TABLE 2— REGISTRANTS REQUESTING AMENDMENTS TO DELETE USES IN CERTAIN PESTICIDE REGISTRATIONS—Continued

Company No.	Company Name and Address
6951	Kanoria Chemicals & Industries Ltd., c/o Jellinek, Schwartz & Connolly, Inc., 1525 Wilson Blvd., Suite 600, Arlington, VA 22209.

III. Existing Stocks Provisions

The Agency has authorized registrants to sell or distribute product under the previously approved labeling for a period of 18 months after approval of the revision, unless other restrictions have been imposed, as in special review actions.

List of Subjects

Environmental protection, Pesticides and pests, Product registrations.

Dated: September 10, 1998.

Linda A. Travers,

Director, Information Resources Services Division, Office of Pesticide Programs.

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ENVIRONMENTAL PROTECTION AGENCY

[PF–828; FRL–6023–7]

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–828, must be received on or before October 30, 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
Ann Sibold	Rm. 212, CM #2, 703–305–6502; e-mail: sibold.ann@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) of the (FFDCA) as amended by the Food Quality Protection Act (FQPA) of 1996 (Pub. L. 104–170); 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–828 (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–828) 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: September 19, 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. American Cyanamid Company

PP 8F4980

EPA has received a pesticide petition (PP 8F4980) from American Cyanamid Company, P.O. Box 400, Princeton, NJ 08543–0400, proposing pursuant to section 408(d) of the FFDCA 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of 4-bromo-2–(4-chlorophenyl)-1-(ethoxymethyl)-5-(trifluoromethyl)-1-pyrrole-3-carbonitrile, (chlorfenapyr) in or on the raw agricultural commodity milk, milk fat, meat, meat fat and meat byproducts at 0.01, 0.03, 0.01, 0.03, and 0.30 parts per million (ppm) respectively, derived from the use of chlorfenapyr ear tags on beef and dairy cattle. 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.* Although not relevant to this use pattern, the Agency has reviewed data submitted in support of pesticide petitions 5F4456, 5G4507, 5G453, 5G4548, and 5G4574 on the metabolism of chlorfenapyr in several plants and concluded that the nature of the residues of chlorfenapyr in plants is adequately understood and that the residue of concern consists of the parent molecule. The metabolic pathway of chlorfenapyr in the laying hen and the lactating goat was also similar to that in laboratory rats.

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 of the limit of detection of the designated method. The gas chromatography analytical methods, M2395.01 and M2398.01, which are proposed as the enforcement method for the residues of chlorfenapyr in milk and muscle/fat, respectively, each have an Limit of Quantification (LOQ) of 0.01 ppm and method M2405, which is proposed as the enforcement method for the residues of chlorfenapyr in liver/kidney tissues has an LOQ of 0.05 ppm. All methods have been validated at the EPA laboratories in Beltsville, MD.

3. *Magnitude of residues.* There is an extensive data base on chlorfenapyr that has been reviewed and accepted by the Agency. A residue depletion study was conducted to determine whether the application of two ear tags containing 30% chlorfenapyr to lactating dairy cattle would result in residues in milk, milk fat or edible tissues (muscle, liver, kidney, and fat). The results of this study indicate that the proposed tolerances for the residues of chlorfenapyr in milk, milk fat, meat, meat fat and meat by-products are more than adequate to cover any residues that may result from this use pattern.

B. Toxicological Profile

1. *Acute toxicity.* Based on the EPA's toxicity category criteria, the acute toxicity category for chlorfenapyr technical and the 3SC formulation is Category II or moderately toxic (signal word WARNING) and the acute toxicity category for the 2SC formulation is Category III or slightly toxic (signal word CAUTION). Males appear to be more sensitive to the effects of chlorfenapyr than females. The acute toxicity profile indicates that absorption by the oral route appears to be greater than by the dermal route. The following are the results from the acute toxicity tests conducted on the technical material:

Rat Oral LD_{50}: 441/1,152 milogram/kilogram body weight (mg/kg b.w.) Male/Female (M/F); Tox. Category II

Rabbit Dermal LD_{50}: >2,000 mg/kg b.w.(M/F); Tox. Category III

Acute Inhal. LC_{50}: 0.83/>2.7 mg/L (M/F); Tox. Category III

Eye Irritation: Moderately Irritating; Tox. Category III

Dermal Irritation: Non-Irritating; Tox. Category IV

Dermal Sensitization: Non-Sensitizer

Acute Neurotoxicity: No-Observed-Adverse-Effect-Level (NOAEL) 45 mg/kg b.w; Not An Acute Neurotoxicant

2. *Genotoxicty.* Chlorfenapyr technical (94.5% active ingredient (a.i.)) was examined in a battery of *in vitro* and *in vivo* tests to assess its genotoxicity and its potential for carcinogenicity. These tests are summarized below.

