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**Toxicology Report No. HEF-S.0059709.14-19, October 2021**
**Toxicology Directorate**

# Toxicology Assessment for Work Unit SAGE 16-01 Qualification of HAP-free Cleaners for Aircraft and Ground Vehicles October 2019-October 2021

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This assessment determines whether the proposed alternatives to hazardous air pollutant (HAP) cleaners for aircraft and ground vehicles exhibit performance comparable with currently utilized cleaners. The objective formulations will eliminate HAPs and result in a product with a reduced human health and environmental impact. The role of the U.S. Army Public Health Center (APHC) in preparing this Toxicology Assessment is to determine whether or not the candidate replacement formulations pose a reduced hazard to human health and the environment. The formulations presented here are mostly considered less toxic than the currently utilized HAP containing formulation. However, no one single formulation appears safer than the others, and full analyses on all of the formulations were not possible due to a lack of existing toxicity data or transparency in the formulation.

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# 1 SUMMARY

## 1.1 Overview

Research, development, testing, training, and use of substances potentially less hazardous to human health and the environment are vital to the readiness of the U.S. Army. Safeguarding the health of Soldiers, Civilians, and the environment requires an assessment of alternatives before they are fielded. Continuous assessments begun early in the Research, Development, Testing and Evaluation (RDT&E) process can save significant time and effort during RDT&E, as well as over the life cycle of the items developed. Residues of pyrotechnics, propellants, explosives, and coatings that were part of mission-essential activities have been found in soil, air, surface, and groundwater samples. Remediation of the contaminated areas has cost the Department of Defense millions of dollars and can interfere with training activities.

## 1.2 Purpose

This assessment determines whether the proposed alternatives to hazardous air pollutant (HAP) cleaners for aircraft and ground vehicles exhibit performance comparable with currently utilized cleaners. The objective formulations will eliminate HAPs and result in a product with a reduced human health and environmental impact. The role of the U.S. Army Public Health Center (APHC) in preparing this Toxicology Assessment is to determine whether or not the candidate replacement formulations pose a reduced hazard to human health and the environment.

## 1.3 Conclusions

Generally, the physical properties of the alternatives' constituents suggest that they are less likely to volatilize at a given temperature compared to the legacy product. Thus, they are inherently less likely to become an airborne threat. Of the products reviewed, none contain constituents listed on the U.S. Environmental Protection Agency (EPA) HAP or Volatile Organic Compound (VOC) lists. However, since "spray" is a considered application process, these products can be considered potentially dangerous from an inhalation perspective. While the above characteristics are generally positive it should be understood that components of the alternatives can have serious toxic effects, particularly if improperly handled.

## 1.4 Recommendations

The formulations presented here are mostly considered less toxic than the currently utilized HAP containing formulation. However, no one single formulation appears safer than the others, and full analyses on all of the formulations were not possible due to a lack of existing toxicity data or transparency in the formulation. Samples of the leading formulations should be submitted for toxicity testing where data are sparse. General precautions in handling should be

followed. Careful attention should also be given to potential physical hazards associated with use (product flammability and stability, for example). Disposal of unused product and hazardous waste should be compliant with all appropriate local, state and Federal regulations. These practices will diminish potential impacts on users and the environment.

## 2. REFERENCES

See Appendix A for list of references; acronyms are listed in the Glossary.

## 3. AUTHORITY

Military Interdepartmental Purchase Request (MIPR) No. W23MWP192458605. This toxicology assessment addresses, in part, the environment, safety, and occupational health (ESOH) requirements outlined in–

- Army Regulation (AR) 200-1, *Environmental Protection and Enhancement*;
- AR 40-5, *Army Public Health Program*;
- AR 40-10, *Health Hazard Assessment Program in Support of the Army Acquisition Process*;
- AR-70-1, *Army Acquisition Policy*;
- Department of Defense Instruction 4715.23, *Pollution Prevention*; and
- The Army Environmental Requirement and Technology Assessment (AERTA) requirements PP-13-12-01*, Securing the Availability of Green, Enhanced Coatings (SAGE Coat)* and PP-4-02-04, *Alternative Products in Cleaning and Degreasing Processes*.

This toxicology assessment was performed as part of an on-going effort by the U.S. Army Safer Alternatives for Readiness (SAFR) program to reduce or eliminate the environmental impact from life-cycle use of new chemical formulations proposed for use in support of weapon systems or platforms. This toxicology assessment is consistent with AERTA requirements for reducing the amount of HAPs/VOCs found in cleaners used on Army weapon systems, regional VOC emission standards on the rework of Army weapon systems, and Aerospace National Emission Standards for Hazardous Air Pollutants (NESHAP) by qualifying cleaners that are compliant with this NESHAP. The Principal Investigator is Mr. Daniel Pope of the Combat Capabilities Development Command, Army Research Laboratory (DEVCOM-ARL), Aberdeen Proving Ground, MD.

## 4. BACKGROUND

Maintenance of U.S. Army vehicles and weapons systems requires cleaning and degreasing in order to keep systems at peak performance capabilities. This cleaning and degreasing process requires the use of solvents in order to solubilize and remove surface contamination. Organic solvents are frequently used throughout the chemical, pharmaceutical, cosmetic, and oil and gas industries, amongst others. Solvents with low boiling points are prone to volatilize at room temperature, which puts them in a class of chemicals known as VOCs. VOCs are regulated by the EPA depending on their potential for photolytic breakdown and subsequent formation of

ozone (EPA 2021b). Some organic compounds do not degrade to ozone and are exempt from EPA regulation. However, there could still be physical, health, or environmental hazards as a result of exposure to these chemicals. These chemicals belong to a class known as HAPs, and are defined as pollutants that are known to or are reasonably anticipated to cause adverse effects to human health or adverse environmental effects (EPA 2021a). Minimizing the use and exposure of maintenance crews to VOCs and HAPs is necessary for overall readiness of the force.

## 5. STATEMENT OF THE PROBLEM

This assessment addresses the qualification of HAP-free solvents for degreasing procedures as the product may be applied through hand-wipe, spray, or immersion. With respect to *routine use*, the point source for generation of toxic airborne chemicals is relatively limited. However, the potential for health hazards increases as incidence of use increases and use occurs in unventilated or poorly-ventilated spaces. As Hazardous *Air* Pollutants, the primary environmental concern is ambient air quality. Environmental water and soil contamination, and potential associated adverse effects, are considered from the perspective of accidental spills or purposeful discharges of the chemicals into these resources.

The U.S. Army strives to comply with all local, state, and federal regulations with respect to identification and mitigation of occupational and environmental hazards. As global agreements arise, regulations change, and new products become available in the marketplace, the Army complies with regulatory changes and seeks to identify safer products for use in its military and civilian workplaces, while maintaining a strong and resilient fighting force. The Army currently uses Eastman™ MPK that contains methyl propyl ketone and methyl isobutyl ketone, which is on the EPA Hazardous Air Pollutants (HAP) list. It is also classified as a Group 2B chemical by the International Agency for Research on Cancer (IARC), which identifies the chemical as "possibly carcinogenic to humans." This assessment reviews the potential toxicities of the currently used product and compares it to potential replacements.

## 6. METHODS

In order to determine the potential human health and environmental impacts of compounds employed in these formulations, it is necessary to identify each compound correctly and to determine its physical, chemical, and toxicological properties. The primary means of identification employed for each compound in this program is its Chemical Abstracts Service Registry Number (CAS RN). While all compounds do not necessarily have a single CAS RN, the CAS RN is an unambiguous way of accessing information for chemical substances. The CAS RN is readily used as a keyword for searching online databases and is often cross-referenced with both systematic and trivial (i.e., "common" or nonsystematic) names for chemical substances. In some cases, synonyms and trade names are also used to identify structures.

Of the list of potential products, only two had insufficient information to develop an assessment. Table 1 lists these individual products and their key ingredients, in addition to the currently utilized Eastman MPK. Also shown are the declared percentage of the formulation (percent by volume) for each of the product ingredients, as provided in the manufacturer's Safety Data

Sheet (SDS). Table 2 lists the other products that have been declared as candidates to replace Eastman MPK but did not have sufficient information to allow for an assessment to be completed. The U.S. Army currently uses Eastman MPK comprised of >90% methyl propyl ketone (CAS RN 107-87-9) and < 10% methyl isobutyl ketone (MIBK) (CAS RN 108-10-1). MIBK is an EPA HAP-listed chemical.

**Table 1. Composition of Formulations**

| Product | Component(s) | Formulation | CAS RN |
|---|---|---|---|
| Eastman™ MPK, UHP | Methyl propyl ketone | 90% | 107-87-9 |
| | Methyl isobutyl ketone | <10% | 108-10-1 |
| Aerogreen® 4015 | 2-butyl cellosolve | 10% | 111-76-2 |
| Aerogreen 4065 | 2-butyl cellosolve | 10% | 111-76-2 |
| Ardrox® JC-5 | Proprietary | 10-20% | -- |
| | Sodium disilicate | 5-10% | 1344-09-8 |
| | Ammonium hydroxide | 0.1-1% | 1336-21-6 |
| Bonderite® C-AK 6871 | Alcohol ($C_{12-15}$) poly(1-6)ethoxylates | 10-30% | Proprietary |
| | Coconut diethanolamide | 10-30% | Proprietary |
| | Diethanolamine | 1-5% | 111-42-2 |
| | Dipropylene glycol monomethyl ether | 1-5% | 34590-94-8 |
| Calla® 804 | Benzalkonium chloride | <1% | 63449-41-2 |
| | Dipropylene glycol monomethyl ether | 1-10% | 34590-94-8 |
| CeeBee R-681 Wipes | Solvent naptha (petroleum), heavy arom. | 1-2.5% | 64742-94-5 |
| | 2-butoxyethanol | 1-2.5% | 111-76-2 |
| | Nonylphenol, ethoxylated | 1-2.5% | 9016-45-9 |
| CeeBee Super Bee 210 | 3-butoxypropan-2-ol | 6-10% | 5131-66-8 |
| | Alcohols, $C_{9-11}$, ethoxylated | 2.5-6% | 68439-46-3 |
| | 4,5-dihydro-1-(2-hydroxyethyl)-1H-imidazoldipropanoic acid | 2.5-6% | 95913-20-5 |
| | Methanol | 0.1-.5% | 67-56-1 |
| Chemsol Wipes | Orange Terpenes | 1-5% | 5989-27-5 |
| | Alcohols, $C_{12-15}$, ethoxylated | 1-5% | 68131-39-5 |
| | Isoparaffinic hydrocarbon/distillates petroleum, hydrotreated light | 1-5% | 64742-47-8 |
| | Dimethyl adipate | 1-5% | 627-93-0 |
| | Diethylhexyl sodium sulfosuccinate | 1-5% | 577-11-7 |
| Eastman Omnia Solvent | Butyl-3-hydroxybutyrate | >98% | 53605-94-0 |
| | Water | < 1% | 7732-18-5 |
| | Proprietary | <1% | |
| Ecolink 250-SS | Decamethylcyclopentasiloxane | 50-80% | 541-02-6 |
| | Octamethylcyclotetrasiloxane | 20-50% | 556-67-2 |
| | Dipropylene glycol dimethyl ether | 2-5% | 111109-77-4 |
| Ecolink NAVSOLVE® | Decamethylcyclopentasiloxane | 40-60% | 541-02-6 |
| | Octamethylcyclotetrasiloxane | 25-50% | 556-67-2 |
| | Dipropylene glycol, n-butyl ether | 2-12% | 29911-28-2 |
| | Hexylene glycol | <1% | 107-41-5 |
| LPS A-151 | Distillates Petroleum, hydrotreated light | 60-70% | 64742-47-8 |
| | Other | 30-40% | |
| Pantheon X-IT® Aircraft Wash | Water | 89-90% | 7732-18-5 |
| | Diethylene glycol monobutyl ether | <8% | 112-34-5 |

**Table 1. Composition of Formulations (continued)**

| Product | Component(s) | Formulation | CAS RN |
|---|---|---|---|
| Pantheon X-IT Carbon Remover and Cleaner | Water<br>Diethylene glycol monobutyl ether | 75-85%<br><14% | 7732-18-5<br>112-34-5 |
| Penair® C-5572 | Water<br>Alcohols, $C_{9-11}$,ethoxylated<br>Capramide DEA | 80-95%<br>10-20%<br>1-10% | 7732-18-5<br>68439-46-3<br>136-26-5 |
| Socomore DS-108 | Ethyl lactate<br>1-propoxy-2-propanol | 60-70%<br>12.5-15% | 97-64-3<br>1569-01-3 |

Legend:
UHP = Ultra High Purity
CAS RN = Chemical Abstracts Service Registry Number

**Table 2. Other Eligible Formulations**

| Product Name |
|---|
| TDA Research SSDX-12 |
| Pantheon Formula 223 |

Note: The reason(s) each compound was excluded from analysis are indicated: (#) comprised entirely of a trade secret chemical.

The properties necessary to assess fate and transport in the environment (FTE) include:

- Molecular weight (MW in grams (g) per mol; g/mol)
- Boiling point(bp) in degrees Celsius $^{O}C$
- Octanol-water partition coefficient (log $K_{OW}$)
- Organic carbon partition coefficient (log $K_{OC}$)
- Water solubility (milligrams (mg) or milliliters (mL) per liter (L) e.g., mg/L or mL/L)
- Henry's Law constant ($K_H$)
- Vapor pressure (vp) in millimeters (mm) of mercury (Hg) - mmHg

Basic physical and chemical properties are usually determined by consulting tertiary sources when such information is available.

