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. Amend § 180.490 as follows:
■ a. Revise the section heading;
■ b. Revise the introductory text in paragraph (a)(1) and add alphabetically the following commodity to the table;
■ c. Revise the introductory text in paragraph (a)(2); and
■ d. Revise the heading in paragraph (c).
The amendments read as follows:

§ 180.490 Imazapic; tolerances for residues.

(a) *General.* (1) Tolerances are established for residues of the herbicide imazapic, including its metabolites and degradates, in or on the commodities listed in the following table. Compliance with the tolerance levels specified is to be determined by measuring the sum of imazapic (2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid) and its metabolites (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-hydroxymethyl-3-pyridinecarboxylic acid and (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-(β-D-glucopyranosyloxy)methyl-3-pyridinecarboxylic acid, calculated as the stoichiometric equivalent of imazapic.

Commodity	Parts per million
* * * * *	
Sugarcane, cane	0.03

(2) Tolerances are established for residues of the herbicide imazapic, including its metabolites and degradates, in or on the commodities listed in the following table. Compliance with the tolerance levels specified is to be determined by measuring the sum of imazapic (2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid) and its metabolite (±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-hydroxymethyl-3-pyridinecarboxylic acid, calculated as the stoichiometric equivalent of imazapic.

* * * * *

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

* * * * *

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2012–0405; FRL–9395–6]

Emamectin; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes a tolerance for residues of emamectin benzoate in or on wine grapes. Syngenta Crop Protection, LLC, requested this tolerance under the Federal Food, Drug, and Cosmetic Act (FFDCA). This document also makes a technical correction to the tolerance expression in the section.

DATES: This regulation is effective August 16, 2013. Objections and requests for hearings must be received on or before October 15, 2013, 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–2012–0405, 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–2012–0405 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 October 15, 2013. 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–2012–0405, 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 August 22, 2012 (77 FR 50661) (FRL–9358–9) 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 2E8018) by Syngenta Crop Protection, LLC, P.O. Box 18300, Greensboro, NC 27419. The petition requested that 40 CFR 180.505 be amended by establishing tolerances for residues of the insecticide emamectin benzoate (a benzoate salt mixture of a minimum of 90% 4′-epi-methylamino-4′- deoxyavermectin B_{1a} and a maximum of 10% 4′-epi-methylamino-4′-deoxyavermectin B_{1b}) resulting from the application of emamectin benzoate in or on imported wine at 0.005 parts per million (ppm). That document referenced a summary of the petition prepared by Syngenta Crop Protection, LLC, the registrant, which is available in the docket, *http://www.regulations.gov.* There were no comments received in response to the notice of filing.

Based upon review of the data supporting the petition, EPA has modified the requested tolerance to emamectin, including its metabolites and degradates, in or on grape, wine at 0.03 ppm. The reason for this change is explained in Unit IV.C.

This final rule also corrects a typographical error (one ''ZB'' missing) in the currently published tolerance expression for § 180.505(a)(2).

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 emamectin benzoate including exposure resulting from the tolerances established by this action. EPA's assessment of exposures and risks associated with emamectin benzoate 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. Emamectin acts by binding to gamma-aminobutyric acid (GABA) gated chloride channels at two different sites, a high affinity binding site that activates the channel and a low-affinity site that blocks the channel. GABA plays a critical role in nervous system development through both non-synaptic and synaptic mechanisms. Consequently, emamectin may have the potential to influence GABA-mediated events important to brain development. Within the mammalian brain, a member of this class of compound (abamectin) has been shown to have widespread binding but particularly abundant in the cerebellum. Through action on the enteric nervous system and induction of longitudinal rhythmic contractions in the isolated ileum, emamectin like abamectin may therefore influence GABA-mediated regulation of metabolism, food intake and body weight at multiple sites. Although GABA receptor mediated neurotoxicity is a solid hypothesis, data in mammalian preparations linking alterations in GABA receptor function to disruptions in neuronal excitability *in vitro* and *in vivo,* and ultimately adverse outcome are currently lacking.

