascending dose study was conducted in human male volunteers to determine the safety and tolerability of Triazamate Technical and to establish a NOEL for adverse clinical toxicity. Single doses of Triazamate Technical, when administered orally by capsule to healthy male subjects, were tolerated up to and including a dose of 1.0 mg/kg. The 3.0 mg/kg dose of triazamate was not clinically tolerated well. Clinically, the NOEL was 0.3 mg/kg of triazamate based on minimal clinical signs at 1.0 mg/kg that were considered possibly related to treatment. Transient decreases in plasma and RBC cholinesterase occurred at doses lower than the dose that elicited adverse clinical signs.

Using its Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), Rohm and Haas Company considers triazamate to be classified as a Group ''E,'' not a likely human carcinogen.

A Reference dose (RfD) of 0.01 mg/kg/day is proposed for humans, based on the clinical NOEL in the human study (0.3 mg/kg) and applying an Uncertainty Factor (UF) of 30. The dose of 0.3 mg/kg was the highest dose in humans that did not produce toxicologically significant adverse effects (i.e., signs of cholinergic toxicity) and is 10 times lower than a dose that produced unequivocal signs of cholinergic toxicity in man. In addition, the clinical NOEL in humans is comparable to the no-observable-adverse-effect level (NOAEL) of 0.42 mg/kg/day following chronic dosing in the dog, the most sensitive laboratory animal species. An Uncertainty Factor of 10 is applied to the clinical NOEL in humans to account for potential variability within humans with respect to sensitivity towards triazamate. An additional Uncertainty Factor of 3 is included, since at 0.03 mg/kg (i.e., 1/10th the dose that was a clinical NOEL) there was a transient but measurable depression in plasma cholinesterase in humans. Although a change in the plasma pseudo-cholinesterase (i.e., butyl-cholinesterase) is not toxicologically significant since this enzyme is not molecularly similar to acetyl-cholinesterase, the additional uncertainty factor of 3 establishes a reference dose at a level where a measurable response of any kind, irrespective of the toxicological significance of the finding, will not plausibly occur.

6. *Animal metabolism.* The absorption, distribution, excretion and metabolism of triazamate in rats, dogs and goats was investigated. Triazamate is rapidly absorbed when given orally (capsule or gavage) but slower following dietary intake. Peak blood levels following dietary administration were 10-fold lower than after gavage administration of an equivalent mg/kg/dose. Elimination is predominately by urinary excretion and triazamate does not accumulate in tissues. The metabolism of triazamate proceeds via ester hydrolysis and then a rapid stepwise cleavage of the carbamoyl group. The free acid, (RH-0422) is the only toxicologically significant metabolite, given that it contains the carbamoyl group. Other metabolites of triazamate, which are seen in other animal and plant metabolism studies, do not contain the carbamoyl group and do not produce cholinesterase inhibition.

7. *Metabolite toxicology.* Common metabolic pathways for triazamate have been identified in both plants (apple, potato, sugar beet) and animals (rat, goat, hen). The metabolic pathway common to both plants and animals involves oxidative demethylation of the carbamoyl group. Extensive degradation and elimination of polar metabolites occurs in animals such that residues are unlikely to accumulate in humans or animals exposed to these residues through the diet.

8. *Endocrine disruption.* The toxicology profile of triazamate shows no evidence of physiological effects characteristic of the disruption of mammalian hormones. In developmental and reproductive studies there was no evidence of developmental or reproductive toxicity. In addition, the molecular structure of triazamate does not suggest that this compound would disrupt the mammalian hormone system. Overall, the weight of evidence provides no indication that triazamate has endocrine activity in vertebrates.

C. Aggregate Exposure

1. *Dietary exposure.* A RfD of 0.01 mg/kg/day is proposed for humans, based on the clinical NOEL in the human study (0.3 mg/kg) and applying an Uncertainty Factor of 30.

2. *Food* — i. *Acute risk.* An acute dietary risk assessment (Dietary Exposure Evaluation Model , Novigen Sciences Inc., 1997) was conducted for triazamate using a Tier 3 Monte Carlo simulations approach using the distribution of residues for apples, pears, head and leaf lettuce, spinach, celery, broccoli, cabbage and mustard greens, the entire distribution of daily food consumption data for pome fruit and leafy and cole crop vegetables and adjustments for percent crop treated. The Margins of Exposure (MOEs) for the 95th percentile exposures were 270 for the U.S. population and 388 for the most sensitive sub-population, Children 1–6 years old. This indicates that acute dietary risk is acceptable because the MOE is greater than 30, and 30 is the appropriate Uncertainty Factor when

the assessment is based on a human clinical study.

ii. *Chronic risk.* Chronic dietary risk assessments (Dietary Exposure Evaluation Model , Novigen Sciences Inc., 1997) were conducted for triazamate using two approaches: (1) using a tolerance levels and assuming 100% of crop is treated, and (2) using anticipated residue concentration levels adjusted for projected market share or percentage of crop treated. The Theoretical Maximum Residue Contribution (TMRC) and Anticipated Residue Contribution (ARC) from these two scenarios represents 35.0% and 3.6%, respectively, of the RfD for the U.S. popultion as a whole. The subgroup with the greatest chronic exposure is Children 1–6 years old for which the TMRC and ARC estimates represents 59.4% and 7.0%, respectively, of the RfD. The chronic dietary risks from these uses do not exceed EPA's level of concern.

