

**ENVIRONMENTAL PROTECTION AGENCY**

**40 CFR Part 372**

[EPA-HQ-TRI-2015-0352; FRL 9935-38-OEI]

**Ethylene Glycol Monobutyl Ether; Community Right-To-Know Toxic Chemical Release Reporting**

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

**ACTION:** Denial of petition.

**SUMMARY:** Environmental Protection Agency (EPA) is denying a petition to remove ethylene glycol monobutyl ether (EGBE) from the category Certain Glycol Ethers under the list of chemicals subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA)

of 1986 and section 6607 of the Pollution Prevention Act (PPA) of 1990. EPA has reviewed the available data on this chemical and has determined that EGBE does not meet the deletion criterion of EPCRA section 313(d)(3). Specifically, EPA is denying this petition because EPA's review of the petition and available information resulted in the conclusion that EGBE meets the listing criterion of EPCRA section 313(d)(2)(B) due to its potential to cause serious or irreversible chronic health effects in humans, specifically, liver toxicity and concerns for hematological effects.

**DATES:** EPA denied this petition on September 24, 2015.

**FOR FURTHER INFORMATION CONTACT:** Daniel R. Bushman, Environmental Analysis Division, Office of Information Analysis and Access (2842T), Environmental Protection Agency, 1200

Pennsylvania Ave. NW., Washington, DC 20460; telephone number: 202-566-0743; fax number: 202-566-0677; email: [bushman.daniel@epa.gov](mailto:bushman.daniel@epa.gov), for specific information on this notice. For general information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Hotline, toll free at (800) 424-9346 (select menu option 3) or (703) 412-9810 in Virginia and Alaska or toll free, TDD (800) 553-7672, <http://www.epa.gov/superfund/contacts/infocenter/>.

**SUPPLEMENTARY INFORMATION:**

**I. General Information**

*A. Does this notice apply to me?*

You may be potentially affected by this action if you manufacture, process, or otherwise use EGBE. Potentially affected categories and entities may include, but are not limited to:

Category	Examples of potentially affected entities
Industry .....	Facilities included in the following NAICS manufacturing codes (corresponding to SIC codes 20 through 39): 311,* 312,* 313,* 314,* 315,* 316, 321, 322, 323,* 324, 325,* 326,* 327, 331, 332, 333, 334,* 335,* 336, 337,* 339,* 111998,* 211112,* 212324,* 212325,* 212393,* 212399,* 488390,* 511110, 511120, 511130, 511140,* 511191, 511199, 512220, 512230,* 519130,* 541712,* or 811490.* *Exceptions and/or limitations exist for these NAICS codes. Facilities included in the following NAICS codes (corresponding to SIC codes other than SIC codes 20 through 39): 212111, 212112, 212113 (correspond to SIC 12, Coal Mining (except 1241)); or 212221, 212222, 212231, 212234, 212299 (correspond to SIC 10, Metal Mining (except 1011, 1081, and 1094)); or 221111, 221112, 221113, 221118, 221121, 221122, 221330 (Limited to facilities that combust coal and/or oil for the purpose of generating power for distribution in commerce) (correspond to SIC 4911, 4931, and 4939, Electric Utilities); or 424690, 425110, 425120 (Limited to facilities previously classified in SIC 5169, Chemicals and Allied Products, Not Elsewhere Classified); or 424710 (corresponds to SIC 5171, Petroleum Bulk Terminals and Plants); or 562112 (Limited to facilities primarily engaged in solvent recovery services on a contract or fee basis (previously classified under SIC 7389, Business Services, NEC)); or 562211, 562212, 562213, 562219, 562920 (Limited to facilities regulated under the Resource Conservation and Recovery Act, subtitle C, 42 U.S.C. 6921 et seq.) (correspond to SIC 4953, Refuse Systems).
Federal Government .....	Federal facilities.

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Some of the entities listed in the table have exemptions and/or limitations regarding coverage, and other types of entities not listed in the table could also be affected. To determine whether your facility would be affected by this action, you should carefully examine the applicability criteria in part 372 subpart B of Title 40 of the Code of Federal Regulations. If you have questions regarding the applicability of this action to a particular entity, consult the person listed in the preceding **FOR FURTHER INFORMATION CONTACT** section.

*B. How can I get copies of this document and other related information?*

1. *Docket.* EPA has established a docket for this action under Docket ID No. EPA-HQ-TRI-2015-0352. Publicly available docket materials are available either electronically in

[www.regulations.gov](http://www.regulations.gov) or in hard copy at the OEI Docket, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave. NW., Washington, DC. This Docket Facility is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744, and the telephone number for the OEI Docket is (202) 566-1752.

2. *Electronic Access.* You may access this **Federal Register** document electronically from the Government Printing Office under the "**Federal Register**" listings at FDSys (<http://www.gpo.gov/fdsys/browse/collection.action?collectionCode=FR>).

**II. Introduction**

Section 313 of EPCRA, 42 U.S.C. 11023, requires certain facilities that manufacture, process, or otherwise use listed toxic chemicals in amounts above reporting threshold levels to report their environmental releases and other waste management quantities of such

chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106. Congress established an initial list of toxic chemicals that comprised more than 300 chemicals and 20 chemical categories.

EPCRA section 313(d) authorizes EPA to add or delete chemicals from the list and sets criteria for these actions. EPCRA section 313(d)(2) states that EPA may add a chemical to the list if any of the listing criteria in Section 313(d)(2) are met. Therefore, to add a chemical, EPA must demonstrate that at least one criterion is met, but need not determine whether any other criterion is met. EPCRA section 313(d)(3) states that a chemical may be deleted if the Administrator determines there is not sufficient evidence to establish any of the criteria described in EPCRA section 313(d)(2)(A)-(C). The EPCRA section 313(d)(2)(A)-(C) criteria are:

- The chemical is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous, or frequently recurring, releases.
- The chemical is known to cause or can reasonably be anticipated to cause in humans:
  - Cancer or teratogenic effects, or
  - serious or irreversible—
    - reproductive dysfunctions,
    - neurological disorders,
    - heritable genetic mutations, or
    - other chronic health effects.
- The chemical is known to cause or can be reasonably anticipated to cause, because of:
  - its toxicity,
  - its toxicity and persistence in the environment, or
  - its toxicity and tendency to bioaccumulate in the environment,

a significant adverse effect on the environment of sufficient seriousness, in the judgment of the Administrator, to warrant reporting under this section.

EPA often refers to the section 313(d)(2)(A) criterion as the “acute human health effects criterion;” the section 313(d)(2)(B) criterion as the “chronic human health effects criterion;” and the section 313(d)(2)(C) criterion as the “environmental effects criterion.”

Under section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479) to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compounds categories. EPA published in the **Federal Register** of November 30, 1994 (59 FR 61432) a statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals.

### III. What is the description of the petition?

On January 23, 2015, EPA received a petition from American Chemistry Council (ACC) Ethylene Glycol Ethers Panel requesting EPA to delete EGBE (Chemical Abstracts Service Registry Number (CASRN) 111-76-2) from the list of chemicals subject to reporting under EPCRA section 313 and PPA section 6607 (Reference (Ref. 1)). EGBE

is not individually listed under EPCRA section 313 but rather is reportable under the Certain Glycol Ethers category. The petitioner contends that the available scientific data show that EGBE has low potential hazard to human health and the environment. Therefore, the petitioner believes that under EPA’s policy for listing decisions under EPCRA section 313, potential exposures should be considered. The petitioner believes that their analysis shows that exposure levels are well below the concern levels for human health and ecological effects.