Integral to its mechanism of action in mammals, this class of compounds is also a substrate for (i.e., binds to) P-glycoprotein (P-gp). P-glycoprotein is a member of the adenosine triphosphate (ATP) binding cassette transporter proteins, which reside in the plasma membrane and function as a transmembrane efflux pump, moving xenobiotics from intracellular to the extracellular domain against a steep concentration gradient with ATP-hydrolysis providing the energy for active transport. P-gp is found in the canallicular surface of hepatocytes, the apical surface of proximal tubular cells in the kidneys, brush border surface of enterocytes, luminal surface of blood capillaries of the brain (blood brain barrier), placenta, ovaries, and the testes. As an efflux transporter, P-gp acts as a protective barrier to keep xenobiotics out of the body by excreting them into bile, urine, and intestinal lumen and prevents accumulation of these compounds in the brain and gonads, as well as the fetus. Therefore, some test animals, in which genetic polymorphisms compromise P-gp expression, are particularly susceptible to abamectin or emamectin-induced neurotoxicity. An example is the CF–1 mouse. Some CF–1 mice are deficient in P-gp and are found to be highly sensitive to the neurotoxicity of abamectin. A small population of humans is also found to be deficient of ATP binding cassette (ABC) transporter proteins due to polymorphism in the gene encoding ABC transporter proteins (Dubin-Johnson Syndrome). In addition, collie dogs have been known to be deficient in P-gp.

Consistent with the mode of action, the main target organ for emamectin is the nervous system; clinical signs (tremors, ptosis, ataxia, and hunched posture) and neuropathology (neuronal degeneration in the brain and in peripheral nerves, muscle fiber degeneration) were found in most of the emamectin studies in rats, dogs, and mice. The dose/response curve was very steep in several studies (most notably with CF–1 mice and dogs), with severe effects (morbid sacrifice and neuropathology) sometimes seen at the lowest-observed-adverse-effect-levels (LOAELs) (0.1 milligram/kiolgram/day (mg/kg/day) with no-observed-adverse-effect-level (NOAEL) of 0.075 mg/kg/day). Although no increased sensitivity was seen in developmental toxicity studies in rats and rabbits, increased qualitative and/or quantitative sensitivity of rat pups was seen in the reproductive toxicity and in the developmental neurotoxicity studies.

The carcinogenicity and mutagenicity studies provide no indication that emamectin is carcinogenic or mutagenic. Emamectin is classified as ''not likely to be carcinogenic to humans.''

The available emamectin data show that there is a difference in species sensitivity, and the data suggest the following order: Rat NOAELs/LOAELs greater than dog NOAELs/LOAELs greater than mouse NOAELs/LOAELs. The toxicity endpoints and points of departure for risk were selected from the results of the 15-day CF–1 mouse oral toxicity study.

Specific information on the studies received and the nature of the adverse effects caused by emamectin benzoate as well as the NOAEL and the LOAEL from the toxicity studies can be found at *http://www.regulations.gov* on pp. 29–35 of the document entitled ''Emamectin Benzoate. Human Health Risk Assessment for a Proposed Tolerance on Imported Wine Grapes'' in docket ID number EPA–HQ–OPP–2012–0405.

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 emamectin benzoate used for human risk assessment is shown in Table 1 of this unit.

TABLE 1—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR EMAMECTIN BENZOATE 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).	NOAEL = 0.075 mg/kg/day UF_A = 10x UF_H = 10x FQPA SF = 3x	Acute RfD = 0.00025 mg/kg/day aPAD = 0.00025 mg/kg/day.	15-day mouse study LOAEL = 0.1 mg/kg/day based on tremors on day 3 of dosing. At the next higher dose (0.3 mg/kg/day), tremors were seen at day 2 of treatment.
Chronic dietary (All populations).	NOAEL= 0.075 mg/kg/day UF_A = 10x UF_H = 10x FQPA SF = 10x	Chronic RfD = 0.000075 mg/kg/day cPAD = 0.000075 mg/kg/day.	15-day mouse study LOAEL = 0.1 mg/kg/day based on moribund sacrifices, clinical signs of neurotoxicity, decreases in body weight and food consumption, and histopathological lesions in the sciatic nerve.

