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Dated: December 14, 1998.

**L. Robert Lake,**

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## ENVIRONMENTAL PROTECTION AGENCY

## 40 CFR Part 180

**[OPP–300702; FRL–6024–5]**

**RIN 2070–AB78**

## Triazamate; Time-Limited Pesticide Tolerance

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Final rule.

**SUMMARY:** This regulation establishes a time-limited tolerance relative to an Experimental Use Permit for combined residues of triazamate (RH–7988) and its metabolite (RH–0422) in or on apples. Rohm and Haas Company requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act (FQPA) of 1996 (Pub. L. 104–170). The tolerance will expire on December 31, 2001.

**DATES:** This regulation is effective December 23, 1999. Objections and requests for hearings must be received by EPA on or before February 22, 1999.

**ADDRESSES:** Written objections and hearing requests, identified by the docket control number, [OPP–300702], must be submitted to: Hearing Clerk (1900), Environmental Protection Agency, Rm. M3708, 401 M St., SW., Washington, DC 20460. Fees accompanying objections and hearing requests shall be labeled ''Tolerance Petition Fees'' and forwarded to: EPA Headquarters Accounting Operations Branch, OPP (Tolerance Fees), P.O. Box 360277M, Pittsburgh, PA 15251. A copy of any objections and hearing requests filed with the Hearing Clerk identified by the docket control number, [OPP–300702], must also be submitted to: Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring a copy of objections and hearing requests to Rm. 119, Crystal Mall (CM) #2, 1921 Jefferson Davis Hwy., Arlington, VA.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically by sending electronic mail (e-mail) to: opp-docket@epamail.epa.gov. Copies of objections and hearing requests must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Copies of objections and hearing requests will also be accepted on disks in WordPerfect 5.1/6.1 file format or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket control number [OPP–300702]. No Confidential Business Information (CBI) should be submitted through e-mail. Electronic copies of objections and hearing requests on this rule may be filed online at many Federal Depository Libraries.

**FOR FURTHER INFORMATION CONTACT:** By mail: Mark Dow, Registration Division 7505C, Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number, and e-mail address: CM #2, 1921 Jefferson Davis Hwy., Arlington, VA, 703 305–5533, e-mail: dow.mark@epamail.epa.gov.

**SUPPLEMENTARY INFORMATION:** In the **Federal Register** of March 6, 1998 (63 FR 11240)(FRL–5777–5), EPA, issued a notice pursuant to section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(e) announcing the filing of a pesticide petition (PP) for tolerance by Rohm and Haas Company, 100 Independence Mall West, Philadelphia, PA 19108–2399. This notice included a summary of the petition prepared by Rohm and Haas Company, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR part 180 be amended by establishing a time-limited tolerance for combined residues of the insecticide triazamate (RH–7988) and its metabolite (RH–0422), in or on apples at 0.1 part per million (ppm). This tolerance will expire on December 31, 2001.

### I. Risk Assessment and Statutory Findings

New section 408(b)(2)(A)(i) of the FFDCA allows EPA to establish a tolerance (the legal limit for a pesticide chemical residue in or on a food) only if EPA determines that the tolerance is ''safe.'' Section 408(b)(2)(A)(ii) defines ''safe'' to mean that ''there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.'' This includes exposure through drinking water and in residential settings, but does not include occupational exposure. Section 408(b)(2)(C) requires EPA to give special consideration to exposure of infants and children to the pesticide chemical residue in establishing a tolerance and to ''ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residue. . . .''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. First, EPA determines the toxicity of pesticides based primarily on toxicological studies using laboratory animals. These studies address many adverse health effects, including (but not limited to) reproductive effects, developmental toxicity, toxicity to the nervous system, and carcinogenicity. Second, EPA examines exposure to the pesticide through the diet (e.g., food and drinking water) and through exposures that occur as a result of pesticide use in residential settings.

#### *A. Toxicity*

1. *Threshold and non-threshold effects.* For many animal studies, a dose response relationship can be determined, which provides a dose that causes adverse effects (threshold effects) and doses causing no observed effects (the ''no-observed effect level'' or ''NOEL'').

Once a study has been evaluated and the observed effects have been determined to be threshold effects, EPA generally divides the NOEL from the study with the lowest NOEL by an uncertainty factor (usually 100 or more) to determine the Reference Dose (RfD). The RfD is a level at or below which daily aggregate exposure over a lifetime will not pose appreciable risks to human health. An uncertainty factor (sometimes called a ''safety factor'') of 100 is commonly used since it is assumed that people may be up to 10 times more sensitive to pesticides than the test animals, and that one person or subgroup of the population (such as infants and children) could be up to 10 times more sensitive to a pesticide than another. In addition, EPA assesses the potential risks to infants and children based on the weight of the evidence of the toxicology studies and determines whether an additional uncertainty factor is warranted. Thus, an aggregate daily exposure to a pesticide residue at or below the RfD (expressed as 100% or less of the RfD) is generally considered acceptable by EPA. EPA generally uses

the RfD to evaluate the chronic risks posed by pesticide exposure. For shorter term risks, EPA calculates a margin of exposure (MOE) by dividing the estimated human exposure into the NOEL from the appropriate animal study. Commonly, EPA finds MOEs lower than 100 to be unacceptable. This 100-fold MOE is based on the same rationale as the 100-fold uncertainty factor.

Lifetime feeding studies in two species of laboratory animals are conducted to screen pesticides for cancer effects. When evidence of increased cancer is noted in these studies, the Agency conducts a weight of the evidence review of all relevant toxicological data including short-term and mutagenicity studies and structure activity relationship. Once a pesticide has been classified as a potential human carcinogen, different types of risk assessments (e.g., linear low dose extrapolations or MOE calculation based on the appropriate NOEL) will be carried out based on the nature of the carcinogenic response and the Agency's knowledge of its mode of action.

2. *Differences in toxic effect due to exposure duration.* The toxicological effects of a pesticide can vary with different exposure durations. EPA considers the entire toxicity data base, and based on the effects seen for different durations and routes of exposure, determines which risk assessments should be done to assure that the public is adequately protected from any pesticide exposure scenario. Both short and long durations of exposure are always considered. Typically, risk assessments include ''acute,'' ''short-term,'' ''intermediate term,'' and ''chronic'' risks. These assessments are defined by the Agency as follows.