Toxicological information needed to estimate potential human health risks includes reported toxicity effects of oral, inhalation, dermal, and ocular exposures; potential for developmental or reproductive toxicity, neurotoxicity, genotoxicity and carcinogenicity; and modes and mechanisms of toxicity. Values reported herein include lethal dose 50% ($LD_{50}$; reported in milligrams per kilogram (mg/kg)), no observed adverse effect level (or concentration) (NOAEL/C), lowest observed adverse effect level (or concentration) (LOAEL/C) reported in mg/kg or mg/liter (mg/L), 50% effect concentration ($EC_{50}$), lethal concentration 50% ($LC_{50}$) typically reported per cubic meter ($m^3$) or mg/L, clinical chemistry values may be reported in deciliters (dL) and some water quality values may be reported in micrograms/liter (µ/L). Toxicological information is derived directly from primary sources whenever possible. Sources used in this search included publications from peer-reviewed journals, official government publications and websites, and tertiary reference sources such as *The Merck Index* (Williams

2013). Commercial suppliers may provide results of in-house research that do not appear in the open literature.

Persistence, bioaccumulation, human health toxicity, and ecotoxicity were assigned to general categories of risk (i.e., low, moderate, or high) based on criteria modified from Howe et al. (2006). Table 3 describes the criteria used in the categorization; the relative proportions of each substance were also factored into the final assessment. Appendix B provides the Globally Harmonized System (GHS) classifications for many of these compounds.

**Table 3. Categorization Criteria Used in the Development of Environmental Safety and Occupational Health Severity[a]**

| | Low | Moderate | High | Unknown |
|---|---|---|---|---|
| PERSISTENCE | Readily biodegrades (<28 days) | Degradation ½ life: water <40 days, soil <120 days | Degradation ½ life: water >40 days soil > 120 days | Data are unavailable, insufficient, or unreliable. |
| TRANSPORT | Water sol. < 10 mg/L log $K_{OC}$ > 2.0 | Water sol. 10–1000 mg/L log $K_{OC}$ 2.0–1.0 | Water sol. > 1000 mg/L log $K_{OC}$ <1.0 | |
| BIOACCUMULATION | log $K_{OW}$ <3.0 | log $K_{OW}$ 3.0–4.5 | log $K_{OW}$ >4.5 | |
| TOXICITY | No evidence of carcinogenicity/ mutagenicity; Subchronic LOAEL > 200 mg/kg-d | Mixed evidence for carcinogenicity/ mutagenicity (B2, 2); Subchronic LOAEL 5–200 mg/kg-d | Positive corroborative evidence for carcinogenicity/ mutagenicity; LOAEL < 5 mg/kg-d | |
| ECOTOXICITY | Acute $LC_{50}/LD_{50}$ >1 mg/L or 1,500 mg/kg; Subchronic $EC_{50}$ >100 µg/L or LOAEL >100 mg/kg-d | Acute $LC_{50}/LD_{50}$ 1-0.1 mg/L or 1,500–150 mg/kg; Subchronic $EC_{50}$ 100-10 µg/L or LOAEL – 10–100 mg/kg-d | Acute $LC_{50}/LD_{50}$<100 µg/L or <150 mg/kg; Subchronic LOAEL <10 mg/kg-d | |

Legend:
mg/L = milligrams per liter; log $K_{OC}$ = Organic carbon partition coefficient
Log $K_{OW}$ = Octanol-water partition coefficient; LOAEL = lowest-observed adverse effect level
$LC_{50}$ = concentration expected to result in 50% lethality to a population of test animals.
$LD_{50}$ = Dose resulting in 50% mortality; mg/kg-d = milligrams per kilogram per day
µg/L = micrograms per liter
Note: [a]Modified from Howe et al. (Howe 2006)

Adverse effects of chemicals are described and classified by the Hazard Communication Standard (HCS) as promulgated by the U.S. Department of Labor Occupational Safety and Health Administration (OSHA) (3); the HCS is congruent with the GHS (4, 5). The EPA uses the GHS and other resources to conduct general hazard data assessments for chemicals that might warrant additional review under the Toxic Substances Control Act. Where applicable, these designators and others (like National Fire Protection Agency) are noted in this report. Physical hazards classifications include those for include flammability, reactivity, and stability under different environmental conditions.

Health hazard endpoints included acute and chronic mammalian toxicity, reproductive toxicity, developmental toxicity, neurotoxicity, carcinogenicity, mutagenicity and genotoxicity, and respiratory sensitization. Environmental considerations include persistence, transport, bioaccumulation and ecotoxicity (effects on vertebrates, invertebrates, algae, and bacteria). If no experimental data were available from literature reviews, toxicity values for various parameters associated with organic chemicals were predicted using quantitative structure activity relationship (QSAR) software when possible with the understanding of physiochemical properties of a chemical substance. Potential effects of inorganic chemicals cannot be predicted through QSAR modelling. QSAR modelling packages used in this review include the EPA's Estimation Programs Interface (EPI) Suite™ 4.11 and Ecological Structure Activity Relationships (ECOSAR)™ 1.1.

# 7 RESULTS

## 7.1 Physical and Chemical Properties

Table C-1 summarizes the physical and chemical properties (Appendix C). When data were not found, "ND" (no data) is inserted. In some cases, the property named is not applicable ("n/a") to the substance being described. For example, if the compound is a nonvolatile solid or an inorganic salt, vapor pressure, $K_{ow}$, $K_{oc}$, and $K_H$ are typically negligible.

## 7.2 Compound Summaries

Table C-2 provides the summaries of mammalian toxicity data (Appendix C). Section 8 discusses the assessments of human health and environmental toxicity for the products. Each characterization is generally based on the criteria set forth in Table 2. The final risk characterization also incorporates assessment of the uncertainty associated with available data, the amount of each compound present in the formulation, and the nature of potential exposure associated with use of the end item.

## 7.3 Eastman MPK, Ultra High Purity

### 7.3.1 Methyl Propyl Ketone [MPK] (CAS 107-87-9)

#### 7.3.1.1 General Information

MPK is a colorless liquid ketone with an odor that resembles acetone. MPK is used as a flavoring agent, in cigarettes, as a solvent both in industry and cosmetics (nail varnish removers), and in paints/coatings. It is found naturally occurring in apples, soya oil, and pineapples. Alternative nomenclature for MPK includes 2-pentanone, ethyl acetone, ethylacetone, methyl n-propyl ketone, 4-methyl-2-butanone, and propyl methyl ketone. Figure 1 shows the chemical structure of the component.

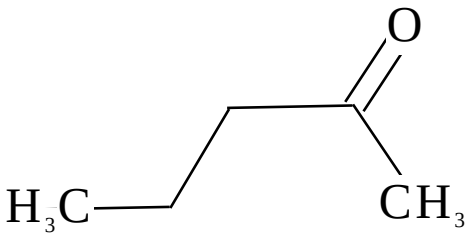


**Figure 1. Methyl propyl ketone (MPK)**

#### 7.3.1.2 Toxicology Data

Probable routes of human exposure include inhalation, ingestion, and skin contact. Acute toxicity is rare. Major effects are rarely observed. Exposure to 150 parts per million (ppm) was associated with a strong odor and with ocular and respiratory tract irritation (NCBI 2020d). Chronic exposure can cause halitosis. Ketones are readily absorbed through the skin and usually readily excreted. Target organs include dermal (skin), neurological (nervous system), ocular (eyes), respiratory (from the nose to the lungs).

##### 7.3.1.2.1 Oral

The acute oral $LD_{50}$ is reported to be 1,600 mg/kg in mouse and rat (PubChem 2020g). MPK is considered to have low to moderate acute oral toxicity. When injected intraperitoneally, the $LD_{50}$ in rat is 800 mg/kg and 1,600 mg/kg in mouse.

MPK administered to rats in drinking water up to 454 mg/kg-day for 13 months caused transient reduced weight gain (9%) early in the study (67 days) but all clinical signs, organ weights, and histopathology were normal at the end of the study (NCBI 2020d).

#### 7.3.1.2.2 Inhalation

The acute inhalation $LC_{50}$ has been reported as 25.5 mg/L (25,500 mg/m$^3$) for a 4-hour exposure in rat (European Chemical Agency (ECHA) 2019). The NOEC following a 13-week occupational exposure was 1,500 ppm (5,284.05 mg/m$^3$).

#### 7.3.1.2.3 Dermal

The dermal $LD_{50}$ in rabbits is 6,500 mg/kg (PubChem 2020g). In general, ketone skin exposures may result in dermatitis and numbness of the exposed areas; contact uticaria has also been reported (PubChem 2020g).

#### 7.3.1.2.4 Ocular

Humans exposed to 1,300–1,500 ppm in air even for a brief exposure reported severe eye irritation that did not produce permanent damage (NCBI 2020d). Human eye irritation at 350 ppm has also been reported (NIOSH 2016).

#### 7.3.1.2.5 Development and Reproduction

No adverse effects on reproduction or development in rats have been noted, with a NOAEC of 5,000 mg/m$^3$ (ECHA 2019).

#### 7.3.1.2.6 Genotoxicity

MPK was not mutagenic either with or without S9 activation in an Ames assay up to 5 mg/plate (the highest dose tested) (ECHA 2019). MEK has been found to be negative in multiple *in vitro* genotoxicity assays to include Ames, L5178/TK lymphoma assay and the BALB/3T3 cell transformation assay (EPA 2003).

#### 7.3.1.2.7 Carcinogenicity

Oral or inhalation cancer studies have not been conducted with MPK. QSAR predicts MPK is unlikely to be carcinogenic (NCBI 2020d).

#### 7.3.1.2.8 Neurotoxicity

Mean daily doses of 144 mg/kg-day for 10 months and 454 mg/kg-day for 13 months in rats revealed no neuropathologic changes in central or peripheral nervous systems (NCBI 2020d).

#### 7.3.1.2.9 Mechanism/Mode of Action

No data were found.

#### 7.3.1.3 Ecological Data

##### 7.3.1.3.1 Fate and Transport

Based on the solubility and log $K_{OC}$, MPK is expected to be mobile in groundwater. It will be unlikely to adsorb to soils, but it is expected to volatilize from surface water and wet/dry soils. MPK is not anticipated to bioaccumulate and will biodegrade in water and aerobic conditions. In the atmosphere, MPK will exist as a vapor and will degrade via photochemical processes. MPK has been detected at very low concentrations (parts per billion range) in some drinking water sources (NCBI 2020d).

##### 7.3.1.3.2 Ecotoxicity

MPK is considered to have low ecotoxicity. In fathead minnow, the acute and subacute $LC_{50}$ is 1,240 mg/L and the NOEC is 1,000 mg/L (EPA 2019a). *Daphnia* are relatively more sensitive with a NOEC of 110 mg/L and an $LC_{50}$ of 1,000 mg/L (EPA 2019a). The 96-hour exposure $EC_{50}$ is 267.3 mg/L for green algae.

##### 7.3.1.3.3 Degradation/Treatment

MPK is biodegraded by microorganisms in soil and sludge; predicted half-life is days to weeks (EPA 2014).

### 7.3.2 Methyl Isobutyl Ketone [MIBK] (CAS 108-10-1)

#### 7.3.2.1 General Information

Methyl isobutyl ketone (MIBK) is a colorless liquid organic solvent with a faint pleasant odor. It is used as a solvent in paints and dry cleaning products. It can be used as a flavoring agent and in food-contact packaging. Synonyms include 4-methyl-2-pentanone, 4-methylpentan-2-one, isopropylacetone, isobutyl methyl ketone, hexone, and 2-methyl-4-pentanone. Figure 2 shows the chemical structure of the component.

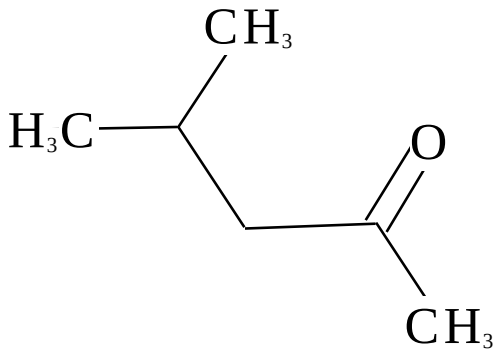


**Figure 2. Methyl Isobutyl Ketone (MIBK)**

#### 7.3.2.2 Toxicology Data

Exposure may occur dermally, orally or via inhalation, and may result in irritation of the eyes and lightheadedness accompanied by weakness, headache, dizziness, vomiting, etc. Long-term exposure can result in similar effects, as well as intestinal pain and potential liver enlargement (PubChem 2020t).

##### 7.3.2.2.1 Oral

The $LD_{50}$ in rat is 2,080 mg/kg, with an $LD_{50}$ in mice of 1,900 mg/kg and guinea pigs of 1,600 mg/kg (PubChem 2020t; ECHA 2020i). In male rats given 1,000 mg/kg-day MIBK for 10 days via oral gavage, a significant increase in accumulation of protein droplets, α2u-globulin and histopathology indicates that MIBK causes renal damage via the α2u-globulin pathway (Borghoff et al. 2009). The Alaska Department of Environmental Conservation (ADEC) has derived a reference dose (RfD) of 0.08 mg/kg-day (Williams et al. 2017).