### IV. What is EPA’s evaluation of the toxicity of EGBE?

EPA’s evaluation of the toxicity of EGBE included a review of the human health and ecological effects data. EPA’s Integrated Risk Information System (IRIS) toxicological review of EGBE (Ref. 2) was the primary source used to determine the human health effects of EGBE. EPA also prepared an assessment of the chemistry, fate, and ecological effects for EGBE (Ref. 3).

#### A. What is EPA’s review of the human health toxicity data for EGBE?

EPA’s evaluation of the toxicity of EGBE included a review (Ref. 4) of the IRIS toxicological review of EGBE (Ref. 2). EPA also reviewed the findings of studies published since the IRIS toxicological review of EGBE, but found no data relevant to include in this evaluation. This Unit outlines the evidence of human health toxicity from the 2010 IRIS toxicological review of EGBE. Unit IV.B. below discusses the conclusions regarding EGBE’s potential human health toxicity.

1. *Toxicokinetics.* In humans, EGBE is absorbed and rapidly distributed following inhalation, ingestion, or dermal exposure (Refs. 5, 6, 7, and 8). Several reviews have described the metabolism of EGBE in detail (Refs. 9, 10, and 11). The principal products from EGBE metabolism are butoxyacetic acid (BAA) (rats and humans) and the glutamine or glycine conjugate of BAA (humans). BAA is excreted in the urine of both rats and humans, which suggests that the creation of BAA through the formation of butoxyacetaldehyde by alcohol dehydrogenase is applicable to rats and humans (Refs. 8, 12, and 13). The other proposed metabolic pathways, however, may only be applicable to rats since the metabolites of these pathways (*i.e.*, ethylene glycol, EGBE glucuronide, and EGBE sulfate) have been observed in the urine of rats (Refs. 14 and 15), but not in humans (Ref. 8). In addition, Corley et al. (Ref. 8) confirmed the finding from

Rettenmeier et al. (Ref. 16) that approximately two-thirds of the BAA formed in humans is conjugated with glutamine and glycine. These pathways, however, have not been observed in the rat.

Several experimental studies have measured the concentration of BAA in human serum and urine following exposure to EGBE. For humans, the elimination kinetics of EGBE and BAA appear to be independent of the route of exposure with an approximate half-life of around one hour for EGBE and an approximate half-life of BAA of 3–4 hours (Refs. 17, 18, and 19).

Several physiologically based pharmacokinetic models for EGBE have been developed. Some older models have described the kinetics of EGBE for acute human exposure and exposure to rats via the ingestion, inhalation, and dermal routes (Refs. 17 and 20 based on data from Refs. 13, 21, and 22). Newer models, however, have extended upon the work of these previous models. Corley et al. (Ref. 7) described the kinetics of EGBE and BAA in both rats and humans. These authors later validated the human dermal exposure model (Ref. 8). Lee et al. (Ref. 23) modeled the kinetics of EGBE and BAA in mice and rats from a National Toxicology Program (NTP) 2-year inhalation bioassay (based on data from Dill et al. (Ref. 24)). Species, gender, age, and exposure concentration-dependent differences in the kinetics of BAA were observed. Corley et al. (Ref. 12) built on the Lee et al. (Ref. 23) model by replacing some model assumptions with experimental data (Note: The Corley et al. (Ref. 12) model, along with the Lee et al. (Ref. 23) rat and mouse model and Corley et al. (Ref. 8) human model were used by EPA to calculate internal doses of EGBE in the 2010 IRIS toxicological review of EGBE (Ref. 2)).

2. *Effects of Acute and Short-Term Exposure.* Hematologic and other effects have been observed in several acute and short-term oral studies of EGBE in rats and mice (Refs. 15, 25, 26, 27, 28, 29, 30, 31, 32, 33, and 34). Varying degrees of hematotoxicity have also been observed in rats and rabbits following dermal application of EGBE (Refs. 14 and 35). Guinea pigs, however, have not demonstrated sensitivity to the hematologic effects of EGBE in acute studies (Refs. 36 and 37). EGBE has also been found to be an ocular irritant when instilled in rabbits (Refs. 38 and 39).

A few *in vitro* studies have investigated EGBE’s potential hemolytic effects in human red blood cells after acute exposures. Bartnik et al. (Ref. 14) reported no hemolysis of human red

blood cells exposed for three hours to BAA levels up to 15 millimolar (mM). Hemolysis was observed in rat red blood cells, however, at BAA levels as low as 1.25 mM. Udden (Ref. 40) incubated human red blood cells with up to 2.0 mM BAA for four hours, and the authors observed none of the morphological changes observed in rat red blood cells at the same concentration. Udden (Ref. 41) reported a significant change in human red blood cell deformability at exposure to 7.5 and 10 mM BAA for 4 hours, whereas deformability in rat red blood cells was significantly increased at 0.05 mM BAA. Mean cellular volume in human blood samples was significantly increased at 10 mM BAA while mean cellular volume in rats was significantly increased at 0.05 mM BAA.

There are a number of case reports of acute ingestion of EGBE with little or no hematologic effects observed (Refs. 42, 43, 44, 45, 46, 47, 48, and 49). Some other observed effects were likely not directly related to hemolysis; however, the cause of the effects cannot be explained based on the limited data available. Also, hemodialysis was employed to remove un-metabolized EGBE in many of the cases.

One experimental study in humans (Ref. 50), observed no effects on red blood cell fragility after exposure of two males and one female to up to 195 part per million (ppm) EGBE for 8 hours.

3. *Carcinogenicity and Mutagenicity.* Under the Guidelines for Carcinogen Risk Assessment (Ref. 51), there is suggestive evidence of EGBE's carcinogenic potential based on a 2-year NTP bioassay in mice and rats (Ref. 52). EGBE has been tested for its potential for genotoxicity both *in vitro* and *in vivo*, and the available data do not demonstrate that EGBE is mutagenic or clastogenic (Refs. 53, 54, 55, 56, 57, and 58).

4. *Reproductive and Developmental Toxicity.* The reproductive and developmental toxicity of EGBE has been investigated in a number of oral and inhalation studies in rats, mice, and rabbits. In a two-generation reproductive toxicity study, fertility was reduced in mice at very high maternally toxic doses ( $\leq 1,000$  milligrams/kilogram (mg/kg)) (Ref. 59), but no other significant reproductive effects were reported in any study (Refs. 26, 52, 60, 61, 62, 63, 64, 65, and 66). Maternal toxicity related to the hematologic effects of EGBE and relatively minor developmental effects have been reported in developmental studies (Refs. 67, 68, 69, and 70). No teratogenic effects were noted in any of the studies. As such, EGBE is not reasonably anticipated to be a reproductive or

developmental toxicant at moderately low to low doses.

5. *Neurotoxicity.* There is no evidence of neurotoxicity in any animal studies of EGBE. One case study patient demonstrated neurologic deficits after ingesting a product with a high dose of EGBE and other chemicals (Ref. 47). Given the general limitations of case studies and the presence of other chemicals, however, EPA cannot draw conclusions about EGBE's potential neurotoxicity from this particular study.

6. *Other Subchronic and Chronic Toxicity.* Hematologic effects and liver toxicity have been observed at low doses of EGBE in several animal studies.

The NTP (Ref. 66) conducted a 13-week study in F344 rats and B6C3F1 mice in which groups of 10 animals/gender/species received EGBE in drinking water at doses of 0, 750, 1,500, 3,000, 4,500, and 6,000 ppm. The corresponding doses based on measured drinking water consumption were: 0, 69, 129, 281, 367, or 452 milligrams/kilogram/day (mg/kg/day) in male rats; 0, 82, 151, 304, 363, or 470 mg/kg/day in female rats; 0, 118, 223, 553, 676, or 694 mg/kg/day in male mice; and 0, 185, 370, 676, 861, or 1,306 mg/kg/day in female mice.

