Express Mail Contract 3 (MC2009–15 and CP2009–21)
Express Mail & Priority Mail Contract 1 (MC2009–6 and CP2009–7)
Express Mail & Priority Mail Contract 2 (MC2009–12 and CP2009–14)
Express Mail & Priority Mail Contract 3 (MC2009–13 and CP2009–17)
Express Mail & Priority Mail Contract 4 (MC2009–17 and CP2009–24)
Express Mail & Priority Mail Contract 5 (MC2009–18 and CP2009–25)
Express Mail & Priority Mail Contract 6 (MC2009–31 and CP2009–42)
Express Mail & Priority Mail Contract 7 (MC2009–32 and CP2009–43)
Parcel Return Service Contract 1 (MC2009–1 and CP2009–2)
Priority Mail Contract 1 (MC2008–8 and CP2008–26)
Priority Mail Contract 2 (MC2009–2 and CP2009–3)
Priority Mail Contract 3 (MC2009–4 and CP2009–5)
Priority Mail Contract 4 (MC2009–5 and CP2009–6)
Priority Mail Contract 5 (MC2009–21 and CP2009–26)
Priority Mail Contract 6 (MC2009–25 and CP2009–30)
Priority Mail Contract 7 (MC2009–25 and CP2009–31)
Priority Mail Contract 8 (MC2009–25 and CP2009–32)
Priority Mail Contract 9 (MC2009–25 and CP2009–33)
Priority Mail Contract 10 (MC2009–25 and CP2009–34)
Priority Mail Contract 11 (MC2009–27 and CP2009–37)
Priority Mail Contract 12 (MC2009–28 and CP2009–38)
Priority Mail Contract 13 (MC2009–29 and CP2009–39)
Priority Mail Contract 14 (MC2009–30 and CP2009–40)
Outbound International
Global Direct Contracts (MC2009–9, CP2009–10, and CP2009–11)
Global Expedited Package Services (GEPS) Contracts
GEPS 1 (CP2008–5, CP2008–11, CP2008–12, and CP2008–13, CP2008–18, CP2008–19, CP2008–20, CP2008–21, CP2008–22, CP2008–23, and CP2008–24)
Global Plus Contracts
Global Plus 1 (CP2008–9 and CP2008–10)
Global Plus 2 (MC2008–7, CP2008–16 and CP2008–17)
Inbound International
Inbound Direct Entry Contracts With Foreign Postal Administrations (MC2008–6, CP2008–14 and CP2008–15)
International Business Reply Service Competitive Contract 1 (MC2009–14 and CP2009–20)

Competitive Product Descriptions

Express Mail
[Reserved for Group Description]
Express Mail
[Reserved for Product Description]
Outbound International Expedited Services
[Reserved for Product Description]
Inbound International Expedited Services
[Reserved for Product Description]
Priority
[Reserved for Product Description]
Priority Mail
[Reserved for Product Description]
Outbound Priority Mail International
[Reserved for Product Description]
Inbound Air Parcel Post
[Reserved for Product Description]
Parcel Select
[Reserved for Group Description]
Parcel Return Service
[Reserved for Group Description]
International
[Reserved for Group Description]
International Priority Airlift (IPA)
[Reserved for Product Description]
International Surface Airlift (ISAL)
[Reserved for Product Description]
International Direct Sacks—M-Bags
[Reserved for Product Description]
Global Customized Shipping Services
[Reserved for Product Description]
International Money Transfer Service
[Reserved for Product Description]
Inbound Surface Parcel Post (at non-UPU rates)
[Reserved for Product Description]
International Ancillary Services
[Reserved for Product Description]
International Certificate of Mailing
[Reserved for Product Description]
International Registered Mail
[Reserved for Product Description]
International Return Receipt
[Reserved for Product Description]
International Restricted Delivery
[Reserved for Product Description]
International Insurance
[Reserved for Product Description]
Negotiated Service Agreements
[Reserved for Group Description]
Domestic
[Reserved for Product Description]
Outbound International
[Reserved for Group Description]

Part C—Glossary of Terms and Conditions [Reserved]

Part D—Country Price Lists for International Mail [Reserved]

[FR Doc. E9–18737 Filed 8–4–09; 8:45 am]

**BILLING CODE 7710–FW–P**

## ENVIRONMENTAL PROTECTION AGENCY

### 40 CFR Part 180

**[EPA–HQ–OPP–2008–0889; FRL–8430–2]**

### Amine Salts of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (Dimethylaminopropylamine, Isopropylamine, Mono-, Di-, and Triethanolamine); Exemption from the Requirement of a Tolerance

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Final rule.

**SUMMARY:** This regulation establishes an exemption from the requirement of a tolerance for residues of amine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (dimethylaminopropylamine, isopropylamine, mono-, di-, and triethanolamine) when used as an inert ingredient in pesticide formulations applied to growing crops and applied to animals. The Joint Inerts Task Force, Cluster Support Team Number 8, submitted a petition to EPA under the Federal Food, Drug, and Cosmetic Act (FFDCA), requesting an exemption from the requirement of a tolerance. This regulation eliminates the need to establish a maximum permissible level for residues of amine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (dimethyl aminopropylamine, isopropylamine, mono-, di-, and triethanolamine).

**DATES:** This regulation is effective August 5, 2009. Objections and requests for hearings must be received on or before October 5, 2009, 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–2008–0889. All documents in the docket are listed in the docket index available at *http://www.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:** Kerry Leifer, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 308–8811; e-mail address: *leifer.kerry@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 electronically available documents 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 e-CFR cite at *http://www.gpoaccess.gov/ecfr*. To access the OPPTS Harmonized Guidelines referenced in this document, go directly to the guidelines at *http://www.epa.gov/opptsfrs/home/guidelin.htm*.

*C. Can I File an Objection or Hearing Request?*

Under section 408(g) of FFDCA, 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–2008–0889 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 5, 2009.