Microbial/Microsome Mutagenicity Assay: Non-mutagenic

Mammalian Cell Chinese Hamster Ovary (CHO)/HGPRT Mutagenicity Assay: Non-mutagenic

In Vivo Micronucleus Assay: Non-genotoxic

In Vitro Chromosome Aberration Assay in CHO: Non-clastogenic

In Vitro Chromosome Aberration Assay in CHLC: Non-clastogenic

Unscheduled DNA Synthesis (UDS) Assay: Non-genotoxic.

3. *Reproductive and developmental toxicity.* Chlorfenapyr is neither a reproductive nor a developmental toxicant and is not a teratogenic agent in the Sprague-Dawley rat or the New Zealand white rabbit. This is demonstrated by the results of the following studies:

Rat Oral Teratology: NOAEL for maternal toxicity 25 mg/kg b.w./day; NOAEL for fetal/develop. toxicity 225 mg/kg b.w./day

Rabbit Oral Teratology: NOAEL for maternal toxicity 5 mg/kg b.w./day

NOAEL for fetal/develop. toxicity 30 mg/kg b.w./day

Rat Two-Generation: NOAEL for parental toxicity /growth and Reproduction offspring development 60 ppm (5 mg/kg b.w./day); NOAEL for reproductive performance 600 ppm (44 mg/kg b.w./day).

4. *Subchronic toxicity.* The following are the results of the subchronic toxicity tests that have been conducted with chlorfenapyr:

28–Day Rabbit Dermal: NOAEL 100 mg/kg b.w./day

28–Day Rat Feeding: NOAEL <600 ppm (< 71.6 mg/kg b.w./day)

28–Day Mouse Feeding: NOAEL <160 ppm (<32 mg/kg b.w./day)

13–Week Rat Dietary: NOAEL 150 ppm (11.7 mg/kg b.w./day)

13–Week Mouse Dietary: NOAEL 40 ppm (8.2 mg/kg b.w./day)

13–Week Dog Dietary: NOAEL 120 ppm (4.2 mg/kg b.w./day).

5. *Chronic toxicity.* Chlorfenapyr is not oncogenic in either Sprague Dawley rats or CD–1 mice and is not likely to be carcinogenic in humans. The following are the results of the chronic toxicity tests that have been conducted with chlorfenapyr:

1–Year Neurotoxicity in Rats: NOAEL 60 ppm (2.6/3.4 mg/kg b.w./day M/F)

1–Year Dog Dietary: NOAEL 120 ppm (4.0/4.5 mg/kg b.w./day M/F)

24–Month Rat Dietary: NOAEL for Chronic Effects 60 ppm (2.9/3.6 mg/kg b.w./day M/F)

NOAEL for Oncogenic Effects 600 ppm (31/37 mg/kg b.w./day M/F)

18–Month Mouse Dietary: NOAEL for Chronic Effects 20 ppm (2.8/3.7 mg/kg b.w./day M/F)

NOAEL for Oncogenic Effects 240 ppm (34.5/44.5 mg/kg b.w./day M/F).

6. *Animal metabolism.* A metabolism study was conducted in Sprague-Dawley rats at approximately 20 and 200 mg/kg b.w using radiolabeled chlorfenapyr. Approximately 65% of the administered dose was eliminated during the first 24 hours (62% in feces and 3% in urine) and by 48 hours following dosing, approximately 85% of the dose had been excreted (80% in feces and 5% in urine). The absorbed chlorfenapyr-related residues were distributed throughout the body and detected in tissues and organs of all

treatment groups. The principal route of elimination was via feces, mainly as unchanged parent plus minor *N*-dealkylated, debrominated and hydroxylated oxidation products.