FQPA SF = Food Quality Protection Act Safety Factor. LOC = level of concern. LOAEL = lowest-observed-adverse-effect-level. mg/kg/day = milligram/kilogram/day. 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 emamectin benzoate, EPA considered exposure under the petitioned-for tolerances as well as all existing emamectin benzoate tolerances in 40 CFR 180.505. EPA assessed dietary exposures from emamectin benzoate 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.

Such effects were identified for emamectin benzoate. In estimating acute dietary exposure, EPA used food consumption information from the U.S. Department of Agriculture (USDA) 2003–2008 National Health and Nutrition Examination Survey, What We Eat in America (NHANES/WWEIA). As to residue levels in food, a probabilistic acute dietary exposure assessment was conducted. The anticipated residue estimates, used for most crops, were based on field trial data. Tolerance-level residues were used for cottonseed oil, tree nuts (including pistachios), and wine. Pesticide Data Program (PDP) monitoring data for years 2009 and 2010 were used for apples since apple juice had a significant impact on exposure. The Dietary Exposure Evaluation Model (DEEM) default processing factors were used except for commodities with chemical-specific processing studies. Percent crop treated (PCT) data were used.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the USDA 2003–2008 NHANES/WWEIA. As to residue levels in food, a somewhat refined chronic dietary exposure assessment was conducted. The anticipated residue estimates, used for most crops, were single-point estimates (averages) based on field trial data. Tolerance-level residues were used for cottonseed oil, tree nuts (including pistachios), and wine. DEEM default processing factors were used except for commodities with chemical-specific processing studies. PCT data were used.

iii. *Cancer.* Based on the data summarized in Unit III.A., EPA has concluded that emamectin benzoate does not pose a cancer risk to humans. Therefore, a dietary exposure assessment for the purpose of assessing cancer risk is unnecessary.

iv. *Anticipated residue and PCT information.* Section 408(b)(2)(E) of FFDCA authorizes EPA to use available data and information on the anticipated residue levels of pesticide residues in food and the actual levels of pesticide residues that have been measured in food. If EPA relies on such information, EPA must require pursuant to FFDCA section 408(f)(1) that data be provided 5 years after the tolerance is established, modified, or left in effect, demonstrating that the levels in food are not above the

levels anticipated. For the present action, EPA will issue such data call-ins as are required by FFDCA section 408(b)(2)(E) and authorized under FFDCA section 408(f)(1). Data will be required to be submitted no later than 5 years from the date of issuance of these tolerances.

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.

For the acute dietary assessment, the Agency estimated the maximum PCT for existing uses as follows: Almonds, 2.5%; apples, 20%; broccoli, 20%; cabbage, 25%; cauliflower, 20%; celery, 40%; cotton, 2.5%; lettuce, 20%; pears, 20%; peppers, 15%; spinach, 10%; and tomatoes, 20%.

For the chronic dietary assessment, the Agency estimated the PCT for existing uses as follows: Almonds, 1%; apples, 10%; broccoli, 5%; cabbage, 10%; cauliflower, 10%; celery, 25%; cotton, 1%; lettuce, 10%; pears, 5%; peppers, 5%; spinach, 5%; and tomatoes, 10%.

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%.

Also for the acute dietary assessment, the Agency used the following PCT estimates for the following recently approved uses: Cantaloupe, 51%; cucumber, 26%; squash, 46%; and watermelon, 21%. For the chronic dietary assessment, the Agency used the following PCT estimates for the following recently approved uses: Cantaloupe, 40%; cucumber, 14%; squash, 29%; and watermelon, 19%.