Indications of mild to moderate anemia were observed in both genders. Statistically significant hematologic effects in female rats included reduced red blood cell counts and hemoglobin concentrations at  $\geq 750$  ppm and increased reticulocytes, decreased platelets, and increased bone marrow cellularity at 3,000 ppm. Liver effects including cytoplasmic alterations, hepatocellular degeneration, and pigmentation were reported in the mid- and high-dose groups ( $\geq 1,500$  ppm for males and females; statistics not reported). Additionally, cytoplasmic alterations of liver hepatocytes were observed in the lowest-dose groups (750 ppm for males and females). The lack of cytoplasmic granularity of the hepatocytes indicates that this response was not due to enzyme induction (Ref. 71). The NTP (Ref. 66) identified a lowest-observed-adverse-effect level (LOAEL) for rats of 750 ppm (approximately 58.6 mg/kg/day calculated using water consumption rates and body weights measured during the last week of exposure and, therefore, slightly different from those reported by the study authors (Ref. 2)) based on decreased red blood cell count and hemoglobin in female rats. A NOAEL was not identified.

A reduction in body weight gain at  $\geq 3,000$  ppm was observed in male and female mice. An increase in relative kidney weight was also observed at all

doses in female mice. Body weight reductions followed decreased water consumption. No histopathologic changes were noted at any dose level, however, relative kidney weights showed a statistically significant increase at 750 and 1,500 ppm in the absence of reduction in body weight gain. The NTP (Ref. 66) identified a LOAEL for mice of 3,000 ppm (approximately, 553–676 mg/kg/day calculated using water consumption rates and body weights measured during the last week of exposure and, therefore, slightly different from those reported by the study authors (Ref. 2)) based on reduced body weight and body weight gain.

Dodd et al. (Ref. 62) conducted a 90-day subchronic inhalation study using F344 rats (16/gender/group) exposed to EGBE for 6 hours/day, 5 days/week at concentrations of 0, 5, 25, and 77 ppm. After 6 weeks, the 77 ppm female rats had statistically significant decreases in red blood cell counts (13%) and hemoglobin concentrations, accompanied by an 11% increase in mean corpuscular hemoglobin. Similar results were observed in males. However, many of these effects had lessened by the end of the study. The authors reported a LOAEL of 77 ppm based on decreases in red blood cell count and hemoglobin concentrations, accompanied by an increase in mean corpuscular hemoglobin in both genders.

The NTP (Ref. 52) conducted a subchronic inhalation study in F344 rats and B6C3F1 mice (10/gender). Rats and mice were exposed to EGBE concentrations of 0, 31, 62.5, 125, 250, and 500 ppm (0, 150, 302, 604, 1,208, and 2,416 milligrams/cubic meter (mg/m<sup>3</sup>)) 6 hours/day, 5 days/week for 14 weeks. The NTP (Ref. 52) identified a LOAEL of 31 ppm in female rats based on decreases in hematocrit, hemoglobin, and red blood cell count and a LOAEL of 62.5 ppm in male rats based on a decrease in red blood cell count. Histopathologic effects were observed in male and female rats. Effects reported in female rats included liver necrosis at 250 ppm and centrilobular degeneration and renal tubular degeneration at 500 ppm. Other effects reported in both genders included: Excessive splenic congestion in the form of extramedullary hematopoiesis (at 250 ppm in male rats and 125 ppm in female rats), hemosiderin accumulation in Kupffer cells (at 125 ppm in male rats and 62.5 ppm in female rats), intracytoplasmic hemoglobin (at 125 ppm in male rats and 31 ppm in female rats), hemosiderin deposition (at 125 ppm in male rats and 62.5 ppm in

female rats), and bone marrow hyperplasia (at 250 ppm in male rats and 62.5 ppm in female rats). The authors identified a LOAEL of 62.5 ppm for mice based on histopathological changes in the forestomach (including: Necrosis, ulceration, inflammation, and epithelial hyperplasia) in both males and females. Signs consistent with the hemolytic effects of EGBE (including: Decreased red blood cell counts, increased reticulocyte counts, and increased mean corpuscular volume) were also observed at 250 and 500 ppm in male and female mice.

The NTP (Ref. 52) also completed a 2-year inhalation study on EGBE in both F344 rats and B6C3F1 mice. In this study, animals were exposed to EGBE 6 hours/day, 5 days/week at concentrations of 0, 31, 62.5, and 125 ppm (0, 150, 302, and 604 mg/m<sup>3</sup>) for groups of 50 F344 rats and 0, 62.5, 125, and 250 ppm (0, 302, 604, and 1,208 mg/m<sup>3</sup>) for groups of 50 B6C3F1 mice. The authors identified a LOAEL of 31 ppm in rats based on decreases in hematocrit, hemoglobin, and red blood cell count in female rats in a satellite group observed at 3 and 6 months. The authors identified 62.5 ppm as the LOAEL for mice based on hemosiderin deposition.

One long-term occupational study of EGBE was identified in the literature. Haufroid et al. (Ref. 72) reported a small decrease in hematocrit and increase in mean corpuscular hemoglobin in a cross sectional study of 31 workers exposed to an average concentration of 0.6 ppm EGBE over 1 to 6 years. The biological significance of these findings, however, is unclear as they were within normal clinical ranges and no other measured parameters were affected by EGBE exposure.

#### *B. What are EPA's conclusions regarding the human hazard potential of EGBE?*

There is evidence to indicate that the human red blood cell response to EGBE exposure is less than that of rodents, however, this conclusion is based on a relatively small number of *in vitro* and short-term human exposure studies with supporting evidence from pharmacokinetic models (Refs. 7, 8, 14, 40, 41, and 50). Little is known of the long-term or repeated exposure responses in humans to EGBE.

In 2010, EPA concluded in the IRIS toxicological review of EGBE that human red blood cells do appear capable of responding similarly to the causative EGBE metabolites, albeit at much higher exposures (Ref. 2). The

IRIS toxicological review of EGBE employed an interspecies uncertainty factor of 1 to derive the reference values for EGBE in part because there was not a preponderance of toxicodynamic data in both animals and humans describing why humans are less sensitive than rats to the hematologic effects in question (Ref. 2). Also, EPA calculated a human equivalent concentration LOAEL (LOAEL<sub>HEC</sub>) for hematologic effects of 271 mg/m<sup>3</sup> (approximately 77 mg/kg/day, assuming constant exposure, an inhalation rate of 20 cubic meters/day (m<sup>3</sup>/day), and a 70 kg human) using pharmacokinetic model estimates (Refs. 7 and 8) of the human internal dose equivalent of the toxic metabolite BAA to that estimated for female rats exposed to 31 ppm EGBE in the NTP (Ref. 52) study (Ref. 2). In its assessment of EGBE, the European Union carried out a slightly different calculation based on the same underlying data and reported a similar, but slightly higher, human equivalent LOAEL of 474 mg/m<sup>3</sup> (approximately 135 mg/kg/day) (Ref. 11).

Additionally, multiple animal studies by the NTP reported liver toxicity (*e.g.*, cytoplasmic alterations of liver hepatocytes at 750 ppm (approximately 69 mg/kg/day) in male rats and 750 ppm (82 mg/kg/day) in female rats (Ref. 66) and liver necrosis at 250 ppm (approximately 243 mg/kg/day) in female rats (Ref. 52)) to which humans do not demonstrate decreased sensitivity. These findings provide further evidence of EGBE's potential toxicity to humans at moderately low to low doses.