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–2008–0889, 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 Facility'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. Background

In the **Federal Register** of March 25, 2009 (74 FR 12856) (FRL–8399–4), 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 8E7472) by The Joint Inerts Task Force (JITF), Cluster Support Team 8 (CST 8), c/o CropLife America, 1156 15th Street, NW., Suite 400, Washington, DC 20005. The petition requested that 40 CFR 180.920 and 40 CFR 180.930 be amended by establishing exemptions from the requirement of a tolerance for residues of the inert ingredient amine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (dimethylaminopro-pylamine, isopro pylamine, mono-, di-, and triethanol amine) (herein referred to in this document as ASABSA) including CAS Reg. Nos. 68953–97–9, 26545–53–9, 877677–48–0, 319926–68–6, 90194–53–9, 55470–69–4, 68910–32–7, 26264–05–1, 157966–96–6, 68584–24–7, 68648–81–7, 68649–00–3, 68953–93–5, 90218–35–2, 27323–41–7, 68584–25–8, 68648–96–4, 68411–31–4, 90194–42–6, and 1093628–27–3, when used as an inert ingredient in pesticide formulations applied to growing crops under 40 CFR 180.920 and applied to animals under 40 CFR 180.930. That notice referenced a summary of the petition prepared by The JITF, CST 8, the petitioner, 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, EPA has modified the exemption requested by limiting the diethanolamine salt of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (CAS Reg. Nos. 26545–53–9 and 68953–97–9) to a maximum of 7% by weight in pesticide formulations intended for application to growing crops and to animals. This limitation is based on the Agency's risk assessment which can be found at *http://www.regulations.gov* in documents ''Dimethylaminopro pylamine, Isopropylamine, Ethanol amine and Triethanolamine Salts of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (JITF CST 8 Inert Ingredients). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations and Diethanolamine Salt of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (DEA - JITF CST 8 Inert Ingredient). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations,'' in docket ID number EPA–HQ–OPP–2008–0889.

This petition was submitted in response to a final rule that was published in the **Federal Register** of August 9, 2006 (71 FR 45415) (FRL–8084–1) in which the Agency revoked, under section 408(e)(1) of FFDCA, the existing exemptions from the requirement of a tolerance for residues of certain inert ingredients because of insufficient data to make the determination of safety required by section 408(b)(2) of FFDCA. The expiration date for the tolerance exemptions subject to revocation was August 9, 2008, which was later extended to August 9, 2009 in the **Federal Register** of August 4, 2008 (73 FR 45317) (FRL–8373–6) to allow for data to be submitted to support the establishment of tolerance exemptions for these inert ingredients prior to the effective date of the tolerance exemption revocation.

## III. Inert Ingredient Definition

Inert ingredients are all ingredients that are not active ingredients as defined in 40 CFR 153.125 and include, but are not limited to, the following types of ingredients (except when they have a pesticidal efficacy of their own): Solvents such as alcohols and hydrocarbons; surfactants such as polyoxyethylene polymers and fatty acids; carriers such as clay and diatomaceous earth; thickeners such as carrageenan and modified cellulose;

wetting, spreading, and dispersing agents; propellants in aerosol dispensers; microencapsulating agents; and emulsifiers. The term ''inert'' is not intended to imply nontoxicity; the ingredient may or may not be chemically active. Generally, EPA has exempted inert ingredients from the requirement of a tolerance based on the low toxicity of the individual inert ingredients.

**IV. Aggregate Risk Assessment and Determination of Safety**

Section 408(b)(2)(A)(i) of FFDCA allows EPA to establish an exemption from the requirement of 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. * * *''

EPA performs a number of analyses to determine the risks from aggregate exposure to pesticide residues. First, EPA determines the toxicity of pesticides. Second, EPA examines exposure to the pesticide through food, drinking water, and through other exposures that occur as a result of pesticide use in residential settings.

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 exemption from the requirement of a tolerance for residues of ASABSA when used as inert ingredients in pesticide formulations applied to growing crops and to animals. 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.

Amine salts of alkyl ($C_8$-$C_{24}$) benzene sulfonic acid readily and fully dissociate to the corresponding amine and alkyl ($C_8$-$C_{24}$) benzenesulfonic acid constituents, therefore the hazard assessment conducted to support the requested exemption from the requirement of a tolerance for ASABSA is primarily based on the hazard assessment for each of the constituents, specifically each associated amine (i.e., dimethylaminopropylamine, isopro pylamine, ethanolamine, diethanol-amine and triethanolamine) and alkyl ($C_8$-$C_{24}$) benzenesulfonic acid.

The hazard profile and endpoints for risk assessment for alkylbenzene sulfonic acid have previously been addressed as part of the tolerance reassessment for tolerance exemptions for alkyl ($C_8$-$C_{24}$) benzenesulfonic acid and its ammonium, calcium, magnesium, potassium, sodium, and zinc salts *http://www.epa.gov/opprd001/inerts/alkylc8.pdf*. The toxicology database for these alkyl benzene sulfonates consists almost entirely of published literature, and is essentially complete and of acceptable quality to assess the potential hazard to humans. The alkylbenzene sulfonates are readily absorbed following oral ingestion, but not following dermal exposure. Following oral exposure, they are readily metabolized, excreted fairly rapidly, and do not accumulate in any tissues. Available acute toxicity data show that alkylbenzene sulfonates are not highly acutely toxic, are irritating to the eye and skin, and are not skin sensitizers. Subchronic and chronic exposures show that the liver, kidney and intestinal tract (following oral exposures) are the major target organs of toxicity. Both *in vitro* and *in vivo* genotoxicity data show that alkylbenzene sulfonates are not genotoxic. The alkylbenzene sulfonates did not cause reproductive or developmental toxicity in acceptable studies. Early (pre Good Laboratory Practice standards) carcinogenicity studies indicate that alkylbenzene sulfonates do not cause an increase in tumor incidence.

The existing toxicology database for the dimethylaminopropylamine, isopropylamine, ethanolamine and triethanolamine salt of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid consists of an OPPTS Harmonized Test Guideline 870.3550 study and acute, subchronic, chronic, carcinogenicity, developmental, and mutagenicity studies on the individual amines. In addition, the petitioner submitted an OPPTS Harmonized Test Guideline 870.3650 combined repeated dose toxicity study with the reproduction/developmental toxicity screening tests on isopropylamine dodecylbenzene sulfonate. The Agency considered these data in its evaluation of amine toxicity. While the test compound for the study is effectively a mixture of the amine and the acid, the study findings do provide some insight into the potential toxicity of the amine constituent.

A summary of the toxicological data considered as part of this action is given below:

1. *Isopropylamine dodecylbenzene sulfonate* (CAS No. 26264–05–1). In an oral gavage OPPTS Harmonized Test Guideline 870.3650 combined repeated dose toxicity study with the reproduction/developmental toxicity screening tests, the parental LOAEL was 320 milligrams/kilograms/day (mg/kg/day) (highest dose tested, (HDT)) based on excessive salivation (both sexes), soft/liquid feces (males), lesions of the forestomach (both sexes). No reproductive or developmental toxicity or neurotoxicity was observed. The NOAEL was 80 mg/kg/day.

2. *Ethanolamine* (CAS No. 141–43–5). Ethanolamine is not acutely toxic in rats by the oral route of exposure but appears to be very acutely toxic by the dermal route of exposure, although this may be a species-specific effect in the rabbit. It is a skin sensitizer and is corrosive to the eye and skin. There is no evidence of mutagenicity in the Ames, Saccharomyces cerevisiae gene conversion, mouse micronucleus, cell transformation, and SCE human lymphocytes tests. In a dermal rat developmental toxicity study conducted with ethanolamine, no maternal or developmental toxicity was observed at 225 mg/kg/day (HDT). Also in a dermal rabbit developmental toxicity study, no maternal or developmental toxicity was observed at 75 mg/kg/day (HDT). In an oral rat developmental toxicity study, the maternal LOAEL was 450 mg/kg/day (HDT) based on decreased body weights during the latter part of gestation and throughout lactation. The developmental LOAEL was 450 mg/kg/day based on decrease body weights in female fetuses on postnatal day (PND) 1 and 4. The maternal/developmental NOAEL was 120 mg/kg/day.