Acute risk, by the Agency's definition, results from 1–day consumption of food and water, and reflects toxicity which could be expressed following a single oral exposure to the pesticide residues. High end exposure to food and water residues are typically assumed.

Short-term risk results from exposure to the pesticide for a period of 1–7 days, and therefore overlaps with the acute risk assessment. Historically, this risk assessment was intended to address primarily dermal and inhalation exposure which could result, for example, from residential pesticide applications. However, since enaction of FQPA, this assessment has been expanded to include both dietary and non-dietary sources of exposure, and will typically consider exposure from food, water, and residential uses when reliable data are available. In this assessment, risks from average food and water exposure, and high-end residential exposure, are aggregated. High-end exposures from all 3 sources are not typically added because of the very low probability of this occurring in most cases, and because the other conservative assumptions built into the assessment assure adequate protection of public health. However, for cases in which high-end exposure can reasonably be expected from multiple sources (e.g. frequent and widespread homeowner use in a specific geographical area), multiple high-end risks will be aggregated and presented as part of the comprehensive risk assessment/characterization. Since the toxicological endpoint considered in this assessment reflects exposure over a period of at least 7 days, an additional degree of conservatism is built into the assessment; i.e., the risk assessment nominally covers 1–7 days exposure, and the toxicological endpoint/NOEL is selected to be adequate for at least 7 days of exposure. (Toxicity results at lower levels when the dosing duration is increased.)

Intermediate-term risk results from exposure for 7 days to several months. This assessment is handled in a manner similar to the short-term risk assessment.

Chronic risk assessment describes risk which could result from several months to a lifetime of exposure. For this assessment, risks are aggregated considering average exposure from all sources for representative population subgroups including infants and children.

*B. Aggregate Exposure*

In examining aggregate exposure, FFDCA section 408 requires that EPA take into account available and reliable information concerning exposure from the pesticide residue in the food in question, residues in other foods for which there are tolerances, residues in groundwater or surface water that is consumed as drinking water, and other non-occupational exposures through pesticide use in gardens, lawns, or buildings (residential and other indoor uses). Dietary exposure to residues of a pesticide in a food commodity are estimated by multiplying the average daily consumption of the food forms of that commodity by the tolerance level or the anticipated pesticide residue level. The Theoretical Maximum Residue Contribution (TMRC) is an estimate of the level of residues consumed daily if each food item contained pesticide residues equal to the tolerance. In evaluating food exposures, EPA takes into account varying consumption patterns of major identifiable subgroups of consumers, including infants and children. The TMRC is a ''worst case'' estimate since it is based on the assumptions that food contains pesticide residues at the tolerance level and that 100% of the crop is treated by pesticides that have established tolerances. If the TMRC exceeds the RfD or poses a lifetime cancer risk that is greater than approximately one in a million, EPA attempts to derive a more accurate exposure estimate for the pesticide by evaluating additional types of information (anticipated residue data and/or percent of crop treated data) which show, generally, that pesticide residues in most foods when they are eaten are well below established tolerances.

Percent of crop treated estimates are derived from federal and private market survey data. Typically, a range of estimates are supplied and the upper end of this range is assumed for the exposure assessment. By using this upper end estimate of percent of crop treated, the Agency is reasonably certain that exposure is not understated for any significant subpopulation group. Further, regional consumption information is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups, to pesticide residues. For this pesticide, ''The chronic dietary risk (food only) for triazamate...does not exceed the Agency's level of concern.''

## II. Aggregate Risk Assessment and Determination of Safety

Consistent with section 408(b)(2)(D), EPA has reviewed the available scientific data and other relevant information in support of this action, EPA has sufficient data to assess the hazards of triazamate and to make a determination on aggregate exposure, consistent with section 408(b)(2), for a time-limited tolerance for combined residues of triazamate (RH–7988) and its metabolite (RH–0422) on apples at 0.1 ppm. EPA's assessment of the dietary exposures and risks associated with establishing the tolerance follows.

*A. Toxicological Profile*

EPA has evaluated the available toxicity data and considered its validity, completeness, and reliability as well as the relationship of the results of the studies to human risk. EPA has also considered available information concerning the variability of the sensitivities of major identifiable subgroups of consumers, including infants and children. The nature of the

toxic effects caused by triazamate are discussed below.

*B. Toxicological Endpoints*

1. *Acute toxicity.* The data base for acute toxicity is considered complete. No additional studies are required at this time. Acute toxicity categories for triazamate are: Acute oral and acute inhalation are toxicity category II; Acute dermal, Occular irritation and Dermal irritation are toxicity category IV; and Dermal sensitization is Not Applicable.

Triazamate produces significant toxicity via the oral and inhalation routes. In the acute oral studies in the rat and the mouse, the $LD_{50}$ values were less than 500 miligrams/kilograms (mg/kg). In the acute inhalation study in the rat, the $LC_{50}$ value was less than 0.5 miligram/liter (mg/L).

2. *Short - and intermediate - term toxicity.* The data base for subchronic toxicity is considered complete.

i. *Thirteen week dietary in rats.* In a subchronic toxicity study, RH–7988 was administered to 10 rats/sex/dose at dietary concentrations of 0, 50, 500, 1,500 or 3,000 ppm (mean measured concentrations of 0, 3, 31, 93 or 192 mg/kg/day for males and 0, 4, 39, 117 or 250 mg/kg/day for females) for 13 weeks. In conjunction with the primary study, 10 additional rats/sex were fed RH–7988 at 0, 50, 500, 1,500 or 3,000 ppm (mean measured concentrations of 0, 3, 31, 95 or 188 mg/kg/day for males and 0, 4, 39, 119 or 250 mg/kg/day for females) for 13 weeks to determine the effects of RH–7988 on cholinesterase activities.

In the primary study, body weights for the 1,500 and 3,000 ppm treatment groups were significantly ($p < 0.05$) depressed for most or all weekly intervals. Body weight gains for the 1,500 and 3,000 ppm treatment groups were 16–23% and 27–37% lower, respectively, than the controls at the end of the study. Decreased food consumption by the 1,500 ppm treatment groups was significant ($p < 0.05$) during the initial 3–4 weeks and at one or several later weekly intervals compared to the controls. The Lowest Observed Adverse Effect Level (LOAEL) for this study is 93.37 mg/kg/day (1,500 ppm) based on decreased body weights and decreased food consumption in both sexes. The NOEL is 31.45 mg/kg/day (500 ppm).