Therefore, the available evidence is sufficient to conclude that EGBE can be reasonably anticipated to demonstrate moderately high to high chronic toxicity in humans based on the EPCRA Section 313 listing criteria (59 FR 61432, November 30, 1994).

#### *C. What is EPA's review of the ecological toxicity of EGBE?*

Based on a review of the available aquatic ecological toxicity data, EGBE does not appear to present a significant concern for adverse effects on the environment. Experimentally measured effects occurred at relatively high concentrations indicating low toxicity (Ref. 3). Such high concentrations are not expected to be observed under typical environmental conditions. Table 1 presents some of the available toxicity data for EGBE, the complete listing of the available toxicity data and more details about the studies can be found in the ecological assessment (Ref. 3).

1. *Acute toxicity.* Toxicity threshold values (duration not specified) of 900 milligrams/liter (mg/L) and 72-hour EC<sub>50</sub> values (*i.e.*, the concentration that is effective in producing a sublethal response in 50% of test organisms) of 911 and 1,840 mg/L for biomass and growth rate, respectively, have been reported for green algae (Refs. 73, 74, and 75). The corresponding 72-hour No-Observed-Effect-Concentration (NOEC) values for biomass and growth rate were 88 and 286 mg/L (Ref. 76). For water fleas (*Daphnia magna*), 24- or 48-hour EC<sub>50</sub> values ranged from 835 to 1,815 mg/L (Refs. 77 and 78). A 48-hour EC<sub>50</sub> value of 164 mg/L in rotifers (reproduction) has also been reported (Refs. 74 and 75).

Acute toxicity values for freshwater fish ranged from an LC<sub>50</sub> (*i.e.*, the concentration that is lethal to 50% of test organisms) of 1,395 mg/L for the golden orfe (*Leuciscus idus*) (duration not specified) (Ref. 79) to a 96-hour LC<sub>50</sub> of 2,137 mg/L for the fathead minnow (*Pimephales promelas*) (Ref. 80). A 96-hour LC<sub>50</sub> value of 1,490 mg/L was available for bluegill sunfish (Ref. 81) and 96-hour LC<sub>50</sub> values for rainbow trout were 1,474 and 1,700 mg/L (Refs. 74, 75, and 82). An LC<sub>50</sub> value (duration not specified) of 1,575 mg/L was also available for golden orfe (*Leuciscus idus*) (Ref. 79) and a 24-hour LC<sub>50</sub> value of 1,700 mg/L was available for goldfish (*Carassius auratus*) (Ref. 83).

A study of the invertebrate *Artemia salina* (brine shrimp) reported a 24-hour LC<sub>50</sub> value of 1,000 mg/L (Ref. 84). Also, an embryo-larval test in which Japanese oyster eggs (*Crassostrea gigas*) were incubated with the test material for 24 hours and then examined for abnormalities indicated an identical 24-hour Lowest-Observed-Effect-Concentration (LOEC) of 1,000 mg/L (Ref. 74). A study of an estuarine/marine fish silverside (*Menidia beryllina*) reported a 96-hour LC<sub>50</sub> value of 1,250 mg/L (Ref. 81).

2. *Chronic toxicity.* Values for chronic toxicity in aquatic plants ranged from an 8-day LOEC (inhibition of cell division) of 35 mg/L for the cyanobacteria *Microcystis aeruginosa* (Refs. 85 and 86) to greater than 1,000 mg/L for a 7-day EC<sub>50</sub> (growth rate) for the green alga *Selenastrum capricornutum* (Ref. 87). Experimental data for the freshwater invertebrate *Daphnia magna* include values that ranged from 100 mg/L for a 21-day NOEC (reproduction) (Refs. 74, 75, and 77) to an EC<sub>50</sub> of 297 mg/L (endpoint not reported) (Ref. 88).

TABLE 1—RANGE OF EXPERIMENTAL ECOLOGICAL TOXICITY VALUES FOR EGBE ON SELECTED TARGET SPECIES

Species	Duration and test endpoint	Experiment type <sup>a</sup>	Value (mg/L)	Reference
<b>Acute aquatic toxicity</b>				
Algae:				
Green algae ( <i>Pseudokirchneriella subcapitata</i> ) .....	72-hour EC <sub>50</sub> (growth) .....	S, M .....	1,840	(Refs. 74 and 75).
Green algae ( <i>Pseudokirchneriella subcapitata</i> ) .....	72-hour NOEC (biomass) ..	S, M .....	88	(Ref. 82).
Freshwater invertebrate:				
Water flea ( <i>Daphnia magna</i> ) .....	48-hour EC <sub>50</sub> .....	S, U, O .....	1,815	(Ref. 78).
Rotifer ( <i>Brachionus calyciflorus</i> ) .....	48-hour EC <sub>50</sub> (reproduction).	S, M .....	164	(Refs. 74 and 75).
Freshwater fish:				
Golden orfe ( <i>Leuciscus idus</i> ) .....	LC <sub>50</sub> .....	NS .....	1,395	(Ref. 79).
Fathead minnow ( <i>Pimephales promelas</i> ) .....	96-hour LC <sub>50</sub> .....	S, O .....	2,137	(Ref. 80).
Estuarine/marine invertebrate:				
Brine shrimp ( <i>Artemia salina</i> ) .....	24-hour LC <sub>50</sub> .....	S, U, C .....	1,000	(Ref. 84).
Japanese oyster eggs ( <i>Crassostrea gigas</i> ) .....	24-hr LOEC (embryotoxicity).	S .....	1,000	(Refs. 74 and 75).
Estuarine/marine fish:				
Silverside ( <i>Menidia beryllina</i> ) .....	96-hour LC <sub>50</sub> .....	S, U .....	1,250	(Ref. 81).
<b>Chronic aquatic toxicity</b>				
Algae:				
Blue-green algae ( <i>Microcystis aeruginosa</i> ) .....	8-day LOEC (cell multiplication inhibition).	S, U .....	35	(Refs. 85 and 86).
Green algae ( <i>Selenastrum capricornutum</i> ) .....	7-day EC <sub>50</sub> (growth rate) ...	S, U .....	>1,000	(Ref. 87).
Freshwater invertebrate:				
Water flea ( <i>Daphnia magna</i> ) .....	21-day NOEC (reproduction).	R, M .....	100	(Refs. 74 and 75).
Water flea ( <i>Daphnia magna</i> ) .....	21-day NOEC .....	R, M .....	100	(Ref. 88).
Water flea ( <i>Daphnia magna</i> ) .....	21-day EC <sub>50</sub> .....	R, M .....	297	(Ref. 88).
Freshwater fish:				
Zebrafish ( <i>Brachydanio rerio</i> ) .....	21-day NOEC (mortality) ...	NS .....	>100	(Ref. 89).

<sup>a</sup>Experiment type: S = static, R = renewal, M = measured, U = unmeasured, O = open test system, NS = not specified

## V. What is EPA's rationale for the denial?

EPA is denying the petition to delete EGBE from the Certain Glycol Ethers category which is subject to reporting under EPCRA section 313. This denial is based on EPA's conclusion that EGBE can reasonably be anticipated to cause serious or irreversible chronic health effects in humans, specifically, liver toxicity and concerns for hematological effects. While EPA acknowledges that there is evidence to indicate that humans are less sensitive than rodents to the hematological effects associated with acute or short-term exposure to EGBE, little is known of the long-term or repeated exposure responses in humans to EGBE. Thus, some concern remains over the potential for hematological effects following a lifetime of exposure to EGBE. Unlike the hematological effects of EGBE, there is no evidence of humans' decreased sensitivity to the reported liver effects relative to rodents. Therefore, EPA has concluded that EGBE meets the EPCRA section 313(d)(2)(B) listing criteria based on the available human health toxicity data.