3. *Triethanolamine* (CAS No. 102–71–6). In acute toxicity studies, triethanolamine is mildly to moderately toxic by the oral and dermal routes of

exposure. It is not irritating in eye and skin irritation studies, and it is not a skin sensitizer. There is no evidence of mutagenicity in the Ames, mouse micronucleus, sex-linked recessive lethal, and Chinese hamster ovary (CHO) cell cytogenetics tests. In a 14–day inhalation study in rat, the NOAEL was 0.25 milligram/liter (mg/L) (approximate oral equivalent dose of 75 mg/kg/day) and the LOAEL was 0.5 mg/L based on increased kidney weights of males and females. In an oral mouse developmental toxicity study (Chernoff-Kavlock screening test), no maternal or developmental toxicity was observed at 1,125 mg/kg/day (only dose tested). In a 13–week dermal study in rat, the NOAEL was 1,000 mg/kg/day and the LOAEL was 2,000 mg/kg/day (HDT) based on reduced body gain and clinical observations (irritation, scaliness, and crustiness of the skin at the site of application). In a 13–week dermal study in mouse, the NOAEL was 2,000 mg/kg/day and the LOAEL was 4,000 mg/kg/day (HDT) based on clinical observations (irritation, scaliness, and discoloration of the skin at the site of application).

4. *Isopropylamine* (CAS No. 75–31–0). In acute toxicity studies, isopropylamine is moderately acutely toxic in rats by the oral route of exposure, but is less toxic by the dermal route and is not toxic by the inhalation route of exposure. Rabbits appear to be more sensitive than rats showing significantly greater acute toxicity by the dermal route. Isopropylamine is not a skin sensitizer. There is no evidence of mutagenicity in the Ames, chromosomal aberrations in human lymphocytes and unscheduled DNA synthesis in rat hepatocytes tests. In a 28–day inhalation study, Sprague-Dawley rats were exposed to inhalation dosage levels of 0, 0.1, 0.5, and 1.35 mg/L for 6 hours/day for 5 days/week. The NOAEL was 0.1 mg/L and the LOAEL was 0.5 mg/L based on microscopic ocular and nasal lesions. In a developmental study, Sprague-Dawley rats were exposed to inhalation dosage levels of 0, 0.1, 0.5, and 1.0 mg/L for 6 hours/day from gestation day (GD) 6 through 15. The maternal toxicity was observed at 1.0 mg/L (HDT) based on decreased body weight and body weight gain. At this dose, no developmental toxicity was observed.

5. *Dimethylaminopropylamine* (CAS No. 109–55–7). Dimethylaminopropylamine is mild to moderately toxic by the oral and inhalation routes of exposure, but it is not a skin sensitizer. There is no evidence of mutagenicity in the Ames and mouse micronucleus tests. Following a 28–day gavage study in Wistar rats, mortality (4/5 females) and clinical signs (males: irregular respiration and respiratory sounds; females: decreased spontaneous activity, stilted gait, swollen abdomen, and impaired respiration) were observed at 250 mg/kg/day (HDT). In an OPPTS Harmonized Test Guideline 870.3550 reproduction and developmental toxicity screening test in Sprague-Dawley rats, parental toxicity was observed at 200 mg/kg/day (HDT) based on decreased body weight gain and clinical signs (respiratory sounds and piloerection). Reproductive and developmental toxicity were not observed at any dose level.

6. *Diethanolamine* (CAS No. 11–42–2). The existing toxicology database for diethanolamine (DEA) consists of several subchronic oral and dermal toxicity studies in rats and mice, carcinogenicity studies in rats and mice, oral and dermal developmental toxicity studies in rats and rabbits, and acute and mutagenicity data. Following repeat oral exposure to DEA, the kidney, liver, and blood are the major target organs. Repeat oral exposure via drinking water resulted in a microcytic anemia that does not involve the bone marrow in rats at 97 mg/kg/day in males and 57 mg/kg/day in females. Increased kidney weights were associated with renal tubular cell necrosis, decreased renal function, increased incidences or severity of nephropathy, and/or mineralization in rats at 97 mg/kg/day (males) and 57 mg/kg/day (females) and in mice at 104 mg/kg/day (lowest dose tested, (LDT)) in males and 142 mg/kg/day (LDT) in females. Increased liver weights were associated with cytoplasmic vacuolization and degeneration of centrilobular hepatocytes in rats and hypertrophy, individual cell necrosis or foci of necrotic hepatocytes in mice. Dose-related decreases in testis and epididymis weights were associated with testicular degeneration, decreased sperm motility, and decreased sperm count in male rats at 97 mg/kg/day. Similar kidney and liver effects were observed following repeat dermal exposure at dose levels of 32/mg/kg/day in rats and 80 mg/kg/day in mice. Demyelination in the brain (medulla oblongata) and spinal cord was observed in rats of both sexes following oral and dermal exposure at dose levels as low as 250 mg/kg/day, with the female being more sensitive. Mortality and neurological symptoms (tremors, stiffness, and ataxia progressing to paresis and paralysis) have been reported following exposure via over-the-counter oral flea treatment (53% DEA) of dogs and cats, however, there are no registered pet care use products containing the DEA salt form of ASABSA.

Developmental toxicity was observed in rats following both oral and dermal exposure to the maternal animal during gestation days (GD) 6-15. Maternal toxicity, as evidenced by decreased body weight/gain and food consumption and/or increased kidney weight, was observed at the same dose levels (125 mg/kg/day) as the developmental effects [an increase in postnatal mortality (PND 0 through 4), an increase in postimplantation loss, and reduced pup body weight following oral exposure. An increased incidence of skeletal variations was observed following dermal exposure at 1500 mg/kg/day (HDT) ]. Developmental toxicity was not observed in rabbits following oral or dermal exposure of the maternal animal during GD 6 through 18.