In the cholinesterase study, both sexes in the 500, 1,500, and 3,000 ppm treatment groups exhibited concentration-dependent decreases in red blood cell (12–41%) and plasma (58–95%) cholinesterase activities compared to the controls. Both sexes in the 1,500 and 3,000 ppm treatment groups had concentration-dependent decreases in brain cholinesterase activities (28–56%) compared to the controls. The LOAEL for this study is 30.96 mg/kg/day (500 ppm) based on decreased plasma cholinesterase activities in both sexes and decreased red blood cell cholinesterase activity in females. The NOEL is 3.09 mg/kg/day (50 ppm).

ii. *Subchronic oral toxicity in mice.* In a 3 month dietary toxicity study, Crl: CD–1 (ICR) BR mice (10 per group per sex) were exposed to triazamate at dose levels of 0, 0.5, 2, 25, 250 or 1,000 ppm (in males 0, 0.09, 0.34, 4.55, 49.75 and 159.43 mg/kg/day; in females 0, 0.13, 0.53, 6.56, 71.82, and 223.19 mg/kg/day). Compound related toxicity was observed at > 25 ppm as evidenced by cholinesterase inhibition in both sexes. Plasma cholinesterase levels were significantly decreased in a dose-dependent manner at 25 ppm in males (11–67% of control) and females (13–73% of control). At these same dose levels, red blood cell cholinesterase levels were significantly decreased in males (72–84% of controls) and in females (84–93% of controls). Brain cholinesterase levels were significantly decreased in males at 1,000 ppm (81% of controls). No other treatment related effects were observed.

Based on plasma cholinesterase inhibition at 25 ppm, the NOEL and LOAEL were 0.34 – 0.53 mg/kg and 4.55 – 6.56 mg/kg, respectively, for both males and females.

iii. *Subchronic dog (non-guideline) 14–day dietary.* In a non-guideline range-finding study, triazamate (99%) was administered to male beagles (4/dose) at dietary levels of 0, 140, 300 or 700 ppm (0, 5.16, 9.64 or 11.25 mg/kg/day) for a period of 2 weeks. Dose levels of 3,500 and 7,000 ppm were initiated but the 3,500 ppm was continued for only one week, with recovery on basal diet (2–week average dose: 8.75 mg/kg/day); animals receiving 7,000 ppm for one day only were fed basal diet for 6 days prior to use as test animals at the 300 ppm level.

There were no unscheduled deaths in this study. The most obvious toxic effect of triazamate is its inhibition of cholinesterase activity in plasma at very low doses (140 ppm, 48% of control; 300 ppm, 54% of control; 700 ppm 54% of control). Other significant effects observed at 140 ppm included only irregular feces. At 300 ppm and above, emesis was reported and decreases were observed in white blood cell count (86% control), alkaline phosphatase activity (67% control) and serum glutamic pyruvic transaminase (SGPT) activity (58% control). Numerous incidences of no fecal output were observed at 70 ppm and above.

From the data presented in this 2–week study, the NOEL for triazamate is < 140 ppm (5.16 mg/kg/day) based on inhibition of plasma cholinesterase and irregular feces (diarrhea, soft stool, mucoid feces, no fecal output). The LOAEL is start ≤ 140 ppm.

iv. *Subchronic oral toxicity-13–week dog.* In a subchronic toxicity study, triazamate (95.3%) was administered to beagle dogs (4/sex/dose) in the diet at dose levels of 0, 1, 10, 100 or 400 ppm (0, 0.03, 0.31, 3.11 or 10.98 mg/kg/day for a period of 13 weeks.

No treatment related clinical signs were observed in the 1 ppm that were related to treatment. In the 10 ppm group, food-like vomitus was observed in 2/4 males. In the 100 ppm, the same observation was made in 2/4 males and 2/4 females. Other observations included fluid vomitus in 1/4 females, bloated abdomen in 1/4 males, 1/4 females was considered thin and 1/4 females had decreased total blood protein (88% control).

Triazamate greatly inhibited the cholinesterase activity in blood plasma at all dose levels but did not appear to do so in red blood cells or brain. No NOEL was established for cholinesterase inhibition.

The LOAEL for inhibition of plasma cholinesterase inhibition was less than 1 ppm (0.03 mg/kg/day) based on inhibition of plasma cholinesterase activity (74% of control) in females receiving this dose level.

The NOEL for systemic effects is 10 ppm (0.31 mg/kg/day) based on vomiting in both sexes, thin appearance in (1/4 females) and bloated abdomen in 1/4 males.

The study satisfied the requirements for a subchronic nonrodent study and is acceptable.

v. *21–day dermal - rat.* In a 21–day dermal study groups of Crl:CD BR rats (6/sex/dose) received 15 repeated dermal applications of triazamate (97%, technical) at doses of 0, 10, 100 and 1,000 mg/kg, 6 hours/day, 5 days /week over a three week period. An other group of 6 male and 6 female rats received repeated dermal applications of a formulation product (50WP, 52% active ingredient (a.i.)) at a dose equivalent to 10 mg a.i./kg/day. Under the conditions of this study, there were no treatment-related clinical signs of toxicity for either product. At 10 mg/kg, there was a biologically significant decrease in plasma cholinesterase for both the technical (females only) and 50WP formulations (both sexes). At 100 mg/kg and at 1,000 mg/kg, there was a statistically significant decrease in

plasma, red cell and brain cholinesterase when compared to controls. At 100 mg/kg, the plasma cholinesterase activity was 50% and 58% of control values for females and males, respectively. The red cell cholinesterase activity was 67% in females and 72% in males and the brain cholinesterase activity was 87% of control activity in both sexes. At 1,000 mg/kg, Plasma cholinesterase activity was 25% in females, and 19% of controls in males; red cell activity was 67% of controls in females and 72% of controls in males and brain cholinesterase activity was 47% in females and 42% in males. Based on the results of this study, for systemic toxicity, the LOAEL was 10 mg/kg based on the biologically significant decreases in plasma cholinesterase activity; a NOEL was not established.

The study satisfied the requirements for a 21–day dermal rat study and is acceptable.

3. *Chronic toxicity*— i. *Oncogenicity*. EPA has established the RfD for triazamate at 0.000164 (0.0002 rounded off) milligrams/kilogram/day (mg/kg/day). This Reference Dose (RfD) is based on a NOEL of 0.0164 mg/kg/day and an uncertainty factor of 100; NOEL established from a combined chronic feeding study in the dog; LOAEL = 0.0236 mg/kg/day.