##### 7.3.2.2.2 Inhalation

The $LC_{50}$ in rat is 100 mg/m$^3$ (PubChem 2020t). In a human exposure study, MIBK was detectable by odor at 10 ppm, but did not cause irritation until 1,800 ppm in the population studied (Dalton et al. 2000). In other human volunteers exposed to up to 200 mg/m$^3$, exposure was irritating and resulted in central nervous system (CNS) symptoms; however, reaction time and simple arithmetic assessment were not affected (Hjelm et al. 1990). Physical responses to inhalation include cough, diarrhea, dizziness, nausea, vomiting, sore throat, and generalized weakness (PubChem 2020t). An inhalation reference concentration of 3 mg/m$^3$ has been derived by ADEC, which is the same as the EPA derived IRIS value (Williams et al. 2017; EPA 2003).

##### 7.3.2.2.3 Dermal

No dermal irritation as detected in white rabbits exposed to MIBK (ECHA 2020i). ADEC has derived a dermal RfD of 0.064 mg/kg-day (Williams et al. 2017).

##### 7.3.2.2.4 Ocular

MIBK is a mild ocular irritant in rabbits (ECHA 2020i).

##### 7.3.2.2.5 Development and Reproduction

In Fischer 344 (F344) rats exposed to up to 3,000 ppm MIBK at gestational days 6 through 15 and sacrificed at day 21, maternal toxicity was observed via decreased body weight and weight gain, increased kidney weight, and decreased food consumption (Tyl et al. 1987). Fetal toxicity manifested in reduced fetal body weight and decreased skeletal ossification. In mice, also exposed to 3,000 ppm MIBK, an increase in deaths occurred in dams, along with an increase in dead fetuses and decreased skeletal ossification. In a two-generation rat reproductive study with doses up to 2,000 ppm, only transient effects on bodyweight and sedative effects following exposure were noted in all generations (Nemec et al. 2004).

#### 7.3.2.2.6 Genotoxicity

MIBK is nonmutagenic in the Ames assay with all strains with and without S9 liver fraction (National Toxicology Program (NTP) 2007). In the mouse lymphoma, BALB/3T3 cell transformation, unscheduled DNA synthesis, and the micronucleus assays, MIBK was not predicted to be mutagenic (O'Donoghue et al. 1988).

#### 7.3.2.2.7 Carcinogenicity

In animals, there is sufficient evidence to consider MIBK a carcinogen, but not to confirm carcinogenicity in humans (IARC 2012). IARC considers MIBK to be a group 2B carcinogen, possibly carcinogenic to humans. The NTP found some evidence of carcinogenicity in rats, particularly in the renal compartment (NTP 2007). Mice had an increased incidence of liver neoplasms.

#### 7.3.2.2.8 Neurotoxicity

There is some evidence of a sedative effect following exposure to MIBK, which is transient (Nemec et al. 2004; Hjelm et al. 1990; PubChem 2020t). No major behavioral effects have been noted.

#### 7.3.2.2.9 Mechanism/Mode of Action

MIBK may result in worsened cholestasis when exposure is combined with manganese-bilirubin or manganese (Vézina and Plaa 1988). MIBK exposure can also prolong the loss of righting reflex in both rats and mice, following ethanol, ketamine, pentobarbital, and thiopental exposure (Sharkawi et al. 1994).

MIBK or its metabolites (4-methyl-2-pentanol and 4-hydroxy-4-methyl-2-pentanone) may interact with constitutive-androstane receptor (CAR) in mice to increase incidence of liver tumors. This is evidenced by a decrease in liver tumors of CAR knock-out mice compared to wild-type mice, both exposed to 1,800 ppm MIBK for 6-hours (h) per day, 5 days per week, for 10 days (Hughes et al. 2016). In kidneys, MIBK interacts with the α2u-globulin pathway, resulting in kidney damage (Borghoff et al. 2009).

### 7.3.2.3 Ecological Data

#### 7.3.2.3.1 Fate and Transport

MIBK is highly water soluble, and with a low log $K_{OC}$, it is expected that MIBK will be highly mobile in groundwater. It will not adsorb to soils, and volatility from moist soils and water will be moderate. It will exist primarily as a vapor and will volatilize from lakes in a matter of days and a river in a matter of hours. It is not expected to bioaccumulate.

#### 7.3.2.3.2 Ecotoxicity

MIBK has low aquatic toxicity. The 24-hour $EC_{50}$ in algae is greater than 1,000 mg/L, while the *Daphnia* 96-hour $EC_{50}$ is greater than 200 mg/L (ECHA 2020i). The $EC_{50}$ for the fathead minnow is greater than 500 mg/L for a 96-hour exposure, while the 48-hour $EC_{50}$ for carp is greater than 700 mg/L. The acute oral $LD_{50}$ in the red-winged blackbird is 100 mg/kg, suggesting high toxicity to birds (PubChem 2020t).

#### 7.3.2.3.3 Degradation/Treatment

MIBK will not be overly persistent in the environment, but it will take days to weeks to remove it from the environment. Removal at waste water treatment plants (WWTP) is marginally successful, with most of the compound volatilizing to air (6.46% of 8.28%).

### 7.4 Aerogreen 4015

### 7.4.1 2-butyl Cellosolve/2-butoxyethanol [2-BE] (CAS 111-76-2)

#### 7.4.1.1 General Information

2-butoxyethanol (2BE) is a clear, liquid with a mild pleasant odor. It is used in food processing as a sanitizer and solvent, in addition to its used as a solvent in paints, surface coatings, cleaning products, and inks. It is also used as a degreaser, dispersant, and as a component in firefighting foams. Synonyms for this compound include butyl cellosolve, ethylene glycol monobutyl ether, butoxyethanol, 2-butyl cellosolve, and butyl glycol. Figure 3 shows the chemical structure of the component.

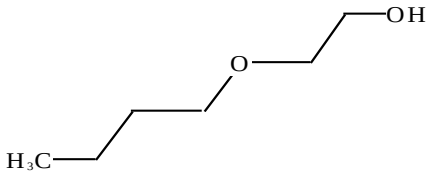


**Figure 3. 2-butoxyethanol (2-BE)**

#### 7.4.1.2 Toxicology Data

Exposure to 2BE is via oral, inhalation, and dermal routes.

#### 7.4.1.2.1 Oral

Ingestion of 2BE can result in abdominal pain, diarrhea, nausea, and vomiting. The rat $LD_{50}$ ranges from 250 – 1,750 mg/kg (ThermoFisherScientific 2007; PubChem 2020e). In mice, the

$LD_{50}$ was found to be 1,230 mg/kg, and behavioral changes included altered sleeping times and general somnolence. The rabbit $LD_{50}$ was 435 mg/kg. Longer exposures show weight loss, onset of gastritis, anemia, and cardiomyopathy in rodents.

A woman exposed to 600 mg/kg 2BE experienced coma, dyspnea, and metabolic acidosis (Rambourg-Schepens et al. 1988).

#### 7.4.1.2.2 Inhalation

Exposure of humans to 2BE by inhalation resulted in nausea and vomiting after an exposure of 195 ppm (942 mg/m$^3$) for 8 hours (PubChem 2020e). The $LC_{50}$ in mice is 3,380 mg/m$^3$-7 hours. In rats, the $LC_{50}$ was 450 ppm (2,175 mg/m$^3$) after a 4-hour exposure, with signs of behavioral ataxia and weight loss.

Repeat exposures can lead to anemia, hypoglycemia, weight loss, etc. (PubChem 2020e).

#### 7.4.1.2.3 Dermal

2BE is a dermal irritant (ThermoFisherScientific 2007).

The dermal $LD_{50}$ in rabbits was 220 mg/kg (ThermoFisherScientific 2007).

#### 7.4.1.2.4 Ocular

2BE is an ocular irritant (ThermoFisherScientific 2007).

#### 7.4.1.2.5 Development and Reproduction

2BE is a reproductive toxicant. Exposure of rats during pregnancy resulted in decreased implantation and an increase in resorbed implants (PubChem 2020e). Musculoskeletal and cardiovascular abnormalities were noted in surviving pups.

#### 7.4.1.2.6 Genotoxicity

2BE is positive in the Ames assay at 19 µmol/plate (Elliott and Ashby 1997).

#### 7.4.1.2.7 Carcinogenicity

Fore stomach and liver are the target organs for tumorigenicity in B6C3F1 mice following inhalation exposure and parenteral exposures to 2BE (Poet et al. 2003). A NTP study found that female B6C3F1 mice had increased incidence of fore stomach squamous cell papilloma or carcinoma, while in males, hemangiosarcomas of the liver were noted (NTP 2000). It may be that 2BE is not a direct carcinogen, but instead causes cancer as a result of increased iron content following hemolysis, caused by 2BE (Park et al. 2002). 2BE is a confirmed animal carcinogen with unknown mechanism of action.

#### 7.4.1.2.8 Neurotoxicity

No data were found.

#### 7.4.1.2.9 Mechanism/Mode of Action

2-BE is metabolized in liver to 2-butoxyacetic acid, which may be responsible for the red cell hemolysis observed following 2BE exposure (Klaunig and Kamendulis 2005). The primary route of elimination is as 2-butoxyacetic acid in urine regardless of exposure method (Ghanayem et al. 1987). Glucuronide and sulfate conjugates of 2-BE have also been noted in rats. The elimination half-life in humans is 30–60 minutes (PubChem 2020e). 2BE partitions to the cytoplasm and extracellular space.

### 7.4.1.3 Ecological Data

#### 7.4.1.3.1 Fate and Transport

With high solubility and a low log $K_{OC}$ (0.882), it is expected that 2BE will transport in groundwater and will be a potential water hazard. It will exist in the air as a vapor and is slightly volatile from surfaces, with a predicted volatilization rate from lakes and rivers of >150 days and 16 days, respectively. It is predicted to oxidize in the atmosphere in 0.455 days. It is not anticipated to adsorb to soils nor is it expected to bioaccumulate.

#### 7.4.1.3.2 Ecotoxicity

The $LC_{50}$ in freshwater fish is 1.5 g/L, while in marine water fish it is 1.25 g/L, with a NOEC of 100 mg/L for up to 21 days of exposure (ECHA 2021). For invertebrates, the $LC_{50}$ is 700 mg/L. In algae, the $EC_{50}$ is 623 mg/L. ECOSAR predicts are similar for fish, *Daphnia*, and green algae as previously stated from the ECHA database.

#### 7.4.1.3.3 Degradation/Treatment

2BE is predicted to biodegrade in the environment, with a predicted half-life of 3.39 days. It will not be removed by WWTP process, with a total of 1.96% removed, and with 1.78% adsorbing to sludge.

## 7.5 Aerogreen 4065

### 7.5.1 2-butoxyethanol/2-butyl cellosolve [2-BE] (CAS 111-76-2)

See paragraph 7.4.1 for toxicity data on 2-BE.

## 7.6 Ardrox JC-5

### 7.6.1 Sodium Disilicate (CAS 1344-09-8)

#### 7.6.1.1 General Information

Sodium disilicate presents as colorless to white or grayish-white crystal-like pieces or amorphous lumps. Silicates are only moderately soluble in cold water, but solutions are strongly alkaline. Commercial uses include lining of Bessemer converters and acid concentrators. Solutions are used as a preservative for eggs, as an additive for fireproofing fabrics, as a detergent in soaps, and as an adhesive, among other uses (O'Neil 2006). Synonyms include sodium metasilicate, disodium monosilicate, and water glass (HSDB 2003a). Sodium disilicate is a component of a wide variety of household products, primarily cleaning agents and personal care products (HSDB 2019). An alternate CAS RN is 6834-92-0 (PubChem 2019b). Figure 4 shows the chemical structure of the component.

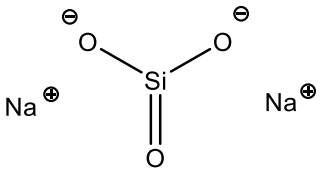


**Figure 4. Sodium Disilicate**

#### 7.6.1.2 Toxicology Data

##### 7.6.1.2.1 Oral

The oral $LD_{50}$ in rats is reported to be 1,960 mg/kg; the oral $LD_{50}$ in mice is reported to be 770 mg/kg (PubChem 2020p). If swallowed, sodium disilicate causes vomiting and diarrhea.

##### 7.6.1.2.2 Inhalation

If inhaled, sodium disilicate causes upper airway irritation, fever/hyperthermia (metal fume fever), and leukocytosis (PubChem 2020p). The GHS single organ specific toxicity for the respiratory system is Category 3 (Fisher 2018).

##### 7.6.1.2.3 Dermal

Sodium disilicate is irritating and caustic to skin. Chronic exposure has been demonstrated to cause contact dermatitis and hives (PubChem 2020p). GHS classification is Category 1B (Fisher 2018).

#### 7.6.1.2.4 Ocular

Sodium disilicate has been observed to damage the corneal epithelium when it comes in contact with the eye (PubChem 2020p). GHS classification is Category 1 (Fisher 2018).

#### 7.6.1.2.5 Development and Reproduction

Sodium disilicate is not a known or suspected reproductive hazard (Fisher 2018).

#### 7.6.1.2.6 Genotoxicity

Sodium disilicate is negative in the Ames mutagenicity test (Sigma-Aldrich 2014).