7. *Metabolism*. The alkyl ($C_8$-$C_{24}$) benzenesulfonic acid amine salts undergo rapid dissociation *in vivo* to form an alkyl ($C_8$-$C_{24}$) benzenesulfonic acid and an amine. The two entities would be absorbed and metabolized independently. The alkyl ($C_8$-$C_{24}$) benzenesulfonic acid should be readily conjugated and rapidly excreted with little alkyl aromatic chain degradation (JITF Submission, 2008, pages 11 and 21). Primary, secondary or tertiary amines should undergo oxidative amine metabolism followed by excretion. Primary aliphatic amines (ethanolamine, isopropylamine) are oxidized to aldehydes/ketones and or acid (glycolic acid or acetone) with release of ammonia. The glycolic acid may further oxidized and or conjugated and excreted. The acetone could be excreted through respiration or further oxidized to methylglyoxyl and then excreted. Secondary aliphatic amines (dimethylaminopropylamine and diethanolamine) may follow various oxidative patterns and some are excreted unchanged. Small molecular weight amines may be exhaled via respiration. Tertiary aliphatic amines (triethanolamine) may be oxidized to amine oxides, which may be excreted in the urine or deaminated with the eventual resultant being release of glycolic acid which may be further oxidized and or conjugated and excreted.

Specific information on the studies received and the nature of the adverse effects caused by ASABSA and its constituents 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 documents ''Dimethylaminopropylamine, Isopropylamine, Ethanolamine and Triethanolamine Salts of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (JITF CST 8 Inert Ingredients). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations and Diethanolamine Salt of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (DEA - JITF CST 8 Inert Ingredient). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations,'' in docket ID number EPA–HQ–OPP–2008–0889 and at *http://www.epa.gov/opprd001/inerts/alkylc8.pdf*.

*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 ASABSA used for human health risk is shown in the following Table 1.

TABLE 1.—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR ASABSA 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) | An effect attributable to a single exposure was not identified. | | |
| Chronic dietary (all populations) dimethyl aminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid. | NOAEL = 50 mg/kg/day<br>$UF_A$ = 10x<br>$UF_H$ = 10x<br>FQPA SF = 1x | Chronic RfD = 0.5 mg/kg/day<br>cPAD = 0.5 mg/kg/day | 28–day oral (gavage) toxicity study in rats with dimethylaminopropylamine<br>NOAEL = 50 mg/kg<br>LOAEL = 250 mg/kg based on mortality (4/5 females) and clinical signs (males: irregular respiration and respiratory sounds; females: decreased spontaneous activity, stilted gait, swollen abdomen, impaired respiration) OECD SIDS. UNEP Publication and BUA Report, October 1996 plus weight of evidence of three studies with alkylbenzene sulfonates:<br>1) Rat reproduction study LOAEL = 250 mg/kg/day based on decreased Day 21 female pup body weight (Buehler, E. et al. 1971. Tox. Appl. Pharmacol.18:83-91)<br>2) 9–month drinking water rat study<br>LOAEL = 145 mg/kg/day based on decreased body weight gain, and serum/ biochemical and enzymatic changes in the liver andkidney (Yoneyama et al. 1976 Ann. Rep. Tokyo Metrop. Res.Lab. Public Health 27(2):105-112)<br>3) 6–month rat dietary study<br>LOAEL = 114 mg/kg/day (0.2%) based on increased caecum weight and slight kidney damage (Yoneyama et al 1972 Ann. Rep. Tokyo Metrop. Res. Lab. Public Health 24:409-440) |

TABLE 1.—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR ASABSA FOR USE IN HUMAN HEALTH RISK ASSESSMENT—Continued

| Exposure/Scenario | Point of Departure and Uncertainty/Safety Factors | RfD, PAD, LOC for Risk Assessment | Study and Toxicological Effects |
|---|---|---|---|
| Chronic dietary (all populations) diethanolamine salt of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid | NOAEL = 48 mg/kg/day<br>$UF_A$ = 10x<br>$UF_H$ = 10x<br>FQPA SF = 10x | Chronic RfD = 0.5 mg/kg/day<br>cPAD = 0.05 mg/kg/day | Subchronic (13–week) oral toxicity study in rats (NTP, 1992)<br>Female LOAEL = 124 mg/kg/day demyelination of the brain and spinal cord<br>Male LOAEL = 97 mg/kg/day, based on decreased testis and epididymis weight associated with degeneration of seminiferous epithelium, decreased numbers of spermatogenic cells, reduced size of seminiferous tubules, decreased sperm, sperm motility, and sperm count |
| Incidental Oral and Inhalation short-term (1 to 30 days) and intermediate-term (1 to 6 months) dimethylaminopropylamine, isopropylamine, ethanol amine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid. | NOAEL = 50 mg/kg/day<br>$UF_A$ = 10x<br>$UF_H$ = 10x<br>FQPA SF = 1x<br>inhalation toxicity is assumed to be equivalent to oral toxicity | Residential LOC for MOE = 100 | 28–day oral (gavage) toxicity study in rats with dim ethylaminopropylamine<br>NOAEL = 50 mg/kg<br>LOAEL = 250 mg/kg based on mortality (4/5 females) and clinical signs (males: irregular respiration and respiratory sounds; females: decreased spontaneous activity, stilted gait, swollen abdomen, impaired respiration) OECD SIDS. UNEP Publication and BUA Report, October 1996 plus weight of evidence of three studies with alkylbenzene sulfonates:<br>1) Rat reproduction study LOAEL = 250 mg/kg/day based on decreased Day 21 female pup body weight (Buehler, E. et al. 1971. Tox. Appl. Pharmacol.18:83-91)<br>2) 9–month drinking water rat study LOAEL = 145 mg/kg/day based on decreased body weight gain, and serum/ biochemical and enzymatic changes in the liver andkidney (Yoneyama et al. 1976 Ann. Rep. Tokyo Metrop. Res. Lab. Public Health 27(2):105-112)<br>3) 6–month rat dietary study LOAEL = 114 mg/kg/day (0.2%) based on increased caecum weight and slight kidney damage (Yoneyama et al 1972 Ann. Rep. Tokyo Metrop. Res. Lab. Public Health 24:409-440) |
| Incidental Oral and Inhalation short-term (1 to 30 days) and intermediate-term (1 to 6 months)--diethanolamine salt of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid. | NOAEL = 48 mg/kg/day<br>$UF_A$ = 10x<br>$UF_H$ = 10x<br>FQPA SF = 10x<br>inhalation toxicity is assumed to be equivalent to oral toxicity | Residential LOC for MOE = 1,000 | Subchronic (13–week) oral toxicity study in rats (NTP, 1992)<br>Female LOAEL = 124 mg/kg/day based on demyelination of the brain and spinal cord<br>Male LOAEL = 97 mg/kg/day, based on decreased testis and epididymis weight associated with degeneration of seminiferous epithelium, decreased numbers of spermatogenic cells, reduced size of seminiferous tubules, decreased sperm, sperm motility, and sperm count |
| Dermal (short- and intermediate-term) -- dimethy laminopropylamine, isopropylamine, ethano lamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid. | No systemic toxicity observed in available dermal toxicity study. Low potential for dermal absorption to ionized amine. No quantitative risk assessment required | | |
| Dermal (short- and intermediate-term) — diethanolamine salt of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid | NOAEL = 125 mg/kg/day<br>$UF_A$ = 10x<br>$UF_H$ = 10x<br>FQPA SF = 10x | Residential LOC for MOE = 1,000 | |