The data base for chronic toxicity and oncogenicity is considered complete.

a. *Chronic nonrodent - 1 year dog.* In a chronic toxicity study triazamate (94.9%) was administered to purebred beagle dogs (4/sex/dose)in the diet at dose levels of 0, 0.1, 0.3, 0.6, 0.9, 15.0 or 150 ppm (corresponding to 0, 0.0025, 0.0078, 0.0164, 0.0236, 0.3904, or 4.42 mg/kg/day) for 52 weeks.

The most significant effect observed was inhibition of plasma, red blood cell and brain cholinesterase activity. Decreases in activity were reported at several dose levels. Plasma cholinesterase activity was decreased (9 to 87% of control value) in both sexes at the two highest dose levels. At 150 ppm red blood cell cholinesterase activity was decreased (64 to 82%) of control values. This finding was not reported at doses equal to and lower than 15 ppm. Brain cholinesterase activity was significantly decreased (53 to 80% of controls) at both the 15 and 150 ppm levels, but statistical significance was only reported for females in the 150 ppm group. Brain cholinesterase activity was decreased (88% of control) for males in the 0.9 ppm group. This decrease in activity is considered biologically significant since the reported decrease is greater than 10% of the control value. Brain cholinesterase inhibition was not observed in animals receiving triazamate at dose levels lower than 0.9 ppm.

The NOEL for cholinesterase inhibition was 0.6 ppm (0.0164 mg/kg/day) based on the inhibition of brain cholinesterase activity (88% of control value) in males at the LOAEL of 0.9 ppm triazamate in the diet (0.0236 mg/kg/day).

No biologically significant treatment related effects were noted with respect to mortality, clinical signs, body weight, food consumption, food efficiency, hematology, clinical chemistry, urinalysis, organ weights, organ/body weight ratios, organ/brain weight ratios, or gross or microscopic pathology. The NOEL for systemic effects is ≧ 150 ppm (4.42 mg/kg/day); the LOAEL is > 150 ppm.

The study is acceptable and satisfies the requirement for a chronic oral non-rodent study.

b. *Chronic oral toxicity/oncogenicity in mice.* In a 78 week oral toxicity/oncogenicity study in mice, groups of 60 CD-1 mice/sex were fed dietary levels of 0, 1, 50, or 1,500 ppm triazamate (equivalent to 0, .13, 6.7, or 210 mg/kg/day for females and 0, 0.17, 8.4 or 262 mg/kg/day for males. At week 55, the highest dose levels were reduced to 1,000 ppm(127 mg/kg and 146 mg/kg for males and females, respectively) due to high mortality. Groups of 10/sex/dose level were included for sacrifice at 12 months.

At 50 ppm, plasma cholinesterase activity was decreased in males (64 to 75%) and in females (69 to 80%) at 6, 12, or 18 months. At the high dose of 1,000/1,500 ppm, a significantly decreased survival rate and a debilitated state of health were observed during the first 12 months in both sexes. Body weight gains overall were depressed compared to controls in males and females (16%), food consumption was slightly decreased in males and marginal decreases in erythrocyte parameters (RBC, HGB and HCT) were observed at 12 and 18 months in males. An increase in the incidence of inhalation pneumonia was observed in both sexes. Inhibition of erythrocyte and brain cholinesterase activity was also observed at 1,000 ppm.

The Lowest Effect Level (LEL) for cholinesterase inhibition is 50 ppm (6.7 and 8.4 mg/kg/day in males and females, respectively) based on plasma cholinesterase activity. The NOEL is for cholinesterase inhibition is 1 ppm (0.13 and 0.17 mg/kg/day, in males and females respectively).

The systemic LEL is 1,000 ppm (127 and 146 mg/kg/day , males and females, respectively) based on decreased body weight gains and inhalation pneumonia. The systemic NOEL was 50 ppm.

There is no evidence of carcinogenic potential. Dosing was excessive at the highest dose (1,000/1,500 ppm) but sufficient numbers of mice were considered available at termination to assess the carcinogenicity at the highest dose. The study is Core Guideline for carcinogenicity and satisfies the requirement for an oncogenicity study in mice as per 83–2(b). For chronic toxicity, the study is core supplementary. No ophthalmoscopic examinations or clinical chemistry determinations were performed, other than for inhibition of cholinesterase activity.

c. *Chronic/carcinogenicity study - rats.* In a combined chronic/oncogenicity study, RH–7988 was administered to 70 Sprague-Dawley rats/sex/dose in the diet at dose levels of 0, 10, 250, or 1,250 ppm (0, 0.45, 11.50, and 59.18 mg/kg/day for males, and 0.58, 14.54, and 73.70 mg/kg/day for females) for 24 months. A total of 10 rats/sex/group were terminated at 12 months and all remaining animals were sacrificed at 24 months of the study.

Chronic toxicity in rats receiving the 1,250 ppm diet was characterized in males by significant decreases in mean body weights (decrease 5–7%; p ≧ 0.05) and body weight gains ( 8–18%; p ≧ 0.05) and by reduced plasma (decrease 71–87%; p ≧ 0.05), erythrocyte (decrease 37–62%; p ≧ 0.05), and brain cholinesterase activities (decrease 26–38%; p ≧ 0.05) in both males and females. In the 250 ppm group animals, reduced plasma (decrease 31–65%; p ≧ 0.05) and erythrocyte (decrease 16–29%; p ≧ 0.05) cholinesterase activities were also observed.

The chronic LOAEL is 250 ppm (11.50 and 14.54 mg/kg/day in males and females, respectively) based on inhibition of plasma and erythrocyte cholinesterase activities in the 250 ppm animals. The chronic NOEL is 10 ppm (0.45 and 0.58 mg/kg/day for males and females, respectively).

Under the conditions of this study, there was no evidence of carcinogenic potential.

Dosing was considered adequate based on decreased body weight and body weight gain in the high-dose males and decreased activity of plasma and Red Blood Cell (RBC) cholinesterase at the mid and high doses and brain cholinesterase at the high dose.

This study is classified as acceptable and satisfies the guideline requirements for a chronic toxicity study (Series 83–1) and a carcinogenicity study (Series 83–2) on the rat.

ii. *Developmental toxicity.* The data base for developmental toxicity is considered complete.

a. *Oral (gavage) developmental toxicity study - rats.* In a developmental toxicity study, RH–7988 (95.7% a.i.) was administered to 25 Crl:CD Br rats/dose by gavage in a corn oil suspension at dose levels of 0, 4, 16, or 64 mg/kg/day from days 6 through 15 of gestation.