Because EPA believes that EGBE has moderately high to high chronic toxicity, EPA does not believe that an exposure assessment is appropriate for determining whether EGBE meets the criteria of EPCRA section 313(d)(2)(B). This determination is consistent with EPA's published statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals (59 FR 61432, November 30, 1994).

## VI. References

EPA has established an official public docket for this action under Docket ID No. EPA-HQ-TRI-2015-0352. The public docket includes information considered by EPA in developing this action, including the documents listed below, which are electronically or physically located in the docket. In addition, interested parties should consult documents that are referenced in the documents that EPA has placed in the docket, regardless of whether these referenced documents are electronically or physically located in the docket. For assistance in locating documents that are referenced in documents that EPA has placed in the docket, but that are not electronically or

physically located in the docket, please consult the person listed in the above **FOR FURTHER INFORMATION CONTACT** section.

- American Chemistry Council. 2014. Petition of the American Chemistry Council's Ethylene Glycol Ethers Panel To Remove Ethylene Glycol Monobutyl Ether From the Toxics Release Inventory Under Section 313 Of The Emergency Planning and Community Right-To-Know Act of 1986. December 29, 2014.
- U.S. EPA. 2010. Toxicological review of Ethylene Glycol Monobutyl Ether (CASRN 111-76-2) in support of summary information on the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency. Washington, DC. <http://www.epa.gov/iris/toxreviews/0500tr.pdf>.
- U.S. EPA. 2009. Technical Review of Ethylene Glycol Monobutyl Ether (EGBE): Chemistry, Environmental Fate and Ecological Toxicity CAS Registry Number 111-76-2. Office of Environmental Information. September 9, 2009.
- U.S. EPA. 2015. Memorandum from Jocelyn Hospital, Toxicologist, Environmental Analysis Division to Megan Carroll, Acting Division Director of the Environmental Analysis Division. July 24, 2015. Subject: Review of the Data in the 2010 Integrated Risk Information System (IRIS) Toxicological

- Review of Ethylene Glycol Monobutyl Ether (EGBE).
5. Kumagai, S., Oda H., Matsunaga I., Kosaka H., Akasaka S. 1999. Uptake of 10 polar organic solvents during short-term respiration. *Toxicol. Sci.* 48: 255–263.
  6. Johanson G., Boman A. 1991. Percutaneous absorption of 2-butoxyethanol vapour in human subjects. *Occup. Environ. Med.* 48: 788–792.
  7. Corley R.A., Bormett G.A., Ghanayem B.I. 1994. Physiologically-based pharmacokinetics of 2-butoxyethanol and its major metabolite 2-butoxyacetic acid, in rats and humans. *Toxicol. Appl. Pharmacol.* 129: 61–79.
  8. Corley R.A., Markham D.A., Banks C., Delorme P., Masterman A., Houle J.M. 1997. Physiologically based pharmacokinetics and the dermal absorption of 2-butoxyethanol vapor by humans. *Fundam. Appl. Toxicol.* 39: 120–130.
  9. Commonwealth of Australia. 1996. National Industrial Chemicals Notification and Assessment Scheme (NICNAS)-priority existing chemical no. 6-2-butoxyethanol in cleaning products. Australian Government Publishing Service. Canberra, Australia. [http://www.nicnas.gov.au/\\_data/assets/pdf\\_file/0003/4368/PEC\\_6\\_2-Butoxyethanol-in-Cleaning-Products\\_Full\\_Report\\_PDF.pdf](http://www.nicnas.gov.au/_data/assets/pdf_file/0003/4368/PEC_6_2-Butoxyethanol-in-Cleaning-Products_Full_Report_PDF.pdf).
  10. ECETOC. 1994. Butoxyethanol criteria document. Special Report No. 7. European Centre for Ecotoxicology and Toxicology of Chemicals. Brussels, Belgium.
  11. E.U. 2006. European Union Risk Assessment Report: 2-butoxyethanol. <http://echa.europa.eu/documents/10162/e74a38e1-b9e1-4568-92c5-615c4b56f92d>.
  12. Corley, R.A., Grant, D.M., Farris, E., Weitz, K.K., Soelberg, J.J., Thrall, K.D., Poet, T.S. 2005. Determination of age and gender differences in biochemical processes affecting the disposition of 2-butoxyethanol and its metabolites in mice and rats to improve PBPK modeling. *Toxicol. Lett.* 156: 127–161.
  13. Medinsky, M.A., Singh, G., Bechtold, W.E., Bond, J.A., Sabourin, P.J., Birnbaum, L.S., Henderson, R.F. 1990. Disposition of three glycol ethers administered in drinking water to male F344/N rats. *Toxicol. Appl. Pharmacol.* 102: 443–455.
  14. Bartnik, F.G., Reddy, A.K., Klecak, G., Zimmermann, V., Hostynek, J.J., Kunstler, K. 1987. Percutaneous absorption, metabolism, and hemolytic activity of n-butoxyethanol. *Fundam. Appl. Toxicol.* 8: 59–70.
  15. Ghanayem, B.I., Blair, P.C., Thompson, M.B., Maronpot, R.R., Matthews, H.B. 1987. Effect of age on the toxicity and metabolism of ethylene glycol monobutyl ether (2-butoxyethanol) in rats. *Toxicol. Appl. Pharmacol.* 91: 222–234.
  16. Rettenmeier, A.W., Hennigs, R., Wodarz, R. 1993. Determination of butoxyacetic acid and N-butoxyacetyl-glutamine in urine of lacquerers exposed to 2-butoxyethanol. *Int. Arch. Occup. Environ. Health.* 65: S151–S153.
  17. Johanson, G. 1986. Physiologically based pharmacokinetic modeling of inhaled 2-butoxyethanol in man. *Toxicol. Lett.* 34: 23–31.
  18. Johanson, G., Johnsson, S. 1991. Gas chromatographic determination of butoxyacetic acid in human blood after exposure to 2-butoxyethanol. *Arch. Toxicol.* 65: 433–435.
  19. Johanson, G., Boman, A., Dynesius, B. 1988. Percutaneous absorption of 2-butoxyethanol in man. *Scand. J. Work Environ. Health.* 14: 101–109.
  20. Shyr, L.J., Sabourin, P.J., Medinsky, M.A., Birnbaum, L.S., Henderson, R.F. 1993. Physiologically based modeling of 2-butoxyethanol disposition in rats following different routes of exposure. *Environ. Res.* 63: 202–218.
  21. Sabourin, P.J., Medinsky, M.A., Birnbaum, L.S., Griffith, W.C., Henderson, R.F. 1992. Effect of exposure concentration on the disposition of inhaled butoxyethanol by F344 rats. *Toxicol. Appl. Pharmacol.* 114: 232–238.
  22. Sabourin, P.J., Medinsky, M.A., Thurmond, F., Birnbaum, L.S., Henderson, R.F. 1993. Erratum to: Effect of dose on the disposition of methoxyethanol, ethoxyethanol, and butoxyethanol administered dermally to male F344/N rats. *Fundamental and Applied Toxicology* 19:124–132. *Fundam. Appl. Toxicol.* 20: 508–510.
  23. Lee, K.M., Dill, J.A., Chou, B.J., Roycroft, J.H. 1998. Physiologically based pharmacokinetic model for chronic inhalation of 2-butoxyethanol. *Toxicol. Appl. Pharmacol.* 153: 211–226.
  24. Dill, J.A., Lee, K.M., Bates, D.J., Anderson, D.J., Johnson, R.E., Chou, B.J., Burka, L.T., Roycroft, J.H. 1998. Toxicokinetics of inhaled 2-butoxyethanol and its major metabolite, 2-butoxyacetic acid, in F344 rats and B6C3F1 mice. *Toxicol. Appl. Pharmacol.* 153: 227–242.
  25. Ghanayem, B.I., Sullivan, C.A. 1993. Assessment of the haemolytic activity of 2-butoxyethanol and its major metabolite, butoxyacetic acid, in various mammals including humans. *Hum. Exp. Toxicol.* 12: 305–311.
  26. Grant, D., Sulsh, S., Jones, H.B., Gangolli, S.D., Butler, W.H. 1985. Acute toxicity and recovery in the hemopoietic system of rats after treatment with ethylene glycol monomethyl and monobutyl ethers. *Toxicol. Appl. Pharmacol.* 77: 187–200.
  27. Ghanayem, B.I., Sanchez, I.M., Matthews, H.B. 1992. Development of tolerance to 2-butoxyethanol-induced hemolytic anemia and studies to elucidate the underlying mechanisms. *Toxicol. Appl. Pharmacol.* 112: 198–206.
  28. Ezov, N., Levin-Harrus, T., Mittelman, M., Redlich, M., Shabat, S., Ward, S.M., Peddada, S., Nyska, M., Yedgar, S., Nyska, A. 2002. A chemically induced rat model of hemolysis with disseminated thrombosis. *Cardiovasc. Toxicol.* 2: 181–194.
  29. Koshkaryev, A., Barshtein, G., Nyska, A., Ezov, N., Levin-Harrus, T., Shabat, S., Nyska, M., Redlich, M., Tsipis, F., Yedgar, S. 2003. 2-Butoxyethanol enhances the adherence of red blood cells. *Arch. Toxicol.* 77: 465–469.
  30. Shabat, S., Nyska, A., Long, P.H., Goelman, G., Abramovitch, R., Ezov, N., Levin-Harrus, T., Peddada, S., Redlich, M., Yedgar, S., Nyska, M. 2004. Osteonecrosis in a chemically induced rat model of human hemolytic disorders associated with thrombosis—a new model for avascular necrosis of bone. *Calcif. Tissue Int.* 74: 220–228.
  31. Redlich, M., Maly, A., Aframian, D., Shabat, S., Ezov, N., Levin-Harrus, T., Nyska, M., Nyska, A. 2004. Histopathologic changes in dental and oral soft tissues in 2-butoxyethanol-induced hemolysis and thrombosis in rats. *J. Oral. Pathol. Med.* 33: 424–429.
  32. Corley, R.A.; Weitz, K.K., Mast, T.J., Miller, R.A., Thrall, B.D. 1999. Short-term studies to evaluate the dosimetry and modes of action of EGBE in B6C3F1 mice [final report]. Battelle Memorial Institute. Richland, WA. Battelle Project No. 29753.
  33. Poet, T.S., Soelberg, J.J., Weitz, K.K., Mast, T.J., Miller, R.A., Thrall, B.D., Corley, R.A. 2003. Mode of action and pharmacokinetic studies of 2-butoxyethanol in the mouse with an emphasis on forestomach dosimetry. *Toxicol. Sci.* 71: 176–189.
  34. Green, T.; Toghiani, A.; Lee, R.; Moore, R.; Foster, J. 2002. The development of forestomach tumors in the mouse following exposure to 2-butoxyethanol by inhalation: Studies on the mode of action and relevance to humans. *Toxicology.* 180: 257–273.
  35. Tyler, T.R. 1984. Acute and subchronic toxicity of ethylene glycol monobutyl ether. *Environ. Health. Perspect.* 57: 185–191.
  36. Shepard, K.P. 1994. Ethylene glycol monobutyl ether: Acute oral toxicity study in the guinea pig. Eastman Kodak Company for Chemical Manufacturers Association. Rochester, NY and Arlington, VA.
  37. Gingell, R., Boatman, R.J., Lewis, S. 1998. Acute toxicity of ethylene glycol monobutyl ether in the guinea pig. *Food Chem. Toxicol.* 36: 825–829.
  38. Jacobs, G.A., Martens, M.A. 1989. An objective method for the evaluation of eye irritation *in vivo*. *Food Chem. Toxicol.* 27: 255–258.
  39. Kennah, H.E. II., Hignet, S., Laux, P.E., Dorko, J.D., Barrow, C.S. 1989. An objective procedure for quantitating eye irritation based upon changes of corneal thickness. *Fundam. Appl. Toxicol.* 12: 258–268.
  40. Udden, M.M. 2000. Rat erythrocyte morphological changes after gavage dosing with 2-butoxyethanol: A comparison with the *in vitro* effects of butoxyacetic acid on rat and human erythrocytes. *J. Appl. Toxicol.* 20: 381–387.
  41. Udden, M.M. 2002. *In vitro* sub-hemolytic effects of butoxyacetic acid on human and rat erythrocytes. *Toxicol. Sci.* 69: 258–264.