#### 7.6.1.2.7 Carcinogenicity

No data were found. Sodium disilicate is not listed as a carcinogen by the IARC, the NTP, the American Conference of Governmental Industrial Hygienists (ACGIH), or OSHA (Fisher 2018).

#### 7.6.1.2.8 Neurotoxicity

No information on neurotoxicity was found.

#### 7.6.1.2.9 Mechanism/Mode of Action

No information on mechanism or mode of action was found.

### 7.6.1.3 Ecological Data

#### 7.6.1.3.1 Fate and Transport

Due to its relative insolubility and tendency to form gels, sodium disilicate is not expected to be mobile in the environment. Disilicates are common substances found in soil. Partition to the atmosphere is not expected due to the inorganic nature of sodium disilicate, and any sodium disilicate found in the atmosphere will be in particulate form. Sodium disilicate is not bioavailable, so it is not expected to bioaccumulate.

#### 7.6.1.3.2 Ecotoxicity

No data were found for toxicity toward green algae. A 96-hour $EC_{50}$ for *Daphnia* is reported to be 216 mg/L, and a 96-hour $LC_{50}$ in zebrafish (*Brachydanio rerio*) is 210 mg/L (Fisher 2018).

#### 7.6.1.3.3 Degradation/Treatment

Due to its inorganic nature, degradation of sodium disilicate in the environment is not expected.

### 7.6.2 Ammonium Hydroxide (CAS 1336-21-6)

#### 7.6.2.1 General Information

Ammonium hydroxide is the hydroxyl salt of the ammonium ion, formed when ammonia reacts with water molecules in solution. Ammonium hydroxide is a colorless solution and ammonia concentrations can range with an upper concentration of about 30%. Ammonia diluted to 10% in water is used as a common household cleaning agent (PubChem 2020h). Ammonium hydroxide is a weak base, and solutions will be alkaline. Uses include as a bleaching or cleaning agent, disinfectant, flame retardant, corrosion inhibitor and anti-scaling agent, as an agricultural fertilizer, and for extracting metals (copper, nickel, molybdenum, etc.) from ores. Figure 5 shows the chemical structure of the component.

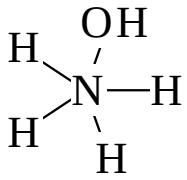

**Figure 5. Ammonium Hydroxide**

#### 7.6.2.2 Toxicology Data

##### 7.6.2.2.1 Oral

The reported lowest $LD_{50}$ concentration for humans is 43 mg/kg; the $LD_{50}$ in rat is 350 mg/kg (PubChem 2020h).

##### 7.6.2.2.2 Inhalation

The reported lowest toxic concentration in humans is 408 ppm (PubChem 2020h). The lowest published toxic concentration of ammonia vapors in humans is 408 ppm. The lowest reported lethal concentration is 5000 ppm in humans, and the immediately dangerous to life or health (IDLH) concentration of ammonia is listed as 300 ppm.

Ammonium hydroxide has an intense, pungent, suffocating odor. Resulting ammonia vapors and aerosols cause respiratory irritation and high concentrations of vapor is corrosive to the respiratory tract, resulting in laryngeal oedema and inflammation. Extreme exposure can cause life-threatening pulmonary edema. Effects of inhalation can be delayed and symptoms of exposure can include cough, sore throat, a burning sensation, labored breathing, and/or shortness of breath (PubChem 2020h).

#### 7.6.2.2.3 Dermal

Ammonium hydroxide causes skin irritation and corrosion, and is listed as GHS category 1 for skin corrosion/irritation. The effects of dermal contact can be delayed, and symptoms of dermal exposure can consist of redness, pain, and blisters. Depending on the concentration, ammonium hydroxide can cause severe chemical burns to the skin (PubChem 2020h).

#### 7.6.2.2.4 Ocular

Ammonium hydroxide as well as ammonia vapors are irritating to the eyes. Concentrated solutions can be corrosive and may cause serious eye damage. Symptoms of ocular exposure include redness, pain, blurred vision, and severe burns. As little as 44 micrograms (µg) caused severe eye irritation in the rabbit (PubChem 2020h).

#### 7.6.2.2.5 Development and Reproduction

No data were found.

#### 7.6.2.2.6 Genotoxicity

The genotoxic effects of ammonia in humans was studied in blood samples obtained from 22 fertilizer factory workers and 42 control workers (ATSDR 2004; Yadav and Kaushik 1997). The results showed an increase in frequency of sister chromatid exchanges, chromosomal aberrations, and mitotic index. The increased frequency of sister chromatid exchanges and chromosomal aberrations corresponded to increased exposure time. A single dose of ammonium administered intraperitoneally (12, 25, or 50 mg/kg) to Swiss albino mice caused an increased frequency of micronuclei (Yadav and Kaushik 1997). Studies in Drosophila melanogaster showed a positive response for mutagenic lethality (Lobasov and Smirnov 1934). Finally, in vitro studies in E. coli (Demerec et al. 1951), chick fibroblasts (Rosenfeld 1932), and mouse fibroblasts (Capuco 1977; Visek et al. 1972) indicated positive responses for genotoxicity. Together, these studies suggest that ammonia/ammonium ions may have clastogenic and mutagenic properties.

#### 7.6.2.2.7 Carcinogenicity

Ammonia is not known to be carcinogenic (ATSDR 2004).

#### 7.6.2.2.8 Neurotoxicity

No data were found.

#### 7.6.2.2.9 Mechanism/Mode of Action

Ammonium hydroxide is a weak base and solutions can be caustic (PubChem, 2020h).

#### 7.6.2.3 Ecological Data

##### 7.6.2.3.1 Fate and Transport

Due to high water solubility, ammonia is expected to be highly mobile if released to groundwater, and may pose a hazard to surface and drinking water. Some ammonia may be removed by the action of plants, for whom ammonia is a basic nutrient. Ammonia will readily partition from water or wet surfaces into the atmosphere, where it is expected to exist as a vapor. Likewise, ammonium hydroxide solutions will release ammonia gas. Ammonia is not expected to bioaccumulate in aquatic species, although plants may use it as a nutrient.

##### 7.6.2.3.2 Ecotoxicity

Ammmonium hydroxide is very toxic to aquatic life (PubChem 2020h). Since the optimal pH range for most aquatic life is 6.5 to 9.0 (Fondriest Environmental 2013), ammonium hydroxide-induced increases in pH levels create an environment that does not support aquatic life. In addition, higher pH levels increase the toxicity of ammonia, with a ten-fold increase in toxicity at pH 8 compared to pH 7 (Lenntech 2020).

Aquatic toxicity data is available for a variety of fish and at various stages of development. For example, the 24-hour LC50 values for adult Rainbow trout and fry (85 days old) are 0.097 and 0.068 mg/L, respectively. Reports for Walking catfish and blue gill (*Lepomis macrochirus*) indicate 48 hour LC50 values of 0.28 and 0.024-0.093 mg/L, respectively. The 96-hour LC50 for Coho salmon is 0.45 mg/L, and for *Salmo clarki* (cutthroat trout fry) is 0.5-0.8 mg/L. In addition, the 48-hour LC50 for the freshwater invertebrate *Daphnia magna* is 0.66 mg/L

##### 7.6.2.3.3 Degradation/Treatment

Ammonium hydroxide is readily biodegradable in water and soil, but no data on degradation of ammonia could be found (LabChem Inc 2016). Ammonia is not expected to be degraded by photolytically-produced hydroxyl radicals.

### 7.7 Bonderite C-AK 6871

#### 7.7.1 Alcohol ($C_{12-15}$) Poly(1-6)ethoxylates

Proprietary component. Not enough information was provided to perform an assessment.

#### 7.7.2 Coconut Diethanolamide

This constituent is listed as a proprietary component of Bonderite C-AK 6871. This constituent is expected to be made up of a variable mixture of various acids, such as lauric acid, myristic acid, palmitic acid, caprylic acid, capric acid, oleic acid, stearic acid, and linoleic acid. These compounds act as surfactants, and as such are expected to be irritating in nature, following both dermal and ocular exposures. Lauric acid, the primary component of coconut diethanolamide, is negative in the Ames assay, is an irritant, and may be a concern for chronic aquatic toxicity (PubChem 2021). However, dermal application of the distillates of coconut diethanolamide did

result in an increase in tumor rate in mice (IARC 2012). Without a better understanding of the makeup of this constituent, it is not feasible to do a more in depth analysis.

### 7.7.3 Diethanolamine [DEA] (CAS 111-42-2)

#### 7.7.3.1 General Information

Diethanolamine (DEA) is an oily, colorless liquid or solid white crystalline compound with a slightly rotten fish or ammonia odor. DEA is used in soaps and as a surfactant in liquid laundry and dishwashing detergents, cosmetics, shampoos, and hair conditioners. It is also used in textile processing, gas purification, as an anticorrosion agent in metalworking fluids, and in preparations of agricultural chemicals. It may also be used as a solvent for numerous intravenous drugs. Synonyms for this substance are Diolamine, Iminodiethanol, 2,2'-Iminodiethanol, and Diethylolamine. Figure 6 shows the chemical structure of the component.

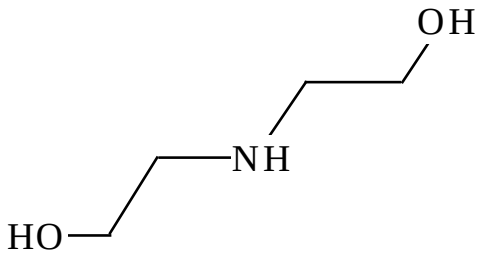


**Figure 6. Diethanolamine**

#### 7.7.3.2 Toxicology Data

##### 7.7.3.2.1 Oral

The rat oral $LD_{50}$ for DEA is 1,600 mg/kg, affecting sense organs, causing lacrimation, and tremors (PubChem 2020m; ECHA 2020h). In mice, the $LD_{50}$ is 3,300 mg/kg. The estimated fatal dose of DEA in humans is 20 g. Ingestion of DEA can result in abdominal pain and a burning sensation.

##### 7.7.3.2.2 Inhalation

Rat 90-day exposures to DEA results in changes to brain weight, liver weight, and ulceration of the stomach at doses as low as 150 mg/m$^3$ for 6-hour exposure periods (Gamer et al. 2008). Fumes are noxious, resulting in coughing, nausea, headache, and a smothering sensation (PubChem 2020m).

#### 7.7.3.2.3 Dermal

The dermal $LD_{50}$ in rabbits is 7.64 mg/kg, while in the guinea pig it was 11.9 mg/kg (PubChem 2020m). DEA exposure on the skin results in moderate dermal irritation, and long-term exposure may cause sensitization (Lessmann et al. 2009).

#### 7.7.3.2.4 Ocular

DEA is a severe eye irritant, resulting in grade 5 lesions in the rabbit (PubChem 2020m).

#### 7.7.3.2.5 Development and Reproduction

Rats fed 200 mg/kg-day DEA from days 6-19 of pregnancy had increased implantation mortality and surviving pups had reduced weight gain. At 125 mg/kg-day, the number of surviving pups was decreased (PubChem 2020m).

#### 7.7.3.2.6 Genotoxicity

DEA is nonmutagenic in the Ames assay, both with and without S9 liver fractions (NTP 1999). There also is no evidence of sister chromatid exchanges or chromosomal aberrations in Chinese Hamster Ovary (CHO) cells, with and without S9. Peripheral blood samples of exposed mice showed no increase in micronucleated erythrocytes.

#### 7.7.3.2.7 Carcinogenicity

According to the IARC, there is inadequate evidence that DEA is a human carcinogen; however, sufficient evidence exists to classify DEA as an animal carcinogen (IARC 2000). IARC classifies DEA as a Group 2B carcinogen (possibly a human carcinogen). The ACGIH classifies DEA as a Group A3 carcinogen, confirmed in animals, but of unknown relevance in humans (ACGIH 2018). Dermal administration for a 2-year bioassay in mice resulted in liver and renal tubule neoplasms (NTP 1999).

#### 7.7.3.2.8 Neurotoxicity

Exposure to DEA can affect brain development and brain weights. Choline deficiencies as a result of DEA exposure, can also affect brain function (IARC 2000; Lehman-McKeeman et al. 2002; Leung et al. 2005).

#### 7.7.3.2.9 Mechanism/Mode of Action

DEA is retained in tissues following oral exposure, with only 30% removed via urine, 3% in feces, and 0.2% exhaled. The remaining DEA was found in liver, kidney, lung, spleen, heart, brain, and muscle following 7 mg/kg-d exposure in F344 rats (IARC 2000). DEA can be incorporated into membrane phospholipids following O-phosphorylation or N-methylation, allowing incorporation into the polar head groups.

Cells exposed to 3 mM DEA showed decreased proliferation and increased apoptosis, with a decrease in choline uptake, thus affecting intracellular choline and phosphocholine levels (Niculescu et al. 2007). Addition of choline to the treatment media did mitigate the effects on proliferation and apoptosis; however, intracellular phosphocholine levels were not increased. Decreases in choline levels can affect neural function as well as alter lipid levels in the liver, leading to fatty liver, and may play a role in carcinogenicity in rodents (PubChem 2020m; Lehman-McKeeman et al. 2002; Leung et al. 2005).