These PCT estimates for recently approved uses represent the upper bound of the use expected during the pesticide's initial 5 years of registration; that is, PCT for new uses of emamectin benzoate 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 for new uses is calculated as the average PCT of the market leader or leaders, (i.e., the pesticide(s) with the greatest PCT) on that site over the 3 most recent years of available data. The PCT recommended for use in the acute dietary assessment for new uses is the maximum observed PCT over the same period. Comparisons are only made among pesticides of the same pesticide types (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/NASS as the source 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 for new uses from other crops, if the production area 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. To evaluate whether the PCT for new uses for emamectin benzoate could be exceeded, EPA considered whether there may be unusually high pest pressure, as indicated in emergency exemption requests for emamectin benzoate; the pest spectrum of the new pesticide in comparison with the market leaders and whether the market leaders are well established for that use; and whether pest resistance issues with past market leaders provide emamectin benzoate with significant market potential. Given currently available information, EPA concludes that it is unlikely that actual PCT for emamectin benzoate will exceed the estimated PCT for new uses during the next 5 years.

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 emamectin benzoate 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 emamectin benzoate in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of emamectin benzoate. 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 Pesticide Root Zone Model/Exposure Analysis Modeling System (PRZM/EXAMS) and Screening Concentration in Ground Water (SCI–GROW) models, the estimated drinking water concentrations (EDWCs) of emamectin benzoate for acute exposures are estimated to be between 0 and 0.465 parts per billion (ppb) for surface water and 0.00054 ppb for ground water, and for chronic exposures are estimated to be 0.150 ppb for surface water and 0.00054 ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For acute dietary risk assessment, a drinking water residue distribution based on the PRZM/EXAMS modeling was used. For chronic dietary risk assessment, the water concentration value of 0.150 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). Emamectin benzoate is not registered for any specific use patterns that would result in residential exposure.

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.''

OPP's *Guidance for Identifying Pesticide Chemicals and Other Substances that have a Common Mechanism of Toxicity* (EPA, 1999) describes the weight of the evidence approach for determining whether or not a group of pesticides share a common mechanism of toxicity. This guidance defines mechanism of toxicity as the major steps leading to a toxic effect following interaction of a pesticide with biological targets. All steps leading to an effect do not need to be specifically understood. Rather, it is the identification of the crucial events following chemical interaction that are required in order to describe a mechanism of toxicity. For example, a mechanism of toxicity may be described by knowing the following: A chemical binds to a given biological target *in vitro,* and causes the receptor-related molecular response; *in vivo* it also leads to the molecular response and causes a number of intervening biological and morphological steps that result in an adverse effect. In this context a common mechanism of toxicity pertains to two or more pesticide chemicals or other substances that cause a common toxic effect to human health by the same, or essentially the same, sequence of major biochemical events. Hence, the underlying basis of the toxicity is the same, or essentially the same, for each chemical. In the case of the macrocyclic lactone pesticides (e.g., abamectin, emamectin, and avermectin), there is a wealth of data on the insecticidal mechanism of action for avermectin: Its insecticidal actions are mediated by interaction with the glutamate-gated chloride channels and $GABA_A$ gated chloride channels. This is presumed to be the insecticidal mechanism of action of emamectin and abamectin as well. Insecticidal mechanism of action does not indicate a common mechanism of toxicity for human health. Further, mammals lack glutamate-gated chloride channels; the toxic actions of avermectin appear to be mediated via interaction with $GABA_A$ and possibly glycine gated chloride channels. There is evidence that avermectin B_{1a} binds to $GABA_A$ receptors and activates Cl flux into neurons (Abalis et al., 1986; Huang and Casida, 1997). However, there is a paucity of data regarding the resultant alterations in cellular excitability of mammalian neurons and neural networks (i.e., changes in cellular excitability and altered network function as documented with pyrethroids), as well as *in vivo* measurements of altered excitability associated with adverse outcomes. Thus, while the downstream steps leading to toxicity via disruption of $GABA_A$ receptor function for avermectin can be postulated, experimental data supporting these actions are lacking. In addition, specific data demonstrating $GABA_A$ receptor interaction in mammalian preparations are lacking for abamectin and emamectin. Moreover, the specificity of such interaction on the adverse outcome would need to be shown experimentally. $GABA_A$ receptors have multiple binding sites which have been proposed to relate to adverse outcomes. For example, Dawson et al (2000) showed for a group of avermectin-like compounds that rank order for anticonvulsant activity did not parallel the rank order for affinity at the [3*H*]-ivermectin site. The authors hypothesized that these findings may be related to differential affinity or efficacy at subtypes of the $GABA_A$ receptor. Other reports have indicated species differences in abamectin effects on $GABA_A$ receptor function in the mouse as compared to the rat (Soderlund et al., 1987).

