tetramethyl-1,3,5,7-tetroxocane, in or on the commodity.

Commodity	Parts per million
Artichoke, globe	0.07
Berry, low growing, subgroup 13–07G	6.25
Bushberry subgroup 13–07B	0.15
Cactus	0.07
Caneberry subgroup 13–07A	0.15
Corn, field, forage	0.30
Corn, field, grain	0.05
Corn, field, stover	0.10
Corn, sweet, forage	0.30
Corn, sweet, kernel plus cob with husks removed	0.05
Corn, sweet, stover	0.10
Fruit, citrus, group 10	0.26
Grass, forage	2.0
Grass, hay	2.0
Leaf petioles subgroup 4B	0.50
Lettuce	1.73
Peppermint, oil	12
Peppermint, tops	4.0
Spearmint, oil	12
Spearmint, tops	4.0
Taro, corm	0.15
Taro, leaves	1.0
Tomato	0.24
Vegetable, brassica, leafy, group 5	2.5
Watercress	3.2

* * * * *

(c) *Tolerances with regional registrations.* Tolerances with a regional registration as defined in § 180.1(l) are established for residues of the molluscicide metaldehyde, including its metabolites and degradates, in or on the following commodities. Compliance with the specified tolerance level is to be determined by measuring only metaldehyde, 2,4,6,8-tetramethyl-1,3,5,7-tetroxocane, in or on the commodity.

Commodity	Parts per million
Soybean, seed	0.05

* * * * *

[FR Doc. 2013–28370 Filed 11–26–13; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2011–0905; FRL–9902–39]

Etofenprox; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for residues of etofenprox in or on multiple commodities which are identified and discussed later in this document. Interregional Research Project Number 4 (IR–4) requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective November 27, 2013. Objections and requests for hearings must be received on or before January 27, 2014, and must be filed in accordance with the instructions provided in 40 CFR part 178 (see also Unit I.C. of the **SUPPLEMENTARY INFORMATION**).

ADDRESSES: The docket for this action, identified by docket identification (ID) number EPA–HQ–OPP–2011–0905, is available at *http://www.regulations.gov* or at the Office of Pesticide Programs Regulatory Public Docket (OPP Docket) in the Environmental Protection Agency Docket Center (EPA/DC), EPA West Bldg., Rm. 3334, 1301 Constitution Ave. NW., Washington, DC 20460–0001. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566–1744, and the telephone number for the OPP Docket is (703) 305–5805. Please review the visitor instructions and additional information about the docket available at *http://www.epa.gov/dockets.*

FOR FURTHER INFORMATION CONTACT: Lois Rossi, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001; telephone number: (703) 305–7090; email address: *RDFRNotices@epa.gov.*

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this action apply to me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. The following list of North American Industrial Classification System (NAICS) codes is not intended to be exhaustive, but rather provides a guide to help readers determine whether this document applies to them. Potentially affected entities may include:

- Crop production (NAICS code 111).
- Animal production (NAICS code 112).
- Food manufacturing (NAICS code 311).
- Pesticide manufacturing (NAICS code 32532).

B. How can I get electronic access to other related information?

You may access a frequently updated electronic version of EPA's tolerance regulations at 40 CFR part 180 through the Government Printing Office's e-CFR site at *http://www.ecfr.gov/cgi-bin/text-idx?&c=ecfr&tpl=/ecfrbrowse/Title40/40tab_02.tpl.*

C. How can I file an objection or hearing request?

Under FFDCA section 408(g), 21 U.S.C. 346a, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. You must file your objection or request a hearing on this regulation in accordance with the instructions provided in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket ID number EPA–HQ–OPP–2011–0905 in the subject line on the first page of your submission. All objections and requests for a hearing must be in writing, and must be received by the Hearing Clerk on or before January 27, 2014. Addresses for mail and hand delivery of objections and hearing requests are provided in 40 CFR 178.25(b).

In addition to filing an objection or hearing request with the Hearing Clerk as described in 40 CFR part 178, please submit a copy of the filing (excluding any Confidential Business Information (CBI)) for inclusion in the public docket. Information not marked confidential pursuant to 40 CFR part 2 may be disclosed publicly by EPA without prior notice. Submit the non-CBI copy of your objection or hearing request, identified by docket ID number EPA–HQ–OPP–2011–0905, by one of the following methods:

- *Federal eRulemaking Portal: http://www.regulations.gov.* Follow the online instructions for submitting comments. Do not submit electronically any information you consider to be CBI or other information whose disclosure is restricted by statute.
- *Mail:* OPP Docket, Environmental Protection Agency Docket Center (EPA/DC), (28221T), 1200 Pennsylvania Ave. NW., Washington, DC 20460–0001.
- *Hand Delivery:* To make special arrangements for hand delivery or delivery of boxed information, please follow the instructions at *http://www.epa.gov/dockets/contacts.html.* Additional instructions on commenting or visiting the docket, along with more information about dockets generally, is available at *http://www.epa.gov/dockets.*

II. Summary of Petitioned-For Tolerance

In the **Federal Register** of December 8, 2011 (76 FR 76674) (FRL–9328–8), EPA issued a document pursuant to FFDCA section 408(d)(3), 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 1E7925) by Interregional Research Project No. 4 (IR–4), 500 College Road East, Suite 201 W, Princeton, NJ 08540. The petition requested that 40 CFR 180.620 be amended by establishing tolerances for residues of the insecticide etofenprox, [2-(4-ethoxyphenyl)-2-methylpropyl 3-phenoxybenzyl ether], in or on food and feed commodities at 0.5 parts per million (ppm). That document referenced a summary of the petition prepared by Mitsui, the registrant, which is available in the docket, *http://www.regulations.gov.* There were no comments received in response to the notice of filing.