#### 7.7.3.3 Ecological Data

##### 7.7.3.3.1 Fate and Transport

With a high solubility and low log $K_{OC}$, DEA is expected to readily transport via groundwater. It is unlikely to adsorb to soils. It will exist in the atmosphere as a vapor/particulate mix and will not readily volatilize from moist soils or water sources. In the atmosphere, it may be susceptible to photodegradation. It is not expected to bioaccumulate.

##### 7.7.3.3.2 Ecotoxicity

In *Oncorhynchus mykiss* (rainbow trout), the 96-hour $LC_{50}$ is 460 mg/L (ECHA 2020h). For the fathead minnow (*Pimephales promelas*), the 96-hour $LC_{50}$ was 1,480 mg/L. The $LC_{50}$ in *Daphnia* is 171 mg/L (48-hour), with an $EC_{50}$ of 30.1 mg/L. For algae, the freshwater $LC_{50}$ was 9.5 mg/L and the marine $LC_{50}$ was 86.96 mg/L.

##### 7.7.3.3.3 Degradation/Treatment

According to EPI Suite™ modeling, WWTP treatment is not expected to remove DEA from waste water, with a total removal following treatment of 1.85%, primarily to sludge. Biodegradation will take days to weeks, and photodegradation will take a matter of hours in the atmosphere.

### 7.7.4 Dipropylene Glycol Monomethyl Ether [DPGME] (CAS 34590-94-8)

#### 7.7.4.1 General Information

Dipropylene glycol monomethyl ether (DPGME) is a colorless liquid with a mild, ether-like odor. DPGME is an important component of a variety of industrial and consumer products that include hydraulic brake fluids, solvents, paints and dyes, cleaning agents, cosmetic fragrances and pesticides. Synonyms for DPGME include 2-methoxymethylethoxypropanol (MMEP), dipropylene glycol methyl ether, PPG-2 methyl ether and various commercial synonyms including arcosolv DPM, dowanol DPM, dowanol-50B, Glysolv DPM, Kino-red, and Ucar solvent 2LM. Figure 7 shows the chemical structure of the component.

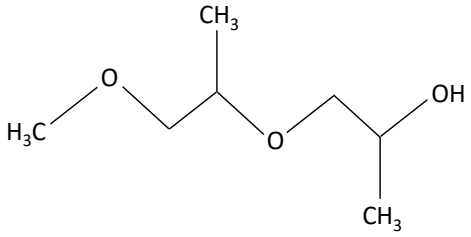


**Fig. 7. Dipropylene Glycol Monomethyl Ether**

### 7.7.4.2 Toxicology Data

Inhalation, dermal contact, and ingestion are the primary routes of exposure to DPGME, which is rapidly absorbed and distributed through the body when introduced by the oral and inhalational routes of exposure (Robinson et al. 2009). It is thought that concentrations above 5000 ppm are unlikely to be encountered in the workplace due in part to its high boiling point, and low vapor pressure.

#### 7.7.4.2.1 Oral

By Toxicity Prediction Komputer Assisted Technology (TOPKAT) modeling, the rat oral $LD_{50}$ for DPGME was estimated as 7.4 g/kg.

The rat oral acute $LD_{50}$ reported as 5.5 mL/kg in males and 5.45 mL/kg in females (Clayton and Clayton 1993) – signs of toxicity were CNS depression (Rowe et al 1954).

While in dogs, the acute oral $LD_{50}$ was estimated as 7.5 mL/kg with effects seen in the lungs and thorax (Shideman and Procita 1951). In a repeated oral administration study, doses of 1.0 g/kg for 35 days caused no observable health effects (Browning 1965). Another dog study concluded that the oral $LD_{50}$ was 7,500 mg/kg with mortality seen within 48 hours. Signs of toxicity included respiratory paralysis (Shideman and Procita 1951).

#### 7.7.4.2.2 Inhalation

The TOPKAT estimate for an acute inhalation $LC_{50}$ in the rat was estimated as >10 g/m$^3$.

Vapor and aerosol exposures to rats, rabbits, guinea pigs, and monkeys results in mild narcosis or CNS depression at doses ranging from 500 ppM DPGME for 7-hours to repeated dose studies of 6 h/day for 9 days or 7 h/day for 6 to 8 months (Rowe et al. 1954, Landry and Yano 1984, Clayton and Clayton 1993). In multiple of these studies, alterations to liver weight were noted, but no histopathological changes were seen.

It should be noted that levels of DPGME in the air of 300 – 400 ppm were described as very disagreeable (Rowe et al. 1954). In addition, the odor threshold and irritation effect level for DPGME were reported as being 35 ppm and 74 ppm, respectively.

#### 7.7.4.2.3 Dermal

TOPKAT does not predict that DPGME will be either a dermal irritant or sensitizer.

The $LD_{50}$ for rats was greater than 20 mL/kg for dermal exposures (ECHA 2020c). In rabbits, the dermal $LD_{50}$ was estimated as 9.5 g/kg.

Human patch tests with DPGME indicated no evidence of either skin irritation or sensitization (Rowe et al. 1954).

#### 7.7.4.2.4 Ocular

TOPKAT does not predict DPGME to be an ocular irritant, which is supported by observation of transient irritation without injury in rabbits (Browning 1965). Constant contact with applications of 5 mL/kg or higher over a period of several weeks caused only mild irritation.

A 20% solution of DPGME (final volume of 0.04 mL) was administered to human volunteers, resulted in a transient (less than 1 minute) stinging sensation and mild transient lachrymation (Ballantyne 1984, 1983).

#### 7.7.4.2.5 Development and Reproduction

TOPKAT does not predict that DPGME will be a developmental or reproductive toxicant. In addition, studies of propylene glycol monomethyl ether in rats do not provice any evidence of toxicity to the fetus (ECHA 2020c).

#### 7.7.4.2.6 Genotoxicity

DPGME was studied for mutagenic activity in *Salmonella typhimurium* strains TA1535, TA1537, TA1538, TA98, and TA100 and for clastogenic activity in CHO cells (ECHA 2020c). In these assays, DPGME was negative both with and without metabolic activation.

#### 7.7.4.2.7 Carcinogenicity

It is unlikely that DPGME is carcinogenic.

#### 7.7.4.2.8 Neurotoxicity

No specific data are available for DPGME. Acute, subacute, and subchronic studies in experimental animal studies indicate that exposure to high concentrations of DPGME, or extensive and prolonged skin contact might produce CNS depression leading to narcosis.

#### 7.7.4.2.9 Mechanism/Mode of Action

Microsomal O-demethylation is a primary route of DPGME biotransformation; the conjugates are considered nontoxic and are rapidly eliminated from the body ((United Nations

Environmental Programme (UNEP) 2001). Dipropylene glycol and sulphates or glucuronides of DPGME have all been identified as main urinary metabolites.

DPGME was evaluated in ToxCast™, where it was active in 17/745 screening assays for nuclear receptor, cell cycle, and DNA binding endpoints, with six scoring above 50% efficacy in the nuclear receptor category. The $EC_{50}$s were between 28 and 74 uM. (Williams et al. 2017).

#### 7.7.4.3 Ecological Data

##### 7.7.4.3.1 Fate and Transport

DPGME is highly soluble and is unlikely to adsorb to soils. Nor is it likely to bioconcentrate. It is likely to volatilize from wet surfaces. It is likely to exist as a particulate in the atmosphere. When DPGME is found in the atmosphere, it is estimated as having a half-life of about 3.4 hours and is thought to be removed from the atmosphere by washout.

##### 7.7.4.3.2 Ecotoxicity

DPGME has been extensively evaluated for aquatic toxicity. The $LC_{50}$ for Daphnids is 1,919 mg/L and 10,000 mg/L for fathead minnow (Williams et al. 2017). The $LC_{50}$ for the emerald shiner (*Notropis atherinoides*) is 150 mg/L. The chronic LOEC is 0.5 mg/L in Daphnids and 5,000 mg/L in diatoms.

##### 7.7.4.3.3 Degradation/Treatment

Biodegradation is the primary means of removal of DPGME from moist soil substrates. In WWTPs, DPGME would not be expected to sorb to sediment substrates or to bioconcentrate, with the major degradation pathway likely to be biodegradation, with minimal contributions from photolytic or hydrolytic degradation pathways.

### 7.8 Calla 804

#### 7.8.1 C8-18-Alkyldimethylbenzyl Ammonium Chlorides [ABAC] (CAS 63449-41-2)

##### 7.8.1.1 General Information

C8-18-Alkyldimethylbenzyl ammonium chlorides (ABAC) is a colorless or yellow powder or gummy amber solid with an aromatic odor and a very bitter taste. ABAC is used as a quaternary compound. Synonyms include Benzyldimethyldecylammonium chloride, benzyl-decyl-dimethylazanium; chloride, and Benzyl(decyl)dimethylammonium chloride. Figure 8 shows the chemical structure of the component.

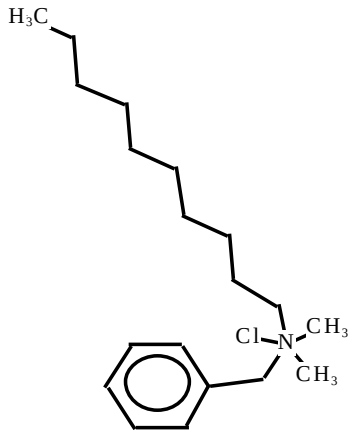


**Figure 8. C8-18 Alkyldimethylbenzyl Ammonium Chlorides**

#### 7.8.1.2 Toxicology Data

Exposure to ABAC can be oral, inhalational, or dermal. It can cause sore throat, skin burns, blurry vision, eye pain, abdominal pain, and diarrhea.

##### 7.8.1.2.1 Oral

The rat oral $LD_{50}$ is 150 mg/kg, with ABAC causing somnolence and hemorrhage (PubChem 2020i).

##### 7.8.1.2.2 Inhalation

No data were found.

##### 7.8.1.2.3 Dermal

No data were found.

##### 7.8.1.2.4 Ocular

No data were found.

##### 7.8.1.2.5 Development and Reproduction

No data were found.

##### 7.8.1.2.6 Genotoxicity

No data were found.

#### 7.8.1.2.7 Carcinogenicity

No data were found.

#### 7.8.1.2.8 Neurotoxicity

No data were found.

#### 7.8.1.2.9 Mechanism/Mode of Action

ABAC is active in estrogen receptor α and retinoid x nuclear receptor α binding according to ToxCast (EPA 2019e).

### 7.8.1.3 Ecological Data

#### 7.8.1.3.1 Fate and Transport

ABAC is moderately soluble and is highly likely to adsorb to soil, so is unlikely to be mobile in groundwater or moist soils. In the air, it will exist as a particulate. It is not expected to easily volatilize from moist soils or bodies of water. It is also not expected to bioconcentrate.

#### 7.8.1.3.2 Ecotoxicity

While ECOSAR predicts low toxicity to ABAC for algae, *Daphnia*, and fish (> 100 mg/L), experimental values indicate that ABAC is extremely toxic to fish and invertebrates, with $LC_{50}$ values ranging from 0.1 to 11.3 mg/L in fish (striped bass, brown trout, and red rasbora) and ≤ 1 mg/L in invertebrates (snail and clam) (EPA 2019e).

#### 7.8.1.3.3 Degradation/Treatment

ABAC will not readily biodegrade; however, photodegradation will be rapid. It will not be easily removed by WWTP.

### 7.8.2 Dipropylene Glycol Monomethyl Ether [DPGME] (CAS 34590-94-8)

See paragraph 7.7.4 for toxicity data on DPGME.

## 7.9 CeeBee R-681 Wipes

### 7.9.1 Solvent Naptha (Petroleum), Heavy Aromatics/Heavy Aromatic Naphtha [HAN] (CAS 64742-94-5)

#### 7.9.1.1 General Information

Heavy aromatic naphtha (HAN) is a mixture of flammable petroleum distillates that is derived from primarily aromatic streams. Naphthalene is a component of this mixture at 5–20% (CISCO 2015). Molecules in this fraction typically have between 9 and 16 carbon atoms and a boiling

range of 165ºC–290ºC (ECHA 2020s). Because this is a complex mixture, HAN is also described as jet fuel and kerosene/kerosine. The toxicological evaluation of hydrocarbon distillates by regulatory agencies involves use of read-across techniques to compare similar mixtures of distillates. Toxicity is believed to be comparable where the carbon chain lengths are similar. The contribution of aromatic and polyaromatic hydrocarbons to toxicity is not consistently evaluated. Figure 9 shows the chemical structure of the component.

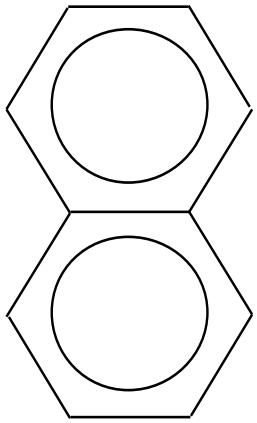

**Figure 9. Solvent Naptha (Petroleum), Heavy Aromatics/Heavy Aromatic Naphtha (HAN)**

### 7.9.1.2 Toxicology Data

#### 7.9.1.2.1 Oral

The acute oral $LD_{50}$ in rats is reported to be 7,050 mg/kg (CISCO 2015).

The chronic NOAEL in rats for a 90-day exposure is 300 mg/kg-day (CISCO 2015).

