notice also contains a correction of a citation in the interim rule.

EFFECTIVE DATE: July 6, 1998.

FOR FURTHER INFORMATION CONTACT: H. Edward Odom, Chief, Legislation and Regulations Division, Visa Services, Department of State, Washington, DC 20520–0106, (202) 663–1204.

SUPPLEMENTARY INFORMATION: An interim rule implementing the new subsection 222(g) of the Immigration and Nationality Act (INA), and requesting comments, was published on January 7, 1998 [63 FR 669]. The period for comments has expired; no comments have been received. The rule will thus stand as originally published, with a correction of the reference to INA 214(k) in 22 CFR 41.101(c)(1) which should read 214(l). As there are now two 214(l)'s in the INA, this reference is to the first one, i.e., the subsection relating to a waiver of the 2-year foreign residence requirement.

As the final regulation is identical to the interim regulation other than for the correction of a citation, it is not being reprinted in full herein.

List of Subjects in 22 CFR Part 41

Aliens, Nonimmigrants, Passports, Visas.

In view of the foregoing, the interim rule amending 22 CFR parts 40 and 41 which was published at 63 FR 669 on January 7, 1998, is adopted as a final rule with the following change:

PART 41—[CORRECTED]

1. The authority citation for part 41 continues to read:

Authority: 8 U.S.C. 1104.

§ 41.101 [Corrected]

2. In § 41.101(c)(1), correct the reference to ''INA 214(k)'' to read ''INA 214(l)''.

Dated: May 20, 1998.

Donna J. Hamilton,

Acting Assistant Secretary for Consular Affairs.

[FR Doc. 98–17735 Filed 7–2–98; 8:45 am]

BILLING CODE 4710–06–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[OPP–300666; FRL–5794–6]

RIN 2070–AB78

Pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for combined residues of pyriproxfen in or on cotton seed and cotton gin byproducts. Valent U.S.A. Corporation requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA), as amended by the Food Quality Protection Act of 1996 (Pub. L. 104-170).

DATES: This regulation is effective July 6, 1998. Objections and requests for hearings must be received by EPA on or before September 4, 1998.

ADDRESSES: Written objections and hearing requests, identified by the docket control number, [OPP–300666], 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–300666], 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, 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–300666]. 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: Joseph Tavano, 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: Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, (703) 305-6411, e-mail: tavano.joseph@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 6F4737) for tolerance by Valent U.S.A. Corporation, 1333 N. California Blvd., Walnut Creek, CA 94596. This notice included a summary of the petition prepared by Valent U.S.A. Corporation, the registrant. There were no comments received in response to the notice of filing.

The petition requested that 40 CFR 180.534 be amended by establishing tolerances for combined residues of the insecticide, pyriproxfen, in or on cotton seed and cotton gin byproducts at 0.05 and 2.0 parts per million (ppm) respectively.

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 three 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 most highly exposed population subgroup was not regionally based.

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 pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine and to make a determination on aggregate exposure, consistent with section 408(b)(2), tolerances for combined residues of pyriproxfen on cotton seed and cotton gin byproducts at 0.05 and 2.0 ppm respectively 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 pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine are discussed below.

1. *Acute toxicity— Acute toxicity studies with technical pyriproxyfen*. Oral LD_{50} in the rat is >5,000 milligram/kilogram (mg/kg) for males and females - Toxicity Category IV; dermal LD_{50} in the rabbit at >2,000 mg/kg - Toxicity Category IV; inhalation LC_{50} in the rat is >1.3 mg/L (highest dose attainable) - Toxicity Category III; primary eye irritation in the rabbit (mild irritatant) - Toxicity Category III; primary dermal irritation in the rabbit (not an irritant: non-irritating to the skin under conditions of test))- Toxicity Category IV. Pyriproxyfen is not a sensitizer.

