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ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180

[EPA–HQ–OPP–2007–0604; FRL–8377–7]

Dichlobenil; Pesticide Tolerances

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for combined residues of dichlobenil and its metabolite, 2,6-dichlorbenzamide, in or on bushberry subgroup 13–07B, caneberry subgroup 13–07A and rhubarb. It also removes existing tolerances on individual members of bushberry subgroup 13–07B (blueberry) and caneberry subgroup 13–07A (blackberry and raspberry) that are superseded by the new crop subgroup tolerances at the same tolerance levels. Interregional Research Project Number 4 (IR–4) requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective August 27, 2008. Objections and requests for hearings must be received on or before October 27, 2008, 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: EPA has established a docket for this action under docket identification (ID) number EPA–HQ–OPP–2007–0604. To access the electronic docket, go to *http://www.regulations.gov*, select ''Advanced Search,'' then ''Docket Search.'' Insert the docket ID number where indicated and select the ''Submit'' button. Follow the instructions on the regulations.gov website to view the docket index or access available documents. All documents in the docket are listed in the docket index available in regulations.gov. Although listed in the index, some information is not publicly available, e.g., Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available in the electronic docket at *http://www.regulations.gov*, or, if only available in hard copy, at the OPP Regulatory Public Docket in Rm. S–4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. The Docket Facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The Docket Facility telephone number is (703) 305–5805.

FOR FURTHER INFORMATION CONTACT: Susan Stanton, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5218; e-mail address: *stanton.susan@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. Potentially affected entities may include, but are not limited to those engaged in the following activities:

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

This listing is not intended to be exhaustive, but rather to provide a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Access Electronic Copies of this Document?

In addition to accessing an electronic copy of this **Federal Register** document through the electronic docket at *http://www.regulations.gov*, you may access this **Federal Register** document electronically through the EPA Internet under the ''**Federal Register**'' listings at *http://www.epa.gov/fedrgstr*. You may also access a frequently updated electronic version of EPA's tolerance regulations at 40 CFR part 180 through the Government Printing Office's pilot e-CFR site at *http://www.gpoaccess.gov/ecfr*.

C. Can I File an Objection or Hearing Request?

Under section 408(g) of FFDCA, 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–2007–0604 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk as required by 40 CFR part 178 on or before October 27, 2008.

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 that does not contain any CBI for inclusion in the public docket that is described in **ADDRESSES**. Information not marked confidential pursuant to 40 CFR part 2 may be disclosed publicly by EPA without prior notice. Submit this copy, identified by docket ID number EPA–HQ–OPP–2007–0604, by one of the following methods:

- *Federal eRulemaking Portal*: *http://www.regulations.gov*. Follow the on-line instructions for submitting comments.
- *Mail*: Office of Pesticide Programs (OPP) Regulatory Public Docket (7502P), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001.
- *Delivery*: OPP Regulatory Public Docket (7502P), Environmental Protection Agency, Rm. S–4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. Deliveries are only accepted during the Docket's normal hours of operation (8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays). Special arrangements should be made for deliveries of boxed information. The Docket Facility telephone number is (703) 305–5805.

II. Petition for Tolerance

In the **Federal Register** of August 22, 2007 (72 FR 47010) (FRL–8142–5), EPA issued a notice pursuant to section 408(d)(3) of FFDCA, 21 U.S.C. 346a(d)(3), announcing the filing of a pesticide petition (PP 7E7230) by Interregional Research Project Number 4 (IR–4), 500 College Road East, Suite 201W, Princeton, NJ 08540–6635. The petition requested that 40 CFR 180.231 be amended by establishing tolerances for combined residues of the herbicide dichlobenil, 2,6-dichlorobenzonitrile, and its metabolite, 2,6-

dichlorobenzamide, in or on rhubarb at 0.15 parts per million (ppm); caneberry, subgroup 13a and wild raspberry at 0.1 ppm; and bushberry, subgroup 13b; aronia berry; bluberry, lowbush; buffalo currant; chilian guava; european barberry; highbush cranberry; honeysuckle; jostaberry; juneberry; lingonberry; native currant; salal; and sea buckthorn at 0.15 ppm. That notice referenced a summary of the petition prepared by Chemtura USA Corporation, the registrant, which is available to the public 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 and recent changes in EPA's crop grouping regulations, EPA has revised the tolerance level for rhubarb and the commodity terms for the berry tolerances. The reasons for these changes are 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....''