#### 7.9.1.2.2 Inhalation

The acute inhalation $LC_{50}$ in rats for heavy aromatic naphtha is reported to be greater than 590 mg/m$^3$ for a 4-hour exposure (PubChem 2020x). The subchronic Lowest Observed Adverse Effect Concentration (LOAEC) in rat is 500 mg/m$^3$. The odor threshold for HAN is reported as <1 ppm (Carpenter et al. 1975).

Heavy aromatic naphtha may be fatal if swallowed and then aspirated; causes respiratory irritation (CISCO 2015).

#### 7.9.1.2.3 Dermal

The dermal $LD_{50}$ for HAN in rabbits is reported to be greater than 2 mL/kg. Exposure at this level results in general depressed activity (somnolence), changes in motor activity, and irritability (PubChem 2020x).

#### 7.9.1.2.4 Ocular

Heavy aromatic naphtha results in damage to eyes from prolonged or repeated exposure (CISCO 2015).

#### 7.9.1.2.5 Development and Reproduction

Members of the kerosene compound class have been tested both for developmental and reproductive toxicity (CISCO 2015). Using jet fuel and kerosene read-across data from reproductive and developmental studies, the NOAEL in female rats is 1,000 mg/kg-day (oral) and the NOAEC is >/= 364 ppm (ECHA 2020s). The LOAEL for fetal effects (reduced fetal weight) is 1,500 mg/kg-day (ECHA 2020s).

#### 7.9.1.2.6 Genotoxicity

Kerosene compounds have been tested in both *in vivo* and *in vitro* genotoxicity assays (ECHA 2020s). The range of *in vitro* assays include bacterial mutagenicity tests modified for testing water-insoluble kerosenes, mutagenicity in mouse lymphoma cells, and sister-chromatid exchange in CHO cells. *In vivo* assays include bone marrow cytogenetic and micronucleus studies in rat and mice. The majority of these studies had negative results and individual constituents have tested negative as well. Thus, the weight of evidence indicates kerosenes and jet fuels are not mutagenic (ECHA 2020s).

#### 7.9.1.2.7 Carcinogenicity

Kerosenes and jet fuels have been shown to not be carcinogenic via the oral or inhalation route. However, chronic dermal exposure to HAN is classified in GHS category 2 for carcinogenicity, possibly carcinogenic to humans (CISCO 2015).

#### 7.9.1.2.8 Neurotoxicity

Inhalation of high concentrations may cause dizziness, anesthesia and other CNS effects. A single exposure can result in GHS category 1 specific target organ toxicity (respiratory irritation) and narcotic effects (CISCO 2015).

#### 7.9.1.2.9 Mechanism/Mode of Action

No information was found on mechanism or mode of action.

#### 7.9.1.3 Ecological Data

##### 7.9.1.3.1 Fate and Transport

HAN is a complex mixture of hydrocarbons that will distribute to the environmental compartments according to the individual constituents' properties. Generally, HAN is expected to demonstrate limited mobility in soil due to lack of aqueous solubility and a probable high log $K_{oc}$ value. Partitioning to the atmosphere from water, wet, or dry soil is expected to be relatively high due to the vapor pressure of this class of hydrocarbons.

##### 7.9.1.3.2 Ecotoxicity

The chemical complexity of HAN limits the utility of aquatic toxicity testing. However, several aquatic toxicity tests have been conducted on kerosenes and jet fuels and these data are available from ECHA and the toxicity values are also found on the CompTox dashboard (ECHA 2020s; EPA 2020). The acute $LC_{50}$ is 2–100 mg/L in rainbow trout and the NOELs ranged from 6.8–10 mg/L (ECHA 2020s). In *Daphnia magna* the immobilization $EC_{50}$ ranges from 1.9–89 mg/L and the NOELs were 0.3–40 mg/L (ECHA 2020s). In a Daphnid 21-day chronic reproductive test, the NOEL was 0.48 mg/L (ECHA 2020s). In algae, the 72-hour growth inhibition NOEL average is 1.0 mg/L and the $EC_{50}$ is 5.0–6.2 mg/L (ECHA 2020s). HAN meets the GHS criteria for classification as Aquatic Chronic II.

Sugar beet seedlings (*Beta vulgaris*) and corn (*Zea mays*) were exposed to heavy aromatic naphtha at concentrations of 100–500 ppm and 0.5% (volume/volume [v/v]) resulting in retardation of growth and deformation of growth, respectively (EPA 2019e).

##### 7.9.1.3.3 Degradation/Treatment

HAN is resistant to hydrolysis due to a lack of suitable hydrolysable functional groups (ECHA 2020s). Kerosenes are biodegradable; naphtha solvents were shown to partially biodegrade within 28 days (ECHA 2020s).

### 7.9.2 2-butoxyethanol/2-butyl Cellosolve [2-BE] (CAS 111-76-2)

See paragraph 7.4.1 for toxicity data on 2-BE.

### 7.9.3 Nonylphenol, Ethoxylated [NPEO] (CAS 9016-45-9)

#### 7.9.3.1 General Information

Nonlyphenol, ethoxylated (NPEO) is a colorless liquid or a white solid with a mild odor. It is used as a non-ionic surfactant, as an emulsifier, and as a metal cleaner. It used to be included in various pesticide formulations; however, it is no longer listed. Synonyms are 2-[2-(4-Nonylphenoxy)ethoxy]ethanol, Polyoxyethylene(10)nonylphenyl ether, CO-630, and (Nonylphenoxy)polyethylene oxide. Figure 10 shows the chemical structure of the component.

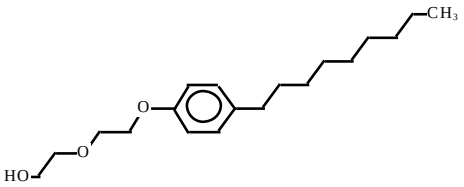


**Figure 10. Nonylphenol, Ethoxylated**

#### 7.9.3.2 Toxicology Data

NPEO is listed as a substance of very high concern within the European Union REACH regulation, and therefore is a restricted substance (ECHA 2020d). NPEO biodegrades to 4-nonylphenyl (4-NP).

##### 7.9.3.2.1 Oral

The rat oral $LD_{50}$ is 1,310 mg/kg (Lewis 1999). The mouse $LD_{50}$ is > 4,000 mg/kg (ECHA). In the rat, a NOEL of 1,000 mg/kg-day has been established, with a NOEL in mice of 600 mg/kg-day (EPA 2019e).

##### 7.9.3.2.2 Inhalation

No data were found

##### 7.9.3.2.3 Dermal

Prolonged contact causes irritation, and the $LD_{50}$ in rabbits is 2,000 mg/kg (Lewis 1999). It has not been found to be a sensitizer (ECHA 2020d).

##### 7.9.3.2.4 Ocular

NPEO causes eye irritation (PubChem 2020d).

##### 7.9.3.2.5 Development and Reproduction

In fathead minnow, continuous flow-through exposure to the NPEO metabolite 4-NP for 42 days to doses as low as 1.1 µg/L negatively affected spermatogenesis and Sertoli cell physiology (Miles-Richardson et al. 1999). Females were unaffected by either NPEoX (the x refers to the number of ethoxylate units) or 4-NP, and secondary sex characteristics were also unchanged with exposure to either compound. Similarly, in another fathead minnow assessment, at concentrations up to 7.9 µg/L NPEO did not show alterations in vitellogenin levels, or affected plasma testosterone or 17β-estradiol concentrations (Nichols et al. 2001).

#### 7.9.3.2.6 Genotoxicity

4-NP is mutagenic as determined in the yeast-based genotoxicity assay (Frassinetti et al. 2011). However, NPEO itself is not mutagenic according to the Ames assay (ECHA 2020d).

#### 7.9.3.2.7 Carcinogenicity

No data were found

#### 7.9.3.2.8 Neurotoxicity

No data were found

#### 7.9.3.2.9 Mechanism/Mode of Action

In *C. elegans*, NPEO modulates gene express related to ROS production, cellular stress, and xenobiotic metabolism (De la Parra-Guerra and Olivero-Verbel 2020). NPEOx is also an endocrine distruptor, as an antagonist to the estrogen receptor α (ERα), androgen receptor, thyroid hormone receptor, and estrogen-related receptor γ.(Ji et al. 2019). It degrades to 4-nonylphenyl.

### 7.9.3.3 Ecological Data

#### 7.9.3.3.1 Fate and Transport

Due to its low solubility and high log $K_{OC}$, NPEO will not transport in groundwater, moderate to strong likelihood of adsorption to soil. NPEO will exist in the air as vapor. The volatility of NPEO is expected to be low, and it will not readily evaporate from moist soils or water surfaces. It is also not expected to bioaccumulate.

#### 7.9.3.3.2 Ecotoxicity

NPEO has been suspected of being an endocrine disruptor, particularly in aquatic species. There does appear to be some effect on reproduction, with $EC_{50}$ values for reproduction below 5 mg/L with great pond snails and fathead minnows (ECOTOX 2009). The $LC_{50}$ in green algae is 12 mg/L, and 9 mg/L in *Daphnia*. In fish, the $LC_{50}$ varies from 1 mg/L to 1000 mg/L depending on the oxygen levels for Scud, bluegill and rainbow trout. In *C. elegans*, the 24-hour $LC_{50}$ was 3215 uM (992 mg/L) (De la Parra-Guerra and Olivero-Verbel 2020). For frogs (*X. laevis*), the $LC_{50}$ was 3.9-5.4 mg/L, with the potential for some effect on metamorphosis following exposure to the breakdown product 4-NP (Mann and Bidwell 2000; Xu et al. 2019).

#### 7.9.3.3.3 Degradation/Treatment

Due to the likelihood of NPEO adsorbing to soil and sludge, NPEO will be mostly removed (85%) by adsorption by WWTP.

## 7.10 CeeBee Super Bee 210

### 7.10.1 3-butoxypropan-2-ol/propylene Glycol N-butyl Ether [PGBE] (CAS 5131-66-8)

#### 7.10.1.1 General Information

Propylene glycol n-butyl ether (PGBE) is a solvent used primarily in surface coatings, leather, pesticides, electrical, industrial cleaners and hard surface cleaners, latex coatings, coupling agents and chemical intermediates for epoxides, solvents and plasticizers (PubChem 2020a). Also known as butoxypropanol, Dowanol-PNB, 3-butoxypropan-2-ol. Figure 11 shows the chemical structure of the component.

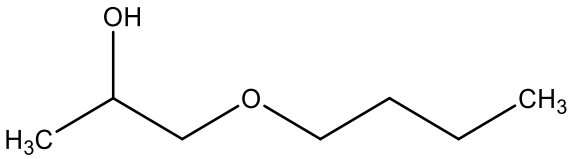


**Figure 11. 3-butoxypropan-2-ol/propylene Glycol N-butyl Ether**

#### 7.10.1.2 Toxicology Data

##### 7.10.1.2.1 Oral

A rat oral $LD_{50}$ for PGBE was reported as 3,300 mg/kg; an earlier study found an $LD_{50}$ of 1,900 mg/kg (UNEP 2003).

Exposure of rats to PGBE in drinking water for 13 weeks at concentrations up to 1,000 mg/kg-day resulted in an increase in liver weights in males and increased kidney weights in females, but only at the highest dose and without associated pathology (United Nations Environmental Programme (UNEP) 2003). Administration of PGBE to rats by oral gavage for 14 consecutive days, at doses up to 400 mg/kg-day resulted in no hematological toxicity (UNEP 2003).

##### 7.10.1.2.2 Inhalation

The inhalation $LC_{50}$ value for PGBE following a 4-hour exposure in rats was 5.83 mg/m$^3$ (ECHA 2020b). No effects were observed in 2-week studies in rats at the highest tested concentrations of 3,244 mg/m$^3$. Overexposure to PGBE may produce dizziness, drowsiness, or nausea (PubChem 2020a).

##### 7.10.1.2.3 Dermal

Contact with PGBE results in redness and pain (ECHA 2020b). Slightly toxic by skin absorption, it may cause irritation with symptoms of reddening and itching. Rats dermally treated with PGBE at doses up to 1 mL/kg-day for up to 13 weeks (5 days/week) had only minor irritation at the site of contact in the highest dose group (UNEP 2003). Similarly, in rabbits treated with up to 100

mg/kg-day PGBE at occupational exposure frequencies (7 hours/day, 5 days/week for 13 weeks), only local irritation effects were observed.

#### 7.10.1.2.4 Ocular

Contact with PGBE results in redness and pain (ECHA 2020b). F344 rats were exposed 6 hours/day for 9 days over an 11-day period. Exposure to 600 ppm PGBE caused fibroblastic proliferation, keratitis, and some degeneration in the cornea. All lesions were reversible (PubChem 2020a).

#### 7.10.1.2.5 Development and Reproduction

There was no effect of PGBE on reproductive rates or in fetal development in a study in pregnant New Zealand White rabbits exposed via topical administrations of concentrations up to 100 mg/kg-day during gestational days 7 to 18 (Gibson et al. 1989). Animals were euthanized and evaluated for pregnancy, number and placement of implantations, early and late resorptions and live fetuses on gestational day 29. There was also no effect on abortion or premature delivery, body weight, or feed consumption. Rats treated in a similar manner with up to 1 mL/kg-day also had no signs of developmental effects (Spencer 2005). Rats were treated on gestational days 6-16.

#### 7.10.1.2.6 Genotoxicity

Results from Ames tests in *Salmonella typhimurium* strains TA98, TA100, TA1535, and TA1537 were negative for mutagenicity with and without S9 activation. PGBE was not mutagenic after exposure of Chinese hamster (K-1) cells (UNEP 2003).

