40 CFR Part 180

[OPP–300455; FRL–5591–5]

RIN No. 2070–AB78

Thiazopyr; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This document establishes tolerances for residues of the herbicide thiazopyr (3-pyridinecarboxylic acid, 2-(difluoromethyl)-5-(4,5-dihydro-2-thiazolyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-, methyl ester) and its metabolites determined as 2-(difluoromethyl)-6-(trifluoromethyl)-3,4,5-pyridinetricarboxylic acid, all expressed as the parent equivalents in or on the raw agricultural commodities orange and grapefruit. Rohm and Haas Company submitted a petition to EPA under the Federal Food, Drug and Cosmetic Act as amended by the Food Quality Protection Act of l996 requesting the tolerances.

EFFECTIVE DATE: This regulation becomes effective March 5, 1997.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300455], may 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 should be identified by the docket control number and submitted to: Public Response and Program Resources Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person, bring copy of objections and hearing requests to: Rm. 1132, CM #2, 1921 Jefferson Davis Highway, Arlington, VA 22202.

A copy of objections and hearing requests filed with the Hearing Clerk may also be submitted electronically to the OPP 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 file format or ASCII file format. All copies of objections and hearing requests in electronic form must be identified by the docket number [OPP–300455].

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. Additional information on electronic submissions can be found below in this document.

FOR FURTHER INFORMATION CONTACT: By mail: Joanne I. Miller, Product Manager (PM) 23, Registration Division (7505C), Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location, telephone number and e-mail address: Rm. 237, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703) 305–6224; e-mail: miller.joanne@epamail.epa.gov.

SUPPLEMENTARY INFORMATION: In the Federal Register of October 21, 1993 (58 FR 54354), EPA issued a notice pursuant to section 408(d) of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a(d), announcing the filing of a pesticide tolerance petition by Monsanto Co., Suite 1100, 700 14th St., NW., Washington, DC 20005. The petition requested that 40 CFR part 180 be amended by adding a regulation for tolerances for combined residues of the herbicide thiazopyr (3-pyridinecarboxylic acid, 2-(difluoromethyl)-5-(4,5-dihydro-2-thiazolyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-, methyl ester) and its metabolites determined as 3-pyridinecarboxylic acid, 5-(aminocarbonyl)-2-(difuoromethyl)-4-(2-methylpropyl)-6-trifluoromethyl-, methyl ester and 3-pyridinecarboxylic acid, 2-(difluoromethyl)-4-(2-methylpropyl)-5-((2-sulfoethyl)amino) carbonyl-6-(trifluoromethyl) and expressed as parent equivalents, in or on the raw agricultural commodities: Citrus, whole fruit at 0.05 ppm; cotton seed at 0.05 ppm and cotton forage at 0.2 ppm. The proposed analytical method for determining residues was gas chromatography with mass spectrometry.

In the Federal Register of August 24, l994 (59 FR 43580) EPA issued a notice of an amendment to the petition. The tolerances requested were changed to residues of thiazopyr (3-pyridinecarboxylic acid, 2-[difluoromethyl]-5-(4,5-dihydro-2-thiazolyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-, methyl ester) and its metabolites determined as 3-pyridinecarboxylic acid, 5-(aminocarbonyl)-2-(difluoromethyl)-4-(2-methylpropyl)-6-trifluoromethyl)-, methyl ester and 3-pyridinecarboxylic acid, 2-(difluoromethyl)-4-(2-methylpropyl)-5-(((2-sulfoethyl) amino) carbonyl)-6-(trifluoromethyl) acid and expressed as parent equivalents, in or on citrus whole fruit at 0.05 ppm, cotton seed at 0.05 ppm and cotton forage at 0.2 ppm. Monsanto Co. requested the petition be amended to read: tolerances of 0.05 ppm for orange, whole fruit and 0.05 for grapefruit, whole fruit. The proposed analytical method for determining residues was mass spectral multiple-ion detection.