TABLE 1.—SUMMARY OF TOXICOLOGICAL DOSES AND ENDPOINTS FOR ASABSA FOR USE IN HUMAN HEALTH RISK ASSESSMENT—Continued

| Exposure/Scenario | Point of Departure and Uncertainty/Safety Factors | RfD, PAD, LOC for Risk Assessment | Study and Toxicological Effects |
|---|---|---|---|
| Cancer (oral, dermal, inhalation) | Classification: Based on SAR analysis, ASABSA is not expected to be carcinogenic. No evidence of carcinogenicity in the available data or SAR analysis for alkyl benzene sulfonates, dimethylaminopropylamine, isopropylamine, ethanolamine, and triethano lamine. No concern for diethanolamine based on SAR analysis, limited evidence in experimental animals; not classifiable as to its carcinogenicity to humans | | |

Point of Departure (POD) = A data point or an estimated point that is derived from observed dose-response data and used to mark the beginning of extrapolation to determine risk associated with lower environmentally relevant human exposures. NOAEL = no observed adverse effect level. LOAEL = lowest observed adverse effect level. UF = uncertainty factor. $UF_A$ = extrapolation from animal to human (interspecies). $UF_H$ = potential variation in sensitivity among members of the human population (intraspecies). PAD = population adjusted dose (a=acute, c=chronic). FQPA SF = FQPA Safety Factor. RfD = reference dose. MOE = margin of exposure. LOC = level of concern. N/A = not applicable.

*C. Exposure Assessment*

Very limited information is available for ASABSA with respect to plant and animal metabolism or environmental degradation. The Agency relied collectively on information provided on the representative chemical structures, the generic cluster structures, the modeled physicochemical information, as well as the structure-activity relationship information. Additionally, information on other surfactants and chemicals of similar size and functionality was considered to determine the residues of concern for these inert ingredients. ASABSA are likely to be fully dissociated in solution. If dissociated amine counter ion or alkylbenzenesulfonic acid residues on plants and livestock undergo any metabolism or hydrolysis, they will likely result as highly polar or conjugated residues, which would not be of concern.

1. *Dietary exposure from food and feed uses.* In evaluating dietary exposure to ASABSA, EPA considered exposure under the petitioned-for exemptions from the requirement of a tolerance. EPA assessed dietary exposures from ASABSA in food as follows:

i. *Acute exposure.* No adverse effects attributable to a single exposure of ASABSA were seen in the toxicity databases. Therefore, an acute dietary risk assessment for ASABSA is not necessary.

ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment for ASABSA, EPA used food consumption information from the U.S. Department of Agriculture (USDA) 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, no residue data were submitted for ASABSA. In the absence of specific residue data, EPA has developed an approach which uses surrogate information to derive upper bound exposure estimates for the subject inert ingredient. Upper bound exposure estimates are based on the highest tolerance for a given commodity from a list of high-use insecticides, herbicides, and fungicides. A complete description of the general approach taken to assess inert ingredient risks in the absence of residue data can be found at *http://www.regulations.gov* in the document ''Alkyl Amines Polyalkoxylates (Cluster 4): Acute and Chronic Aggregate (Food and Drinking Water) Dietary Exposure and Risk Assessments for the Inerts'', in docket ID number EPA–HQ–OPP–2008–0738.

In the dietary exposure assessment, the Agency assumed that the residue level of the inert ingredient would be no higher than the highest tolerance for a given commodity. Implicit in this assumption is that there would be similar rates of degradation (if any) between the active and inert ingredient and that the concentration of inert ingredient in the scenarios leading to these highest of tolerances would be no higher than the concentration of the active ingredient.

The Agency believes the assumptions used to estimate dietary exposures lead to an extremely conservative assessment of dietary risk due to a series of compounded conservatisms. First, assuming that the level of residue for an inert ingredient is equal to the level of residue for the active ingredient will overstate exposure. The concentrations of active ingredient in agricultural products are generally at least 50 percent of the product and often can be much higher. Further, pesticide products rarely have a single inert ingredient; rather there is generally a combination of different inert ingredients used which additionally reduces the concentration of any single inert ingredient in the pesticide product relative to that of the active ingredient. EPA made a specific adjustment to the dietary exposure assessment to account for the use limitations of the amount of diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid that may be in formulations (no more than 7%, which corresponds to a concentration of 2% diethanolamine) and assumed that the diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid are at the maximum limitations rather than at equal quantities with the active ingredient. This remains a very conservative assumption because surfactants are generally used at levels far below these percentages. For example, EPA examined several of the pesticide products associated with the tolerance/commodity combination which are the driver of the risk assessment and found that these products did not contain surfactants at levels greater than 2.25% and that none of the surfactants were diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid.

Second, the conservatism of this methodology is compounded by EPA's decision to assume that, for each commodity, the active ingredient which will serve as a guide to the potential level of inert ingredient residues is the active ingredient with the highest tolerance level. This assumption overstates residue values because it would be highly unlikely, given the high number of inert ingredients, that a single inert ingredient or class of ingredients would be present at the level of the active ingredient in the highest tolerance for every commodity. Finally, a third compounding conservatism is EPA's assumption that all foods contain the inert ingredient at the highest tolerance level. In other words, EPA assumed 100 percent of all foods are treated with the inert ingredient at the rate and manner necessary to produce the highest residue legally possible for an active ingredient.

In summary, EPA chose a very conservative method for estimating what level of inert residue could be on food, and then used this methodology to choose the highest possible residue that could be found on food and assumed that all food contained this residue. No consideration was given to potential degradation between harvest and consumption even though monitoring data shows that tolerance level residues are typically one to two orders of magnitude higher than actual residues in food when distributed in commerce.

Accordingly, although sufficient information to quantify actual residue levels in food is not available, the compounding of these conservative assumptions will lead to a significant exaggeration of actual exposures. EPA does not believe that this approach underestimates exposure in the absence of residue data.

iii. *Cancer*. The Agency used a qualitative structure activity relationship (SAR) database, DEREK11, to determine if there were structural alerts suggestive of carcinogenicity. No structural alerts for carcinogenicity were identified. Additionally, there is not evidence of carcinogenicity of the ASABSA amine or alkylbenzenesulfonic acid constituents. Therefore, a cancer dietary exposure assessment is not necessary to assess cancer risk.

iv. *Anticipated residue and percent crop treated (PCT) information*. EPA did not use anticipated residue and/or PCT information in the dietary assessment for ASABSA. Tolerance level residues and/or 100% crop treated were assumed for all food commodities.