2,6-Dichlorobenzamide (BAM) is a common metabolite and soil degradate of dichlobenil and the fungicide fluopicolide. BAM is the major residue detected in plants following dichlobenil use and is, therefore, a residue of concern. For this reason, aggregate exposure and risk associated with BAM were assessed separately from dichlobenil. In assessing aggregate exposure and risk for BAM, EPA considered exposures associated with both dichlobenil and fluopicolide uses.

Consistent with section 408(b)(2)(D) of FFDCA, and the factors specified in section 408(b)(2)(D) of FFDCA, 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 the petitioned-for tolerances for combined residues of dichlobenil and its metabolite, 2,6-dichlorobenzamide (BAM) on bushberry subgroup 13–07B, caneberry subgroup 13–07A and rhubarb at 0.15 ppm, 0.10 ppm and 0.06 ppm, respectively. EPA's assessment of exposures and risks associated with establishing tolerances 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 acute toxicity tests, dichlobenil demonstrated moderate acute toxicity via the oral, dermal and inhalation routes. It is neither a dermal irritant, eye irritant, nor a dermal sensitizer. In the subchronic and chronic oral toxicity studies in hamsters, rats and dogs, the liver was the primary target organ. For example, in a 90-day oral toxicity study in rats, inflammation and necrosis were observed in the liver of males, and increased liver weight and liver histopathology (swelling and vacuolation of hepatocytes) were observed in females. In a 90-day oral toxicity study in hamsters, increased liver weight, enlarged liver (with rough surface) and swollen hepatocytes were observed in females. In addition, decreased weight of the prostate and mineralization of the prostate were reported in males. Increased liver weights and hepatic enzymes, as well as liver histopathology, were observed at lower doses in two chronic dog toxicity studies, as well as in the combined chronic toxicity/carcinogenicity study in the rat.

In addition to the liver, the nose is considered a target organ for dichlobenil. Olfactory toxicity was observed following dermal and inhalation exposures in toxicity studies that were either published in the open literature (dermal) or submitted to the Agency (inhalation). In each study, degeneration of the olfactory epithelium, which is composed of olfactory sensory neurons, was observed. Olfactory toxicity was not observed in the chronic oral (capsule) toxicity study in the dog. No other evidence of neurotoxicity was seen in the toxicity studies for dichlobenil.

EPA classified dichlobenil as a Group C (possible human) carcinogen based on the results of carcinogenicity studies in hamsters and rats and its structural similarity to bromoxynil and thiobenzamide, which are associated with hepatocellular tumors in rodents. In a high-dose hamster study, there was a treatment-related increase in liver adenomas and combined adenomas/carcinomas in males at the highest dose tested; however, this dose was considered excessive, based on decreased body weight gains and severe hepatotoxicity. In a second hamster study, performed at lower, but adequate doses, there was no treatment-related increase in the incidence of any tumor type. In the rat study, there was a treatment-related increase in the incidence of hepatocellular tumors in females only. Based on the weight of the evidence, EPA classified dichlobenil as a possible human carcinogen but determined that the chronic dietary risk assessment based on the cPAD would be protective of any potential cancer effects for the following reasons: The liver tumors seen in male hamsters occurred only at an excessively high dose. The increases in liver tumors in the rat were statistically significant in only one sex (females), while tumors were predominantly benign adenomas and supporting evidence was weak at best. Although the tumor type (hepatocellular) is considered unusual for the strain of rat tested, tumors did not occur to an unusal degree or with an early onset. Further, dichlobenil was determined to be non-mutagenic in bacteria and mammalian cells, as well as non-clastogenic in several mammalian assays (*in vitro* and *in vivo*).

In the rat prenatal developmental toxicity study, maternal effects (decreased body weight gain, food consumption and food efficiency) were seen at the mid- and high doses, whereas no prenatal developmental effects occurred at any dose. In the rabbit developmental toxicity study, prenatal effects (an increase in total resorptions/dam, post-implantation loss, as well as external, visceral, and skeletal anomalies) occurred in the presence of maternal toxicity (severe decreases in body weight gain (120%) and food consumption (30%)). In the rat reproduction study, effects in the pups (decreased body weight during weaning in both F1 (16–23%) and F2 (19–22%) generation pups) occurred at a lower dose than that which resulted in parental toxicity (decreases in premating and gestation body weight gain and premating food consumption

in both parental and F1 generation adults), indicating increased quantitative susceptibility of the pups.