2. *Subchronic toxicity*— i. *Rats*. In the subchronic feeding study in rats, the no-observed effect level (NOEL) was 27.68 mg/kg/day. The lowest oberved effect level (LOEL) was 141.28 mg/kg/day, based upon higher mean total cholesteral and phospholipids, decreased mean RBCs, hematocrit and hemoglobin counts and increased relative liver weight.

ii. *Dogs*. In the subchronic feeding study in dogs, the NOEL was 100 mg/kg/day and the LOEL was 300 mg/kg/day. The effects were based on increased absolute and relative liver weight in males and hepatocellular hypertrophy in females. These findings were also observed at 1,000 mg/kg/day and may represent adaptive changes at both 300 mg/kg/day and the limit dose of 1,000 mg/kg/day.

iii. *Dermal study - Rats*. In a 21-day dermal study in rats, the NOEL for systemic effects was >1,000 mg/kg/day (limit dose). The LOEL for systemic effects was not established in this study. No dermal or systemic toxicity was observed at any dose tested.

3. *Chronic toxicity/carcinogenicity*— i. *Dogs*. In a one-year chronic feeding study in dogs, the NOEL was 100 mg/kg/day. The LOEL was 300 mg/kg/day based on decreased weight gain, increased absolute and relative liver weight, mild anemia, increased cholesterol and triglycerides.

ii. *Mice*. The oncogenicity study in mice the NOEL and LOEL for systemic toxicity in males are 600 ppm and 3,000 ppm, respectively, based on an renal lesions in males. The technical grade test material was given to male and female CD-1 mice in diet for 18 months at 0, 120, 600, or 3,000 ppm. No statistically significant increase in tumor incidence relative to controls were observed in either sex at any does up to 3,000 ppm (highest dose tested).

iii. *Rats*. In the chronic feeding/oncogenicity study in rats, the NOEL (systemic) was 35.1 mg/kg/day and the LOEL (systemic) was 182.7 mg/kg/day. The technical grade test material was administered to male and female Sprague-Dawley rats in diet for 24 months at 0, 120, 600, or 3,000 ppm. A decrease of 16.9% in bogy weight gain in females at 3,000 ppm (182.7 mg/kg/day) was basis for the systemic LOEL.

4. *Developmental toxicity*— i. *Rabbits*. In the developmental study in rabbits, the maternal NOEL/LOEL for maternal toxicity were 100 and 300 mg/kg/day based on premature delivery/abortions, soft stools, emaciation, decreased activity and bradypnea. The developmental NOEL was determined to be 300 mg/kg/day and developmental LOEL was determined to be undetermined; no dose related anomalies occurred in the 4 remaining litters studied at 1,000 mg/kg/day.

ii. *Rats*. In the developmental study in rats, a maternal NOEL/LOEL were determined to be 100 mg/kg/day and 300 mg/kg/day, respectively. These findings were based on increased incidences in mortality and clinical signs at 1,000 mg/kg/day with decreases in food consumption, body weight, and body weight gain together with increases in water consumption at 300 and 1,000 mg/kg/day. The developmental NOEL/LOEL were 100 mg/kg/day and 300 mg/kg/day based on the increase of skeletal variations at 300 mg/kg/day and above.

5. *Reproductive toxicity*. In a two-generation reproduction study in rats, the systemic NOEL was 1,000 ppm (87 mg/kg/day). The LOEL for sytemic toxicity was 5,000 ppm (453 mg/kg/day). Effects were based on decreased body weight, weight gain and food consumption in both sexes and both generations, and increased liver weights in both sexes associated with liver and kidney histopathology in males. The reproductive NOEL was 5,000 ppm. A reproductive LOEL was not established.

6. *Mutagenicity*. Studies on gene mutation and other genotoxic effects: In a Gene Mutation Assay (Ames Test)/Reverse Mutation, finding were determined as negative for induction of gene mutation measured as the reversion to histine protrophy of 5 *S.typhimurium* strains and *E.Coli* WP2 uvra at doses from 10 to 5,000 µg/plate with & without S-9 activation. The highest dose was insoluble. A Gene Mutation assay in Mammalian Cells was found to be negative f or mutagencity in CHO (Chinese hamster ovary) V79 cells with and without metabolic activation up to cytotoxic doses (300 µg/mL). In a Structural Chromosomal Aberration Assay *in vivo*, findings proved nonclastogenic in CHO cells both with and without S-9 activation up to cytotoxic doses (300 µg/mL). In Other Genotoxicity Assays, an increase in unscheduled DNA synthesis was not induced both with and without activation in HeLa cells exposed up to insoluble doses ranging to 6.4 µg/mL (without activation) and 51.2 µg/mL (with activation).