Currently there are two products that contain etofenprox registered for mosquito control. However, the existing registrations do not allow treatments on or over agricultural areas. IR–4 submitted this petition to establish tolerances for residues of etofenprox in or on food and feed commodities so that the registration can be modified to allow repeated applications (aerial and ground) over agricultural crops, pasture and rangeland.

Based upon review of the data supporting the petition, EPA has modified the level at which tolerances are being established. The reason for this change is explained in Unit IV.C.

III. Aggregate Risk Assessment and Determination of Safety

Section 408(b)(2)(A)(i) of 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) of FFDCA 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) of FFDCA 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. . . .''

Consistent with FFDCA section 408(b)(2)(D), and the factors specified in FFDCA 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 and to make a determination on aggregate exposure for etofenprox including exposure resulting from the tolerances established by this action. EPA's assessment of exposures and risks associated with etofenprox 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.

In mammals, the major targets of etofenprox are the liver, thyroid, kidney, and hematopoietic system. Results from subchronic and chronic feeding studies in rats indicate that males may be more sensitive to treatment-related effects of etofenprox than females. All subchronic and chronic toxicity including carcinogenicity studies showed adverse effects (organ weights, histopathology, biochemistry, hematology, and clinical chemistry) in two or more of the target organs/systems. Additionally, decreases in body weights and food consumption were observed in most of the studies.

In a mouse carcinogenicity study, the kidney was the most sensitive target organ, especially in males, and many deaths were attributed to renal lesions. Males showed a positive trend in renal cortical adenomas alone and in combined carcinomas and adenomas; however, tumor incidence was within the historical control range. Other effects included decreased body and thymus gland weights, and increased liver, spleen, and pituitary gland weights. Microscopic changes included centrilobular hepatocyte enlargement.

Relevant toxicity studies showed no quantitative or qualitative evidence of increased susceptibility in offspring. A prenatal developmental toxicity study in rabbits showed no quantitative or qualitative evidence of increased susceptibility in offspring, in that the developmental effects were seen at doses that resulted in maternal toxicity, including death. There was no indication of increased susceptibility of offspring in the 1-generation/developmental study in rats. In the developmental portion of the study, effects were seen in maternal animals, while no effects were observed in the offspring. In the 2-generation reproductive toxicity study in rats, there was also no evidence of increased susceptibility of offspring.

Although etofenprox exposure does result in some neurotoxic effects, these effects only occur at high doses. An acute neurotoxicity study in the adult rat revealed no treatment-related effects. The subchronic neurotoxicity study in the rat showed decreased body weight gains, increased liver weights in all dose groups, and increased incidence of rearing behavior in males and abnormal gait in females. The developmental neurotoxicity study in rats showed increased rearing behavior in mothers at the highest dose tested (HDT). In offspring, eye abnormalities were observed at the high-dose level and effects on motor/locomotor activity and auditory startle response observed at the high-dose level.

The immunotoxicity studies in the rat and mouse were both negative for immunotoxicity.

The cancer classification for etofenprox is ''Not likely to be carcinogenic to humans at doses that do not alter rat thyroid hormone homeostasis.'' This decision was based on the following considerations:

i. Treatment-related thyroid follicular cell tumors were seen in both male and female rats at a dose level considered to be adequate, and not excessive, to assess carcinogenicity;

ii. No treatment-related tumors were seen in male or female mice when tested at a dose that was considered adequate to assess carcinogenicity;

iii. There is no mutagenicity concern for etofenprox based *in vivo* or *in vitro* assays;

iv. The non-neoplastic toxicological evidence (*i.e.,* thyroid growth and thyroid hormonal changes) indicates that etofenprox disrupts the thyroid-pituitary hormonal status; and

v. Rats are substantially more sensitive than humans to the development of thyroid follicular cell tumors in response to thyroid hormone imbalance. The overall weight-of-the-evidence was considered sufficient to indicate that etofenprox induced thyroid follicular tumors through an antithyroid mode of action.

Specific information on the studies received and the nature of the adverse effects caused by etofenprox as well as the no-observed-adverse-effect-level (NOAEL) and the lowest-observed-adverse-effect-level (LOAEL) from the toxicity studies can be found at *http://www.regulations.gov* in the document

titled ''Etofenprox: Section 3 Aggregate Human Health Risk Assessment for a Label Amendment to Remove Application Restriction Over Crop, Range, and Pasture land,'' pp. 36–41 docket ID number EPA–HQ–OPP–2011–0905.