Delayed maturity of the uterus was observed in all high-dose females tested in the chronic oral (capsule) toxicity study in the dog. A marked decrease in mean uterine weight at the high dose confirmed this finding. Ovarian weights were also decreased in high-dose females, but no alterations were observed microscopically. These results are suggestive of modulation of the female endocrine system in this study; however, the dose utilized in the dichlobenil risk assessment for the chronic RfD is almost forty times lower than that at which the effects were observed and is considered protective of any potential endocrine modulation.

BAM demonstrated moderate acute toxicity via the oral route of exposure. In subchronic and chronic toxicity studies, the primary oral effects seen in the rat and dog were body weight changes. Adverse liver effects were also observed but at doses of BAM that were higher than those of dichlobenil. There is no evidence that BAM is either mutagenic or clastogenic; nor is there evidence of endocrine mediated toxicity. BAM is considered to be neurotoxic, based on clinical signs of neurotoxicity following oral exposure in several short-term assays, in addition to toxicity to the olfactory sensory neurons observed following single intraperitoneal exposures of mice to BAM. In the absence of carcinogenicity study data for a second species (a rat study is available), the EPA has assumed that BAM's carcinogenic potential is similar to that of dichlobenil, the parent compound having the greatest carcinogenicity potential. Dichlobenil is classified as a ''group C, possible human carcinogen.'' Quantification of cancer risk is based on the cPAD approach which requires comparison of the chronic exposure to the cPAD. Using this methodology will adequately account for all chronic toxic effects, including carcinogenicity, likely to result from exposure to dichlobenil and, therefore, to BAM.

Specific information on the studies received and the nature of the adverse effects caused by dichlobenil and BAM, 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 documents *Dichlobenil; Human Health Risk Assessment for Proposed Uses on Rhubarb; Caneberry, Subgroup 13–07A; and Bushberry, Subgroup 13–07B*, page 37 and *2,6-Dichlorobenzamide (BAM); 2,6-Dichlorobenzamide (BAM) as a Metabolite/Degradate of Fluopicolide and Dichlobenil. Human Health Risk Assessment for Proposed Uses of Rhubarb, Dichlobenil on Caneberries (Subgroup 13–07A), and Bushberries (Subgroup 13–07B*, page 17 in docket ID number EPA–HQ–OPP–2007–0604.

B. Toxicological Endpoints

For hazards that have a threshold below which there is no appreciable risk, a toxicological point of departure (POD) is identified as the basis for derivation of reference values for risk assessment. The POD may be defined as the highest dose at which no adverse effects are observed (the NOAEL) in the toxicology study identified as appropriate for use in risk assessment. However, if a NOAEL cannot be determined, the lowest dose at which adverse effects of concern are identified (the LOAEL) or a Benchmark Dose (BMD) approach is sometimes used for risk assessment. Uncertainty/safety factors (UFs) are used in conjunction with the POD to take into account uncertainties inherent in the extrapolation from laboratory animal data to humans and in the variations in sensitivity among members of the human population as well as other unknowns. Safety is assessed for acute and chronic dietary risks by comparing aggregate food and water exposure to the pesticide to the acute population adjusted dose (aPAD) and chronic population adjusted dose (cPAD). The aPAD and cPAD are calculated by dividing the POD by all applicable UFs. Aggregate short-, intermediate-, and chronic-term risks are evaluated by comparing food, water, and residential exposure to the POD to ensure that the margin of exposure (MOE) called for by the product of all applicable UFs is not exceeded. This latter value is referred to as the Level of Concern (LOC).

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 greater than that 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 dichlobenil and BAM used for human risk assessment can be found at *http://www.regulations.gov* in the documents *Dichlobenil; Human Health Risk Assessment for Proposed Uses on Rhubarb; Caneberry, Subgroup 13–07A; and Bushberry, Subgroup 13–07B*, page 19 and *2,6-Dichlorobenzamide (BAM); 2,6-Dichlorobenzamide (BAM) as a Metabolite/Degradate of Fluopicolide and Dichlobenil. Human Health Risk Assessment for Proposed Uses of Rhubarb, Dichlobenil on Caneberries (Subgroup 13–07A), and Bushberries (Subgroup 13–07B*, page 5 in docket ID number EPA–HQ–OPP–2007–0604.