7. *Metabolism*. The results of the metabolism studies are as follows:

Acceptable: Rats were orally dosed with ^{14}C-labeled pyriproxyfen at 2 or

1,000 mg/kg and at repeated oral doses (14 daily doses) of unlabeled pyriproxyfen at 2 mg/kg followed by administration of a single oral dose of labeled pyriproxyfen at 2 mg/kg. Most radioactivity was excreted in the feces (81-92%) and urine (5-12%) over a 7 day collection period. Expired air was not detected. Tissue radioactivity levels were very low (less than 0.3%) except for fat. Examination of urine, feces, liver, kidney, bile and blood metabolites yielded numerous (>20) identified metabolites when compared to synthetic standards. The major biotransformation reactions of pyriproxyfen include: (i) Oxidation of the 4′ - position of the terminal phenyl group; (ii) oxidation at the 5′ - position of pyridine; and (iii) cleavage of the ether linkage and conjugation of the resultant phenols with sulfuric acid.

8. *Neurotoxicity*. Neurotoxicity has not been observed in any of the acute, subchronic, chronic, developmental or reproductive studies performed with pyriproxyfen.

B. Toxicological Endpoints

1. *Acute toxicity*. An acute dietary dose and endpoint was not identified in the database. The Agency concludes that there is a reasonable certainty of no harm from acute dietary exposure.

2. *Short - and intermediate - term toxicity*. Doses and endpoints were not identified for short and intermediate-term dermal and inhalation exposure. The Agency concludes that there are reasonable certainties of no harm from these exposures.

3. *Chronic toxicity*. EPA has established the RfD for pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine at 0.35 mg/kg/day. This RfD is based on a NOEL of 35.1 mg/kg/day and an uncertainty factor (UF) of 100. The NOEL was established from the combined chronic feeding/oncogenicity study in rats where the LOEL was 3,000 ppm, based on a 16.9% decrease in body weight gain in females when compared to controls.

4. *Carcinogenicity*. Pyriproxyfen is classified as Category E: not carcinogenic in two acceptable animal studies.

C. Exposures and Risks

1. *From food and feed uses*. In today's action tolerances will be established (40 CFR 180.534) for the combined residues of pyriproxfen, in or on the raw agricultural commodities: cotton seed and cotton gin byproducts at 0.05 and 2.0 ppm respectively. Risk assessments were conducted by EPA to assess dietary exposures and risks from pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine as follows:

i. *Acute exposure and risk*. Acute dietary risk assessments are performed for a food-use pesticide if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a one day or single exposure. No acute dietary endpoint and dose was identified in the toxicology data base for pyriproxyfen, therefore the Agency concludes that there is a reasonable certainty of no harm from acute dietary exposure.

ii. *Chronic exposure and risk*. The chronic dietary exposure analysis from food sources was conducted using the RfD of 0.35 mg/kg/day. The RfD is based on the NOEL of 35.1 mg/kg/day in male and female rats from the Chronic Feeding/Oncogenicity study in rats, and an uncertainty factor of 100 applicable to all population subgroups.

In conducting this chronic dietary risk assessment, EPA has made very conservative assumptions: 100% of cottonseed having pyriproxyfen tolerances will contain pyriproxyfen residues and those residues will be at the level of the established tolerance. This results in an overestimate of human dietary exposure. Thus, in making a safety determination for this tolerance, EPA is taking into account this conservative exposure assessment.

The existing pyriproxyfen tolerances (published, pending, and including the necessary Section 18 tolerances) result in a Theoretical Maximum Residue Contribution (TMRC) that is equivalent to the following percentages of the RfD: U.S. population (48 states) 0.00029%; Nursing infants (< 1 year old) 0.00003%; Non-nursing infants (< 1 year old) 0.00009%; Children (1-6 years old) 0.00053%; Children (7-12 years old) 0.00045%; Non-Hispanic Whites 0.00030%; Males (13-19 years old) 0.00032%.

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).