B. Toxicological Points of Departure/ Levels of Concern

Once a pesticide's toxicological profile is determined, EPA identifies toxicological points of departure (POD) and levels of concern to use in evaluating the risk posed by human exposure to the pesticide. For hazards that have a threshold below which there is no appreciable risk, the toxicological POD is used as the basis for derivation of reference values for risk assessment. PODs are developed based on a careful analysis of the doses in each toxicological study to determine the dose at which no adverse effects are observed (the NOAEL) and the lowest dose at which adverse effects of concern are identified (the LOAEL). Uncertainty/ safety factors are used in conjunction with the POD to calculate a safe exposure level—generally referred to as a population-adjusted dose (PAD) or a reference dose (RfD)—and a safe margin of exposure (MOE). For non-threshold risks, the Agency assumes that any amount of exposure will lead to some degree of risk. Thus, the Agency estimates risk in terms of the probability of an occurrence of the adverse effect expected in a lifetime. For more information on the general principles EPA uses in risk characterization and a complete description of the risk assessment process, see *http://www.epa.gov/pesticides/factsheets/riskassess.htm*.

A summary of the toxicological endpoints for etofenprox used for human risk assessment is shown in the Table of this unit.

TABLE—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR ETOFENPROX FOR USE IN HUMAN HEALTH RISK ASSESSMENT

Exposure/scenario	Point of departure and uncertainty/safety factors	RfD, PAD, LOC for risk assessment	Study and toxicological effects
Acute dietary (All populations) ..	No adverse effects attributable to a single dose were observed in oral toxicity studies, including developmental toxicity studies in rats and rabbits. Therefore, an acute reference dose was not established.		
Chronic dietary (All populations)	NOAEL = 3.7 mg/kg/day. UF_A = 10x UF_H = 10x FQPA SF = 1x	cRfD = 0.037 mg/kg/day. cPAD = 0.037 mg/kg/day	Combined Chronic Toxicity/Carcinogenicity Study in Rat. LOAEL = 25.5 mg/kg/day based on increased thyroid weights. Related to increased liver weights and histopathology changes in liver and thyroid that occurred at the higher dose.
Incidental oral short- and intermediate-term (1 to 30 days and 1 to 6 months).	NOAEL = 20 mg/kg/day. UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	Subchronic Oral Toxicity in Rat. LOAEL = 120 mg/kg/day based on decreased body weight gain, increased liver and thyroid weights with corresponding histopathology, changes in hematology and clinical chemistry.
Incidental oral long-term (> 6 months).	NOAEL = 3.7 mg/kg/day. UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	Combined Chronic Toxicity/Carcinogenicity Study in Rat. LOAEL = 25.5 mg/kg/day based on increased thyroid weights. Related to increased liver weights and histopathology changes in liver and thyroid that occurred at the higher dose.
Inhalation short- and intermediate-term (1 to 30 days and 1 to 6 months).	Inhalation study ..... NOAEL = 10.6 mg/kg/day. UF_A = 10x UF_H = 10x FQPA SF = 1x	LOC for MOE = 100	13-Week Inhalation Toxicity in Rat. LOAEL = 52.3 mg/kg/day based on organ weight changes and histopathological changes in liver, adrenals and thyroid.
Cancer (Oral, dermal, inhalation).	Classification: ''Not likely to be carcinogenic to humans at doses that do not alter rat thyroid hormone homeostasis.''		

FQPA SF = Food Quality Protection Act Safety Factor. LOAEL = lowest-observed-adverse-effect-level. LOC = level of concern. mg/kg/day = milligram/kilogram/day. MOE = margin of exposure. NOAEL = no-observed-adverse-effect-level. PAD = population adjusted dose (a = acute, c = chronic). RfD = reference dose. UF = uncertainty factor. UF_A = extrapolation from animal to human (interspecies). UF_H = potential variation in sensitivity among members of the human population (intraspecies).

C. Exposure Assessment

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to etofenprox, EPA considered exposure under the petitioned-for tolerances as well as all existing etofenprox tolerances in 40 CFR 180.620. EPA assessed dietary exposures from etofenprox in food as follows:

i. *Acute exposure.* Quantitative acute dietary exposure and 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 1-day or single exposure. No such effects were identified in the toxicological studies for etofenprox; therefore, a quantitative acute dietary exposure assessment is unnecessary.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the U.S. Department of Agriculture's (USDA's) National Health and Nutrition Examination Survey, What We Eat in America (NHANES/ WWEIA). The assessment assumed tolerance level residues for all commodities, incorporated estimated percent crop treated (PCT) values, and used the Dietary Exposure Evaluation Model Food Commodity Intake Database (DEEM–FCID) default processing factors. The submitted crop field trial data were conducted at a rate (0.07 lb ai/A) 10X greater than the proposed application rate of at 0.007 lb ai/A per site for mosquito control. The number and locations of field trials were in accordance with the initial

recommendations put forth by the EPA. EPA recommended field trials be conducted at the 1x and 10x rates and indicated that if there were residues detected in the samples collected above the limit of quantification (LOQ) at both 1x and 10x rates, then a tolerance would be required at the level observed at the 1x rate. However, the available crop field trial data do not reflect the number of applications proposed or the use of ground application equipment. Therefore, the Agency considered an analysis submitted by IR–4 of different modeled runs to estimate the residues resulting from multiple aerial applications using the Terrestrial Residue Exposure (TREX) model following repeated ultra low volume (ULV) applications to estimate an upper bound tolerance value. The EPA also evaluated the proposed multiple application scenarios using AGricultural DISPersal (AGDISP) 8.25 and assumed the same application parameters (*e.g.,* drop size distribution, application material, and application height) as considered in the TREX analysis. A deposition rate of 33% was assumed for aerial and ground ULV applications, which corresponds to a residue value of 4.8 ppm (to represent the worst case) with a wind speed of 1 mph. These analysis result in estimated an upper bound value of 4.77 ppm for ground and aerial applications. Therefore, the EPA determined that a tolerance of 5 ppm, which is based on conservative assumptions, is adequate to cover the expected residues. The proposed tolerance of 5 ppm on food and feed commodities significantly increases the dietary burdens of etofenprox in livestock and necessitates establishing tolerances on livestock commodities.