7. *Metabolite toxicology.* The parent molecule is the only moiety of toxicological significance which needs regulation in plant and animal commodities.

8. *Endocrine disruption.* Collective organ weights and histopathological findings from the two-generation rat reproduction study, as well as from the subchronic and chronic toxicity studies in two or more animal species, demonstrate no apparent estrogenic effects or effects on the endocrine system. There is no information available which suggests that chlorfenapyr would be associated with endocrine effects.

C. Aggregate Exposure

1. *Food.* For purposes of assessing the potential dietary exposure, a Theoretical Maximum Residue Contribution (TMRC) has been calculated from the proposed tolerance of chlorfenapyr in milk at 0.01 ppm, milk fat at 0.03 ppm, meat at 0.01 ppm, meat fat at 0.03 ppm and meat by-products at 0.30 ppm. As there are no other established U.S. permanent tolerances for chlorfenapyr, the only dietary exposure to residues of chlorfenapyr in or on food will be limited to residues in milk, milk fat, meat, meat fat and meat byproducts derived from cattle. The contribution of all these tolerances to the daily consumption will be insignificant for the overall U.S. population (utilizing only 0.23% of the reference dose (RfD) as well as all sensitive subpopulations including children aged 1–6 (0.52% of RfD utilized) and non-nursing infants (utilization of 0.47% of RfD).

2. *Drinking water.* There is no available information about chlorfenapyr exposures via levels in drinking water. There is no concern for exposure to residues of chlorfenapyr in drinking water because of this use pattern on ear tags. Moreover, because of its extremely low water solubility (120 parts per billion (ppb) at 25° C). Chlorfenapyr is also immobile in soil and does not leach because it is strongly adsorbed to all common soil types. In addition, the label explicitly prohibits applications near aquatic areas. There is a reasonable certainty that no harm will result from dietary exposure to chlorfenapyr, because dietary exposure to residues on food will use only a small fraction of the Reference Dose (RfD) (including exposure of sensitive subpopulations), and exposure through drinking water is expected to be insignificant.

3. *Non-dietary exposure.* Chlorfenapyr is currently not registered for use in residential indoor or outdoor uses. However, based on the physico-chemical characteristics of the compound, the proposed use pattern as an ear tag and available information concerning its environmental fate, non-dietary exposure is expected to be negligible. The vapor pressure of chlorfenapyr is 4.05×10^{-8} mm of mercury; therefore, the potential for non-occupational exposure by inhalation is insignificant. Moreover, the current proposed registration is for outdoor, terrestrial uses which severely limit the potential for non-occupational exposure.

D. Cumulative Effects

The pyrrole insecticides represent a new class of chemistry with a unique mechanism of action. The parent molecule, AC 303,630 is a pro-insecticide which is converted to the active form, CL 303,268, via rapid metabolism by mixed function oxidases (MFOs). The active form uncouples oxidative phosphorylation in the insect mitochondria by disrupting the proton gradient across the mitochondrial membrane. The production of Adenosine Triphosphate (ATP) is inhibited resulting in the cessation of all cellular functions. Because of this unique mechanism of action, it is highly unlikely that toxic effects produced by chlorfenapyr would be cumulative with those of any other pesticide chemical.

In mammals, there is a lower titer of MFOs, and chlorfenapyr is metabolized by different pathways (including dehalogenation, oxidation, and ring hydroxylation) to other polar metabolites without any significant accumulation of the potent uncoupler, CL 303,268. In the rat, appoximately 85% of the administered dose is excreted in the feces within 48 hours, thereby reducing the levels of AC 303,630 and CL 303,268 that are capable of reaching the mitochondria. This differential metabolism of AC 303,630 to CL 303,268 in insects versus to other polar metabolites in mammals is responsible for the selective insect toxicity of the pyrroles.

E. Safety Determination

1. *U.S. population.* The RfD of 0.03 mg/kg b.w./day for the residues of chlorfenapyr in milk, milk fat, meat, meat fat, and meat byproducts, is calculated by applying a 100–fold safety factor to the overall NOAEL of 3 mg/kg b.w./day. This NOAEL is based on the results of the chronic feeding studies in the rat and mouse and the 2–generation reproduction study in the rat (see *B. Toxicological Profile*). Therefore, the combined TMRC for the proposed chlorfenapyr tolerances in milk, milk fat, meat, meat fat and meat byproducts (0.0000681 mg/kg b.w./day) will utilize approximately 0.23% of the RfD for the general US population.