C. Exposure Assessment

1. *Dietary exposure from food and feed uses*. In evaluating dietary exposure to dichlobenil, EPA considered exposure under the petitioned-for tolerances as well as all existing dichlobenil tolerances in 40 CFR 180.231. In evaluating dietary exposure to BAM, EPA considered exposure resulting from all proposed and registered uses of dichlobenil and fluopicolide. EPA assessed dietary exposures from dichlobenil and BAM 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.

a. *Dichlobenil*. An effect of concern attributable to a single exposure was not identified for the general population, including infants and children; however, such effects (an increase in total resorptions/dam, post-implantation loss, as well as external, visceral, and skeletal anomalies) were identified for the population subgroup females, 13 to 49 years old. In estimating acute dietary exposure of females, 13 to 49 years old, EPA used food consumption information from the United States Department of Agriculture (USDA) 1994–1996 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA assumed that 100 percent of all crops with established or pending tolerances are treated with dichlobenil and contain tolerance-level residues.

b. *BAM*. EPA identified an effect of concern attributable to a single exposure for the general population (lethargy after a single dose in a dose range finding assay for an *in vivo* mouse erythrocyte micronucleus assay) and for females 13 to 49 years old (increased incidences of late abortion and skeletal and visceral anomalies in a rabbit developmental toxicity study). In estimating acute dietary exposure, EPA used food consumption information from the United States Department of Agriculture (USDA) 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA used

maximum residues of BAM from fluopicolide and dichlobenil field trials on food commodities with established/pending tolerances. The assessments assumed 100 percent crop treated (PCT) for all commodities except apples, blueberries, cherries, peaches, pears and raspberries.

ii. *Chronic exposure*. a. *Dichlobenil*. In conducting the chronic dietary exposure assessment, EPA used food consumption information from the United States Department of Agriculture (USDA) 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA assumed that 100 percent of all crops with established or pending tolerances are treated with dichlobenil and contain tolerance-level residues.

b. *BAM*. In conducting the chronic dietary exposure assessment, EPA used food consumption information from the United States Department of Agriculture (USDA) 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA used maximum residues of BAM from fluopicolide and dichlobenil field trials on food commodities with established/pending tolerances. The assessments assumed 100 PCT for all commodities except apples, blueberries, cherries, cranberries, peaches, pears and raspberries.

iii. *Cancer*. EPA classified dichlobenil as a Group C, possible human, carcinogen but determined that the chronic dietary risk assessment based on the cPAD would be protective of any potential cancer effects. The weight of the evidence supporting this determination is discussed in unit III.A. (Toxicological Profile). EPA has assumed that BAM's carcinogenic potential is similar to that of dichlobenil, the parent compound having the greatest carcinogenicity potential. As with dichlobenil, the chronic dietary risk assessment based on the cPAD is expected to protect for any potential cancer effects. Separate cancer exposure assessments are not needed for dichlobenil or BAM.

iv. *Anticipated residue and PCT information*. EPA did not use anticipated residues in the dietary risk assessments for dichlobenil but did use anticipated residues (maximum field trial residues) for BAM. 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.

The Agency used PCT information as follows:

The Agency did not use PCT information in the dichlobenil dietary risk assessments. For the BAM acute assessment, maximum PCT estimates were used for the following commodities: Apples, blueberries, cherries, peaches and pears, each at 2.5%; and raspberries at 5%. For the BAM chronic assessment, average PCT estimates were used for the following commodities: Apples, blueberries, cherries, peaches and pears, each at 1%; raspberries at 5%; and cranberries at 45%.

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 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 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 dichlobenil 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 assessments for dichlobenil and BAM in drinking water. These simulation models take into account data on the physical, chemical, and fate/transport characteristics of dichlobenil and BAM. 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 dichlobenil for acute exposures are estimated to be 298 parts per billion (ppb) for surface water and 0.93 ppb for ground water. The estimated drinking water concentrations (EDWCs) of dichlobenil for chronic exposures for non-cancer assessments are estimated to

be 4.6 ppb for surface water and 0.93 ppb for ground water.

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 BAM for acute exposures are estimated to be 21 parts ppb for surface water and 56.2 ppb for ground water. The estimated drinking water concentrations (EDWCs) of BAM for chronic exposures for non-cancer assessments are estimated to be 8.6 ppb for surface water and 56.2 ppb for ground water.

Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For acute dietary risk assessment for dichlobenil, the water concentration value of 298 ppb was used to assess the contribution to drinking water. For chronic dietary risk assessment for dichlobenil, the water concentration value of 4.6 ppb was used to assess the contribution to drinking water. For acute and chronic dietary risk assessment for BAM, the water concentration value of 56.2 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).

There are several dichlobenil products that may be used around roses and other woody ornamentals in established residential plantings. Since they are approved for professional applicator use only, residential handler exposures are not expected. Post-application exposure of adults and children to dichlobenil and BAM from the use of dichlobenil products on ornamental plantings is expected to be negligible and, therefore, was not assessed.

Fluopicolide is currently registered for the following uses that could result in residential exposure to the metabolite/degradate BAM: Residential turfgrass and recreational sites. EPA assessed residential exposure to BAM using the following assumptions: Residential handler exposure was not evaluated for turf uses, because the metabolite BAM is believed to form slowly in plants and soil after the product containing parent fluopicolide has been applied. Residential post-application exposure via the dermal route is likely for adults and children entering treated lawns; however, post-application exposure via the inhalation route is expected to be negligible. Toddlers may also be exposed via incidental ingestion (i.e., hand-to-mouth, object-to-mouth (turfgrass), and soil ingestion) during post-application activities on treated turf. Post-application exposures are expected to be of short and intermediate duration.

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 dichlobenil (parent) or its metabolite BAM and any other substances. For the purposes of this tolerance action, therefore, EPA has not assumed that dichlobenil (parent) or its metabolite BAM has a common mechanism of toxicity with other substances. EPA has aggregated BAM exposure from both use of dichlobenil and fluopicolide. 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 website 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*. The pre- and postnatal toxicology database for dichlobenil includes rat and rabbit developmental toxicity studies and a 2-generation reproduction toxicity study in rats. There was no evidence of increased qualitative or quantitative susceptibility of *in utero* rats or rabbits in the developmental toxicity studies for dichlobenil. In the rat reproduction study, effects in the pups (decreased body weight during weaning) occurred at a lower dose than that which resulted in parental toxicity (decreases in premating and gestation body weight gain and premating food consumption), indicating increased quantitative susceptibility of the pups. However, the degree of concern for the body weight changes in pups is low. There are clear NOAELs for effects in both the pups and parental animals; and EPA is using the pup NOAEL, which is 6-fold lower than the dose at which decreased pup body weight was observed, to assess incidental oral exposure of children.

There was no evidence of increased prenatal susceptibility in the rabbit developmental toxicity study for BAM. In this study, an increase in the incidences of late abortion, as well as visceral and skeletal anomalies, was observed at the high dose. However, severe maternal toxicity (severely decreased body weight gain and food consumption and late abortion) was also observed at the same dose.

3. *Conclusion–i. Dichlobenil*. EPA has determined that the 10X FQPA SF must be retained for all prechronic (i.e., acute and subchronic) oral exposure scenarios. EPA has also determined that reliable data show the safety of infants and children would be adequately protected if the FQPA SF were reduced to 1X for all other (i.e., chronic, dermal or inhalation) exposure scenarios. These decisions are based on the following findings:

a. The dichlobenil database is incomplete to the extent that the existing data have not assayed the potential for dichlobenil to induce olfactory toxicity following short-term (prechronic) oral exposure. Olfactory toxicity has been assayed and demonstrated after dermal, inhalation and intraperitoneal exposure of rodents to dichlobenil. No oral studies, to date, have reported olfactory toxicity for dichlobenil; however, olfactory toxicity was assayed in only one study – a chronic dog study – submitted to the Agency. In the chronic dietary dog study, no effects on the nasal epithelium from long term exposure were observed. Due to the uncertainty regarding the potential for dichlobenil to induce olfactory toxicity following oral exposure of prechronic duration, EPA has retained the 10X FQPA SF. For chronic exposures and prechronic dermal and inhalation exposure scenarios, the 10X SF is not needed to account for database uncertainty. Olfactory toxicity was not observed in the chronic oral dog study, and the

doses selected for dermal and inhalation exposure risk assessments are based on a very sensitive and conservative endpoint (olfactory histopathology – epithelial damage). This is a conservative endpoint because it is unknown whether this olfactory histopathology would have an adverse effect on the function of the sense of smell.