Specific information on the TREX and AGDISP analyses can be found at *http://www.regulations.gov* in the document titled ''Spray Drift Analysis for the Etofenprox Label Amendment (Petition No. 1E7925)'' docket ID number EPA–HQ–OPP–2011–0905.

iii. *Cancer.* Based on the data summarized in Unit III.A., EPA has concluded that etofenprox does not pose a cancer risk to humans at doses that do not alter rat thyroid hormone homeostasis. Because the cPAD is protective of etofenprox's effect on thyroid hormones and dietary exposure to etofenprox for the purpose of assessing cancer risk would be the same or lower than dietary exposure relevant to other chronic endpoints, a dietary exposure assessment for the purpose of assessing cancer risk is unnecessary.

iv. *Anticipated residue and PCT information.* Section 408(b)(2)(F) of FFDCA states that the Agency may use data on the actual percent of food treated for assessing chronic dietary risk only if:

- Condition a: The data used are reliable and provide a valid basis to show what percentage of the food derived from such crop is likely to contain the pesticide residue.
- Condition b: The exposure estimate does not underestimate exposure for any significant subpopulation group.
- Condition c: Data are available on pesticide use and food consumption in a particular area, the exposure estimate does not understate exposure for the population in such area.

In addition, the Agency must provide for periodic evaluation of any estimates used. To provide for the periodic evaluation of the estimate of PCT as required by FFDCA section 408(b)(2)(F), EPA may require registrants to submit data on PCT.

In most cases, EPA uses available data from United States Department of Agriculture/National Agricultural Statistics Service (USDA/NASS), proprietary market surveys, and the National Pesticide Use Database for the chemical/crop combination for the most recent 6–7 years. EPA uses an average PCT for chronic dietary risk analysis. The average PCT figure for each existing use is derived by combining available public and private market survey data for that use, averaging across all observations, and rounding to the nearest 5%, except for those situations in which the average PCT is less than one. In those cases, 1% is used as the average PCT and 2.5% is used as the maximum PCT. EPA uses a maximum PCT for acute dietary risk analysis. The maximum PCT figure is the highest observed maximum value reported within the recent 6 years of available public and private market survey data for the existing use and rounded up to the nearest multiple of 5%.

The Agency estimated the PCT for proposed uses of etofenprox as a mosquito adulticide which may result in residues on food and feed commodities. The PCT estimates are for 35 agricultural crops which may be exposed to mosquito adulticide applications of etofenprox. The agricultural crops included in the analysis are apples, pears, oranges, rice, field com, wheat, and 29 crops grown predominantly in California. The EPA relied on national and state level usage data for the most widely used mosquito insecticides to develop percent crop treated estimates for new uses. The general approach to estimating PCT was to assume that all etofenprox mosquito adulticide applications will be made randomly across the landscape without regard to land use patterns. Except for area wide vector control programs, this approach is highly conservative in that mosquito adulticide applications are generally made to populated urban and suburban areas. However, because of the inherent drift of mosquito adulticides into non-target areas, it is realistic to assume that some residues of etofenprox may be found on agricultural crops in the urban-agricultural interface. Using this approach, PCT estimates including residues on rice, which is a registered use, are as follows:

Apples: 1%; almonds: 5%; apricots: 5%; artichokes: 5%; avocados: 5%; broccoli: 5%; Brussels sprouts: 5%; carrots: 5%; cauliflower: 5%; celery: 5%; chicory: 5%; dates: 5%; field corn: 1%; figs: 5%; garlic: 5%; grapes: 5%; honeydew melon: 5%; kiwifruit: 5%; lemons: 5%; nectarines: 5%; olives: 5%; oranges: 15%; pears: 1%; persimmons: 5%; pistachios: 5%; plums: 5%; pluots: 5%; pomegranates: 5%; prunes: 5%; raisins: 5%; rice: 3%; tomatoes: 5%; walnuts: 5%; wheat: 1%; all other crops: (including livestock commodities, milk, and eggs) 3%.

The Agency used the market leader approach to develop upper bound percent crop treated estimates for this new use. Under the market leader approach, this upper bound is estimated as the percent of the crop treated by the most widely used pesticide for the new use. The EPA's usual application of the market leader approach for deriving PCT traditionally focuses on broad categories of pesticides (e.g., insecticides, fungicides, or herbicides) applied directly to crops for control of agricultural pests. In this case, however, EPA determined that this would not be appropriate because mosquito adulticides fill a unique niche in the pesticide marketplace. The amount of general insecticide use on crops has no rational relationship to the amount of mosquito adulticide use. Instead of using the insecticides applied directly on these crops, EPA chose the most widely used mosquito adulticide in the states/regions that the crop is grown in. For occasional area wide vector control programs for West Nile Virus (WNV) or Vector-borne encephalitis (Western Equine Encephalitis, Eastern Equine Encephalitis, or St. Louis Encephalitis) this approach provides an accurate estimate of the PCT for agricultural crops.