#### 7.10.1.2.7 Carcinogenicity

No data were found.

#### 7.10.1.2.8 Neurotoxicity

No data were found.

#### 7.10.1.2.9 Mechanism/Mode of Action

PGBE is primarily metabolized in the liver via mixed function oxidation (ECHA 2020b). This results in cleavage of the ether and yields propylene glycol and an alcohol. Further metabolism results in the production of carbon dioxide ($CO_2$) and water. Conjugation may occur with glucuronide, sulfate, or glutathione to assist in excretion via the urine. There is evidence that PGBE may interact with ERα and NRF2, which binds the antioxidant response element involved in oxidative stress response (Williams et al. 2017).

#### 7.10.1.3 Ecological Data

##### 7.10.1.3.1 Fate and Transport

With an estimated water solubility of 4.21 x 104 mg/L and a log $K_{OC}$ of 0.965, PGBE is predicted to have high mobility in ground water, and could pose a hazard to surface or drinking water.

The estimated vapor pressure of 0.397 mmHg indicates that PGBE will be found in the vapor phase in the atmosphere; however, the Henry's Law Constant of 4.8 x 10-8 atm-$m^3$/mol indicatives that PGBE is essentially nonvolatile from aqueous systems.

The calculated log bioconcentration factor (BCF) of 0.500, based on an estimated log $K_{OW}$ of 0.980 suggests that PGBE has a low potential for bioaccumulation in aquatic organisms and will not concentrate in the food chain.

##### 7.10.1.3.2 Ecotoxicity

PGBE appears to have a low ecotoxicity, where algae growth was inhibited 42% at 1,000 mg/L, and EPI Suite modeling predicts a 96-hr $EC_{50}$ of 524.7 mg/L (UNEP 2003). An $LC_{50}$ of >1,000 mg/L was found in *Daphnia*, while the $LC_{50}$ in the guppy was found to be between 560 and 100 mg/L.

PGBE was applied to monocotyledon and dicotyledon plants, and phytotoxicity was observed at a NOEC of 25%, indicating low toxicity (UNEP 2003).

##### 7.10.1.3.3 Degradation/Treatment

EPI Suite modeling predicts that less than 2% of PGBE will be degraded in WWTP, primarily due to sludge adsorption. PGBE is moderately biodegradable, where in one test, 60% of the chemical was removed after 28-days, as measured by $CO_2$ evolution, and in another, >90% of the chemical was removed after 28-days as measured by dissolved organic carbon (DOC) removal (UNEP 2003). The half-life of PGBE as a result of photolysis is approximately 4-hours.

### 7.10.2 Alcohols, $C_{9-11}$, Ethoxylated (CAS 68439-46-3)

#### 7.10.2.1 General Information

Ethoxylated alcohols are colorless liquids with a mild odor (Haz-Map 2018a). Synonyms include $C_{9-11}$ Pareth-3, Pareth-91-3, ($C_9$-$C_{11}$) Alkyl alcohol ethoxylate; Ethoxylated $C_{9-11}$ alcohols; Neodol 91-6. These compounds are used as emulsifiers and surfactants. Figure 12 shows the chemical structure of the component.

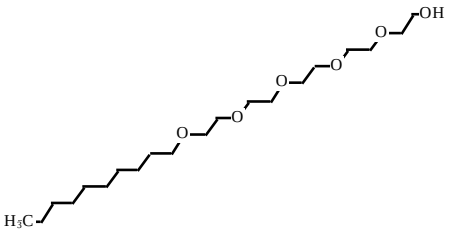


**Figure 12. Alcohols, $C_{9\text{-}11}$, Ethoxylated**

#### 7.10.2.2 Toxicology Data

Clinical signs observed during acute toxicity tests include somnolence, ataxia, and diarrhea in rat oral $LD_{50}$ studies and rabbit dermal $LD_{50}$ studies (Haz-Map 2018a).

##### 7.10.2.2.1 Oral

The acute oral $LD_{50}$ in rat ranges from 1,378 – 5,130 mg/kg. Symptoms of overexposure include ataxia, somnolence (general depressed activity), gastrointestinal hypermotility, and diarrhea (ChemIDPlus 2018).

##### 7.10.2.2.2 Inhalation

The $LC_{50}$ is >1,600 mg/m$^3$ for a 4 hour exposure in rats (ECHA 2020k). Based upon effects observed on ingestion of ethoxylated alcohols, inhalation effects are likely to include ataxia and general depressed activity.

##### 7.10.2.2.3 Dermal

The dermal $LD_{50}$ in rabbits is greater than 2,000 mg/kg (ChemIDPlus 2018).Ethoxylated alcoholscause weight loss and changes in phosphorous and potassium in 13-week intermittent dermal studies of rats (Haz-Map 2018b).

The undiluted compound is severely irritating to the skin (Gingell and Lu 1991). The threshold for irritation in rats is above 10% weight/volume (w/v) aqueous solution; at 25% w/v solution, flaking and hyperkeratosis was observed.

##### 7.10.2.2.4 Ocular

Based upon the observed dermal effects, ethoxylated alcohols are likely to be ocular irritants.

##### 7.10.2.2.5 Development and Reproduction

Potential reproductive toxicity was evaluated in a two-generation dermal toxicity study in F344 rats. The highest dose tested was equivalent to 250 mg/kg-day (ECHA 2020k). Although some sporadic changes in body and organ weights were observed, the NOAEL was established at

250 mg/kg-day. A structurally similar compound (C14-15AE7; CAS 68951-67-7) was tested in a 2-year dietary two-generation reproduction study (ECHA 2020k). The highest dose tested, 250 mg/kg-day was the NOAEL, which indicates developmental and reproductive toxicity is low.

#### 7.10.2.2.6 Genotoxicity

There was no evidence of mutagenicity in the Ames assay either with or without metabolic activation. Concentration data were not given (Gingell and Lu 1991).

#### 7.10.2.2.7 Carcinogenicity

Ethoxylated alcohols are not likely to be carcinogenic.

#### 7.10.2.2.8 Neurotoxicity

Rabbits challenged with ethoxylated alcohols exhibit neurological symptoms to include ataxia, somnolence (general depressed activity) and gastrointestinal hypermotility and diarrhea (ChemIDPlus 2018).

#### 7.10.2.2.9 Mechanism/Mode of Action

No data were found regarding the mechanism or mode of action.

### 7.10.2.3 Ecological Data

#### 7.10.2.3.1 Fate and Transport

If released to soils, ethoxylated alcohols are expected to be moderately mobile and may pose a hazard to groundwater, surface, and drinking water. Partition of ethoxylated alcohols to the atmosphere from water or wet soil is not predicted to be significant; however, vaporization from dry surfaces is likely. Any material present in the atmosphere will be present in vapor form, and subject to oxidation by ultraviolet (UV)-generated hydroxyl radicals. Ethoxylated alcohols are expected to have a low tendency to bioaccumulate based on a log $K_{ow}$ of less than 3.0.

#### 7.10.2.3.2 Ecotoxicity

Ethoxylated alcohol has been tested in numerous aquatic species. The 96-hour $LC_{50}$ is 5-7 mg/L for rainbow trout (*O. mykiss*), the 48-hour $EC_{50}$ is 2.5 mg/L for *D. magna*, and the 96-hour $EC_{50}$ is 1.4 mg/L for green algae (*S. capricornutum*) (ECHA 2020k).

#### 7.10.2.3.3 Degradation/Treatment

The U.S. EPA's EPI Suite program predicts ethoxylated alcohols will be readily degraded in the environment, with persistence of days to weeks. Air oxidation is predicted to have a half-time of only 1.46 hours.

Ethoxylated alcohols are predicted to be poorly removed (<3%) by physical waste water treatment processes.

### 7.10.3 1H-Imidazoledipropanoic Acid, 4,5-Dihydro-1-(2-Hydroxyethyl)-, 2-Norcoco Alkyl Derivs, di-Me Esters, Phosphates (Esters), Sodium Salts [IDPA Ester Salts] (CAS 95913-20-5)

#### 7.10.3.1 General Information

IDPA ester salts is a surfactant used in cleaning solutions. Synonyms include: 4,5-dihydro-1-(2-hydroxyethyl)-1H-imidazoledipropanoic acid alykyl derivs, esters, sodium salts.

#### 7.10.3.2 Toxicology Data

Few data were found on IDPA ester salts.

##### 7.10.3.2.1 Oral

A surfactant comprised of IDPA ester salts and methanol is listed as practically nontoxic for oral exposure. As methanol appears to be a minor component in the mixture, IDPA ester salts are not expected to be hazardous (Colonial Chemical 2014).

##### 7.10.3.2.2 Inhalation

No data were found.

##### 7.10.3.2.3 Dermal

No data were found.

##### 7.10.3.2.4 Ocular

No data were found.

##### 7.10.3.2.5 Development and Reproduction

No data were found.

##### 7.10.3.2.6 Genotoxicity

No data were found.

##### 7.10.3.2.7 Carcinogenicity

No data were found.

#### 7.10.3.2.8 Neurotoxicity

No data were found.

#### 7.10.3.2.9 Mechanism/Mode of Action

No data were found.

### 7.10.3.3 Ecological Data

#### 7.10.3.3.1 Fate and Transport

No data were found.

#### 7.10.3.3.2 Ecotoxicity

No data were found.

#### 7.10.3.3.3 Degradation/Treatment

No data were found.

### 7.10.4 Methanol (CAS 67-56-1)

#### 7.10.4.1 General Information

Methanol is a colorless liquid with a slightly alcoholic odor. Industrial uses include as a raw material for making formaldehyde and methyl esters of organic and inorganic acids, antifreeze for automotive radiators and air brakes, ingredient of gasoline and diesel oil antifreezes, octane booster in gasoline, and multiple other uses. Synonyms include methyl alcohol, carbinol, wood spirit, and wood alcohol (O'Neil 2006). Figure 13 shows the chemical structure of the component.

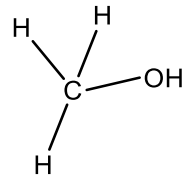


**Figure 13. Methanol**

### 7.10.4.2 Toxicology Data

Potential symptoms of overexposure are irritation of the eyes, skin, and upper respiratory system; dermatitis; headache, drowsiness, dizziness, vertigo, light headedness; nausea, vomiting, anorexia; weakness, fatigue; abdominal, back and leg pain; visual disturbances, dimness of vision, dilated pupils; optic nerve damage, and bilateral blindness (O'Neil 2006).

Methanol occurs naturally in humans, animals, and plants. Methanol is metabolized primarily in the liver by sequential oxidation to formaldehyde and then formate or formic acid. Conversion to $CO_2$ completes the detoxification of methanol. The acute and short-term toxicity of methanol varies greatly among different species, toxicity being highest in species with a relatively poor ability to metabolize formate. Poor metabolism of formate results in metabolic acidosis and neuronal toxicity, whereas in animals that readily metabolize formate consequences of CNS depression (coma, respiratory failure, etc.) are usually the cause of death (NCBI 2020b).

#### 7.10.4.2.1 Oral

The minimal lethal dose of methanol in humans has not been determined; ingestion of about 1 g/kg can cause death if the individual is untreated and has not consumed ethanol (NCBI 2020b). Oral toxicity in humans most often results from drinking of adulterated or denatured alcoholic beverages.

The oral $LD_{50}$ in rats is reported to be 5,628 mg/kg, 7,300 mg/kg in mice, 8,000 mg/kg in dogs, 14,400 mg/kg in rabbits and 2-3 g/kg in monkeys (NCBI 2020b).

#### 7.10.4.2.2 Inhalation

The 4-hour inhalation $LC_{50}$ in rats is reported to be 64,000 ppm, and 87.5 mg/L for a 6-hour exposure. In cats, the $LC_{50}$ is 85.41 mg/L for a 4.5-hour exposure and 43.68 mg/L for a 6-hour exposure (NCBI 2020b).

Conversion factor: 1 mg/L = 764 ppm = 1.31 $mg/m^3$ (NCBI 2020b).

#### 7.10.4.2.3 Dermal

Methanol is a dermal irritant (NCBI 2020b).

The dermal $LD_{50}$ in rabbits is reported to be approximately 15,800 mg/kg body weight (NCBI 2020b).

#### 7.10.4.2.4 Ocular

Methanol is an ocular irritant (NCBI 2020b).

The most noted health consequences of longer term exposure to lower levels of methanol is a broad range of ocular effects (NCBI 2020b).

#### 7.10.4.2.5 Development and Reproduction

Methanol is teratogenic in rats at high concentrations (NCBI 2020b).

Hansen et al. (2005) used rat and mouse whole embryo cultures to distinguish the toxicity of methanol and its metabolites. Mouse embryos were more sensitive, but for both mice and rats, embryonic viability, dysmorphogenesis (increased), and growth parameters (decreased) were affected in a dose-dependent manner. In both rats and mice, neural tube closure was delayed, along with incomplete axial rotation, decreased rate of growth, blood pooling in the head and visceral yolk sac and accumulation of necrotic matter in the amnion. In the CD-1 dams, reduced body weights and transient neurological signs were noted in 20% of the animals exposed up to 19,500 mg/m$^3$ methanol during gestational days 6–15. Additionally, increased resorptions, reduced fetal weights and increased fetal malformations were noted at 13,000 and 19,000 mg/m$^3$. Other malformations included neural and ocular defects, cleft palate, hydronephrosis and deformed tails and limb anomalies, dependent upon when in gestation exposure occurred (Hansen et al. 2005).