In the Federal Register of November 22, l996 (61 FR 59440)(FRL–5573–8) EPA issued a third notice of filing to amend the petition to bring the petition in conformity with the Food Quality Protection Act (FQPA) of l996. The notice contained a summary of the petition prepared by the petitioner and this summary contained conclusions and arguments to support its conclusion that the petition complied with FQPA. In this instance the petitioner proposed to amend 40 CFR part 180 by establishing a regulation for tolerances for residues of thiazopyr in or on orange and grapefruit at 0.05 ppm on the whole fruit, the same as proposed in the previous EPA notices of filing.

There were no comments or requests for referral to an advisory committee received in response to the notices of filing.

The data submitted in the petition and other relevant material have been evaluated. The toxicology data listed below were considered in support of these tolerances.

I. Toxicological Profile

1. A battery of acute toxicity studies placing technical thiazopyr in Toxicity Categories III and IV.

2. A 3–month feeding study in rats at dietary intakes of 0, 0.07, 0.67, 6.60, 68, or 201 milligrams per kilogram per day (mg/kg/day) (males) and 0.08, 0.79, 8.0, 79 or 227 mg/kg/day (females) with a no observed effect level (NOEL) of 6.6 mg/kg/day, based on increased liver, thyroid and kidney weights, changes in clinical chemistry and hematological parameters and on gross and microscopic changes observed in the liver and thyroid at dose levels of 68 mg/kg/day and higher. At the 201 mg/kg/day dose diffuse thyroid follicular cell hypertrophy/hyperplasia was observed.

3. A 3–month feeding study in dogs at 0, 3, 6, 35 and 175 mg/kg/day (males) and 0, 2, 3, 35 and 160 mg/kg/day (females) with a NOEL of 2 mg/kg/day, based on decreased body weight gain and increased SGPT levels at 3 and 6 mg/kg/day for males and females, respectively and above; decreased total protein and albumin concentration and

albumin/globulin ratio, increased AP, hepatocytic hypertrophy, oval cell proliferation and increased hepatocytic fatty content at 35 mg/kg/day and above; and decreased calcium concentration which is thought to be related to the hypoalbuminemia, decreased cholesterol and triglyceride concentrations, slightly increased GGT and SGPT, follicular hyperplasia of thyroid, increased colloid content in follicles and increased relative thyroid weight at 175 mg/kg/day.

4. A 3–week dermal study in rabbits at 0, 100, 500 and 1,000 mg/kg/day with a NOEL of 100 mg/kg/day. The effects were increased mean absolute and relative kidney weights and minimal multifocal or periportal hepatocyte vacuolation.

5. A 1–year feeding study in dogs at 0, 0.8, 7.8, 86 mg/kg/day (males) and 0, 0.8, 8.8, and 78 mg/kg/day (females). The NOEL was 0.8 mg/kg/day and the LOEL is 7.8 mg/kg/day based upon hepatocellular hypertrophy/hyperplasia, which was observed at 7.8 to 8.8 mg/kg/day for males and females, respectively, and above. In addition, an increase of approximately 10% in prothrombin time was observed at 8.6 and 7.8 mg/kg/day for males and females, respectively with both sexes, as well as increased SGOT, SGPT, GGT and ALK and decreases in cholesterol, albumin, total protein and calcium levels. An increase in absolute and relative liver weights were also observed at 2,000 ppm. Enlargement and/or discoloration in some of the high dose animals provided additional evidence of hepatotoxicity.

6. A developmental toxicity study in rats at 0, 10, 100 and 250 mg/kg/day. The maternal NOEL is 100 mg/kg/day and the maternal lowest observed effect level (LOEL) is 250 mg/kg/day based on increased liver weights, salivation, decreased body weight gains and food consumption. The developmental NOEL was 100 mg/kg/day and the developmental LOEL was 250 mg/kg/day based on increased incidences of unossified sternebra(e) and 7th cervical rib variations.

7. A developmental toxicity study in rabbits at 0, 10, 75 and 175 mg/kg/day. The maternal NOEL was 75 mg/kg/day based on reduced body weight gain and food consumption. The developmental NOEL was 175 mg/kg/day the highest dose tested. No effects were observed.