42. Bauer, P., Weber, M., Mur, J.M., Protois, J.C., Bollaert, P.E., Condi, A., Larcen, A., Lambert, H. 1992. Transient non-cardiogenic pulmonary edema following massive ingestion of ethylene glycol butyl ether. *Intensive Care Med.* 18: 250–251.
43. Gijzenbergh, F.P., Jenco, M., Veulemans, H., Groeseneken, D., Verberckmoes, R., Delooz, H.H. 1989. Acute butylglycol intoxication: A case report. *Hum. Toxicol.* 8: 243–245.
44. Gualtieri, J.F., Harris, C.R., Roy, R., Corley, R.A., Manderfield, C. 1995. Multiple 2-butoxyethanol intoxications in the same patient: Clinical findings, pharmacokinetics, and therapy. *J. Toxicol. Clin. Toxicol.* 33: 550–551.
45. Gualtieri, J.F., DeBoer, L., Harris, C.R., Corley, R. 2003. Repeated ingestion of 2-butoxyethanol: Case report and literature review. *J. Toxicol. Clin. Toxicol.* 41: 57–62.
46. Rambourg-Schepens, M.O., Buffet, M., Bertault, R., Jaussaud, M., Journe, B., Fay, R., Lamiabie, D. 1988. Severe ethylene glycol butyl ether poisoning. Kinetics and metabolic pattern. *Hum. Toxicol.* 7: 187–189.
47. Burkhart, K.K., Donovan, J.W. 1998. Hemodialysis following butoxyethanol ingestion. *Clin. Toxicol.* 36: 723–725.
48. Osterhoudt, K.C. 2002. Fomepizole therapy for pediatric butoxyethanol intoxication. *J. Toxicol. Clin. Toxicol.* 40: 929–930.
49. Dean, B.S., Krenzelok, E.P. 1991. Critical evaluation of pediatric ethylene glycol monobutyl ether poisonings. *Vet. Hum. Toxicol.* 33: 362.
50. Carpenter, C.P., Pozzani, U.C., Weil, C.S., Nair III, J.H., Keck, G.A., Smyth Jr., H.F. 1956. The toxicity of butyl cellosolve solvent. *AMA Arch. Ind. Health.* 14: 114–131.
51. U.S. EPA. 2005. Guidelines for carcinogen risk assessment, Final Report. Risk Assessment Forum, U.S. Environmental Protection Agency. Washington, DC. EPA/630/P-03/001F. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=116283>.
52. NTP. 2000. NTP technical report on the toxicology and carcinogenesis studies of 2-butoxyethanol (CAS No. 111–76–2) in F344/N rats and B6C3F1 mice (inhalation studies). National Toxicology Program. Research Triangle Park, NC. NTP TR 484. <http://ntp.niehs.nih.gov/?objectid=070AC403-B110-CA79-3A23AF79DE7B752A>.
53. Zeiger, E., Anderson, B., Haworth, S., Lawlor, T., Mortelmans, K. 1992. Salmonella mutagenicity tests: V Results from the testing of 311 chemicals. *Environ. Mol. Mutagen.* 19: 2–141.
54. Gollapudi, B.B., Barber, E.D., Lawlor, T.E., Lewis, S.A. 1996. Re-examination of the mutagenicity of ethylene glycol monobutyl ether to Salmonella tester strain TA97a. *Mutat. Res.* 370: 61–64.
55. Chiewchanwit, T., Au, W.W. 1995. Mutagenicity and cytotoxicity of 2-butoxyethanol and its metabolite, 2-butoxyacetaldehyde, in Chinese hamster ovary (CHO–AS52) cells. *Mutat. Res.* 334: 341–346.
56. Klaunig, J.E., Kamendulis, L.M. 2005. Mode of action of butoxyethanol-induced mouse liver hemangiosarcomas and hepatocellular carcinomas. *Toxicol. Lett.* 156: 107–115.
57. NTP. 1996. Toxicology and carcinogenesis studies of acetonitrile (CAS No 75–05–8) in F344/N rats and B6C3F1 mice (inhalation studies). National Toxicology Program. Research Triangle Park, NC. <http://ehp.niehs.nih.gov/ntp/docs/4004xxdoc.html>.
58. Keith, G., Coulais, C., Ederh, A., Bottin, M.C., Rihn, B. 1996. Ethylene glycol monobutyl ether has neither epigenetic nor genotoxic effects in acute treated rats and in subchronic treated v-HA-ras transgenic mice. *Occup. Hyg.* 2: 237–249.
59. Heindel, J.J., Gulati, D.K., Russell, V.S., Reel, J.R., Lawton, A.D., Lamb IV, J.C. 1990. Assessment of ethylene glycol monobutyl and monophenyl ether reproductive toxicity using a continuous breeding protocol in Swiss CD–1 Mice. *Fundam. Appl. Toxicol.* 15: 683–696.
60. Nagano, K., Nakayama, E., Koyano, M., Oobayashi, H., Adachi, H., Yamada, T. 1979. Testicular atrophy of mice induced by ethylene glycol mono alkyl ethers (author's translation). *Sangyo Igaku/Jap. J. Ind. Health.* 21: 29–35.
61. Nagano, K., Nakayama, E., Oobayashi, H., Nishizawa, T., Okuda, H., Yamazaki, K. 1984. Experimental studies on toxicity of ethylene glycol alkyl ethers in Japan. *Environ. Health Perspect.* 57: 75–84.
62. Dodd, D.E., Snellings, W.M., Maronpot, R.R., Ballantyne, B. 1983. Ethylene glycol monobutyl ether: Acute, 9-day, and 90-day vapor inhalation studies in Fischer 344 rats. *Toxicol. Appl. Pharmacol.* 68: 405–414.
63. Doe, J.E. 1984. Further studies on the toxicology of the glycol ethers with emphasis on rapid screening and hazard assessment. *Environ. Health Perspect.* 57: 199–206.
64. Foster, P.M., Lloyd, S.C., Blackburn, D.M. 1987. Comparison of the *in vivo* and *in vitro* testicular effects produced by methoxy-, ethoxy- and n-butoxy acetic acids in the rat. *Toxicology.* 43: 17–30.
65. Exon, J.H., Mather, G.G., Bussiere, J.L., Olson, D.P., Talcott, P.A. 31991. Effects of subchronic exposure of rats to 2-methoxyethanol or 2-butoxyethanol: Thymic atrophy and immunotoxicity. *Fundam. Appl. Toxicol.* 16: 830–840.
66. NTP. 1993. NTP technical report on toxicity studies of ethylene glycol ethers: 2-methoxyethanol, 2-ethoxyethanol, 2-butoxyethanol (CAS Nos. 109–86–4, 110–80–5, 111–76–2) administered in drinking water to F344/N rats and B6C3F1 mice. National Toxicology Program. Research Triangle Park, NC. 26; NIH Publication 93–3349.
67. Nelson, B.K., Setzer, J.V., Brightwell, W.S., Mathinos, P.R., Kuczuk, M.H., Weaver, T.E., Goad, P.T. 1984. Comparative inhalation teratogenicity of four glycol ether solvents and an amino derivative in rats. *Environ. Health Perspect.* 57: 261–271.
68. Tyl, R.W., Millicovsky, G., Dodd, D.E., Pritts, I.M., France, K.A., Fisher, L.C. 1984. Teratologic evaluation of ethylene glycol monobutyl ether in Fischer 344 rats and New Zealand white rabbits following inhalation exposure. *Environ. Health Perspect.* 57: 47–68.
69. Hardin, B.D., Goad, P.T., Burg, J.R. 1984. Developmental toxicity of four glycol ethers applied cutaneously to rats. *Environ. Health Perspect.* 57: 69–74.
70. Wier, P.J., Lewis, S.C., Traul, K.A. 1987. A comparison of developmental toxicity evident at term to postnatal growth and survival using ethylene glycol monoethyl ether, ethylene glycol monobutyl ether, and ethanol. *Teratog. Carcinog. Mutagen.* 7: 55–64.
71. Greaves, P. 2000. Hepatocellular hypertrophy and hyperplasia. In *Histopathology of preclinical toxicity studies: Interpretation and relevance in drug safety evaluation* (pp. 445–448). New York, NY: Elsevier.
72. Haufroid, V., Thirion, F., Mertens, P., Buchet, J.P., Lison, D. 1997. Biological monitoring of workers exposed to low levels of 2-butoxyethanol. *Int. Arch. Occup. Environ. Health.* 70: 232–236.
73. Bringmann, G., Kuhn, R. 1977. Limiting values for the damaging action of water pollutants to bacteria (*Pseudomonas putida*) and green algae (*Scenedesmus quadricauda*) in the cell multiplication inhibition test. *Z. Wasser Abwasser Forsch.* 10(3/4): 87–98. (In German)
74. Devillers, J., Chezeau, A., Thybaud, E., Poulsen, V., Procher, J.-M., Graff, L., Vasseur, P., Mouchet, F., Ferrier, V., Quiniou, F. 2002. Ecotoxicity of ethylene glycol monobutyl ether and its acetate. *Toxicology Mechanisms and Methods.* 12: 255–263.
75. Devillers, J., Chezeau, A., Thybaud, E., Poulsen, J.-M., Graff, L., Vasseur, P., Chenon, P., Mouchet, F., Ferrier, V., Quiniou, F. 2002. Ecotoxicity of ethylene glycol monomethyl ether and its acetate. *Toxicology Mechanisms and Methods.* 12: 241–254.
76. INERIS. 1999. Détermination de la toxicité chronique du 2-butoxyethanol vis-à-vis de l'algue d'eau douce *Pseudokirchneriella subcapitata*, Ba746d-CGR21427. Verneuil-en-Halatte, France, 14 december 1999, INERIS: 14. As cited in Ref. 77.
77. ECB (European Chemicals Bureau). 2006. European Union Risk Assessment Report for 2-Butoxyethanol (EGBE). Vol. 68. European Commission.
78. Bringmann, G., Kuhn, R. 1982. Results of the toxic action of water pollutants on *Daphnia magna* in an improved standardized procedure. *Z. Wasser Abwasser Forsch.* 15(1): 1–6. (In German)
79. Juhnke, I., Luedemann, D. 1978. Results of the study of 200 chemical compounds on acute fish toxicity using the Golden Orfe test. *Z. Wasser Abwasser Forsch.* 11(5): 161–164. (In German)
80. Dow Chemical Co. 1979. Toxicity of Dowanol EB to freshwater organisms (redactor: Bartlett), 31 August 1979. As cited in Ref. 77.
81. Dawson, G.W., Jennings, A.L., Drozdowski, D., Rider, E. 1975. The acute toxicity of 47 industrial chemicals