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

A screening level drinking water analysis, based on the Pesticide Root Zone Model/Exposure Analysis Modeling System (PRZM/EXAMS) was performed to calculate the estimated drinking water concentrations (EDWCs) of ASABSA. Modeling runs on four surrogate inert ingredients using a range of physical chemical properties that would bracket those of ASABSA were conducted. Modeled acute drinking water values ranged from 0.001 parts per billion (ppb) to 41 ppb. Modeled chronic drinking water values ranged from 0.0002 ppb to 19 ppb. Further details of this drinking water analysis can be found at *http://www.regulations.gov* in the documents ''Dimethylaminopropylamine, Isopropylamine, Ethanolamine and Triethanolamine Salts of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (JITF CST 8 Inert Ingredients). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations and Diethanolamine Salt of Alkyl ($C_8$-$C_{24}$) Benzenesulfonic Acid (DEA - JITF CST 8 Inert Ingredient). Human Health Risk Assessment to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations,'' in docket ID number EPA–HQ–OPP–2008–0889.

For the purpose of the screening level dietary risk assessment to support this request for an exemption from the requirement of a tolerance for ASABSA, a conservative drinking water concentration value of 100 ppb based on screening level modeling was used to assess the contribution to drinking water for chronic dietary risk assessments for the parent compounds and for the metabolites of concern. These values were directly entered into the dietary exposure model.

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). ASABSA may be used as inert ingredients in pesticide products that are registered for specific uses that may result in outdoor residential exposures. A screening level residential exposure and risk assessment was completed for pesticide products containing ASABSA as inert ingredients. In this assessment, representative scenarios, based on end-use product application methods and labeled application rates, were selected. For each of the use scenarios, the Agency assessed residential handler (applicator) inhalation and dermal exposure for use scenarios with high exposure potential (i.e., exposure scenarios with high-end unit exposure values) to serve as a screening assessment for all potential residential pesticides containing ASABSA. Similarly, residential postapplication dermal and oral exposure assessments were also performed utilizing high-end exposure scenarios. Further details of this residential exposure and risk analysis can be found at *http://www.regulations.gov* in the document ''JITF Inert Ingredients. Residential and Occupational Exposure Assessment Algorithms and Assumptions Appendix for the Human Health Risk Assessments to Support Proposed Exemption from the Requirement of a Tolerance When Used as Inert Ingredients in Pesticide Formulations,'' in docket ID number EPA–HQ–OPP–2008–0710.

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.''

EPA has not found ASABSA to share a common mechanism of toxicity with any other substances, and ASABSA do not appear to produce a toxic metabolite produced by other substances. For the purposes of this tolerance action, therefore, EPA has assumed that ASABSA do not have a common mechanism of toxicity with other substances. 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*.—i. *Dimethylaminopropylamine, isopropylamine, ethanol amine, and triethanolamine salts of alkyl* ($C_8$-$C_{24}$) *benzenesulfonic acid*. The available mammalian toxicology database for dimethylaminopro pylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic is complete with respect to assessing the increased susceptibility to infants and

children as required by FQPA for the dimethylaminopropylamine, isopropylamine, ethanolamine and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzene sulfonic acid. There was no increased susceptibility to the offspring of rats following prenatal and postnatal exposure in the OPPTS Harmonized Test Guidelines 870.3550 and 870.3650 reproductive/developmental screening studies, and developmental effects studies.

There was no increased susceptibility to the offspring of rats following prenatal and postnatal exposure in the OPPTS Harmonized Test Guideline 870.3650 study with isopropylamine dodecylbenzene sulfonate. Developmental toxicity was not observed, whereas parental toxicity was manifested as excessive salivation in both sexes, soft feces in males, and lesions of the forestomach in both sexes. No increased susceptibility was observed in offspring of rats following exposure in the OPPTS Harmonized Test Guideline 870.3550 study with dimethylaminopropylamine. Developmental toxicity was not observed, whereas parental toxicity was manifested as decreased body-weight gain and clinical signs. Susceptibility was not demonstrated in the offspring in a rat developmental toxicity study with isopropylamine following inhalation exposure. Developmental toxicity was not observed, whereas parental toxicity was manifested as decreased body weight and body-weight gain. In developmental toxicity studies with ethanolamine following dermal (rat and rabbit) exposure, developmental and maternal toxicity were not observed. In a developmental toxicity study, increased susceptibility to the offspring was not observed following oral exposure to ethanolamine. Developmental toxicity was observed (decreased body weight in female fetuses on PND 1-4) at the same dose level where maternal toxicity was observed (decreased body weight during the latter part of gestation and throughout lactation). Since a clear NOAEL of 120 mg/kg/day was identified for offspring effects, and the selected point of departure of 50 mg/kg/day (mortality and clinical signs) for the dietary and inhalation risk assessments is protective of the offspring effects, there are no residual concerns.

There is no evidence in the available toxicity studies or scientific literature to indicate neurotoxic effects of these amines in laboratory animals. The clinical signs observed in females in the 28–day study with dimethylaminopropylamine (stilted gait and decreased spontaneous activity are considered agonal in nature.

The prenatal developmental and reproduction studies with alkylbenzene sulfonates showed no qualitative or quantitative evidence of increased susceptibility. Several reproduction and many developmental studies have been performed with alkylbenzene sulfonates in a number of animal species. In the developmental studies, whenever toxicity was observed in adults, it was generally for mild effects (slight body weight changes, intestinal disturbances) except for severe dermal irritation effects in dermal developmental studies. Any developmental toxicity observed in these same studies included minor increases in visceral/skeletal anomalies and some fetal losses; but only at maternally toxic doses. In one reproduction study, there were slight changes in hematology and histopathology (both within historical control ranges) and slight decreases in body weight in the offspring at the highest dose of 250 mg/kg/day (at which there were no effects on the parental generation). There were no effects in either the parents or offspring in the other two alkyl benzensulfonate reproductive toxicity studies at the high dose tested of 70 and 170 mg/kg/day, respectively.

ii. *Diethanolamine salt of alkyl* ($C_8$-$C_{24}$) *benzenesulfonic acid (DEA).* There is no OPPTS Harmonized Test Guideline 870.3650 combined repeated dose toxicity study with the reproduction/developmental toxicity screening test available on DEA. The toxicology database on DEA consists of open literature studies that include oral and dermal exposure developmental toxicity studies in rats and a dermal exposure developmental toxicity study in rabbits. There are no reproductive toxicity or neurotoxicity studies available on DEA.