2. *From drinking water*— i. *Acute exposure and risk*. As previously stated, no acute dietary endpoint was identified for assessment of acute dietary risk. Thus the risk from acute exposure is considered to be negligible.

ii. *Chronic exposure and risk*. No monitoring data is available to perform a quantitative drinking water risk assessment for pyriproxyfen at this time. Thus, the GENEEC model and the SCI-GROW model were run to produce estimates of pyriproxyfen concentrations in surface and ground water respectively. The primary use of these models is to provide a coarse screen for sorting out pesticides for which OPP has a high degree of confidence that the true levels of the pesticide in drinking water will be less than the human health drinking water levels of concern (DWLOCs). A human health DWLOC is the concentration of a pesticide in drinking water which would result in unacceptable aggregate risk, after having already factored in all food exposures and other non-occupational exposures for which OPP has reliable data.

For chronic (non-cancer) exposure to pyriproxyfen in surface and ground water, the drinking water levels of concern are 12,250 g/L for males (13 yrs+), 10,500 g/L for females (13 yrs+) and 3,500 g/L for children (1-6 yrs). To calculate the DWLOC for chronic (non-cancer) exposure relative to a chronic toxicity endpoint, the chronic dietary food exposure (from DRES) was subtracted from the RfD to obtain the acceptable chronic (non-cancer) exposure to pyriproxyfen in drinking water. DWLOCs were then calculated using default body weights and drinking consumption figures.

Estimated average concentrations of pyriproxyfen in surface and ground water are 0.011 ppb (after adjustment for the highly conservative nature of the GENEEC model and 0.006 ppb, respectively). The estimated average concentrations of pyriproxyfen in surface and ground water are less than OPP's level of concern for pyriproxyfen in drinking water as a contribution to chronic aggregate exposure. Therefore, taking into account present uses and uses proposed in this action, OPP concludes with reasonable certainty that residues of pyriproxyfen in drinking water (when considered along with other sources of exposure for which OPP has reliable data) would not result in unacceptable levels of aggregate human health risk at this time.

3. *From non-dietary exposure*. Pyriproxyfen is the active ingredient in many registered residential (indoor, non-food) products for flea and tick control. Formulations include foggers, aerosol sprays, emulsifiable concentrates, and impregnated materials (pet collars). Pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine is currently registered for use on the following residential non-food sites: indoor premise, pet bedding, dogs and cats.

i. *Acute exposure and risk*. An acute dietary dose and endpoint was not

identified. Thus the risk from acute aggregate exposure is considered to be negligible.

ii. *Chronic exposure and risk.* Long-term exposure to pyriproxyfen in residential use products is not expected. Therefore there is no chronic risk. Consumer use of these products typically results in short-term, intermittent exposures.

iii. *Short- and intermediate-term exposure and risk.* The Agency concludes that there is reasonable certainty of no harm from short term and intermediate-term dermal and inhalation occupational and residential exposure due to the lack of significant toxicological effects observed.

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.'' 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 pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine 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, pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine 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 pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine has a common mechanism of toxicity with other substances.

D. Aggregate Risks and Determination of Safety for U.S. Population

1. *Acute risk.* An acute dietary dose and endpoint was not identified. Thus the risk from acute aggregate exposure is considered to be negligible.

2. *Chronic risk.* Using the TMRC exposure assumptions described above, EPA has concluded that aggregate exposure to pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine from food will utilize 0.0003% of the RfD for the U.S. population. The major identifiable subgroup with the highest aggregate exposure is children (1-6 years old). See discussion below. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. There are currently no chronic residential scenarios. The estimated average concentrations of pyriproxyfen in surface and ground water are less than OPP's level of concern for pyriproxyfen in drinking water as a contribution to chronic aggregate exposure. Therefore, EPA concludes with reasonable certainty that residues of pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine in drinking water do not contribute significantly to the aggregate chronic human health risk at the present time when considering the present uses and uses proposed by this action.

E. Aggregate Cancer Risk for U.S. Population

Pyriproxyfen is classified as Category E: not carcinogenic in two acceptable animal studies.