Maternal toxicity was demonstrated at 64 mg/kg/day by treatment-related clinical signs of toxicity and decreased body weights (days 8, 10, 13, 16, and 20, decrease 5–6%, $p > 0.05$), body weight gains (overall treatment period, decrease 25%, $p > 0.05$), and feed consumption (decrease 25 and 12%, $p > 0.05$, days 6–10 and 10–16, respectively). Clinical signs of toxicity noted during the treatment period in the high-dose group included fasciculations, salivation, rapid breathing, diarrhea, mucoid feces, tan stained perineum, and red stained nose. Body weights, body weight gains, feed consumption, and clinical signs of toxicity were unaffected by treatment at dose levels of 4 and 16 mg/kg/day. Cesarean section parameters were similar between the controls and all treated groups. No treatment-related changes were noted in mortality or gross pathology at any dose level. The maternal LOAEL is 64 mg/kg/day, based on treatment-related clinical signs of toxicity and decreased body weights, body weight gains, and feed consumption. The maternal NOEL is 16 mg/kg/day.

There were no treatment-related effects in developmental parameters at any administered dose level. The developmental LOAEL was not observed. The developmental NOEL is 64 mg/kg/day.

b. *Developmental toxicity - rabbits.* In a developmental toxicity study, 21 New Zealand White rabbits per group received RH–7988 (triazamate, 94.9%) by gavage on gestational days 7–19 at dose levels of 0, 0.05, 0.5 or 10 mg/kg/day. Corn oil served as the control substance and vehicle for the test article. The study authors did not indicate if doses were adjusted for concentration of active ingredient. Analytical chemistry results demonstrated that the lowest dose was 136% of target, i.e. 0.068 mg/kg/day.

Maternal toxicity was observed at 10 and 0.5 mg/kg/day as evidenced by increased incidences of clinical signs (soiled perineum, diarrhea and scant/no feces), significantly decreased body weight gain and food consumption during the entire gestational period. Based on these results, the maternal toxicity NOEL is 0.068 mg/kg/day and the maternal toxicity LOAEL is 0.5 mg/kg/day.

Developmental toxicity was not observed in this study, therefore, the developmental NOEL was 10 mg/kg, the developmental LOAEL was not determined.

iii. *Reproductive toxicity* The data base for reproductive toxicity is considered complete.

*Two generation reproduction study in rats.* In a two-generation reproduction study, Crl: CDBR rats (25/group) received RH–7988 (triazamate, 94.9%) at dietary levels of 0, 10, 250, or 1,500 ppm (equal to 0, 0.8, 19.9 or 116.8 mg/kg/day for females and 0, 0.7, 17.0, or 101.4 mg/kg/day for males) during premating.

The NOEL for systemic toxicity was 10 ppm. The LOAEL was 250 ppm based on decreased red blood cell and plasma cholinesterase activity in males and females in both generations.

At 250 ppm, plasma cholinesterase activity was 25 to 38% of control value and at 1,500 ppm the plasma cholinesterase activity was 6 to 13% of control level. Red blood cell activity was 65 to 80% of control at 250 ppm and 53–57% of control at 1,500 ppm. Additional findings at 1,500 ppm included decreased body weight ($F_0$ males, $F_1$ males and $F_1$ females), decreased food consumption ($F_0$ males, $F_1$ males and $F_1$ females) and an increased incidence of clinical signs (soft feces, small irregular shaped feces) in males in the $F_0$ and both sexes in the $F_1$.

The NOEL for reproductive toxicity was 250 ppm (17 – 19.9 mg/kg). The LOAEL was 1,500 ppm (101.4 – 116.8 mg/kg) based on decreased pup body weight on lactation days 14 and 21 in both generations.

iv. *Neurotoxicity.* Adequacy of data base for neurotoxicity (Series 81–8, 82–5): This chemical is not an OP and hen studies were not performed or required. Because of the cholinesterase inhibiting properties of the compound, acute and subchronic neurotoxicity studies were conducted. The data base for neurotoxicity is considered to be complete. No additional studies are required at this time.

In an Acute neurotoxicity study, RH–7988 was administered to Crl CD:BR rats of both sexes (10/sex/dose) by gavage at single doses of 0, 5, 25 or 75 mg/kg. There was no neuropathology reported on brain, spinal cord (and ganglia) and peripheral nerves. There were no treatment related mortalities. Cholinesterase activity was not assessed.

Based on study results the NOEL is 5 mg/kg. A threshold NOEL could be considered at 25 mg/kg due to the marginal effects observed in males, only at that dose level. This guideline [Series 81–8] acute neurotoxicity study is not yet classified because a formal review has not yet been done. The NOEL and LOAEL are tentative at this time.

In a Subchronic neurotoxicity study RH7988 was administered to Crl CD:BR (Sprague-Dawley) rats of both sexes at dietary levels of 0, 10, 250 or 1,500 ppm (0, 0.6, 14.3 or 86.8 mg/kg/day, respectively for males and 0, 0.7, 17.1 or 103.5 mg/kg/day for females). There was no effect on motor activity when dosed groups were compared to controls and no treatment related deaths were reported. Necropsy and histopathology did not reveal any lesions that could be correlated to treatment with the test material. Brain weights were comparable between groups.

Based on the results reported, the NOEL is 10 ppm (0.6/ 0.7 mg/kg/day[M/F]). The LOAEL is 250 ppm (14.3/17.1 mg/kg/day[M/F]) based on statistically and biologically significant decreases in plasma and red blood cell cholinesterase activity. This guideline [Series 82–5] subchronic neurotoxicity study is not yet classified because a formal review has not yet been done. The NOEL and LOAEL are tentative at this time.

v. *Mutagenicity.* The data base for Mutagenicity is considered adequate.

vi. *Metabolism.* The data base for metabolism is considered to be complete.

Groups of male and female Wistar rats were dosed with $^{14}C$-labeled RH–7988 at oral doses of 0.3 or 30 mg/kg and at 14–day repeated oral doses of RH–7988 at 3 ppm followed by a single oral dose of $^{14}C$–RH–7988 at 0.3 mg/kg. In addition, groups of rats were subjected to dietary administration of $^{14}C$–RH–7988 at 300 ppm (males only) and 3,000 ppm (females only). The excretion of radioactivity into urine and feces was rapid and complete in all groups tested and most of the test compound administered was excreted in the urine (67–109%) and feces (10–33%) from the animals. Total recovery of radioactivity ranged between 101% and 128% of the administered dose for all tested groups within 3 to 4 days after dosing. No marked sex-related difference was observed in the excretion patterns.