No evidence of increased susceptibility to the offspring of rats or rabbits following prenatal dermal exposure was located. There was qualitative prenatal susceptibility in the rat oral developmental toxicity study. The developmental findings with a NOAEL of 50 mg/kg/day were well-characterized and included increased developmental sensitivity in the form of increased postnatal day (PND) 0 through 4 mortality and post implantation loss, and reduced pup body weight at 125 mg/kg/day (developmental LOAEL). The maternal toxicity NOAEL/LOAEL of 50/125 mg/kg/day was based on increased absolute liver weight. Developmental toxicity was demonstrated in the rat following dermal exposure to the maternal animal during gestation days (GD) 6 through 15, as evidenced by increased incidence of skeletal variations at 1500 mg/kg/day (HDT). The NOAEL for developmental toxicity was 500 mg/kg/day; the LOAEL for maternal toxicity was 150 mg/kg (LDT) based on microcytic anemia with abnormal red blooc cell morphology. The degree of concern for the increased qualitative susceptibility seen in the oral developmental toxicity study in rats (prenatal exposure) is low since a clear NOAEL/LOAEL was established for oral developmental toxicity and since a more sensitive endpoint of concern (48 mg/kg/day, the NOAEL from the rat subchronic toxicity study) has been utilized in assessing the risks from incidential and chronic oral exposure to the diethanolamine salt of alkyl (C8-C24) benzenesulfonic acid.

Demyelination has been observed in the brain (medulla) and spinal cord of rats following oral and dermal exposure, and decreased testis and epididymis weights associated with degeneration of seminiferous epithelium, decreased numbers of spermatogenic cells, reduced size of seminiferous tubules, decreased sperm; decreased sperm motility and sperm count have been observed in male rats following oral exposure.

DEA is structurally related to the essential nutrient choline, and choline deficiency during pregnancy has been shown to reduce neurogenesis and increase apoptosis in rat and mouse fetal hippocampus. In the open literature, DEA has been shown to alter neurogenesis and induce apoptosis in fetal mouse hippocampus following dermal exposure of the maternal animal to DEA during pregnancy.

The existing toxicology database is not adequate for assessing the sensitivity of infants and children to DEA exposure because a reproduction study is not available and in light of the findings in adult animals (demyelination in the brain and spinal cord and degeneration of the seminiferous tubules of the testis) that suggest the potential for developmental, reproductive, and/or neurodevelopmental toxicity in the young animal. The particular findings in the parental animals lead to uncertainties for the offspring. There is a concern for neurodevelopment since this is not addressed in the currently available database.

3. *Conclusion.*—i. *Dimethylaminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl* ($C_8$-$C_{24}$) *benzenesulfonic acid.* EPA has determined that reliable data show that the safety of infants and children would be adequately protected if the

FQPA SF were reduced to 1X. That decision is based on the following findings:

a. The toxicity database for dimethyl aminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid is considered adequate for assessing the risks to infants and children to dimethyl aminopropylamine, isopropylamine, ethanolamine and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid exposures (the available studies are described in Unit IV.D.2.).

b. No susceptibility was demonstrated in the offspring in the OPPTS Harmonized Guideline 870.3650 combined repeated dose toxicity study with the reproduction/developmental toxicity screening test in rats with isopropylamine dodecylbenzene sulfonate following prenatal and postnatal exposure.

c. No susceptibility was demonstrated in the offspring in the OPPTS Harmonized Guideline 870.3550 reproduction/developmental toxicity screening test with dimethylaminopropylamine following prenatal and postnatal exposure.

d. No susceptibility was demonstrated in the offspring in an inhalation developmental toxicity study with isopropylamine.

e. The prenatal developmental and reproduction studies with alkylbenzene sulfonates showed no qualitative or quantitative evidence of increased susceptibility. Slight changes in hematology and histopathology (both within historical control ranges) and slight decreases in body weight in the offspring at the highest dose of 250 mg/kg/day (at which there were no effects on the parental generation) were seen with alkylbenzenesulfonate in one reproduction study, however there were no effects in either the parents or offspring in the other two alkyl benzensulfonate reproductive toxicity studies at the high dose tested of 70 mg/kg/day and 170 mg/kg/day, respectively. Since the selected point of departure of 50 mg/kg/day (mortality and clinical signs) for the dietary and inhalation risk assessments is protective of the offspring effects, there are no residual concerns.

f. No susceptibility was demonstrated in the offspring in dermal (rat and rabbit) and oral (rat) developmental toxicity studies with ethanolamine. Developmental toxicity was observed following oral exposure with ethanolamine at the same dose level where maternal toxicity was observed. Since a clear NOAEL of 120 mg/kg/day was identified for offspring effects, and the selected point of departure of 50 mg/kg/day (mortality and clinical signs) for the dietary and inhalation risk assessments is protective of the offspring effects, there are no residual concerns.

g. No evidence of neurotoxicity was demonstrated in the database for alkylbenzene sulfonates, dimethylaminopropylamine, isopropylamine, ethanolamine, and triethanolamine and isopropylamine salt of dodecylbenzenesulfonic acid and thus there is no need for a developmental neurotoxicity study or additional UFs to account for neurotoxicity.

h. There are no residual uncertainties identified in the exposure databases. The food and drinking water assessment is not likely to underestimate exposure to any subpopulation, including those comprised of infants and children. The food exposure assessments are considered to be highly conservative as they are based on the use of the highest tolerance level from the surrogate pesticides for every food and 100 PCT is assumed for all crops. EPA also made conservative (protective) assumptions in the ground and surface water modeling used to assess exposure to ASABSA 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 ASABSA.

ii. *Diethanolamine salts of alkyl* ($C_8$-$C_{24}$) *benzenesulfonic acid*. EPA has determined that the FQPA SF should be retained. That decision is based on the following findings:

a. Although no increased susceptibility was demonstrated in the offspring in the available dermal studies in rats and rabbits following prenatal exposure to DEA, and the degree of concern is low for the increased qualitative susceptibility seen in the oral developmental toxicity study in rats, considering the limited data in the literature on DEA, which indicate a potential for developmental and/or reproductive and/or developmental neurotoxicity effects, the toxicology database for DEA is not considered adequate for assessing the sensitivity of infants and children to DEA when used as an inert ingredient (the available studies are described in Unit IV.D.2.).

b. There are no neurotoxicity studies available on DEA.

c. There are no reproductive toxicity studies available on DEA.

d. There are no developmental toxicity studies available on DEA that assess neurodevelopment.

*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*.There was no hazard attributable to a single exposure seen in the toxicity database for ASABSA. Therefore, ASABSA are not expected to pose an acute risk.

2. *Chronic risk*. A chronic aggregate risk assessment takes into account exposure estimates from chronic dietary consumption of food and drinking water. Using the exposure assumptions discussed in this unit for chronic exposure, including the limitation of use of diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid to not more than 7% of the pesticide product, the chronic dietary exposure from food and water to dimethylaminopropylamine, isopropylamine, ethanolamine and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid, is 23% of the cPAD for the U.S. population and 75% of the cPAD for children 1 to 2 years old, the most highly exposed population subgroup. The chronic dietary exposure from food and water to diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid is 19% of the cPAD for the U.S. population and 56% of the cPAD for children 1 to 2 years old, the most highly exposed population subgroup.