b. Apart from the degenerative effects of dichlobenil on olfactory sensory neurons, there are no other indications of neurotoxicity in any of the studies available for dichlobenil. The 10X FQPA SF being retained for prechronic oral exposure scenarios is adequate to account for olfactory neurotoxicity. For dermal and inhalation exposure scenarios, EPA is using a very sensitive endpoint that should be protective of all populations, including infants and children.

c. There is no evidence that dichlobenil results in increased susceptibility in *in utero* rats or rabbits in the prenatal developmental toxicity studies. Although there is evidence of quantitative susceptibility in the 2-generation reproduction study in rats, the degree of concern is low, and the Agency did not identify any residual uncertainties after establishing toxicity endpoints and traditional UFs to be used in the risk assessment of dichlobenil.

d. There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessments were performed assuming 100 PCT and tolerance-level residues. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to dichlobenil in drinking water. Residential exposure of infants and children to dichlobenil is expected to be negligible. These assessments will not underestimate the exposure and risks posed by dichlobenil.

ii. *BAM:* EPA has retained the 10X FQPA SF for BAM for those exposure scenarios that do not rely on dichlobenil toxicity data. These scenarios are acute dietary for the general population (including infants and children) and females 13–49 years of age; chronic dietary; and incidental oral non-dietary. Although EPA has developmental, reproduction, and subchronic and chronic toxicity studies for the metabolite BAM, and a structure activity analysis indicates EPA has identified its principal toxicological effects and level of toxicity, EPA is retaining the FQPA 10X SF due to remaining questions regarding the systemic neurotoxic potential of BAM, including olfactory toxicity via the oral route of exposure and the use of a LOAEL in assessing acute dietary risk for the general population. For the dermal and inhalation routes of exposures, for which the Agency is relying on dichlobenil toxicity data, EPA has reduced the FQPA SF for BAM toxicity to 1X, based on a comparison of toxicity via the intraperitoneal route of exposure showing that higher doses of BAM are needed to induce levels of olfactory toxicity that are similar to those caused by dichlobenil. Olfactory toxicity, the most sensitive endpoint, was the endpoint chosen for these exposure scenarios. Other factors EPA considered in the FQPA SF decisions for BAM include the following:

a. To compensate for deficiencies in the toxicology database for BAM, EPA performed a comparative analysis of the toxicity of BAM and the parent compounds, dichlobenil and fluopicolide, using the available animal data and DEREK analysis. DEREK is a toxicology application that uses structure-activity relationships to predict a broad range of toxicological properties based on a comprehensive analysis of a compound's molecular structure. Based on the available animal data and Derek analyses, BAM does not appear to cause different organ specific toxicities compared to fluopicolide and dichlobenil. The kidney and liver toxicities are common to all three compounds. With respect to relative toxicity, conclusions from the evaluation of the animal studies appear to confirm that both fluopicolide and dichlobenil appear to be more or equally toxic compared to BAM. A full discussion of EPA's comparative toxicity analysis of BAM, dichlobenil and fluopicolide can be found at *http://www.regulations.gov* in the document *Comparative Toxicity using Derek analysis for Dichlobenil, Fluopicolide and BAM* in docket ID number EPA–HQ–OPP–2007–0604. Based on the results of the available animal data and the DEREK analysis, EPA concludes that the safety factors discussed in the previous paragraph are adequate.

b. There is no evidence that BAM results in increased susceptibility of *in utero* rabbits in the prenatal developmental toxicity study.

c. There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessments were refined using reliable PCT information and anticipated residue values calculated from residue field trial results. EPA made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to BAM 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 BAM.

E. Aggregate Risks and Determination of Safety

EPA determines whether acute and chronic pesticide exposures are safe by comparing aggregate exposure estimates to the aPAD and cPAD. The aPAD and cPAD represent the highest safe exposures, taking into account all appropriate SFs. EPA calculates the aPAD and cPAD by dividing the POD by all applicable UFs. For linear cancer risks, EPA calculates the probability of additional cancer cases 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 POD to ensure that the MOE called for by the product of all applicable UFs is not exceeded.

1. *Acute risk.* An acute aggregate risk assessment takes into account exposure estimates from acute dietary consumption of food and drinking water. Using the exposure assumptions discussed in this unit for acute exposure, the acute dietary exposure from food and water to dichlobenil will occupy 33% of the aPAD for females, 13 to 49 years old, the only subpopulation at risk from acute exposure to dichlobenil.