Peak plasma/whole blood $^{14}C$-concentration was attained 5–15 minutes after oral dosing (0.3 or 30 mg/kg/day) and 12–24 hours after dietary administration (300 ppm or 3,000 ppm).

At 3 days after oral administration of a low-dose (0.3 mg/kg, single or 14–day repeated dosing) or single high-dose (30 mg/kg), 0.6–4% of the administered

radioactivity remained in the tissues (0.1–0.2%) and carcass (0.4–4%). There were no sex-dependent differences in retention or distribution of the test article. The greatest amount of radioactivity (expressed as percent of the administered dose) was associated with the fat, liver, and muscle. At 3 days after oral administration of a single low- or high-dose of RH–7988, thyroid contained the highest tissue $^{14}$C-concentration (expressed as ppm equivalent/tissue).

High Pressure Liquid Chromatography (HPLC) analysis of urine and feces from rats after oral administration of 30 mg/kg of $^{14}$C–RH–7988 showed four $^{14}$C containing metabolites. Parent was not detected in any sample analyzed. The urine contained Metabolite 1 (35.5–49.4% of the dose), Metabolite 2 (9.5–13.7%), Metabolite 3 (0.9–2.7%) and a trace of Metabolite 4. The feces contained only Metabolite 1 (16.7–19.8%) and a trace of Metabolite 4. Most of the metabolites are cleavage products of RH–7988 either at the carbamoyl functionality or at the ester. The authors provided a proposed metabolic pathway that is consistent with the available data.

vii. *Dermal absorption* . In a dermal absorption study $^{14}$C triazamate was administered to male Crl:CDBR rats at a single dermal application at 0.5, 0.05 or 0.005 mg/centimeter (cm). The fur was removed from the intrascapular region of the back 24 hours prior to the administration of the test material. Dermal absorption at the highest concentration was less than 2% at 1, 10 and 24 hours. At the mid concentration, the dermal absorption ranged from less than 1% at 1 hour to approximately 13% after 24 hours. At the lowest concentration of 0.005 mg/cm, the highest percentage of absorption (19%) was reported at 24 hours; at 1 hour, the absorption was less than 1%.

Dermal Absorption Factor: A dermal absorption factor of 10% should be used for correcting oral dosing to dermal dosing.

*C. Exposures and Risks*

1. *From food and feed uses.* Risk assessments were conducted by EPA to assess dietary exposures and risks from RH–7988 and RH–0422 as follows:

i. *Acute dietary 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.

For assessment of acute dietary risk, a maternal NOEL of 0.068 mg/kg/day is used from a developmental toxicity study on rabbits. The selected endpoint is based on clinical signs and decreases in body weight gain and food consumption at a dose level of 0.5 mg/kg/day.

The Margin of Exposure (MOE) is a measure of how closely the anticipated exposure comes to the NOEL and is calculated as a ratio of the NOEL to the exposure (NOEL/exposure = MOE). The Agency is not generally concerned unless the MOE is below 100 when the NOEL is based upon data generated in animal studies. The 100 factor is to take into account interspecies extrapolation and intraspecies variability. For triazamate, the Agency's level of concern is for MOEs that are below 100.

A dietary risk evaluation system (DRES) analysis assuming 100% crop treated and using the proposed tolerance level of 0.05 ppm for apples and average residue concentrations from field trial data for apple juice was conducted. Average residues for apple juice were derived The resulting MOEs for triazamate are summarized below.

| Subgroup | NOEL mg/kg/day | MOE |
|---|---|---|
| General U.S. Population | 0.068 | 68 |
| Infants (< 1 yr) | 0.068 | 34 |
| Children (1-6 yrs) | 0.068 | 45 |
| Females (13+ yrs) | 0.068 | 226 |
| Males (13+ yrs) | 0.068 | 226 |

As shown above, the MOEs for adult males and females are greater than 100 and MOEs for the subgroups General U.S. Population, Infants (< 1 year), and Children (1–6 years old) are below 100. However, the Agency determined that in reality, the MOEs will be above a level of concern (>100) because of the following factors: 1) While the DRES analysis assumes 100% crop treated, less than 5% of the crop is ''actually'' treated with triazamate; 2) the acreage treated is approximately 3,000 acres, in 20 states over a 2–year period; 3) the field trial data show non-detectable residue levels ( < 0.01 ppm) after a post-treatment interval of 21 days; and 4) the unlikely leaching of this chemical due to its physical and chemical properties.

ii. *Chronic exposure and risk.* (Anticipated Residue Contribution or ARC) The chronic dietary exposure analysis was conducted using a RfD of 0.0002 mg/kg/day. The RfD is based on the NOEL for cholinesterase inhibition of 0.0164 mg/kg/day in male dogs from the chronic toxicity study in beagle dogs and an uncertainty factor of 100, applicable to all population subgroups.

In conducting this chronic dietary risk assessment, EPA is assuming that triazamate will be applied under the experimental use permit directions for use: 2,107.5 lbs ai to be applied on 2,810 acres over a 2–year period. Under these assumptions, the crop may contain triazamate residues when approximately 1% of the crop are treated. Anticipated residue values of 0.05 ppm derived from field trial data were used. There are no other published, pending, or section 18 tolerances for triazamate.

The resulting ARCs are equivalent to the following percents of the RfD for the subgroups listed below.

| Subgroup | %Rfd |
|---|---|
| U.S. Population (48 states) | 0.045% |
| Northeast Region | 0.056% |
| Western Region | 0.054% |
| Hispanics | 0.048% |
| Non-Hispanic Whites | 0.047% |
| Non-Hispanic Others | 0.047% |
| Nursing Infants (< 1 yr) | 0.329% |
| Non-Nursing Infants (< 1yr) | 0.0442% |
| Children (1–6 yrs) | 0.034% |
| Children (7–12 yrs) | 0.060% |

The subgroups listed above are: (1) the U.S. population (48 states); (2) those for infants and children; and (3) the other subgroups for which the percentage of the RfD occupied is greater than that occupied by the subgroup U.S. population (48) states.

The chronic dietary risk (food only) for triazamate therefore, does not exceed the Agency's level of concern.