In conclusion, although $GABA_A$ receptor mediated neurotoxicity may be a common mechanism endpoint for the macrocyclic lactone pesticides, data demonstrating the interactions of emamectin and abamectin with mammalian $GABA_A$ receptors are not available, and data in mammalian preparations linking alterations in $GABA_A$ receptor function to disruptions in neuronal excitability *in vitro* and *in vivo,* and ultimately adverse outcome, are also currently lacking for this class of compounds. In the absence of such data, the key biological steps leading to the adverse outcome (i.e., the mammalian mechanism of action) cannot be established and by extension a common mechanism of toxicity (CMT) cannot be established.

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 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 Food Quality Protection Act Safety Factor (FQPA 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.* Although no increased sensitivity was seen in developmental toxicity studies in rats and rabbits, increased qualitative and/or quantitative sensitivity of rat pups was seen in the reproductive toxicity study and in the developmental neurotoxicity study. In the reproduction study, whole body tremors, hind limb extension, and hind limb splay were seen in the F_1 and F_2 pups while these clinical signs were not seen in F_0 parental animals at similar dose levels. In addition, a greater incidence of decreased fertility was seen in the F_1 parental females than in the F_0 females. In the developmental neurotoxicity study, no maternal effect was seen at the highest dose tested whereas dose-related decrease in open-field motor activity was seen in the mid-dose in pups on postnatal day 17. Body tremors, hind-limb extension, and auditory startle were also found in the high-dose pups.

3. *Conclusion.* Based on currently available data, EPA is retaining the 10X FQPA SF for chronic assessments and is using a 3X FQPA SF for acute assessments. This decision is based on the following findings:

i. *Completeness of the toxicity database.* The toxicology database used to assess pre- and postnatal exposure to emamectin contains all required studies with exception of an immunotoxicity study and a subchronic inhalation toxicity study, which are data gaps.

The Agency evaluated subchronic, chronic, carcinogenicity, developmental, and reproduction studies as well as acute and subchronic neurotoxicity studies for any effects that might indicate that emamectin induced changes in the organs generally associated with immunological toxicity. In the studies evaluated, only the 14-week oral toxicity study in dogs showed an increase in the incidence of thymus atrophy at 1 mg/kg/day. In the 1-year feeding study in dog, thymus atrophy was not reported at similar dose levels tested. Currently, the point of departure for risk assessment is 0.075 mg/kg/day, which is more than 10 times less than the dose where thymus atrophy had been reported. Therefore, since the acute and chronic RfD's are 0.00025 mg/kg/day and 0.000075 mg/kg/day, respectively, the Agency does not believe an immunotoxicity study will result in a lower POD than that which is currently in use for overall risk assessment. As such, a database uncertainty factor is not necessary to account for the lack of an immunotoxicity study.