F. Aggregate Risks and Determination of Safety for Infants and Children

1. *Safety factor for infants and children*— i. *In general* . In assessing the potential for additional sensitivity of infants and children to residues of pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine, EPA considered data from developmental toxicity studies in the rat and rabbit and a two-generation reproduction study in the rat. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for pre-and post-natal toxicity and the completeness of the database unless EPA determines that a different margin of safety will be safe for infants and children. Margins of safety are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard uncertainty factor (usually 100 for combined inter- and intra-species variability)) and not the additional tenfold MOE/uncertainty factor when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/safety factor.

ii. *Developmental toxicity studies.* In the rat developmental study, the developmental NOEL was 100 mg/kg/day and the maternal NOEL was 100 mg/kg/day. Therefore, there was no prenatal developmental toxicity in the presence of maternal toxicity. Similarly in rabbits, the prenatal developmental NOEL was 300 mg/kg/day and the maternal NOEL was 300 mg/kg/day. Therefore, prenatally exposed fetuses were not more sensitive to the effects of pyriproxyfen than maternal animals.

iii. *Reproductive toxicity study*. In the rat reproduction study, the parental NOEL of 1,000 ppm was identical to the pup NOEL of 1,000 ppm and decreased body weight was seen in both pup and parental animals. This finding demonstrates that there are no extra sensitivities with respect to pre- and post-natal toxicity between adult and infant animals.

iv. *Pre- and post-natal sensitivity*. The oral perinatal and prenatal data demonstrated no indication of increased sensitivity of rats or rabbits to *in utero* and postnatal exposure to pyriproxyfen.

v. *Conclusion*. The 10x factor for infants and children (as required by FQPA) was removed, since there was no special sensitivity for infants and children and the data base is complete. For chronic dietary risk assessment, a UF of 100 is adequate for protection from exposure to pyriproxyfen.

2. *Acute risk*. An acute dietary dose and endpoint was not identified. Thus the risk from acute aggregate exposure is considered to be negligible.

3. *Chronic risk*. Using the conservative exposure assumptions described above, EPA has concluded that aggregate exposure to pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine from food will utilize 0.00053% of the RfD for infants and children. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. There are currently no chronic residential scenarios. The estimated average concentrations of pyriproxyfen in surface and ground water are less than OPP's level of concern for pyriproxyfen in drinking water as a contribution to chronic aggregate exposure. Therefore, OPP concludes with reasonable certainty that residues of pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine in drinking water do not contribute significantly to the aggregate chronic human health risk at the present time when considering the present uses and uses proposed by this action. EPA concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to pyriproxyfen (2-[1-methyl-2-(4-phenoxyphenoxy)ethoxy]pyridine residues.

4. *Short- or intermediate-term risk*. Short-term and intermediate-term dermal and inhalation risk assessments for residential exposure are not required due to the lack of significant toxicological effects observed.

III. Other Considerations

A. Metabolism In Plants and Animals

EPA considers the nature of the residue in cotton to be adequately understood. Metabolism of pyriproxyfen in cotton proceeds through hydroxylation and cleavage of the phenoxy ether linkage, with additional metabolism by oxidation and conjugation reactions. Much of the metabolized pyriproxyfen is reincorporated into natural products. The HED Metabolism Committee previously issued a tentative conclusion (15-JUL-1996) that the residue of concern in plants is pyriproxyfen *per se*. A meeting of the Chemistry Science Advisory Council (25-FEB-1998) confirmed this conclusion for cotton and determined that future food uses involving pyriproxyfen should be reviewed by the HED Metabolism Committee. Metabolism of phenyl-^{14}C pyriproxyfen in poultry proceeds through hydroxylation of the phenoxyphenyl ring, sulfation of the 4′-OH phenoxyphenyl moiety, hydroxylation of the pyridyl ring, and cleavage of the ether linkage. Metabolism of pyridyl-^{14}C pyriproxyfen in poultry proceeds through hydroxylation of the phenoxyphenyl ring, sulfation of the 4′-OH phenoxyphenyl moiety, hydroxylation of the pyridyl ring, cleavage of the ether linkage and oxidation of the side chain. EPA concludes that the nature of the residue in poultry is adequately understood, and that tolerances are not needed.

Metabolism of phenyl-^{14}C pyriproxyfen in goats proceeds through hydroxylation of the phenoxyphenyl and pyridyl rings, sulfation of the 4′-OH phenoxyphenyl moiety, and cleavage of the ether linkage. Metabolism of pyridyl-^{14}C pyriproxyfen in goats proceeds through hydroxylation of the phenoxyphenyl and pyridyl rings, sulfation of the 4′-OH phenoxyphenyl moiety, cleavage of the ether linkage and oxidation of the side chain. EPA concludes that the nature of the residue in ruminants is adequately understood for this present use and that tolerances are not required.