2. *Infants and children.* The TMRC in milk, milk fat, meat, meat fat and meat byproducts consumed by a non-nursing infant (<1 year of age) is 0.000141 mg/kg b.w./day. This will use 0.47% of the RfD for non-nursing infants. The TMRC for the proposed chlorfenapyr tolerances in milk, milk fat, meat, meat fat and meat byproducts consumed by a child 1–6 years of age is 0.000156 mg/kg b.w./day, which is less than 1% (actual 0.52%) of the RfD. Therefore, the results of the toxicology and metabolism studies support both the safety of chlorfenapyr to humans based on the intended use as cattle ear tag and the granting of the requested tolerances in milk, milk fat, meat, meat fat and meat by-products.

F. International Tolerances

Section 408 (b)(4) of the amended FFDCA requires EPA to determine whether a maximum residue level has been established for the pesticide chemical by the Codex Alimentarius Commission.

There is neither a Codex proposal, nor Canadian or Mexican tolerances/limits for residues of chlorfenapyr in meat and meat byproducts. Therefore, a compatibility issue is not relevant to the proposed tolerance. (Ann Sibold)

2. Rohm and Haas Company

PP 7F4894

EPA has received a pesticide petition (PP 7F4894) 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; ethyl (3-tert-butyl-1-dimethylcarbamoyl–1*H*–1,2,4-triazol-5-ylthio) acetate in or on the raw agricultural commodity apples at 0.1 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The metabolism of triazamate in plants (apples) is adequately understood for the purposes of this tolerance. The metabolism of triazamate involves hydrolysis of the ester and oxidative demethylation of the carbamoyl group. Parent compound is rapidly metabolized and is either not found or found at trace levels in pome fruit. The majority of the residue which may remain on the fruit is present as non-cholinesterase inhibiting metabolites whose structures do not contain the dimethylcarbamoyl moiety. The metabolism of triazamate in goats proceeds along the same metabolic pathway as observed in plants. Because apple pomace is not fed to poultry, there is no reasonable expectation that measurable residues of triazamate or any of its metabolites will occur in eggs, poultry meat or poultry meat by-products. The transfer of residues into milk and meat was minimal in the goat metabolism and the majority of the residue which was found in the milk and tissues was non-cholinesterase metabolites. Because of this low transfer rate and the low measurable residues present in apple pomace, there is no reasonable expectation of finding measurable residues of triazamate or any of its metabolites in milk, meat or meat by-products.

2. *Analytical method.* An analytical method using chemical derivitization followed by gas chromatography (GC) using Nitrogen-Phosphorous detection has been developed and validated for residues of triazamate and its cholinesterase-inhibiting metabolite (RH–0422) for pome fruit and processed apple fractions. For all matrices, the methods involve Soxhlet extraction of the residue from fruit samples with solvents, purification of the extracts by liquid-liquid partitioning, derivitization of the metabolite with diazomethane, and final purification of the residues using solid phase extraction column chromatography. The limit of quantitation (LOQ) of the methods is 0.01 ppm for pome fruit, apple juice, sauce and wet apple pomace.

3. *Magnitude of residues.* —i. *Acute risk.* An acute dietary risk assessment (Dietary Exposure Evaluation Model, Novigen Sciences Inc., 1997) was conducted for triazamate using two approaches: (1) a Tier 1 approach using a tolerance level residue of 0.10 ppm and (2) Monte Carlo simulations using an entire distribution of field trial residues for pome fruit and adjusted for percent crop treated (Tier 3). Using the Tier 1 approach margins of exposure (MOEs) at the 95th and 99th percentiles of exposure for the overall U.S. population were 572 and 199, respectively. Using the Tier 3 procedure in which residues were adjusted for percent crop treated, the MOEs for the 95th and 99th percentiles were 8,769 and 1,511, respectively. Acute exposure was also estimated for non-nursing infants, the most sensitive sub-population. For this population, MOEs at the 95th and 99th percentiles of exposure were 113 and 83, respectively. Using the Tier 3 method, MOEs were 909 and 396, respectively. Acute dietary risk is considered acceptable if the MOE is greater than 30, an appropriate safety factor when based on a human clinical study. Even under the conservative assumptions presented here, the more realistic estimates of dietary exposure (Tier 3 analyses) clearly demonstrate adequate MOEs up to the 99th percentile of exposure for all population subgroups.