8. A two-generation reproductive study in rats at 0, 0.72, 7.33 and 72.9 mg/kg/day (males) and 0, 0.86, 8.49, 81.3 mg/kg/day (females). The parental/systemic NOEL was 0.72 mg/kg/day. The toxic effects were increased absolute and relative liver weight, hepatic discoloration, histologic evidence of hepatic hypertrophy and vacuolization in females in both generations. The reproductive NOEL was 72.9 mg/kg/day, the highest dose tested. There were no reproductive effects.

9. A mouse carcinogenicity study at doses of 0, 0.17, 1.6, 16.9 66.3 and 128.4 mg/kg/day (males) and 0,0.24, 2.6, 26.8, 108.1 and 215.9 mg/kg/day (female). The systemic NOEL was 1.6 mg/kg/day. The effects were hepatocellular hypertrophy and amyloid deposition. At 66.3 mg/kg/day the same lesions plus increased liver weights, random and periportal hepatocellular vacuolation were observed. At 128.4 mg/kg/day the same lesions plus distended abdomen, slight increase in ALP, SGOT and SGPT, abnormal coloration and enlargement of liver, decrease in absolute and relative spleen weights, increase in absolute and relative kidney weights, increase in eosinophilia in hepatocytes, kidney nephropathy and lymphocytic hyperplasia of the nesenteric lymph nodes were observed. There were no increases in neoplastic lesions in any of the treated groups.

10. A 2–year rat carcinogenicity study at doses of 0, 0.04, 0.4, 4.4, 44.2 or 136.4 mg/kg/day (males) and 0, 0.06, 0.6, 5.6, 56.3 or 177.1 mg/kg/day (female) with a systemic NOEL of 4.4 mg/kg/day. The effects were protruding eyes, evidence of mild anemia, increased GGT and cholesterol, increased absolute and relative liver, kidney and thyroid weights and significant increase in microscopic lesions in the liver (hypertrophy and vacuolar changes), kidney (nephropathy) and thyroid (hypertrophy and hyperplasia); decreased mean body weight and body weight gain and food consumption. A statistically significant increase in thyroid follicular cell adenomas/cystadenomas were observed in males at 44.2 and 136.4 mg/kg/day. A nonsignificant increase in renal tubular adenomas in high-dose females was considered to be equivocal.

The EPA's Health Effects Division Carcinogenicity Peer Review Committee classified thiazopyr as a Group C, possible human carcinogen and recommended that for the purpose of risk characterization a Margin of Exposure (MOE) approach should be used in evaluation of the consequences of human exposure.

11. An acceptable study for inducing reverse mutation in Ames Salmonella strains of bacteria exposed with or without activation at doses up to 10,000 micrograms per plate. The study showed negative results.

12. An acceptable study for inducing micronuclei in bone marrow cells of mice treated up to a lethal dose of 800 mg/kg. The study showed negative results.

13. A mutagenic study with Chinese hamster ovary cells exposed *in vitro* with or without activation to doses up to 1,000 micrograms, the highest dose tested. The study showed negative results for inducing forward mutation at the hypoxanthine guanine phosphoribosyl transferase locus (HGPRT). On the basis of the studies on mutagenicity and genotoxicity, it is concluded that thiazopyr is not a mutagenic or genotoxic chemical.

14. An acute neurotoxicity in rats at doses of 0, 500, 1,000 and 2,000 mg/kg with a NOEL of 500 mg/kg. The effects were transient differences in functional observational battery and motor activity compared to control groups. The results of the study were considered to be inconclusive for neurotoxicity. At the highest dose (2,000 mg/kg) it was not possible to distinguish between neurotoxicity and general systemic toxicity.