These estimates represent the upper bound of use expected during the pesticide's initial five years of registration; that is, PCT for etofenprox is a threshold of use that EPA is reasonably certain will not be exceeded

for each registered use site. The PCT recommended for use in the chronic dietary assessment is calculated as the average PCT of the market leader or leaders, (i.e., the one(s) with the greatest PCT) on that site over the three most recent years of available data. The comparisons are only made among pesticides of the same pesticide type (e.g., the market leader for insecticides on the use site is selected for comparison with a new insecticide). The market leader included in the estimation may not be the same for each year since different pesticides may dominate at different times. Typically, EPA uses USDA National Agricultural Statistics Service (NASS) as the source of data because it is publicly available and directly reports values for PCT. When a specific use site is not reported by USDA/NASS, EPA uses proprietary data and calculates the PCT given reported data on acres treated and acres grown. If no data are available, EPA may extrapolate PCT from other crops (proxies), if the crop management and pest spectrum are substantially similar. A retrospective analysis to validate this approach shows few cases where the PCT for the market leaders were exceeded. Further review of these cases identified factors contributing to the exceptionally high use of a new pesticide. Given the results of this review, to evaluate whether the PCT for etofenprox could be exceeded, EPA considered whether there may be unusually high mosquito pressure or disease transmission potential; whether the market leaders are well established for that use; and whether pest resistance issues with past market leaders provide etofenprox with significant market potential. Given currently available information, EPA concludes it is unlikely that actual PCT for etofenprox will exceed the estimated PCT for new uses during the next five years.

Specific information on the methodology to estimate PCT can be found at *http://www.regulations.gov* in the document titled ''BEAD Estimate of the Percent Crop Treated for New Use (PCTn) of Etofenprox when used as a Mosquito Adulticide in Agricultural Areas'' docket ID number EPA–HQ–OPP–2011–0905.

The Agency believes that the three conditions discussed in Unit III.C.1.iv. have been met. With respect to Condition a, PCT estimates are derived from Federal and private market survey data, which are reliable and have a valid basis. The Agency is reasonably certain that the percentage of the food treated is not likely to be an underestimation. As to Conditions b and c, regional consumption information and consumption information for significant subpopulations is taken into account through EPA's computer-based model for evaluating the exposure of significant subpopulations including several regional groups. Use of this consumption information in EPA's risk assessment process ensures that EPA's exposure estimate does not understate exposure for any significant subpopulation group and allows the Agency to be reasonably certain that no regional population is exposed to residue levels higher than those estimated by the Agency. Other than the data available through national food consumption surveys, EPA does not have available reliable information on the regional consumption of food to which etofenprox may be applied in a particular area.

2. *Dietary exposure from drinking water.* The Agency used screening level water exposure models in the dietary exposure analysis and risk assessment for etofenprox in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of etofenprox. Further information regarding EPA drinking water models used in pesticide exposure assessment can be found at *http://www.epa.gov/oppefed1/models/water/index.htm.*

Based on the Tier 1 Rice Model and Screening Concentration in Ground Water (SCI–GROW) models, the estimated drinking water concentrations (EDWCs) of etofenprox for chronic exposures are estimated to be 1.2 ppb for surface water and 3.0×10^{-3} ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For chronic dietary risk assessment, the water concentration of value 1.2 ppb was used to assess the contribution to drinking water.

3. *From non-dietary exposure.* The term ''residential exposure'' is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiticides, and flea and tick control on pets). Etofenprox is currently registered for the following uses that could result in residential exposures: Cat and dog spot-on treatments, as a bed bug treatment, as indoor space and crack and crevice sprays, and as indoor and outdoor foggers. EPA assessed residential exposure using the following assumptions: Adults can potentially be exposed to etofenprox residues during residential application of etofenprox, including indoor surface-directed and aerosol space spray and outdoor fogger use. Handler exposure is expected to be short-term in duration and because there was no adverse dermal effect identified for etofenprox, risk was assessed only for exposure via the inhalation route. There is also potential for post-application exposure for individuals as a result of being in an environment that has been previously treated with etofenprox. Because of the registered indoor uses, intermediate-term post application exposures are possible. However, since the short- and intermediate-term endpoints and PODs for inhalation and oral routes are the same, the short-term exposure and risk estimates are considered to be protective of potential intermediate-term exposure and risk. Because adverse dermal toxicity effects were not identified for etofenprox, only short- and intermediate-term post-application inhalation exposures were assessed for adults and short- and intermediate-term post-application inhalation and incidental oral exposures were assessed for children. Additionally, long-term post-application incidental oral exposure to children from petting treated cats or dogs was also assessed.