*2. Drinking water risk (acute and chronic).* Drinking water levels of concern (DWLOC) are the concentrations of triazamate in drinking water which would result in unacceptable aggregate risk, after factoring in all food exposures and other non-occupational for which the Agency has reliable data. To calculate the DWLOC for acute exposure relative to an acute dietary toxicity endpoint, the acute dietary food exposure is subtracted from the ratio of the acute NOEL (used for acute dietary assessments) to the MOE.

However, for triazamate, the acute DWLOC could not be calculated because this ratio is less than the food exposure.

To calculate the DWLOC for chronic (non-cancer) exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure (from DRES) is subtracted from the RfD to obtain the acceptable chronic (non-cancer) exposure to triazamate in drinking water. DWLOCs were then calculated using default body weights and drinking water consumption figures.

The DWLOCs for triazamate are 6.97 μ/L for adults and 1.99 μ/L for children (1–6 years old) which are higher than the estimated average concentrations for triazamate in surface (0.25 μ/L) and ground water (0.000063 μ/L). Therefore, for the use proposed in this action, the Agency concludes with reasonable certainty that residues of triazamate in drinking water would not result in unacceptable levels of aggregate health risk at this time.

*D. Statement of the Adequacy of the Residential Exposure Data- base to Assess Infants' and Children's Exposures*

There are no residential uses associated with this product, therefore exposures and risks for children from such uses are not a concern.

*E. Cumulative Exposure to Substances with Common Mechanism of Toxicity*

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

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

EPA does not have, at this time, available data to determine whether triazamate 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, triazamate does not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that triazamate has a common mechanism of toxicity with other substances.

*F. Aggregate Exposure and Risk Assessment/Characterization*

1. *Acute aggregate exposure and risk.* As indicated from the acute dietary (food only) risk assessment, a high-end exposure estimate was calculated for these subgroups: general U.S. population, infants (< 1 year old), children (1–6 years old), females 13+ years, and males 13+.

Three of the population subgroups, general U.S. population, infants (<1 year old) and children (1–6 years old), yielded MOEs below 100%. However, given the limited acreage proposed for use in this action, the low percent crop actually treated, and the physical and chemical properties of this chemical (e.g.,it does not leach, is not persistent, degrades rapidly, etc.), and based on best scientific judgement, the Agency concludes with reasonable confidence that residues of triazamate in drinking water will not contribute significantly to the aggregate acute human health risk when considering the use proposed by this action.

2. *Short- and intermediate- term aggregate exposure and risk.* Triazamate is not currently registered for any residential uses. Therefore, a risk assessment for short- and intermediate-term aggregate risk is not required.

3. *Chronic aggregate exposure and risk.* For the U.S. population, 0.045% of the RfD is occupied by dietary (food) exposure. Triazamate is not currently registered for residential uses, thus, no chronic residential exposure is anticipated. The estimated average concentrations (EECs) of triazamate for the U.S. population and for children (1–6 years old) in surface and ground water are less than OPP's levels of concern for triazamate in drinking water as a contribution to chronic aggregate exposure when considering the use proposed by this action.

4. *Determination of safety (U.S. population, infants, and children).* Triazamate has been classified as a ''not likely'' human carcinogen, based on a lack of evidence of carcinogenicity in mice and rats at dose levels judged to be adequate to assess the carcinogenic potential. Thus, a cancer risk assessment is not required. Triazamate does not have residential uses; therefore, no residential risk assessment is required.

Acute dietary (food + water) risk estimates do exceed the Agency's level of concern for the U.S. population and for infants and children. Chronic dietary (food + water) risk for the U.S. population and for infants and children do not exceed the Agency's level of concern. However, given the limited acreage proposed for use in this action, the low percent crop actually treated, and the physical and chemical properties of this chemical (e.g., it does not leach, is not persistent, degrades rapidly, etc.), and based on best scientific judgement, the Agency concludes with reasonable confidence that residues of triazamate in drinking

water will not contribute significantly to the aggregate acute and chronic human health risk when considering the use proposed by this action.

## III. Other Considerations

*Endocrine Disruption.* EPA is required to develop a screening program to determine whether certain substances (including all pesticides and inerts) ''may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other endocrine effect....'' The Agency is currently working with interested stakeholders, including other government agencies, public interest groups, industry and research scientists in developing a screening and testing program and a priority setting scheme to implement this program. Congress has allowed 3 years from the passage of FQPA (August 3, 1999) to implement this program. At that time, EPA may require further testing of this active ingredient and end use products for endrocrine disrupter effects.

## IV. International Tolerances

There are no approved CODEX maximum residue levels (MRLs) established for residues of triazamate. No previous Experimental Use Permits have been requested for triazamate and no permanent or temporary tolerances have been established for residues of triazamate or its metabolites in/on raw agricultural or animal commodities.

## V. Analytical Method

Nitrogen phosphorus detector/gas liquid chromatography (NPD/GLC) (Method TR–34–89–37) has been submitted and validated.

## VI. Conclusion

Therefore, the tolerance is established for combined residues of triazamate (RH–7988) and its metabolite (RH–0422) in or on apples at 0.1 ppm.

## VII. Objections and Hearing Requests

The new FFDCA section 408(g) provides essentially the same process for persons to ''object'' to a tolerance regulation issued by EPA under new section 408(e) and (l)(6) as was provided in the old section 408 and in section 409. However, the period for filing objections is 60 days, rather than 30 days. EPA currently has procedural regulations which govern the submission of objections and hearing requests. These regulations will require some modification to reflect the new law. However, until those modifications can be made, EPA will continue to use those procedural regulations with appropriate adjustments to reflect the new law.

Any person may, by February 22, 1999, file written objections to any aspect of this regulation and may also request a hearing on those objections. Objections and hearing requests must be filed with the Hearing Clerk, at the address given above (40 CFR 178.20). A copy of the objections and/or hearing requests filed with the Hearing Clerk should be submitted to the OPP docket for this rulemaking. The objections submitted must specify the provisions of the regulation deemed objectionable and the grounds for the objections (40 CFR 178.25). Each objection must be accompanied by the fee prescribed by 40 CFR 180.33(i). If a hearing is requested, the objections must include a statement of the factual issues on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the requestor (40 CFR 178.27). A request for a hearing will be granted if the Administrator determines that the material submitted shows the following: There is genuine and substantial issue of fact; there is a reasonable possibility that available evidence identified by the requestor would, if established, resolve one or more of such issues in favor of the requestor, taking into account uncontested claims or facts to the contrary; and resolution of the factual issues in the manner sought by the requestor would be adequate to justify the action requested (40 CFR 178.32). Information submitted in connection with an objection or hearing request may be claimed confidential 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. A copy of the information 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.