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 level residue of 0.10 ppm assuming 100% of crop is treated and (2) using a tolerance level residue of 0.10 ppm adjusted for projected percent crop treated. The Theoretical Maximum Residue Contribution (TMRC) from the proposed pome fruit tolerance represents 0.91% of the RfD for the U.S. population as a whole. The subgroup with the greatest chronic exposure is non-nursing infants (less than 1 year old), for which the TMRC estimate represents 6.3% of the RfD. The chronic dietary risks from this use do not exceed EPA's level of concern.

B. Toxicological Profile

1. *Acute toxicity.* Triazamate is a moderately toxic cholinesterase inhibitor belonging to the carbamoyl triazole 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 0f > 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 No-Observed-Adverse-Effect-Level (NOAEL) 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.* Triazamate Technical is not a developmental or reproductive toxicant:

i. In a developmental toxicity study in rats with Triazamate Technical, the NOAEL for developmental toxicity was 64 mg/kg (highest dose tested). The NOAEL for maternal toxicity was 16 mg/kg based on clinical signs of cholinergic toxicity at 64 mg/kg.

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

iii. In a 2–generation reproduction study in rats with Triazamate Technical, the NOAEL for reproductive effects was 1,500 ppm (101 and 132 mg/kg/day for males and females, respectively; highest dose tested). The NOAEL 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).

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 NOAEL 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 NOAEL 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 NOAEL 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 NOAEL 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 NOAEL for blood cholinesterase inhibition was 2 ppm (0.4 and 0.5 mg/kg/day for males and females, respectively) based on based on decreases in plasma cholinesterase activity at 25 ppm (4 and 6 mg/kg/day for males and females, respectively). The NOAEL 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 NOAEL 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 NOAEL 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 NOAEL for 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*. 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.

i. 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; highest dose tested). The NOAEL 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 NOAEL 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).

ii. 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; highest dose tested). The NOAEL 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 NOAEL 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).

iii. In a chronic dietary toxicity study (12 months) in dogs with Triazamate Technical, the NOAEL 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 NOAEL 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).

6. *Animal metabolism*. The adsorption, 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 metabolite (RH–0422) is the only toxicologically significant metabolite, given that it contains the dimethylcarbamoyl 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) and animals (rat, goat). The metabolic pathway common to both plants and animals involves hydrolysis of the ester and oxidative demethylation of the carbamoyl group. Extensive degradation and elimination of polar metabolites occurs in animals such that residue are unlikely to accumulate in humans or animals exposed to these residues through the diet.

8. *Endocrine disruption*. The toxicology profile of 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*. Tolerances for residues of triazamate should be expressed as the total residue from triazamate [acetic acid, [(1-((dimethylamino) carbonyl)-3–(1,1-dimethylethyl)-1*H*–1,2,4-triazol-5-yl) thio]-, ethyl ester] and its cholinesterase inhibiting metabolite acetic acid, [(1-((dimethylamino) carbonyl)-3-(1,1-dimethylethyl)–1*H*–1,2,4-triazol-5-yl) thio]. No other tolerances currently exist for residues of triazamate on food crops.

i. *Acute risk*. An acute dietary risk assessment (Dietary Exposure Evaluation Model, Novigen Sciences Inc., 1997) was conducted for triazamate using two approaches: (a) A Tier 1 approach using a tolerance level residue of 0.10 ppm. (b) Monte Carlo simulations using an entire distribution of field trial residues for pome fruit and adjusted for percent crop treated (Tier 3).