15. Two metabolism studies were conducted in rats with radio-labeled thiazopyr. One with the ^{14}C at the 4 position of the pyridine ring and one with the ^{14}C at the 4′ and 5′ positions of the thiazole ring. The absorption of an orally administered dose was about 90%. The overall radiolabel recovery for all study groups was 88.9, plus or minus 0.65%. No significant sex-related differences were observed in the total percent recovery. However, the distribution of recovery was sex-related. There was little radiolabel detected in tissues at study termination. Preferential sites for localization of the radiolabel included liver, adipose tissue, muscle and bone. The metabolic pathway is essentially an oxidative pathway. Vulnerable sites of the molecule are the thiazoline ring, the isobutyric side chain and the pyridine rings. Thiazopyr appears to be rapidly and extensively eliminated with low amounts of residues remaining in the tissues and carcasses. The percentage of radiolabel remaining in the carcasses following feeding thiazoline-labeled thiazopyr was between 6.9 and 10.8%.

16. Special mechanistic studies for mode of toxic action on thyroid function. The results of three studies on the effects of thiazopyr on thyroid function and mechanisms involved in the disposition of T4 in rats were reviewed. These studies are described below:

a. Thiazopyr was administered through the diet, in rats, at 0 and 150 mg/kg/day to determine the subchronic effect on hormone level and other biochemical endpoints. Animals were

assayed at 7, 14, 28, 56 or 90 days. Significant decreases in body weight gain were observed at 90 days. Early in the study the treated rats showed increases in TSH (ranging from 133 to 200% of controls) and decreases in T4 (ranging from 43% to 76% of controls). In addition there were increases in liver and thyroid weights and increases in thyroid follicular cell hypertrophy/hyperplasia. Reverse T3 was increased at 28 days, and T3 was either not affected or increased. There were indications of increases in hepatic UDPGT activity and significant increases in T4 UDPGT activity. Hepatic 5′-monodeiodinase activity was either not affected or decreased. The effects observed in this study were supportive of the theory that thiazopyr may induce thyroid tumors through a disruption in the thyroid-pituitary hormonal feedback mechanisms.

b. A second study on the effects of thiazopyr on the biochemical mechanisms of thyroid toxicity in rats at doses of 0, 0.5, 1.5, 5, 15, 50 or 150 mg/kg/day was conducted. Dose response effects on various biochemical parameters were observed. Two groups of the rats in the study were observed for reversibility of effects observed up to 56 and 112 days. Doses at 15, 50 and 150 mg/kg/day significantly increased the liver weights. Thyroid weights were increased at doses of 50 and 150 mg/kg/day. There was no significant effect on body weight or body weight gains during the study. The T4 UDPGT levels were increased by 117 and 376% above controls at the 50 and 150 mg/kg/day dosages, respectively. Effects of 150 mg/kg/day were increases in T3, TSH and rT3 serum concentrations, and increased incidence of follicular cell hypertrophy/hyperplasia at the 150 mg/kg/day dose. A NOEL of 1.5 mg/kg/day was determined based on liver weight increases. Thyroid weight was the only parameter that did not return to those similar to the controls. At the 56 and 112 day recovery periods the thyroid weights were 120 and 123% of control values, respectively.

c. A third thyroid function study on the biochemical mechanisms involved with disposition of T4 in rats fed dosages of 0 and 150 mg/kg/day for 56 days was conducted. Rats feed thiazopyr had increase T4 UDPGT activity and total deiodinase activity in their livers. There was also a two-fold increase in mixed function oxidase enzyme activity. Results of the three studies suggest that increased glucuronidation, deiodination of T4 and T3, and increased rate of clearance of T4 from the blood and excretion of the hormone and its metabolites in the bile could significantly reduce the level of circulating T4 in the male rat.

Results of these studies support the hypothesis that thiazopyr may induce thyroid tumors through a disruption of the thyroid-pituitary hormonal feedback mechanism circulating T4 in the male rat.