B. Analytical Enforcement Methodology

Residue analytical method RM-33P-2 has undergone validation in EPA laboratories and is suitable to gather residue data and to enforce tolerances.

The multiresidue method will serve as a confirmatory method for residues of pyriproxyfen.

C. Magnitude of Residues

Based on the radioactive metabolic studies and the calculated dietary burden, EPA concludes that the proposed uses on cotton fall under 40 CFR 180.6(a)(3) since there is no reasonable expectation of finite residues in meat, milk, poultry, and eggs and thus tolerances are not required at this time. If additional uses are sought that could result in greater livestock dietary exposure from feedstuffs, the need for milk, meat, poultry and eggs tolerances will be reassessed.

D. International Residue Limits

There are no CODEX, Canadian, or Mexican tolerances for pyriproxyfen residues on cottonseed or cotton gin byproducts. Therefore, international harmonization is not an issue at this time. Pyriproxyfen is scheduled as a new compound for JMPR review (both toxicology and residue chemistry) in 1999.

E. Rotational Crop Restrictions

An acceptable confined accumulation in rotational crops study with Ph-^{14}C and Py-^{14}C pyriproxyfen was submitted . The study showed no significant uptake (<0.01 ppm) of radioactive residues (pyriproxyfen) by lettuce, radish, or wheat. The majority of the ^{14}C was found in the unextractable material in the post extraction solids. These findings indicated that the ^{14}C has been reincorporated in other, non-pyriproxyfen related compounds. Therefore a plant back interval is not necessary for cotton treated with pyriproxyfen.

IV. Conclusion

Therefore, tolerances are established for combined residues of pyriproxfen in cotton seed and cotton gin byproducts at 0.05 and 2.0 ppm respectively.

V. 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 September 4, 1998, 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.

VI. Public Record and Electronic Submissions

EPA has established a record for this rulemaking under docket control number [OPP–300666] (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 Room 119 of the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Crystal Mall #2, 1921 Jefferson Davis Hwy., 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.

VII. Regulatory Assessment Requirements

This final rule establishes tolerances 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).

In addition, since these tolerances and exemptions that are established on the basis of a petition under FFDCA section 408(d), such as the tolerances 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.

VIII. 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: June 18, 1998.

Stephen L. Johnson,

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 180.534 is added to subpart C to read as follows:

§ 180.534 Pyriproxyfen; tolerances for residues.

(a) *General.* Tolerances are established for combined residues of the insecticide pyriproxyfen in or on the following agricultural commodities:

Commodity	Parts per million
Cotton gin byproducts ...	2.0

Commodity	Parts per million
Cottonseed	0.05

(b) *Section 18 emergency exemptions.* [Reserved]

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

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

[FR Doc. 98–17729 Filed 7-2-98; 8:45 am]

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DEPARTMENT OF TRANSPORTATION

Research and Special Programs Administration

49 CFR Part 195

[Docket No. RSPA–97–2362; Amdt. 195–62]

RIN 2137—AD05

Pipeline Safety: Incorporation by Reference of Industry Standard on Leak Detection

AGENCY: Research and Special Programs Administration (RSPA).

ACTION: Final rule.

SUMMARY: This rule adopts as a referenced document an industry publication for pipeline leak detection, API 1130, ''Computational Pipeline Monitoring,'' published by the American Petroleum Institute (API). This rule requires that an operator of a hazardous liquid pipeline use API 1130 in conjunction with other information, in designing, evaluating, operating, maintaining, and testing its software-based leak detection system. The use of this document will significantly advance the acceptance of leak detection technology on hazardous liquid pipelines. However, this rule does not require operators to install such systems.

DATES: This final rule takes effect July 6, 1999.

FOR FURTHER INFORMATION CONTACT: Lloyd W. Ulrich, telephone: (202) 366–4556, FAX: (202) 366–4566, e-mail: lloyd.ulrich@rspa.dot.gov regarding the subject matter of this final rule, or Dockets Unit, (202) 366–4453, for copies of this final rule or other material in the docket. Further information can be obtained by accessing OPS' Internet Home Page at: ops.dot.gov.

