6560-50-P

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, 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 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 EBGE (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 BBA 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 (>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 ≥ 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 (≥ 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 ≥ 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^3)) 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^3) for groups of 50 F344 rats and 0, 62.5, 125, and 250 ppm (0, 302, 604, and 1,208 mg/m^3) 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 ($LOAEL_{HEC}$) for hematologic effects of 271 mg/m^3 (approximately 77 mg/kg/day, assuming constant exposure, an inhalation rate of 20 cubic meters/day (m^3/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^3 (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_{50} 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_{50} values ranged from 835 to 1,815 mg/L (Refs. 77 and 78). A 48-hour EC_{50} 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_{50} (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_{50} of 2,137 mg/L for the fathead minnow (*Pimephales promelas*) (Ref. 80). A 96-hour LC_{50} value of 1,490 mg/L was available for bluegill sunfish (Ref. 81) and 96-hour LC_{50} values for rainbow trout were 1,474 and 1,700 mg/L (Refs. 74, 75, and 82). An LC_{50} value (duration not specified) of 1,575 mg/L was also available for golden orfe (*Leuciscus idus*) (Ref. 79) and a 24-hour LC_{50} 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_{50} 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_{50} 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_{50} (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_{50} 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**[a]	**Value (mg/L)**	**Reference**
Acute aquatic toxicity				
Algae				
Green algae (*Pseudokirchneriella subcapitata*)	72-hour EC_{50} (growth)	S, M	1,840	(Refs. 74 and 75)
Green algae (*Pseudokirchneriella subcapitata*)	72-hour NOEC (biomass)	S, M	88	(Ref. 82)
Freshwater invertebrate				
Water flea (*Daphnia magna*)	48-hour EC_{50}	S, U, O	1,815	(Ref. 78)
Rotifer (*Brachionus calyciflorus*)	48-hour EC_{50} (reproduction)	S, M	164	(Refs. 74 and 75)
Freshwater fish				
Golden orfe (*Leuciscus idus*)	LC_{50}	NS	1,395	(Ref. 79)
Fathead minnow (*Pimephales promelas*)	96-hour LC_{50}	S, O	2,137	(Ref. 80)
Estuarine/marine invertebrate				
Brine shrimp (*Artemia salina*)	24-hour LC_{50}	S, U, C	1,000	(Ref. 84)

Japanese oyster eggs (*Crassostrea gigas*)	24-hr LOEC (embryotoxicity)	S	1,000	(Refs. 74 and 75)
Estuarine/marine fish				
Silverside (*Menidia beryllina*)	96-hour LC_{50}	S, U	1,250	(Ref. 81)
Chronic aquatic toxicity				
Algae				
Blue-green algae (*Microcystis aeruginosa*)	8-day LOEC (cell multiplication inhibition)	S, U	35	(Refs. 85 and 86)
Green algae (*Selenastrum capricornutum*)	7-day EC_{50} (growth rate)	S, U	>1,000	(Ref. 87)
Freshwater invertebrate				
Water flea (*Daphnia magna*)	21-day NOEC (reproduction)	R, M	100	(Refs. 74 and 75)
Water flea (*Daphnia magna*)	21-day NOEC	R, M	100	(Ref. 88)
Water flea (*Daphnia magna*)	21-day EC_{50}	R, M	297	(Ref. 88)
Freshwater fish				
Zebrafish (*Brachydanio rerio*)	21-day NOEC (mortality)	NS	>100	(Ref. 89)

[a]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.

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List of Subjects in 40 CFR Part 372

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

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