3. *Short-term risk*. Short-term aggregate exposure takes into account short-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

ASABSA are used as inert ingredients in pesticide products that are currently registered for uses that could result in short-term residential exposure and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with short-term residential exposures to ASABSA. Using the exposure assumptions described in this unit, EPA has concluded that the combined short-

term aggregated food, water, and residential exposures result in aggregate MOEs of 220 and 260 for adult males and females, respectively. Adult residential exposure combines high end outdoor dermal and inhalation handler exposure with a high end post application dermal exposure from contact with treated lawns. EPA has concluded the combined short-term aggregated food, water, and residential exposures result in an aggregate MOE of 110 for children. Children's residential exposure includes total exposures associated with contact with treated lawns (dermal and hand-to-mouth exposures). As the level of concern is for MOEs that are lower than 100, these MOEs are not of concern.

4. *Intermediate-term risk*. Intermediate-term aggregate exposure takes into account intermediate-term residential exposure plus chronic exposure to food and water (considered to be a background exposure level).

ASABSA are used as inert ingredients in pesticide products that are currently registered for uses that could result in intermediate-term residential exposure and the Agency has determined that it is appropriate to aggregate chronic exposure through food and water with intermediate-term residential exposures to ASABSA. Using the exposure assumptions described in this unit, EPA has concluded that the combined intermediate-term aggregated food, water, and residential exposures result in aggregate MOEs of 540 and 570 for adult males and females, respectively. Adult residential exposure includes high end post application dermal exposure from contact with treated lawns. EPA has concluded that the combined intermediate-term aggregated food, water, and residential exposures result in an aggregate MOE of 110 for children. Children's residential exposure includes total exposures associated with contact with treated lawns (dermal and hand-to-mouth exposures). As the level of concern is for MOEs that are lower than 100, these MOEs are not of concern.

5. *Aggregate cancer risk for U.S. population*. The Agency has not identified any concerns for carcinogenicity relating to ASABSA.

6. *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 residues of ASABSA.

## V. Other Considerations

*A. Analytical Enforcement Methodology*

An analytical method is not required for enforcement purposes since the Agency is establishing an exemption from the requirement of a tolerance without any numerical limitation.

*B. International Residue Limits*

The Agency is not aware of any country requiring a tolerance for ASABSA nor have any CODEX Maximum Residue Levels been established for any food crops at this time.

## VI. Conclusion

Therefore, an exemption from the requirement of a tolerance is established for residues of dimethylaminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid when used as an inert ingredient in pesticide formulations applied to growing crops under 40 CFR 180.920 and to animals under 40 CFR 180.930 and to diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid when used as an inert ingredient at levels not to exceed 7% by weight in pesticide formulations applied to growing crops under 40 CFR 180.920 and to animals under 40 CFR 180.930.

## VII. Statutory and Executive Order Reviews

This final rule establishes an exemption from the requirement of 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, 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 section 408(d) of FFDCA, such as the exemptions 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).

## VIII. 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: July 30, 2009.

**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 asfollows:

**Authority:** 21 U.S.C. 321(q), 346a and 371.

■ 2. In § 180.920, the table is amended by adding alphabetically the following inert ingredients:

**§ 180.920 Inert ingredients used pre-harvest; exemptions from the requirement of a tolerance.**

* * * * *

| Inert Ingredients | Limits | Uses |
|---|---|---|
| * * * * * * | * | |
| Diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (CAS Reg. Nos. 26545–53–9 and 68953–97–9). | Not to exceed 7% of pesticide formulation. | Surfactants, related adjuvants of surfactants |
| * * * * * * | * | |
| Dimethylaminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (CAS Reg. Nos. 26264–05–1, 27323–41–7, 55470–69–4, 68411–31–4, 68584–24–7, 68584–25–8, 68648–81–7, 68648–96–4, 68649–00–3, 68910–32–7, 68953–93–5, 90194–42–6, 90194–53–9, 90218–35–2, 157966–96–6, 319926–68–6, 877677–48–0, 1093628–27–3). | | Surfactants, related adjuvants of surfactants |
| * * * * * * | * | |

■ 3. In §180.930, the table is amended by adding alphabetically the following inert ingredients:

**§ 180.930 Inert ingredients applied to animals; exemptions from the requirement of a tolerance.**

* * * * *

| Inert Ingredients | Limits | Uses |
|---|---|---|
| * * * * * * | * | |
| Diethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (CAS Reg. Nos. 26545–53–9 and 68953–97–9). | Not to exceed 7% of pesticide formulation. | Surfactants, related adjuvants of surfactants |
| * * * * * * | * | |
| Dimethylaminopropylamine, isopropylamine, ethanolamine, and triethanolamine salts of alkyl ($C_8$-$C_{24}$) benzenesulfonic acid (CAS Reg. Nos. 26264–05–1, 27323–41–7, 55470–69–4, 68411–31–4, 68584–24–7, 68584–25–8, 68648–81–7, 68648–96–4, 68649–00–3, 68910–32–7, 68953–93–5, 90194–42–6, 90194–53–9, 90218–35–2, 157966–96–6, 319926–68–6, 877677–48–0, 1093628–27–3). | | Surfactants, related adjuvants of surfactants |
| * * * * * * | * | |

[FR Doc. E9–18698 Filed 8–4–09; 8:45 am]

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## ENVIRONMENTAL PROTECTION AGENCY

## 40 CFR Part 180

**[EPA–HQ–OPP–2009–0145; FRL–8430–1]**

## Alkyl Alcohol Alkoxylates; Exemption from the Requirement of a Tolerance

**AGENCY:** Environmental Protection Agency (EPA).

**ACTION:** Final rule.

**SUMMARY:** This regulation establishes an exemption from the requirement of a tolerance for [residues] of α-alkyl-ω-hydroxypoly (oxypropylene) and/or poly (oxyethylene) polymers where the alkyl chain contains a minimum of six carbons when used as an inert ingredient in pesticide formulations. The Joint Inerts Task Force (JITF), Cluster Support Team Number 1, submitted a petition to EPA under the Federal Food, Drug, and Cosmetic Act (FFDCA), requesting an exemption from the requirement of a tolerance. This regulation eliminates the need to establish a maximum permissible level for residues of α-alkyl-ω-hydroxypoly (oxypropylene) and/or poly (oxyethylene) polymers where the alkyl chain contains a minimum of six carbons.

**DATES:** This regulation is effective August 5, 2009. Objections and requests for hearings must be received on or before October 5, 2009, 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–2009–0145. All documents in the docket are listed in the docket index available at *http://www.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:** Kerry Leifer, Registration Division (7505P), Office of Pesticide Programs,