The worst-case residential short-term exposure for adults is from post-application inhalation exposure from treatment of flying insects. The worst-case residential short-term exposure for children 1 to 2 years old is from combined inhalation and oral hand-to-mouth post-application exposures from treatment of flying insects. EPA typically combines exposures for treatments to control the same pests (e.g. flea treatment on surfaces and on pets) because such treatments could reasonably be expected to occur on the same day. But a similar presumption is not generally followed for exposures for treatments to control different pests. For etofenprox, EPA has not combined short-term exposures from use of etofenprox to control flying insects and its use to control fleas, ticks, and bed bugs. Several factors support this approach for etofenprox. First, EPA's manner of estimating short-term residential exposures is very conservative. When assessing individual short-term residential post-application exposure scenarios, EPA assumes exposure occurs at the level of zero-day residues (i.e., day of application residues) on each day of the short-term exposure period (1–30 days), instead of incorporating information on residue decline values. EPA also assumes that an individual performs the same post-application activities, intended to represent high-end exposures as described in the Residential SOPs, for

the same amount of time every day over the short-term exposure period, rather than averaging post-application activity levels and exposures over that period. Second, these exposure estimates are then compared to points of departure that are typically based on weeks of dosing in test animals. Longer exposure periods generally produce lower points of departure. For etofenprox, the short-term risk assessment is particularly conservative because the point of departure for the short-term (1 to 30-days) risk assessment is based on a toxicity study involving continuous exposure over 90 days. Third, usage survey data indicate that concurrent use of separate pesticide products that contain the same active ingredient to treat the same or different pests does not typically occur. Combining conservative exposure estimates with a conservative point of departure for an event that is itself improbable (co-occurrence of use of the same pesticide to control different pests) would unrealistically overstate exposure.

Further information regarding EPA standard assumptions and generic inputs for residential exposures may be found at *http://www.epa.gov/pesticides/trac/science/trac6a05.pdf.*

4. *Cumulative effects from substances with a common mechanism of toxicity.* Section 408(b)(2)(D)(v) of FFDCA 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.''

Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, EPA has not made a common mechanism of toxicity finding as to etofenprox. Although etofenprox shares some structural characteristics with synthetic pyrethroids, it is not included in the pyrethroid cumulative assessment. Naturally occurring pyrethrins and the synthetic pyrethroids (collectively called 'pyrethroids') are grouped for purposes of cumulative risk assessed based on the following shared characteristics:

i. *Common structure.* Pyrethrins and pyrethroids share a common structure; acid and alcohol moieties joined through an ether linkage;

ii. *Sodium channel disruption. In vitro* studies demonstrate the ability of pyrethroids to modify mammalian sodium channel kinetics, leading to alterations in membrane excitability and firing potentials;

iii. *Neurotoxic effects.* Pyrethroid toxicity is manifested through neurological syndromes described as either T (fine tremors), CS (choreoathetosis and salivation), or some combination thereof, depending on the structure. Open literature supports a correlation between the modification in sodium channel kinetics and the resulting syndrome.

Etofenprox is not included in the pyrethroid common mechanism grouping or included in the cumulative risk assessment because etofenprox does not exhibit these key characteristics. Etofenprox is an ether compound; pyrethroids are esters. Etofenprox exposure does not result in the neurotoxic syndromes typical of pyrethroids and no available data suggest the molecular target for etofenprox is the sodium channel.

For the purposes of this tolerance action, therefore, EPA has not assumed that etofenprox has a common mechanism of toxicity with other pyrethroids. For information regarding EPA's efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the policy statements released by EPA's Office of Pesticide Programs concerning common mechanism determinations and procedures for cumulating effects from substances found to have a common mechanism on EPA's Web site at *http://www.epa.gov/pesticides/cumulative/.*

D. Safety Factor for Infants and Children

1. *In general.* Section 408(b)(2)(C) of FFDCA provides that EPA shall apply an additional tenfold (10X) margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the database on toxicity and exposure unless EPA determines based on reliable data that a different margin of safety will be safe for infants and children. This additional margin of safety is commonly referred to as the FQPA Safety Factor (SF). In applying this provision, EPA either retains the default value of 10X, or uses a different additional safety factor when reliable data available to EPA support the choice of a different factor.

2. *Prenatal and postnatal sensitivity.* There is no indication of increased quantitative or qualitative susceptibility of the developing offspring in toxicology database for etofenprox. Developmental effects were seen at doses that caused maternal toxicity. No developmental effects were seen in the rat 1-generation/developmental study. In the 2-generation reproduction toxicity study, toxicity in the offspring occurred at the level of parental toxicity (increased organs weights and associated pathological changes occurred in both the pups and parents). In the developmental neurotoxicity study in rats, the observed eye abnormalities associated with body injuries could not be disassociated from possible altered, treatment-related maternal behavior.

3. *Conclusion.* EPA has determined that reliable data show the safety of infants and children would be adequately protected if the FQPA SF were reduced to 1x. That decision is based on the following findings:

i. The toxicity database for etofenprox is complete.

ii. An acute neurotoxicity study in the adult rat revealed no treatment-related effects. The subchronic neurotoxicity study in the rat showed decreased body weight gains, increased liver weights in all dose groups, and increased incidence of rearing behavior and abnormal gait, all in the absence of histopathological changes. The developmental neurotoxicity study in rats showed increased rearing behavior in mothers. In offspring, eye lesions (including sclera and lens hemorrhage), which are sometimes associated with aggressive maternal behavior, were observed prior to weaning at the highest dose tested. Effects on motor/locomotor activity and auditory startle response were also observed in the high-dose treatment groups on PND 58. These latter isolated, post-ontogenic effects of treatment are not presumed to occur following a single dose.