## VIII. Public Record and Electronic Submissions

EPA has established a record for this rulemaking under docket control number [OPP–300702] (including any comments and data submitted electronically). 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 public record is located in Rm. 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Electronic comments may 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.

The official record for this rulemaking, as well as the public version, as described above will be kept in paper form. Accordingly, EPA will transfer any copies of objections and hearing requests received electronically into printed, paper form as they are received and will place the paper copies in the official rulemaking record which will also include all comments submitted directly in writing. The official rulemaking record is the paper record maintained at the Virginia address in ''ADDRESSES'' at the beginning of this document.

## IX. Regulatory Assessment Requirements

### *A. Other Acts and Executive Orders*

This final rule establishes a tolerance under FFDCA section 408(d) in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled Regulatory Planning and Review (58 FR 51735, October 4, 1993). This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 *et seq.*, or impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Pub. L. 104–4). Nor does it require any prior consultation as specified by Executive Order 12875, entitled Enhancing the Intergovernmental Partnership (58 FR 58093, October 28, 1993), or special considerations as required by Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994), or require OMB review in accordance with Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 19885, April 23, 1997).

### *B. Executive Order 12875*

Under Executive Order 12875, entitled Enhancing Intergovernmental

Partnerships (58 FR 58093, October 28, 1993), EPA may not issue a regulation that is not required by statute and that creates a mandate upon a State, local or tribal government, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by those governments. If the mandate is unfunded, EPA must provide to the Office of Management and Budget (OMB) a description of the extent of EPA's prior consultation with representatives of affected State, local and tribal governments, the nature of their concerns, copies of any written communications from the governments, and a statement supporting the need to issue the regulation. In addition, Executive Order 12875 requires EPA to develop an effective process permitting elected officials and other representatives of State, local and tribal governments ''to provide meaningful and timely input in the development of regulatory proposals containing significant unfunded mandates.''

Today's rule does not create an unfunded federal mandate on State, local or tribal governments. The rule does not impose any enforceable duties on these entities. Accordingly, the requirements of section 1(a) of Executive Order 12875 do not apply to this rule.

*Executive Order 13084*

Under Executive Order 13084, entitled Consultation and Coordination with Indian Tribal Governments (63 FR 27655, May 19,1998), EPA may not issue a regulation that is not required by statute, that significantly or uniquely affects the communities of Indian tribal governments, and that imposes substantial direct compliance costs on those communities, unless the Federal government provides the funds necessary to pay the direct compliance costs incurred by the tribal governments. If the mandate is unfunded, EPA must provide OMB, in a separately identified section of the preamble to the rule, a description of the extent of EPA's prior consultation with representatives of affected tribal governments, a summary of the nature of their concerns, and a statement supporting the need to issue the regulation. In addition, Executive Order 13084 requires EPA to develop an effective process permitting elected and other representatives of Indian tribal governments ''to provide meaningful and timely input in the development of regulatory policies on matters that significantly or uniquely affect their communities.''

Today's rule does not significantly or uniquely affect the communities of Indian tribal governments. This action does not involve or impose any requirements that affect Indian Tribes. Accordingly, the requirements of section 3(b) of Executive Order 13084 do not apply to this rule.

In addition, since these tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 *et seq.*) do not apply. Nevertheless, the Agency has previously assessed whether establishing tolerances, exemptions from tolerances, raising tolerance levels or expanding exemptions might adversely impact small entities and concluded, as a generic matter, that there is no adverse economic impact. The factual basis for the Agency's generic certification for tolerance actions published on May 4, 1981 (46 FR 24950) and was provided to the Chief Counsel for Advocacy of the Small Business Administration.

## X. Submission to Congress and the Comptroller General

The Congressional Review Act, 5 U.S.C. 801 *et seq.*, as added by the Small Business Regulatory Enforcement Fairness Act of 1996, generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report, which includes a copy of the rule, to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. house of Representatives, and the Comptroller General of the United States prior to publication of the rule in the **Federal Register**. This rule is not a ''major rule'' as defined by 5 U.S.C. 804(2).

## List of Subjects in 40 CFR part 180

Environmental protection, Administrative practice and procedure, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: November 18, 1998.

**Joseph J. Merenda,**

*Acting Director, Office of Pesticide Programs.*

Therefore, 40 CFR chapter I is amended as follows:

## PART 180 —— [AMENDED]

1. The authority citation for part 180 continues to read as follows:

**Authority:** 21 U.S.C. 346a and 371.

2. Section 180.536 is added to read as follows:

### § 180.536 Triazamate; tolerances for residues.

(a) *General.* Time-limited tolerances are established for the combined residues of triazamate (RH-7988) ethyl(3-tert-butyl-1-dimethylcarbamoyl-1*H*-1,2,4-triazol-5-ylthio)acetate and its metabolite (RH0422) in or on the following commoditie(s):

| Commodity | Parts per million | Expiration/ Revocation Date |
|---|---|---|
| Apples ..... | 0.1 | 12/31/01 |

(b) *Section 18 emergency exemptions.*

(c) *Tolerances with regional registrations.* [Reserved]

(d) *Indirect or inadvertent residues.* [Reserved]

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## FEDERAL EMERGENCY MANAGEMENT AGENCY

## 44 CFR Part 206

## Federal Disaster Assistance for Disasters Declared On or After November 23, 1988

*CFR Correction*

In title 44 of the Code of Federal Regulations, revised as of October 1, 1998, on page 471, § 206.207 was inadvertently removed. The removed text should read as follows:

### § 206.207 Administrative and audit requirements.

(a) *General.* Uniform administrative requirements which are set forth in 44 CFR part 13 apply to all disaster assistance grants and subgrants.

(b) *State administrative plan.* (1) The State shall develop a plan for the administration of the Public Assistance program that includes at a minimum, the items listed below:

(i) The designation of the State agency or agencies which will have the responsibility for program administration.

(ii) The identification of staffing functions in the Public Assistance program, the sources of staff to fill these functions, and the management and oversight responsibilities of each.

(iii) Procedures for:

(A) Notifying potential applicants of the availability of the program;