EPA performed two different acute risk assessments for BAM – one focusing on females 13 to 49 years old and designed to protect against prenatal effects and the other focusing on acute effects relevant to all other population groups. The more sensitive acute endpoint was seen as to prenatal effects rather than other acute effects. For females 13 to 49 years old, the acute dietary exposure from food and water will occupy 28% of the aPAD addressing prenatal effects. As to acute effects other than prenatal effects, the acute dietary exposure from food and water to BAM will occupy 28% of the aPAD for infants less than 1 year old, the population subgroup with the highest estimated acute dietary exposure to BAM.

2. *Chronic risk.* Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that chronic exposure to dichlobenil from food and water will utilize 30% of the cPAD for children, 1 to 2 years old, the population group receiving the greatest dichlobenil exposure. Chronic exposure to BAM from food and water

will utilize 93% of the cPAD for infants, less than 1 year old, the population group receiving the greatest BAM exposure. Based on the explanation in Unit III.C.3., regarding residential use patterns, chronic residential exposure to residues of dichlobenil or BAM is not expected.

3. *Short-/intermediate-term risk*. Short- and intermediate-term aggregate exposure takes into account short- or intermediate-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level). Although dichlobenil is registered for use on ornamentals in residential areas, residential handler exposures are not expected and post-application exposures of adults and children are expected to be negligible. Therefore, the short-term aggregate risk is the sum of the risk from exposure to dichlobenil through food and water and will not be greater than the chronic aggregate risk.

Fluopicolide is currently registered for uses that could result in short- and intermediate-term residential exposure to its metabolite, BAM, and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with short- and intermediate-term oral residential exposures to BAM. It is not appropriate to aggregate dietary (i.e., oral) exposures and dermal exposures because the toxic effects identified for the oral and dermal exposure pathways differ. Using the exposure assumptions described in this unit for short- and intermediate-term exposures, EPA has concluded the combined short-term food, water, and residential exposures aggregated result in aggregate MOEs of 3,200 for infants and 5,400 for children, 1 to 2 years old. The aggregate MOEs for infants and children include food and drinking water exposures to BAM from all existing and new uses of dichlobenil and fluopicolide, as well as post-application incidental oral exposures from activities on lawns treated with fluopicolide. MOEs for dermal exposures on treated lawns are 10,000 for adults and 6,000 for infants/children. As noted above, it is not appropriate to aggregate chronic exposure from food and water with oral exposures. Post-application inhalation exposure of adults and children is expected to be negligible.

4. *Aggregate cancer risk for U.S. population*. The Agency has determined that quantification of human cancer risk is not necessary for dichlobenil or BAM and that the chronic risk assessments based on the established cPADs are protective of potential cancer effects. Based on the results of the chronic risk assessments discussed in Unit III.E.2, EPA concludes that dichlobenil and BAM are not expected to pose a cancer risk.

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 dichlobenil or BAM residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (Pesticide Analytical Manual (PAM) Vol. II, Method A, a gas-liquid chromatography/electroconductivity detector (GLC/ECD) method) is available to enforce the tolerance expression. In addition, dichlobenil is completely recovered using the multiresidue methods in PAM Vol. I Sections 302 and 304. BAM is completely recovered using Section 302.

B. International Residue Limits

No CODEX, Canadian or Mexican maximum residue limits (MRLs) have been established for dichlobenil.

C. Revisions to Petitioned-For Tolerances

Based upon review of the data supporting the petition and recent changes in EPA's crop grouping regulations, EPA has revised the tolerance level for rhubarb and the commodity terms for the berry tolerances. The tolerance for rhubarb was reduced from 0.15 ppm to 0.06 ppm, the lower limit of method validation (LLMV), based on the absence of detectable residues in the field trials.

IR–4 petitioned for individual tolerances on caneberry, subgroup 13a and wild raspberry; bushberry, subgroup 13b; aronia berry; bluberry, lowbush; buffalo currant; chilian guava; european barberry; highbush cranberry; honeysuckle; jostaberry; juneberry; lingonberry; native currant; salal; and sea buckthorn. In the **Federal Register** of December 7, 2007 (72 FR 69150) (FRL–8340–6), EPA issued a final rule that revised the crop grouping regulations. As part of this action, EPA expanded and revised berries group 13. Changes to crop group 13 (berries) included adding new commodities, revising existing subgroups and creating new subgroups (including Caneberry subgroup 13–07A and Bushberry subgroup 13–07B, which include the berry commodities requested in IR–4's petition and cultivars, varieties, and/or hybrids of these).