Evidence of neurotoxicity was also observed in other studies. In a subchronic mouse study piloerection, hunched posture, lethargy, body tremors, and an unsteady gait were noted in both sexes above the limit dose. The rat developmental study showed increased salivation in all treatment groups of the F_0 generation and decreased (non-statistically significant) mobility (both sexes) and rearing behavior (males) in the F_1 generation. In the 2-generation reproduction study F_1 pups exhibited clinical signs of body tremors, lethargy, unsteady gait, and abnormal movements during most of the lactation period at the high dose.

However, residual concern for neurotoxicity is low based on the following:

a. Signs of neurotoxicity in the database occur only at the high dose level in each study;

b. The studies show clear and well-defined NOAELs;

c. The signs of neurotoxicity are well-characterized in terms of their effects in offspring; and

d. The PODs used for risk assessment are protective of neurotoxicity seen in the database.

No systemic toxicity was observed in the 28-day dermal study in rabbits up to 1,000 mg/kg/day. In this study, clinical signs were evaluated and signs such as piloerection, hunched posture, lethargy, body tremors, an unsteady gait and salivation, seen in the oral repeated dose studies discussed in this unit, were not observed. With neurotoxic signs occurring only at high doses in the oral studies and a dermal absorption factor (DAF) of 7% for etofenprox, neurotoxic manifestations via the dermal route are not expected below the limit dose. Therefore, concern for neurotoxicity following dermal exposure is low.

iii. As discussed in this unit, there is no indication of increased quantitative or qualitative susceptibility of the developing offspring in the toxicology database for etofenprox.

iv. There are no residual uncertainties identified in the exposure databases. The chronic dietary exposure assessment utilizes tolerance residue levels for all commodities based on conservative modeled estimates. The residue level of 5 ppm is considered an upper bound estimate for both ground and aerial applications that assume the conservative deposition onto surrounding crops following a ULV mosquito adulticide application. The dietary assessment also assumes conservative, upper-bound PCT estimates for the proposed uses. By using these screening level assessments, actual exposures/risks are not expected to be underestimated. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to etofenprox in drinking water. EPA used similarly conservative assumptions to assess post-application exposure of children as well as incidental oral exposure of toddlers. These assessments will not underestimate the exposure and risks posed by etofenprox.

E. Aggregate Risks and Determination of Safety

EPA determines whether acute and chronic dietary pesticide exposures are safe by comparing aggregate exposure estimates to the acute PAD (aPAD) and chronic PAD (cPAD). For linear cancer risks, EPA calculates the lifetime probability of acquiring cancer given the estimated aggregate exposure. Short-, intermediate-, and chronic-term risks are evaluated by comparing the estimated aggregate food, water, and residential exposure to the appropriate PODs to ensure that an adequate MOE exists.

1. *Acute risk.* An acute aggregate risk assessment takes into account acute exposure estimates from dietary consumption of food and drinking water. No adverse effect resulting from a single oral exposure was identified and no acute dietary endpoint was selected. Therefore, etofenprox is not expected to pose an acute risk.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to etofenprox from food and water will utilize 32% of the cPAD for children 1–2 years old, the population group receiving the greatest exposure.

There is potential chronic/long-term exposure to etofenprox via dietary (which is considered background exposure) and residential (which is considered primary) exposure pathways for children 1 to < 2 years old. Chronic/long-term exposure to etofenprox for adults is expected via the dietary (background exposure) and residential (primary) exposure pathways; however, there is no dermal hazard identified for etofenprox, incidental oral exposure is not expected for adults, and inhalation exposure is not expected for adults from treating pets; therefore, chronic/long-term risk is best represented by the risk from dietary exposure described in this unit.

The aggregate long-term MOE for children 1 to < 2 years old, including dietary exposure (food and water) and incidental oral exposures from contact with treated pets is 180. Because EPA's level of concern for etofenprox is a MOE of 100 or below, this MOE is not of concern.

3. *Short- and intermediate-term risk.* Short- and intermediate-term aggregate exposure takes into account short- and intermediate-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level). Etofenprox is currently registered for uses that could result in short- and intermediate-term residential exposure, and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with short- and intermediate-term residential exposures to etofenprox.

As noted in Unit III.C.3., because the short- and intermediate-term endpoints and PODs for inhalation and oral routes are the same, the short-term exposure and risk estimates are considered to be protective of potential intermediate-term exposure and risk.

Using the exposure assumptions described in this unit for short-term exposures, EPA has concluded the combined short-term food, water, and residential exposures result in aggregate MOEs of 420 for children 1- < 2 years old, and 1,700 for adults. Because EPA's level of concern for etofenprox is a MOE of 100 or below, these MOEs are not of concern.

4. *Aggregate cancer risk for U.S. population.* Based on the data summarized in Units III.A. and III.C.1.iii., EPA has concluded that etofenprox does not pose a cancer risk to humans.

5. *Determination of safety.* Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to the general population or to infants and children from aggregate exposure to etofenprox residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

For crop commodities, adequate enforcement methodology (liquid chromatography/mass spectrometry/mass spectrometry (LC/MS/MS)) is available to enforce the tolerance expression. For livestock commodities, adequate enforcement methodology (gas chromatography/mass spectrometry (GC/MS)) is available to enforce the tolerance expression.