Using the Tier 1 approach margins of exposure (MOEs) at the 95th and 99th percentiles of exposure for the overall U.S. population were 572 and 199, respectively. Using the Tier 3 procedure in which residues were adjusted for

percent crop treated, the MOEs for the 95th and 99th percentiles were 8,769 and 1,511, respectively. Acute exposure was also estimated for non-nursing infants, the most sensitive sub-population. For this population, MOEs at the 95th and 99th percentiles of exposure were 113 and 83, respectively. Using the Tier 3 method, MOEs were 909 and 396, respectively. Acute dietary risk is considered acceptable if the MOE is greater than 30, an appropriate safety factor when based on a human clinical study. Even under the conservative assumptions presented here, the more realistic estimates of dietary exposure (Tier 3 analyses) clearly demonstrate adequate MOEs up to the 99th percentile of exposure for all population subgroups.

ii. *Chronic risk*. Chronic dietary risk assessments (Dietary Exposure Evaluation Model, Novigen Sciences Inc., 1997) were conducted for triazamate using two approaches: (a) Using a tolerance level residue of 0.10 ppm assuming 100% of crop is treated and (b) Using a tolerance level residue of 0.10 ppm adjusted for projected percent crop treated. The Theoretical Maximum Residue Contribution (TMRC) from the proposed pome fruit tolerance represents 0.91% of the RfD for the U.S. population as a whole. The subgroup with the greatest chronic exposure is non-nursing infants (less than 1 year old), for which the TMRC estimate represents 6.3% of the RfD. The chronic dietary risks from this use do not exceed EPA's level of concern.

2. *Drinking water*. An additional potential source of dietary exposure to residues of pesticides are residues in drinking water. Pesticides may reach drinking water either by leaching to groundwater or by runoff to surface water. Both triazamate and its cholinesterase-inhibiting metabolite 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 could be found in ground or surface water when triazamate is applied according to label directions. The negligible potential for mobility was confirmed in four outdoor field dissipation studies and two outdoor lysimeter studies. There is no established Maximum Concentration Level (MCL) for residues of triazamate in drinking water. No drinking water health advisory levels have been established for triazamate. Significant exposure from cholinesterase-inhibiting residues of triazamate in drinking water is not anticipated.

3. *Non-dietary exposure*. Triazamate is not registered for either indoor or outdoor residential use. 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 appears to be structurally related to the carbamate class of insecticides which 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 there is no reliable data to indicate that the toxic effects caused by triazamate would be cumulative with those of any other compound, including carbamates. Based on these points, Rohm and Haas Company has considered only the potential risks of triazamate in it's exposure assessment.

E. Safety Determination

1. *U.S. population*. The acute and chronic dietary exposure to triazamate and its metabolite from the proposed use on pome fruit were evaluated. Exposure to triazamate and its toxicologically significant metabolite on pome fruit does not pose an unreasonable health risk to consumers including the sensitive subgroup non-nursing infants. In Tier 1 and Tier 3 acute analyses for the 95th percentile exposures, MOEs were greater than 100 for both the general U.S. population and non-nursing infants. Using the TMRC and assuming 100% of crop treated, the most conservative chronic approach), chronic dietary exposures represents 0.6% of the RfD for the U.S. population and 6.3% for non-nursing infants under 1 year old. 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 in *C. Aggregate Exposure* 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 and non-nursing infants.

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.

Developmental toxicity was not observed in developmental studies using rats and rabbits. The NOAEL 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 NOAEL was 101–132 mg/kg/day. These NOAELs are 10–fold or higher than those observed for systemic toxicity, i.e., cholinesterase inhibition. Rohm and Haas Company concludes that there is a reasonable certainty that no harm will occur to infants and children from aggregate exposure to residues of triazamate.

F. International Tolerances

There are no approved CODEX maximum residue levels (MRLs) established for residues of triazamate. (Mark Dow)

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ENVIRONMENTAL PROTECTION AGENCY

[PF–617A; FRL–6028–1]

EcoScience Corp; Withdrawal of Pesticide Petition

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the withdrawal of pesticide petition (PP) 4F4397 without prejudice to future filing.

FOR FURTHER INFORMATION CONTACT: By mail: Shanaz Bacchus, c/o Product Manager (PM) 90, Biopesticides and Pollution Prevention Division (7511C), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number and e-mail address: Rm. 902W34, CM #2, 1921 Jefferson Davis Highway, Arlington, VA 22202, (703) 308–8097, e-mail: bacchus.shanaz@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the **Federal Register** of February 8, 1995, 60