- to fresh and saltwater fishes. *Journal of Hazardous Materials*. 1: 303–318.
82. INERIS. 1999. Détermination de la toxicité aiguë du 2-butoxyethanol vis-à-vis de *Oncorhynchus mykiss*, unpublished, Ba746f-CGR21427. Verneuil-en-Halatte, France, 14 december 1999, INERIS: 10. As cited in Ref. 77.
83. Bridie, A.L., Wolff, C.J.M., Winter, M. 1979. The acute toxicity of some petrochemicals to goldfish. *Water Res.* 13(7): 623–626.
84. Price, K.S., Waggy, G.T., Conway, R.A. 1974. Brine shrimp bioassay and seawater BOD of petrochemicals. *Journal WPCF*. 46(1): 63–76.
85. Bringmann, G., Kuhn, R. 1978. Threshold Values of Substances Harmful to Water for Blue Algae (*Microcystis aeruginosa*) and Green Algae (*Scenedesmus quadricauda*) in Tests Measuring the Inhibition of Cellular Propagation. *Vom Wasser*. 50:45 60 (in German) (English Abstract), Tr 80 0201, Literature Research Company: 22 p.
86. Bringmann, G., Kuhn, R. 1978. Testing of Substances for Their Toxicity Threshold: Model Organisms *Microcystis (Diplocystis) aeruginosa* and *Scenedesmus quadricauda*. *Mitt. Int. Ver. Theor. Angew. Limnol.* 21: 275 284.
87. Dill, DC, Milazzo, D.P. 1988. Dowanol PM Glycol Ether: Evaluation of the toxicity to the green alga, *Selenastrum capricornutum* Printz. Dow Chemical Company. EPA Document Control Number 86–890001160. 18 pages.
88. INERIS. 1999. Détermination de la toxicité chronique du 2-butoxyethanol vis-à-vis de *Daphnia magna*, Ba746a-CGR21427. Verneuil-en-Halatte, France, 15 december 1999, INERIS: 13. As cited in Ref. 77.
89. INERIS. 2001. Essai poisson 21 jours, *Danio rerio*, unpublished report, N° 22685, 05.11.2001. As cited in Ref. 77.