EPA indicated in the December 7, 2007 final rule as well as the earlier May 23, 2007 proposed rule (72 FR 28920 (FRL–8126–1) that, for existing petitions for which a Notice of Filing had been published, the Agency would attempt to conform these petitions to the rule. Therefore, consistent with this rule, EPA is establishing tolerances on Caneberry subgroup 13–07A and Bushberry subgroup 13–07B. All of the berry commodities for which IR–4 requested tolerances are included in these revised subgroups.

EPA concludes it is reasonable to revise the petitioned-for tolerances so that they agree with the recent crop grouping revisions because:

1. Although the new crop groups/subgroups include several new commodities, the added commodities are closely related minor crops which contribute little to overall dietary or aggregate exposure and risk; and dichlobenil/BAM exposure from these added commodities was considered when EPA conducted the dietary and aggregate risk assessments supporting this action; and

2. The representative commodities for the revised crop group/subgroups have not changed.

V. Conclusion

Therefore, tolerances are established for combined residues of dichlobenil, 2,6-dichlorobenzonitrile, and its metabolite, 2,6-dichlorobenzamide, in or on bushberry subgroup 13–07B at 0.15 ppm; caneberry subgroup 13–07A at 0.10 ppm; and rhubarb at 0.06 ppm. The existing tolerances on individual members of bushberry subgroup 13–07B (blueberry) and caneberry subgroup 13–07A (blackberry and raspberry) that are superseded by the new crop subgroup tolerances at the same tolerance levels are being removed.

VI. Statutory and Executive Order Reviews

This final rule establishes tolerances under section 408(d) of FFDCA 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, *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 section 408(d) of FFDCA, 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 section 408(n)(4) of FFDCA. 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) (Public Law 104–4).

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), Public Law 104–113, section 12(d) (15 U.S.C. 272 note).

VII. Congressional Review Act

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

List of Subjects in 40 CFR Part 180

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

Dated: August 15, 2008.

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. Section 180.231 is amended by removing the commodities Blackberry, Blueberry and Raspberry and alphabetically adding the following commodities to the table in paragraph (a) to read as follows:

§ 180.231 Dichlobenil; tolerances for residues.

(a) * * *

Commodity	Parts per million
* * * * *	*
Bushberry subgroup 13–07B	0.15
Caneberry subgroup 13–07A	0.10
* * * * *	*
Rhubarb	0.06

* * * * *

[FR Doc. E8–19859 Filed 8–26–08; 8:45 am]

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FEDERAL COMMUNICATIONS COMMISSION

47 CFR Part 27

[WT Docket No. 02–353; FCC 03–251]

Service Rules for Advanced Wireless Services

AGENCY: Federal Communications Commission.

ACTION: Final rule; correction.

SUMMARY: In this document the Federal Communications Commission corrects an inadvertent error that occurred when the Commission adopted final rules for the Advanced Wireless Services in the 1710–1755 MHz and 2110–2155 MHz bands, including provisions for application, licensing, operating and technical rules, and for competitive bidding. These rules were published in the **Federal Register** on Friday, February 6, 2004 (69 FR 5711). Specifically, the error occurred in a table to the rules concerning interference protection at certain Federal Government operations in the 1710–1755 MHz band. As a result of this correction, the table will be amended as intended by the Commission.

DATES: Effective August 27, 2008.

FOR FURTHER INFORMATION CONTACT: John Spencer at 202–418–2487.

SUPPLEMENTARY INFORMATION: This is a correction to a summary of the Commission's Report and Order in WT Docket No. 02–353, FCC 03–251, adopted on October 16, 2003 and released on November 25, 2003. The Report and Order adopted licensing, technical, and competitive bidding rules to govern the use of the spectrum at 1710–1755 MHz and 2110–2155 MHz, which had previously been allocated for advanced wireless services, in a manner that would enable service providers to put this spectrum to use for any purpose consistent with its allocation.

Need for Correction

As published, the final rules contain an error in § 27.1134 in Table 1. The Commission inadvertently omitted the abbreviation for the word kilometers (km) after the category heading 'Radius of Operation' in Table 1: Protected Department of Defense Facilities. This correction restores the information that was inadvertently omitted.