3. *Drinking water.* Both triazamate and its cholinesterase-inhibiting metabolite RH-0422 are degraded rapidly in soil This rapid degradation has been observed in both laboratory and field studies and makes it highly unlikely that measurable residues of either compound would be found in ground or surface water when triazamate is applied according to the proposed label use directions.

4. *Non-dietary exposure.* Triazamate is not registered for either indoor or outdoor residential uses. Non-occupational exposure to the general population is therefore not expected and not considered in aggregate exposure estimates.

D. Cumulative Effects

The potential for cumulative effects of triazamate with other substances that have a common mechanism of toxicity was considered. It is recognized the triazamate, although structurally a pseudo-carbamate, exhibits toxicity similar to the carbamate class of insecticides, and that these compounds produce a reversible inhibition of the enzyme cholinesterase. However, Rohm and Haas Company concludes that consideration of a common mechanism of toxicity is not appropriate at this time since EPA does not have the methodology to resolve this complex scientific issue concerning common mechanisms of toxicity. Based on these points, Rohm and Haas Company has considered only the potential risks of triazamate and RH-0422 in its cumulative exposure assessment.

E. Safety Determination

1. *U.S. population.* The acute and chronic dietary exposures to triazamate and its metabolite from the proposed use on leafy and cole crop vegetables were evaluated. Exposure to triazamate and its toxicologically significant metabolite in or on pome fruit or leafy and cole crop vegetables does not pose an unreasonable health risk to consumers including the sensitive subgroup non-nursing infants. In Tier 3 acute analyses for the 95th percentile exposures, MOEs were 270 for the general U.S. population. Using the TMRC and assuming 100% of crop treated, the most conservative chronic approach, chronic dietary exposures represents 35.0% of the RfD for the U.S. population. 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.

Using the two conservative exposure assessments described above and taking into account the completeness and reliability of the toxicity data, Rohm and Haas Company concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of triazamate and its toxicologically significant metabolite to the U.S. population.

2. *Infants and children.* In assessing the potential for additional sensitivity of infants and children to residues of triazamate, data from developmental toxicity studies in the rat and rabbit and two two–generation reproduction studies in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development to one or both parents. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA may apply an additional Uncertainty Factor for infants and children in the case of threshold effects to account for pre-and post- natal effects and the completeness of the toxicity database. Based on current toxicological data requirements, the toxicology database for triazamate relative to pre- and post-natal effects is complete. For triazamate, developmental toxicity was not observed in developmental studies using rats and rabbits. The NOEL for developmental effects in rats was 64 mg/kg/day and rabbits was 10 mg/kg/day. In the two–generation reproductive toxicity study in the rat, the reproductive/developmental toxicity NOEL was 101–132 mg/kg/day. These NOELs are 10–fold or higher than those observed for systemic toxicity, i.e., cholinesterase inhibition.

In Tier 3 acute dietary analyses for the 95th percentile exposures, MOEs were 388 for Children 1–6 years old. Using the TMRC and assuming 100% of crop treated, the most conservative chronic approach, chronic dietary exposures represents 59.4% of the RfD for Children 1–6 years old. Using the ARC and adjusted for an anticipated market share or percentage of crop treated, the chronic dietary exposure to this subgroup represents 7.0% of the RfD. Therefore Rohm and Haas Company concludes that there is a reasonable certainty that no harm will result from aggregate exposure to residues of triazamate and its toxicologically significant metabolite to infants and children.

F. International Tolerances

There are no approved CODEX maximum residue levels (MRLs) established for residues of triazamate. MRLs have been established for vegetables at 0.05 ppm in Italy, for sugar beets at 0.05 ppm in the Czech Republic and 0.15 ppm in the U.K., for potatoes at 0.02 ppm in France, for cabbage at 0.1 ppm in Hungary, and for peas at 0.05 ppm in the Czech Republic and 0.02 ppm in Hungary and for green peas at 0.05 ppm in Hungary. (Mark Dow)

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ENVIRONMENTAL PROTECTION AGENCY

[FRL–6152–3]

Settlement Under Section 122(h) of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA); In the Matter of Agate Lake Scrap Yard, Nisswa, Minnesota

AGENCY: Environmental Protection Agency (EPA).

ACTION: Settlement of CERCLA section 107 Cost Recovery Matter.

SUMMARY: EPA is proposing to settle a cost recovery claim with two potentially responsible parties (PRPs) with regard to past costs at the Agate Lake Scrap Yard site (the Site) in Nisswa, Minnesota. The EPA is authorized under section 122(h) of the CERCLA to enter into this administrative settlement.

Response costs totaling $264,423 were incurred by EPA in connection with the remedial action at the Site. On July 25,