In regards to the inhalation toxicity study, there are currently no residential uses registered for emamectin benzoate, and therefore, lack of this study does not impact the Agency's assessment of pre- and postnatal exposure.

Another completeness issue with regard to the toxicity database is that EPA is using a short-term study for long-term risk assessment. The data submitted show that CF–1 mice, which lack P-gp, are the most sensitive species/strand of animal tested. EPA only has data on CF–1 mice in short-term studies. Longer-term studies used CD–1 mice. Hence a short-term study in CF–1 mice was used to choose the chronic POD. The extrapolation from a short-term study in CF–1 mice to a long-term POD introduces additional uncertainty into the risk assessment process.

ii. *Potential pre- and postnatal toxicity.* Although no increased sensitivity was seen in developmental toxicity studies in rats and rabbits, increased qualitative and/or quantitative sensitivity of rat pups was seen in the reproductive toxicity study and in the developmental neurotoxicity study. A degree-of-concern analysis was conducted to determine whether or not an additional safety factor is needed to account for the increased susceptibility in pups; it was concluded that the degree-of-concern was low for both 2-generation reproduction and developmental neurotoxicity studies. The reasons are as follows:

a. For the 2-generation reproduction study:

- There was a clear NOAEL for the offspring toxicity.
- The decreased fertility seen in F_1 adults might have been due to histopathological lesions in the brain and central nervous system (seen in both F_0 and F_1 generations), rather than due to a direct effect on the reproductive system.

b. For the developmental neurotoxicity study:

- Although multiple offspring effects (including decreased pup body weight, head and body tremors, hindlimb extension and splay, changes in motor activity and auditory startle) were seen at the highest dose, and no maternal effects were seen at any dose, there was a clear NOAEL for offspring toxicity at the low dose.
- The offspring LOAEL (at the mid dose) is based on a single effect seen on only 1 day (decreased motor activity on PND 17) and no other offspring toxicity was seen at the LOAEL.

Two other considerations raise residual concerns about whether the traditional safety factors are protective of potential pre- and postnatal toxicity. First, the steepness of the dose-response curve means that there is a small margin of error provided by reliance on the study NOAEL. Second, the severity of effects at the LOAEL (death and neuropathology), exacerbate the concern raised by the steep dose response curve.

iii. *The completeness of the exposure database.* The assessment for food incorporates somewhat refined anticipated residue estimates for most commodities that were derived from field trial data and PCT. The availability and use of monitoring data and food preparation-reduction factors for washing, cooking, etc., may have resulted in a more refined estimate of dietary exposure. Therefore, exposures to residues in food are not expected to be exceeded.

The dietary drinking water assessment utilizes water concentration values generated by model and associated modeling parameters which are designed to provide conservative, health protective, high-end estimates of water concentrations which will not likely be exceeded.

Taking all of these findings into account, EPA has concluded that there are not reliable data supporting lowering of the default 10X FQPA SF for chronic exposures. Specifically, EPA does not have reliable data showing that infants and children will be adequately protected using the traditional inter- and intra-species safety factors due to the steepness of the dose-response curve, the severity of effects at the LOAEL (death and neuropathology), and the use of a short-term study for long-term risk assessment. The Agency did not use a chronic study for the point of departure because the chronic studies were conducted in rats, dogs, and CD–1 mice.

Taking all of these findings into account, for acute exposures, EPA has concluded that there are reliable data supporting lowering the default 10X FQPA SF to 3X. Although the steepness of the dose-response curve and the severity of the effects at the LOAEL introduce uncertainty with regard to whether the inter- and intra-species safety factors are protective of infants and children from acute effects, EPA has concluded that use of the 15-day neurotoxicity CF–1 mouse study provides reliable data to reduce the FQPA SF for acute assessments from 10X to 3X. The Agency determined that a 3X FQPA SF is adequate for assessing acute dietary risk based on the following weight of evidence considerations:

- An endpoint of concern attributable to a single exposure was not identified for *in utero* effects since there was no concern for developmental toxicity and there was no indication of increased susceptibility (qualitative or quantitative) of rat or rabbit fetuses to *in utero* exposure to emamectin.
- Although there was evidence of increased susceptibility in the developmental neurotoxicity (DNT) study, an endpoint of concern was not identified for acute dietary risk assessment for prenatal exposures because the adverse effect at the LOAEL (i.e., decrease in open-field motor activity) was seen only on postnatal day 17 and not seen after a single exposure.
- The POD selected for acute dietary risk assessment is a NOAEL (with a clear LOAEL) seen after repeated dosing but is used for assessing acute risk (i.e., a very conservative approach).

Therefore, the Agency is confident that the retention of a 3X FQPA SF (to account for the steepness of the dose response curve) will not underestimate risk and provides reasonable certainty of no harm from exposure to emamectin benzoate.

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.* Using the exposure assumptions discussed in this unit for acute exposure, the acute dietary exposure from food and drinking water to emamectin benzoate will occupy 91% of the aPAD for females 13–49 years old, the population group receiving the greatest exposure.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to emamectin benzoate from food and water will utilize 16% of the cPAD for all infants less than 1 year old, the population group receiving the greatest exposure. There are no residential uses for emamectin benzoate.

3. *Short-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).

Both short- and intermediate-term adverse effects were identified; however, emamectin benzoate is not registered for any use patterns that would result in either short- or intermediate-term residential exposure. Short- and intermediate-term risk is assessed based on short- and intermediate-term residential exposure plus chronic dietary exposure. Because there is no short- or intermediate-term residential exposure and chronic dietary exposure has already been assessed under the appropriately protective cPAD (which is at least as protective as the POD used to assess short- or intermediate-term risk), no further assessment of short- or intermediate-term risk is necessary, and EPA relies on the chronic dietary risk assessment for evaluating short- and intermediate-term risk for emamectin benzoate.

4. *Aggregate cancer risk for U.S. population.* Based on the lack of evidence of carcinogenicity in two adequate rodent carcinogenicity studies, emamectin benzoate is not expected to 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 emamectin benzoate residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (high performance liquid chromatography with fluorescence detection (HPLC/FLD)) is available to enforce the tolerance expression.

The method 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.

Harmonization issues regarding the tolerance expression are associated with this petition. There is a Codex MRL for grapes of 0.03 ppm. The Codex residue definition for the MRL and for the risk assessment is emamectin B_{1a} benzoate. The recommended U.S. tolerance is 0.03 ppm to harmonize with Codex but the U.S. residue definition includes additional analytes.

C. Revisions to Petitioned-For Tolerances

The difference in the proposed tolerance level of 0.005 ppm and the recommended tolerance level of 0.03 ppm is because EPA does not set tolerances on wine but rather on the raw commodity wine grapes. The recommended tolerance level reflects the harmonized residue values in the raw commodity as described in Unit IV.B.

EPA has revised the tolerance expression to clarify:

1. That, as provided in FFDCA section 408(a)(3), the tolerance covers metabolites and degradates of emamectin benzoate not specifically mentioned.

2. That compliance with the specified tolerance levels is to be determined by measuring only the specific compounds mentioned in the tolerance expression.

V. Conclusion

Therefore, a tolerance is established for residues of emamectin, including its metabolites and degradates, in or on grape, wine at 0.03 ppm. Compliance with the tolerance levels specified is to be determined by measuring only the sum of emamectin (a mixture of a minimum of 90% 4′-epi-methylamino-4′-deoxyavermectin B_{1a} and maximum of 10% 4′-epi-methylamino-4′-deoxyavermectin B_{1b}) and its metabolites 8,9-isomer of the B_{1a} and B_{1b} component of the parent (8,9-ZMA), or 4′-deoxy-4′-epi-amino-avermectin B_{1a} and 4′-deoxy-4′-epi-amino-avermectin B_{1b}; 4′-deoxy-4′-epi-amino avermectin B_{1a} (AB_{1a}); 4′-deoxy-4′-epi-(*N*-formyl-*N*-methyl)amino-avermectin (MFB_{1a}); and 4′-deoxy-4′-epi-(*N*-formyl)amino-avermectin B_{1a} (FAB_{1a}), calculated as the stoichiometric equivalent of emamectin.