SUPPLEMENTARY INFORMATION:

I. Background on Requiring Leak Detection Equipment

A. Congressional Mandate To Issue Regulations

Congress, in section 212 of the Pipeline Safety Act of 1992 (codified at 49 U.S.C. 60102(j)), required the Secretary of Transportation, by October 24, 1994, to survey and assess the effectiveness of emergency flow restricting devices (EFRDs) and other procedures, systems, and equipment used to detect and locate hazardous liquid pipeline ruptures and minimize product releases from hazardous liquid pipeline facilities. Congress further mandated that the Secretary issue regulations two years after completing the survey and assessment (no later than October 24, 1996). These regulations would prescribe the circumstances under which hazardous liquid pipeline operators would use EFRDs or other procedures, systems, and equipment used to detect and locate pipeline ruptures and minimize product releases from pipeline facilities. The Secretary delegated this authority to the Research and Special Programs Administration (RSPA).

B. Advance Notice of Proposed Rulemaking, Volpe Center Report and Public Workshop

RSPA used several means to gather information on EFRDs and leak detection equipment. We issued an advance notice of proposed rulemaking (ANPRM) (59 FR 2802, Jan. 19, 1994) to solicit information primarily from hazardous liquid pipeline operators about operational data and costs related to EFRDs and about the performance of leak detection systems to detect and locate hazardous liquid pipeline ruptures and minimize product release. The ANPRM also sought information to help determine which critical pipeline locations should be protected from product releases. Commenters provided limited usable data and generally opposed requiring leak detection equipment and EFRDs.

We contracted with the Volpe National Transportation Systems Center (Volpe Center) to conduct a research study on SCADA [1] systems, including leak detection systems. Its report, ''Remote Control Spill Reduction Technology: A Survey and Analysis of Applications for Liquid Pipeline Systems'' (September 29, 1996), found that because of the pipeline industry's diversity, each system used for leak detection must be custom configured for a particular pipeline system, that SCADA and leak detection systems were dependent on the sophistication of the host computer and how rapidly and diverse remote field data can be collected, and that operators have invested in SCADA systems, but have invested much less in software-based leak detection systems.

RSPA also held a public workshop on October 19, 1995, to obtain more data on EFRDs and leak detection systems. Participants confirmed the Volpe Center report's finding that each leak detection system is unique to the pipeline on which it is installed. Discussions included operational and economic problems with leak detection systems, as well as their operational, economic and environmental benefits.

Detailed discussion of the ANPRM, Volpe Center report, and workshop can be found at 62 FR 56141; October 29, 1997.

C. Development of API 1130

In 1994, the API formed a task force to develop a document on computational pipeline monitoring (CPM). The task force produced API 1130, entitled ''Computational Pipeline Monitoring,'' which addressed the use of software-based leak detection equipment. API 1130 defines computational pipeline monitoring as ''an algorithmic monitoring tool that allows the pipeline controller to respond to a pipeline operating anomaly which may be indicative of a commodity release.'' The document's stated purpose is to assist the pipeline operator in selecting, implementing, testing, and operating a CPM system, and to help to identify the complexities, limitations, and other implications of detecting anomalies on liquid pipelines using CPM systems.

RSPA and the Volpe Center staff monitored the task force's work. Minutes of the task force meetings, and copies of final drafts of API 1130, are available in Docket No. PS–133.

D. Definition of Areas Unusually Sensitive to Environmental Damage

Congress required that in prescribing standards, RSPA identify the circumstances where EFRDs and other equipment must be installed. RSPA's current policy is to base regulations on risk assessment. We believe that a

[1] SCADA is an acronym for Supervisory Control and Data Acquisition. SCADA systems utilize computer technology to continuously gather data (e.g., pressure, temperature, and delivery flow rates) from remote locations on the pipeline. Dispatchers use SCADA systems to assist in day-to-day operating decisions on the pipeline. SCADA systems can also provide input for real-time models of the pipeline operation. Such models compare current operating conditions with calculated data values. A deviation may indicate the possibility of a leak.