II. Aggregate Exposures

1. *Food and feed uses.* The primary source for human exposure to thiazopyr will be from ingestion of both raw and processed agricultural commodities as proposed in the November 22, 1996 notice of filing cited above. Based on tolerances of 0.05 ppm in or on orange and grapefruit, the Theoretical Maximum Residue Contributions (TMRC) for the U.S. adult population and for U.S. children (1 to 6 years of age) were determined. In deriving the dietary exposure to thiazopyr and its metabolites, EPA assumed that 100% of the orange and grapefruit crops were cultured with the aid of this herbicide. A chronic exposure was used to estimate the TMRC. The TMRC for the U.S. population was estimated to be 0.000118 mg/kg/day. The TMRC for children, 1 to 6 years of age was 0.000324 mg/kg/day. The TMRC for children, 7 to 12 years of age was 0.000173 mg/kg/day.

2. *Potable water.* There is presently no EPA Lifetime Health Advisory level for thiazopyr and its degradates as drinking water contaminates. Thiazopyr has not been found in ground water. A monoacid degradate was found in wells at concentrations of up to 7.6 parts per billion (ppb). The wells were being monitored as part of a prospective ground water study in the state of Florida. Using a standard potable water ingestion of 2 liters per day by adults and 1 liter per day by children, the exposure from potable water to adults was determined to be 0.000217 mg/kg/day. Exposure to children was determined to be 0.00076 mg/kg/day.

3. *Non-dietary uses.* There are no non-dietary uses registered under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), as amended.

4. 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.'' While 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 capability to resolve the scientific issues concerning common mechanism of toxicity in a meaningful way. EPA is commencing 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 enable the Agency to apply common mechanism issues to its pesticide risk assessments. At present, however, the Agency does not know how to apply the information in its files concerning common mechanism issues to risk assessments, and therefore believes that in most cases there is no ''available information'' concerning common mechanism that can be scientifically applied to tolerance decisions. Where it is clear that a particular pesticide may share a significant common mechanism with other chemicals, or where it is clear that a pesticide does not share a common mechanism with other chemicals, a tolerance decision may be affected by common mechanism issues. The Agency expects that most tolerance decisions will fall into the area in between, where EPA can not reasonably determine whether a pesticide does or does not share a common mechanism of toxicity with other chemicals (and, if so, how that common mechanism should be factored into a risk assessment). In such circumstances, the Agency will reach a tolerance decision based on the best, currently available and usable information, without regard to common mechanism issues. However, the Agency will also revisit such decisions when the Agency learns how to apply common mechanism information to pesticide risk assessments.

In the case of thiazopyr, EPA has determined that it does not now have the capability to apply the information in its files to a resolution of common mechanism issues in a manner that would be useful in a risk assessment. This tolerance determination therefore does not take into account common mechanism issues. The Agency will reexamine the tolerances for thiazopyr, if reexamination is appropriate, after the Agency has determined how to apply common mechanism issues to its pesticide risk assessments.

III. Determination of Safety for U.S. Population and Non-nursing Infants

1. *The U.S. population.* Based on a NOEL of 0.8000 milligrams per kilogram of body weight per day (mg/kg bwt/day) from a 2–year dog feeding study that showed a liver effect of hepatocellular hypertrophy and hyperplasia, and using a safety or uncertainty factor of 100 to

account for the interspecies extrapolation and intraspecies variability, the Agency has determined a Reference Dose (RfD) of 0.008 mg/kg bwt/day for this assessment of risk. Based on the available toxicity data and the available exposure data identified above, the proposed tolerances will utilize 1.5% of the RfD for the U.S. population. Including an estimated exposure of 7.6 ppb in potable water, the dietary exposure for the U.S. adult population, assuming the ingestion of 2 liters of water per day, increases to 0.000335 mg/kg/day and utilizes 4.6% of the RfD.

2. *Non-nursing infants.* Using the RfD of 0.008 mg/kg/bwt/day as described above and the TMRC of 0.000251 mg/kg/day determined of non-nursing infants, the proposed tolerances utilize 13.97% (3.1% dietary and 10.87% potable water) of the RfD.

3. *Nonfood uses.* There are no nonfood uses of thiazopyr registered under FIFRA, as amended.