#### List of Subjects in 40 CFR Part 372

Environmental protection, Community right-to-know, Reporting and recordkeeping requirements, and Toxic chemicals.

Dated: September 24, 2015.

**Arnold E. Layne,**

*Director, Office of Information Analysis and Access.*

[FR Doc. 2015–25674 Filed 10–7–15; 8:45 am]

**BILLING CODE 6560–50–P**

## FEDERAL COMMUNICATIONS COMMISSION

### 47 CFR Part 1

[MD Docket No. 15–121; FCC 15–108]

### Assessment and Collection of Regulatory Fees for Fiscal Year 2015

**AGENCY:** Federal Communications Commission.

**ACTION:** Proposed rule.

**SUMMARY:** In this document the Commission revises its Schedule of Regulatory Fees to recover an amount of \$339,844,000 that Congress has required the Commission to collect for fiscal year 2015. Section 9 of the Communications Act of 1934, as amended, provides for the annual assessment and collection of regulatory fees under sections 9(b)(2) and 9(b)(3), respectively, for annual “Mandatory Adjustments” and “Permitted Amendments” to the Schedule of Regulatory Fees.

**DATES:** Comments are due November 9, 2015 and Reply Comments are due December 7, 2015.

**FOR FURTHER INFORMATION CONTACT:** Roland Helvajian, Office of Managing Director at (202) 418–0444.

**SUPPLEMENTARY INFORMATION:** This is a summary of the Commission’s Further Notice of Proposed Rulemaking (FNPRM), FCC 15–108, MD Docket No. 15–121, adopted on September 1, 2015 and released on September 2, 2015.

#### I. Administrative Matters

##### A. Initial Regulatory Flexibility Analysis

1. As required by the Regulatory Flexibility Act of 1980 (RFA),<sup>1</sup> the Commission has prepared an Initial Regulatory Flexibility Analysis (IRFA) relating to this Further Notice of Proposed Rulemaking.

##### B. Initial Paperwork Reduction Act of 1995 Analysis

2. This document does not contain new or modified information collection requirements subject to the Paperwork Reduction Act of 1995 (PRA), Public Law 104–13. In addition, therefore, it does not contain any new or modified information collection burden for small business concerns with fewer than 25 employees, pursuant to the Small Business Paperwork Relief Act of 2002, Public Law 107–198, *see* 44 U.S.C. 3506(c)(4).

##### C. Filing Instructions

3. Pursuant to sections 1.415 and 1.419 of the Commission’s rules, 47 CFR 1.415, 1.419, interested parties may file comments and reply comments on or before the dates indicated on the first page of this document. Comments may be filed using the Commission’s Electronic Comment Filing System (ECFS). *See Electronic Filing of*

<sup>1</sup> *See* 5 U.S.C. 603. The RFA, *see* 5 U.S.C. 601–612, has been amended by the Small Business Regulatory Enforcement Fairness Act of 1996 (SBREFA), Public Law 104–121, Title II, 110 Stat. 847 (1996). The SBREFA was enacted as Title II of the Contract with America Advancement Act of 1996 (CWAAA).

*Documents in Rulemaking Proceedings*, 63 FR 24121 (1998).

• **Electronic Filers:** Comments may be filed electronically using the Internet by accessing the ECFS.

• **Paper Filers:** Parties who choose to file by paper must file an original and one copy of each filing. If more than one docket or rulemaking number appears in the caption of this proceeding, filers must submit two additional copies for each additional docket or rulemaking number.

○ Filings can be sent by hand or messenger delivery, by commercial overnight courier, or by first-class or overnight U.S. Postal Service mail. All filings must be addressed to the Commission’s Secretary, Office of the Secretary, Federal Communications Commission.

○ All hand-delivered or messenger-delivered paper filings for the Commission’s Secretary must be delivered to FCC Headquarters at 445 12th St. SW., Room TW–A325, Washington, DC 20554. The filing hours are 8:00 a.m. to 7:00 p.m. All hand deliveries must be held together with rubber bands or fasteners. Any envelopes and boxes must be disposed of before entering the building.

○ Commercial overnight mail (other than U.S. Postal Service Express Mail and Priority Mail) must be sent to 9300 East Hampton Drive, Capitol Heights, MD 20743.

○ U.S. Postal Service first-class, Express, and Priority mail must be addressed to 445 12th Street SW., Washington, DC 20554.

4. People with Disabilities: To request materials in accessible formats for people with disabilities (braille, large print, electronic files, audio format), send an email to [fcc504@fcc.gov](mailto:fcc504@fcc.gov) or call the Consumer & Governmental Affairs Bureau at 202–418–0530 (voice), 202–418–0432 (tty).

##### D. Ex Parte Information

5. This proceeding shall be treated as a “permit-but-disclose” proceeding in accordance with the Commission’s ex parte rules. Persons making ex parte presentations must file a copy of any written presentation or a memorandum summarizing any oral presentation within two business days after the presentation (unless a different deadline applicable to the Sunshine period applies). Persons making oral ex parte presentations are reminded that memoranda summarizing the presentation must list all persons attending or otherwise participating in the meeting at which the ex parte presentation was made, and summarize all data presented and arguments made