The methods may be requested from: Chief, Analytical Chemistry Branch, Environmental Science Center, 701 Mapes Rd., Ft. Meade, MD 20755–5350; telephone number: (410) 305–2905; email address: *residuemethods@epa.gov*.

B. International Residue Limits

In making its tolerance decisions, EPA seeks to harmonize U.S. tolerances with international standards whenever possible, consistent with U.S. food safety standards and agricultural practices. EPA considers the international maximum residue limits (MRLs) established by the Codex Alimentarius Commission (Codex), as required by FFDCA section 408(b)(4). The Codex Alimentarius is a joint United Nations Food and Agriculture Organization/World Health Organization food standards program, and it is recognized as an international food safety standards-setting organization in trade agreements to which the United States is a party. EPA may establish a tolerance that is different from a Codex MRL; however, FFDCA section 408(b)(4) requires that EPA explain the reasons for departing from the Codex level.

Codex has established etofenprox MRLs on several crop and livestock commodities at levels that range from 0.01–8.0 ppm. These MRLs are different than the tolerances established for etofenprox in the United States. Codex and U.S. MRLs/tolerances could not be harmonized due to differences in the use pattern used to derive the tolerances. Codex MRLs were based on field trial data from foliar and granular use of etofenprox to kill crop pests in agricultural fields whereas the U.S. tolerances were based on aerial application over crops to kill mosquitoes. Different application amounts, frequencies, and techniques are used for these different use patterns and thus harmonization with Codex cannot be achieved.

C. Revisions to Petitioned-For Tolerances

The proposed tolerance at 0.5 ppm was estimated using limited field trial data. These data were determined to be insufficient to support the proposed use pattern. Subsequently, the applicant submitted modeling results using the Terrestrial Residue Exposure Model (TREX) which estimated residues following repeated ULV applications and concluded residues were likely to peak at 1.5 ppm following repeated aerial applications to agricultural crops. EPA estimated an upper-bound crop residue estimate of 5.0 ppm following repeated ULV aerial and ground applications. In addition, based on the Agency review, it was determined that tolerances were required on livestock commodities as well.

V. Conclusion

Therefore, tolerances are established for residues of etofenprox, [2-(4-ethoxyphenyl)-2-methylpropyl 3-phenoxybenzyl ether], in or on food commodities at 5.0 ppm; feed commodities at 5.0 ppm; eggs at 0.40 ppm; hog fat at 4.0 ppm; hog meat at 0.20 ppm; hog, meat byproducts at 4.0 ppm; fat of cattle, goat, horse, and sheep at 10.0 ppm; meat of cattle, goat, horse, and sheep at 0.40 ppm; meat byproducts of cattle, goat, horse, and sheep at 10.0 ppm; milk at 0.60 ppm; poultry, fat at 1.0 ppm; poultry, meat at 0.01 ppm; and poultry, meat byproducts at 1.0 ppm.

VI. Statutory and Executive Order Reviews

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). Because this final rule has been exempted from review under Executive Order 12866, this final rule is not subject to Executive Order 13211, entitled ''Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use'' (66 FR 28355, May 22, 2001) or Executive Order 13045, entitled ''Protection of Children from Environmental Health Risks and Safety Risks'' (62 FR 19885, April 23, 1997). 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.*), nor does it require any special considerations under Executive Order 12898, entitled ''Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations'' (59 FR 7629, February 16, 1994).

Since 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.

This final rule directly regulates growers, food processors, food handlers, and food retailers, not States or tribes, nor does this action alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of FFDCA section 408(n)(4). As such, the Agency has determined that this action will not have a substantial direct effect on States or tribal governments, on the relationship between the national government and the States or tribal governments, or on the distribution of power and responsibilities among the various levels of government or between the Federal Government and Indian tribes. Thus, the Agency has determined that Executive Order 13132, entitled ''Federalism'' (64 FR 43255, August 10, 1999) and Executive Order 13175, entitled ''Consultation and Coordination with Indian Tribal Governments'' (65 FR 67249, November 9, 2000) do not apply to this final rule. In addition, this final rule does not impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (2 U.S.C. 1501 *et seq.*).

This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA) (15 U.S.C. 272 note).

VII. Congressional Review Act

Pursuant to the Congressional Review Act (5 U.S.C. 801 *et seq.*), 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 action 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 13, 2013.

G. Jeffrey Herndon,

Acting Director, Registration Division, 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. 321(q), 346a and 371.

■ 2. In § 180.620, revise the table in paragraph (a) to read as follows:

§ 180.620 Etofenprox; tolerances for residues.

(a) * * *

Commodity	Parts per million
Cattle, fat	10.0
Cattle, meat	0.40
Cattle, meat byproducts	10.0
Egg	0.40
All food commodities (including feed commodities) not otherwise listed in this subsection	5.0
Goat, fat	10.0
Goat, meat	0.40
Goat, meat byproducts	10.0
Hog, fat	4.0
Hog, meat	0.20
Hog, meat byproducts	4.0
Horse, fat	10.0
Horse, meat	0.40
Horse, meat byproducts	10.0
Milk	0.60
Poultry, fat	1.0
Poultry, meat	0.01
Poultry, meat byproducts	1.0
Rice, grain	0.01
Sheep, fat	10.0
Sheep, meat	0.40
Sheep, meat byproducts	10.0

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