IV. Determination of Safety for Infants and Children

Risk to infants and children was determined by use of two developmental toxicity studies. One study in rats had a NOEL for developmental toxicity of 100 mg/kg/day, based on an increase in the incidence of unossified sternebrae and 7th cervical rib variations. The maternal NOEL was also 100 mg/kg/day based on toxic effects of increased liver weights, salivation, decreased body weight gains and food consumption. Fetal toxicity was only observed at maternally toxic doses. No malformations were observed at any dose. A second study in rabbits had a maternal NOEL of 75 mg/kg/day based on effects in reducing body weight gain and food consumption. There were no development effects at l75 mg/kg/day, the highest dose tested.

In a reproduction study in rats, the parental NOEL was 0.72 to 8.1 mg/kg/day. The reproductive toxicity NOEL was 72.9 to 81.3 mg/kg/day. There were no treatment-related effects on any reproductive parameter in the adults or their offspring. Overall, thiazopyr was not associated with significant developmental or reproductive effects below maternally toxic doses.

FFDCA section 408 provides that EPA shall apply an additional safety factor for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the database unless EPA determines that such additional factor is not necessary to protect the safety of infants and children. EPA believes that reliable data support using a different safety factor (usually 100x) and not the additional safety factor when EPA has a complete data base and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the traditional safety factors.

The toxicological database for evaluating pre- and post-natal toxicity for thiazopyr is mostly complete. Available data indicate that no developmental toxicity was observed in the rabbit study at the highest dose tested (175 mg/kg/day). Maternal toxicity was observed in the rabbit in the 175 mg/kg/day dose group which consisted of reductions in body weight gain and food consumption. In the rat developmental study, a reduction in maternal body weight gain and body weight was observed at the highest dose tested (250 mg/kg/day). Developmental toxicity was observed in the high dose (250 mg/kg/day) as increased incidences of unossified sternebra and 7th cervial rib variations.

The NOEL for systemic (parental) toxicity is 0.72 mg/kg/day. The NOEL for reproductive toxicity is 72.9 mg/kg/day (highest dose tested). There were no reproductive effects noted in the study. These data taken together suggest minimal concern for developmental or reproductive toxicity and do not indicate any increased pre- or post-natal sensitivity in the offspring; no additional uncertainty factor for increased sensitivity in infants and children is appropriate.

The percent of the RfD that will be utilized by the aggregate exposure to thiazopyr will range from 7.148% for non-nursing infants, up to 13.55% for children (1 to 6 years of age). Therefore, EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure.

V. Other Considerations

A. Endocrine Effects

An evaluation of the potential effects on the endocrine systems of mammals was partially determined by chronic toxicology studies described above. There were observed pathology of the endocrine organs in those studies. Three supplemental rat studies were conducted to determine the mode of toxic action of thiazopyr on thyroid function. The mode of toxic action as indicated by effects of thiazopyr on serum hormone levels, hepatic enzyme activity, and thyroid-pituitary hormonal feedback mechanisms.

B. Metabolism in Plants and Animals

The metabolism of thiazopyr in plants and animals is adequately understood for the purposes of these tolerances. There were no crop residues found after the preemergence use in the culture of orange and grapefruit. The metabolites that were identified in a radiolabeled thiazopyr study and converted to two common entities: amide ester and sulfonic diacid. However, the Agency has accepted enforcement analytical methodology that uses only one common entity to determine greater than 70% of the expected thiazopyr residues.

C. Analytical Method

There is a practical analytical method for detecting and measuring levels of thiazopyr and its metabolites in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set in these tolerances. The proposed analytical method for determining residues is gas-liquid chromatography with mass selective detection. Thiazopyr and its metabolites are converted to a common moiety which is quantified. The quantitation limit of this method is 0.015 ppm for whole orange fruit. EPA has provided information on this method to FDA. Because of the long lead time from establishing these tolerances to publication, the enforcement methodology is being made available in the interim to anyone interested in pesticide enforcement when requested by mail from: Calvin Furlow, Public Response Branch, Field Operations Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. Office location and telephone umber: Rm. 1130A, CM #2, 1921 Jefferson Davis Hwy., Arlington, VA 22202, (703)–305–5937.