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: August 7, 2013.

Lois Rossi,

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 § 180.505:

■ a. Add alphabetically the following commodity and footnote 1 to the table in paragraph (a)(1).

■ b. Revise the introductory text of paragraph (a)(2).

The amendments read as follows:

§ 180.505 Emamectin; tolerances for residues.

(a) * * *

(1) * * *

Commodity	Parts per million
* * * * *	
Grape, wine [1]	0.03
* * * * *	

[1] There are no U.S. registrations for use of emamectin on grape, wine.

(2) Tolerances are established for emamectin, including its metabolites and degradates, in or on the commodities in the following table. Compliance with the tolerance levels specified in the following table is to be determined by measuring only the sum of emamectin (MAB_{1a} + MAB_{1b} isomers) and the associated 8,9-Z isomers (8,9-ZB_{1a} and 8,9-ZB_{1b}).

* * * * *

[FR Doc. 2013–19863 Filed 8–15–13; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 300

[EPA–HQ–SFUND–1986–0005; FRL–9846–4]

National Oil and Hazardous Substances Pollution Contingency Plan; National Priorities List: Partial Deletion of the Torch Lake Superfund Site

AGENCY: U.S. Environmental Protection Agency.

ACTION: Direct final rule.

SUMMARY: The U.S. Environmental Protection Agency Region 5 is publishing a direct final Notice of Deletion of the Quincy Smelter and Calumet Lake parcels of Operable Unit 3 (OU3) of the Torch Lake Superfund Site (Site), located in Houghton County, Michigan, from the National Priorities List (NPL). The NPL, promulgated pursuant to Section 105 of the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, as amended, is an appendix of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP). This direct final partial deletion is being published by EPA with the concurrence of the State of Michigan, through the Michigan Department of Environmental Quality (MDEQ), because EPA has determined that all appropriate response actions at these identified parcels under CERCLA, other than operation, maintenance, and five-year reviews, have been completed. However, this partial deletion does not preclude future actions under Superfund.

This partial deletion pertains to the surface tailings and slag deposits of the Quincy Smelter and Calumet Lake parcels of OU3. The following parcels or areas will remain on the NPL and are not being considered for deletion as part of this action: Dollar Bay, Point Mills, Boston Pond, and North Entry.

DATES: This direct final partial deletion is effective October 15, 2013 unless EPA receives adverse comments by September 16, 2013. If adverse comments are received, EPA will publish a timely withdrawal of the direct final partial deletion in the **Federal Register** informing the public that the deletion will not take effect.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA–HQ–SFUND–1986–0005, by one of the following methods:

- *http://www.regulations.gov:* Follow online instructions for submitting comments.
- *Email:* Nefertiti DiCosmo, Remedial Project Manager, at *dicosmo.nefertiti@epa.gov* or Dave Novak, Community Involvement Coordinator, at *novak.dave@epa.gov.*
- *Fax:* Gladys Beard at (312) 697–2077.
- *Mail:* Nefertiti DiCosmo, Remedial Project Manager, U.S. Environmental Protection Agency (SR–6J), 77 West Jackson Boulevard, Chicago, IL 60604, (312) 886–6148 or Dave Novak, Community Involvement Coordinator, U.S. Environmental Protection Agency (SI–7J), 77 West Jackson Boulevard, Chicago, IL 60604, (312) 886–7478 or toll free at 1 (800) 621–8431.
- *Hand delivery:* Dave Novak, Community Involvement Coordinator, U.S. Environmental Protection Agency