D. International Tolerances

There are no Codex Alimentarius Commission (Codex) Maximum Residue Levels (MRLs) for thiazopyr.

E. Summary of Findings

The analysis for thiazopyr using tolerance level residues shows that the proposed uses in the culture of orange and grapefruit will not cause exposure to exceed the levels at which the Agency believes there is an appreciable risk. All population subgroups examined by EPA are exposed to thiazopyr residues at levels below 100 percent of the RfD for chronic effects.

Based on the information cited above, the Agency has determined that the establishment of these tolerances by adding a new section to 40 CFR part 180

will be safe; therefore, the tolerances are established as set forth below.

VI. 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 (1)(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 governs 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 May 5, 1997, file written objections to any aspect of this regulation (including the automatic revocation provision) 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 issue(s) on which a hearing is requested, the requestor's contentions on such issues, and a summary of any evidence relied upon by the objector (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 a 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 issue(s) 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 Confidential Business Information (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.

VII. Public Docket

A record has been established for this rulemaking under docket number [OPP–300455]. A public version of this record, 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 Room 1132 of the Public Response and Program Resources Branch, Field Operation Division (7506C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Highway, Arlington, VA. EPA has also established a special record for post-FQPA tolerances which contains documents of general applicability. This record can be found in the same location.

The official record for this rulemaking, as well as the public version, as described above, is kept in paper form. Accordingly, in the event there are objections and hearing requests, 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. The official rulemaking record is the paper record maintained at the Virginia address in ''ADDRESSES'' at the beginning of this document.

VIII. Regulatory Assessment Requirements

Under Executive Order 12866 (58 FR 51735, Oct. 4, 1993), this action is not a ''significant regulatory action'' and since this action does not impose any information collection requirements subject to approval under the Paperwork Reduction Act, 44 U.S.C. 3501 et seq., it is not subject to review by the Office of Management and Budget. In addition, this action does not impose any enforceable duty, or contain any ''unfunded mandates'' as described in Title II of the Unfunded Mandates Reform Act of 1995 (Pub. L. 104–4), or require prior consultation as specified by Executive Order 12875 (58 FR 58093, October 28, l993), or special considerations as required by Executive Order 12898 (59 FR 7629, February 16, l994).

Because tolerances established on the basis of a petition under section 408(d) of FFDCA do not require issuance of a proposed rule, the regulatory flexibility analysis requirements of the Regulatory Flexibility Act (RFA), 5 U.S.C. 604(a), do not apply. Prior to the recent amendment of the FFDCA, EPA had treated such rulemakings as subject to the RFA; however, the amendments to the FFDCA clarify that no proposal is required for such rulemakings and hence that the RFA is inapplicable.

Under 5 U.S.C. 801(a)(1)(A) of the Administrative Procedure Act (APA) as amended by the Small Business Regulatory Enforcement Fairness Act of 1996 (Title II of Pub. L. 104–121, 110 Stat. 847), EPA submitted 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 General Accounting Office prior to publication of the rule in today's Federal Register. This rule is not a ''major rule'' as defined by 5 U.S.C. 804(2) of the APA as amended.

List of Subjects in 40 CFR Part 180

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

Dated: February 19, 1997.

Stephanie R. Irene,

Acting Director, Office of Pesticide Programs.

Therefore, 40 CFR part 180 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. By adding § 180.496 to read as folllows:

§ 180.496 Thiazopyr; tolerances for residues.

Tolerances are established for combined residues of the herbicide thiazopyr (3-pyridinecaroxylic acid, 2-(difluoromethyl)-5-(4,5-dihydro-2-thiazolyl)-4-(2-methylpropyl)-6-(trifluoromethyl)-, methyl ester) and its metabolites determined as 2-(difluoromethyl)-6-(trifluoromethyl)-3,4,5-pyridinetricarboxylic acid, all expressed as the parent equivalents in or on the following raw agricultural commodities:

Commodities	Parts per million
Grapefruit	0.05
Orange	0.05

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