#### 7.10.4.2.6 Genotoxicity

In the Ames bacterial reversion assay for mutagenicity, methanol was not mutagenic to *Salmonella* strains TA97, TA98, TA1535, TA 1537, and TA1538 with or without metabolic activation (NCBI 2020b). Methanol was equivocal *Salmonella* strain TA102 in the presence of metabolic activation. Methanol was not mutagenic in a DNA-repair test using various strains of *E. coli WP2* and in a forward mutation assay using *Schizosaccharomyces pombe*.

The micronucleus assay was negative in mice exposed by inhalation to 800 or 4,000 ppm of methanol for 5 days (NCBI 2020b). Additionally, no sister chromatid exchanges or chromosomal aberrations were found in lung cells; or synaptosomal complex damage in spermatocytes.

#### 7.10.4.2.7 Carcinogenicity

Methanol is not considered to be carcinogenic to humans (NCBI 2020b).

#### 7.10.4.2.8 Neurotoxicity

Long-Evans-hooded rats were exposed to 0 or 4,500 ppm methanol for 6hours/day starting at gestational day 6 up to postnatal day 21 (NCBI 2020b). Offspring did not display an effect in suckling or an odor aversion test following methanol exposure; however, a transient decrease in motor activity was noted at post-natal day 18, which increased by day 25. Offspring were tested in two operant conditioning paradigms: a running wheel test and a lever press test (stochastic spatial discrimination learning test). Adult males had decreased rate of running compared to baseline; females had increased rates of running. Performance was impaired in the lever test in both males and females in the exposed groups.

#### 7.10.4.2.9 Mechanism/Mode of Action

Upon ingestion, methanol is metabolized along the same pathway as alcohol, by alcohol and aldehyde dehydrogenase in the liver (PubChem 2020s). Metabolism of methanol by these enzymes results in the formation of formaldehyde and formic acid, which can lead to toxic effects in the central nervous system, eyes, and gastrointestinal tract. Detoxification is either through inhibition of the aldehyde dehydrogenase by introducing competing ethanol or via folate which can detoxify formic acid.

### 7.10.4.3 Ecological Data

#### 7.10.4.3.1 Fate and Transport

Based upon high aqueous solubility and low $K_{oc}$, methanol is expected to have high mobility in groundwater if released to soil. Volatilization from either wet or dry soil surfaces is expected to be significant based upon the Henry's Law constant and vapor pressure. Methanol is expected to exist in the atmosphere solely as a vapor. Bioaccumulation of methanol is not expected, and metabolism by a number of organisms is likely (NCBI 2020b).

#### 7.10.4.3.2 Ecotoxicity

Kaviraj et al. (2004) evaluated the impact of methanol on fish and aquatic invertebrates. The $LC_{50}$ for a 96-hour exposure in the crustacean *Moina micrura* was 15.32 g/L and was the most sensitive of the species evaluated. Fish exposed to lethal concentrations of methanol showed impaired respiration and swimming, as well as feeding rate and growth and reproduction (up to 1,527 mg/L). The 90-day exposures resulted in growth reduction, a negative effect on the maturity index and fecundity of fish at treatments greater than or equal to 47.49 mg/L. A NOEC of 23.75 mg/L was derived for freshwater aquatic ecosystems (Kaviraj et al. 2004). The 48-hour exposures in green algae resulted in an $EC_{50}$ of 3 mg/L for physiology and photosynthesis and greater than 60.4 mg/L for population growth rate. The 24-hour $EC_{50}$ in *Daphnia* was reported to be >10,000 mg/L. The fathead minnow had an $LC_{50}$ of 28,100 mg/L (NCBI 2020b).

#### 7.10.4.3.3 Degradation/Treatment

Biodegradation is expected to be a significant fate process for methanol, but hydrolysis and direct photodegradation are not expected to be important fate processes. Degradation by photolytically produced hydroxide radicals is relatively rapid, with a half-life of 13 days (NCBI 2020b).

## 7.11 Chemsol Wipes

### 7.11.1 Orange Terpenes (CAS 5989-27-5)

#### 7.11.1.1 General Information

Orange terpenes is a clear, colorless, oily liquid with a lemon-like odor. It can be used as a dietary supplement and is a major component of citrus peel extracts. It has been postulated to

have chemopreventative and antitumor activities. Additionally, it has been utilized as a solvent of cholesterol to dissolve cholesterol-containing gallstones and can be used to relieve heartburn and gallstones (Pubchem). It is used a topical insecticide on tablecloths or insect-repellent strips in food- or feed-handling establishments. Synonyms include Orange terpenes, (+)-Limonene, (R)-(+)-Limonene, (+)-carvene, Citrene and several others. Figure 14 shows the chemical structure of the component.

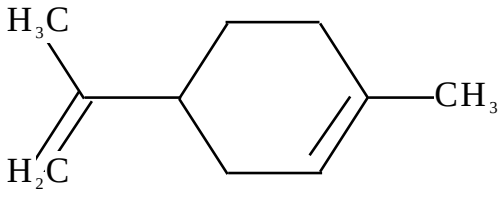


**Figure 14. Orange Terpenes**

#### 7.11.1.2 Toxicology Data

Direct infusion of orange terpenes into the bile system causes pain in the upper abdomen, nausea, vomiting, and diarrhea. Additional symptoms of exposure include irritation of the skin and eyes, skin sensitization, dizziness, rapid and shallow breathing, and tachycardia.

##### 7.11.1.2.1 Oral

The mouse oral $LD_{50}$ ranged from 5.6-6.6 g/kg, in the rat it is 5 g/kg (PubChem 2020k). Oral exposure in rats can result in nephrotoxicity and ultimately resulting in renal tumor formation. This effect is only noted in male rats and requires the presence of alpha-2u-globulin and is an effect only found in rats, thus is not a concern for human exposures (EPA 1991). Long-term exposure in other species can result in decreased bodyweight gain and altered kidney weights. A LOAEL (6 month) was determined to be 1,000 mg/kg-body weight (bw) in dogs (ECHA 2020j).

##### 7.11.1.2.2 Inhalation

Respiratory depression was noted at 570 $mg/m^3$ for 1 month in mice (PubChem 2020k).

##### 7.11.1.2.3 Dermal

Dermal exposure results in redness and pain (PubChem 2020k). The dermal $LD_{50}$ is greater than 5 g/kg for a 24-hour application. Patch exposures in human resulted in positive reactions for 2% of subjects tested, which is equivalent to other common sensitizers (PubChem 2020k). Orange terpenes has also tested positive for skin sensitization in the local lymph node assay (LLNA) (ECHA 2020j).

#### 7.11.1.2.4 Ocular

Ocular exposure to orange terpenes results in redness (PubChem 2020k).

#### 7.11.1.2.5 Development and Reproduction

In pregnant rats administered orange terpenes between days 7 and 15 of gestation, neonates showed reduced weight gain and musculoskeletal defects at doses up to 2800 mg/kg (PubChem 2020k). In a rabbit reproductive study, no effect was seen in fetuses as low as 1,000 mg/kg-d and at 250 mg/kg-d in dams for decreased weight gain.

#### 7.11.1.2.6 Genotoxicity

Orange terpenes is not mutagenic in the Ames assay in TA98, TA100, TA1535, and TA1537 both with and without S9 liver fraction (PubChem 2020k). It also did not induce chromosomal aberrations or sister chromatid exchanges in CHO cells.

#### 7.11.1.2.7 Carcinogenicity

According to IARC, there is inadequate evidence in humans for carcinogenicity of orange terpenes; therefore, it is not classifiable (group 3) (IARC 1993). In laboratory animals, there is sufficient evidence of carcinogenicity due to the appearance of renal tumors in male rats.

#### 7.11.1.2.8 Neurotoxicity

No data were found.

#### 7.11.1.2.9 Mechanism/Mode of Action

Orange terpenes may exert cell growth inhibition by a p21-dependent signaling pathway and may induce apoptosis via the TGF-β signaling pathway (PubChem 2020k). It is via these mechanisms that its antitumor effects are suspected to operate. It is not well-absorbed across the dermal layer and is quickly excreted by the body primarily in urine (80% in rats), with the remainder in the feces. Orange terpenes binds alpha-2u-globulin in the male rat kidney, resulting in the formation of hyaline droplets and nephrotoxicity, eventually leading to renal tumor formation.

### 7.11.1.3 Ecological Data

#### 7.11.1.3.1 Fate and Transport

Orange terpenes is not likely to transport in groundwater due to its low solubility and high log $K_{OC}$; however, it is likely to adsorb to soil. It is anticipated to exist as vapor is expected to be readily volatile. Volatilization from a river is expected in a matter of hours and approximately 4.5 days from a lake. It is not expected to bioaccumulate.

#### 7.11.1.3.2 Ecotoxicity

ECOSAR predicted that for green algae, *Daphnia*, and fish, the $EC_{50}/LC_{50}$ would be <0.6 mg/L. This is reflected in experimental toxicities of 0.702 mg/L in the fathead minnow, 0.577 mg/L in *Daphnia*, and <1.5 mg/L in green algae (PubChem 2020k). The $LC_{50}$ in the earthworm is 60 ppm, with a NOEL of 42.1 ppm (EPA 2019e).

#### 7.11.1.3.3 Degradation/Treatment

Orange terpenes is not expected to be overly persistent in the environment. It is expected to biodegrade in soil, with 74% removal by 28 days (ECHA 2020j). EPI Suite predicted a half-life of approximately 2 weeks. It will rapidly degrade in the air. Sewage treatment is also effective, with 94% removal by WWTP, 31.45% to sludge and 62.83% to air.

### 7.11.2 Alcohols, C12-C15, Ethoxylated (CAS 68131-39-5)

#### 7.11.2.1 General Information

Ethoxylated alcohols is a variable mixture that exists as a hazy liquid with a mild odor. They are permitted for use as an inert ingredient in nonfood pesticide products. Uses include as a detergent or surfactant in laundry detergents, surface cleaners, cosmetics, pesticides, textiles and paint. They are commercially marketed under a variety of names including Pareth, Dobanol, Neodol, Serdox, Slovasol, Tergitol and others (ECHA 2020l). Figure 15 shows the chemical structure of the component.

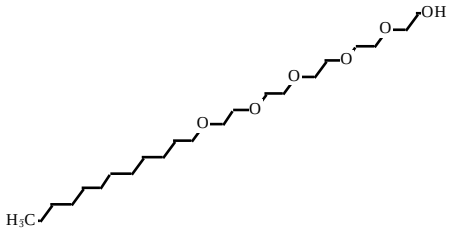


**Figure 15. Alcohols, C12-C15, Ethoxylated**

#### 7.11.2.2 Toxicology Data

#### 7.11.2.2.1 Oral

The acute oral $LD_{50}$ has been reported as greater than 5,000 mg/kg (ECHA 2020l). Ethoxylated alcohols cause ataxia in oral lethal dose studies (Gingell and Lu 1991). This level of toxicity corresponds to GHS Category 5 or uncategorized.

#### 7.11.2.2.2 Inhalation

No adverse effects due to inhalation are expected; however, overexposure may result in ataxia (Gingell and Lu 1991).

#### 7.11.2.2.3 Dermal

Ethoxylated alcohols are not a dermal irritant or a skin sensitizer (ECHA 2020l).

#### 7.11.2.2.4 Ocular

Ethoxylated alcohols are not an eye irritant (ECHA 2020l).

#### 7.11.2.2.5 Development and Reproduction

Ethoxylated alcohols are not expected to cause developmental or reproductive effects (ECHA 2020l).

An alcohol ethoxylate (AE) and alcohol ethoxy sulfate (AES) demonstrated both teratogenic and toxic effects in *Xenopus laevis* embryos and tadpoles. The AE compound produced greater effects than the AES compound. (Cardellini and Ometto 2001).

#### 7.11.2.2.6 Genotoxicity

Ethoxylated alcohols were negative in the *in vitro* mammalian chromosome aberration test and the *in vivo* erythrocyte micronucleus test (ECHA 2020l).

#### 7.11.2.2.7 Carcinogenicity

Ethoxylated alcohol is not expected to be a carcinogen (ECHA 2020l).

#### 7.11.2.2.8 Neurotoxicity

Ataxia is a reported symptom of exposure (Gingell and Lu 1991).

#### 7.11.2.2.9 Mechanism/Mode of Action

No data were found.

### 7.11.2.3 Ecological Data

#### 7.11.2.3.1 Fate and Transport

Due to high aqueous solubility, ethoxylated alcohols are expected to be readily transported in groundwater, and may pose a hazard to surface and drinking water. Partition between water and wet surfaces is a low possibility due to the high aqueous solubility of these compounds,

while vaporization from a dry surface is possible, but likely slow due to their chemical nature. There is a moderate hazard of bioaccumulation in aquatic species.

#### 7.11.2.3.2 Ecotoxicity

Aquatic endpoints reported include a 72-hour $EC_{50}$ in algae of 0.75 mg/L, a 48-hour $EC_{50}$ in *Daphnia* of 0.14 mg/L, and a 96-hour $LC_{50}$ in fish of 1.3 mg/L (ECHA 2020l).

The 72-hour $LC_{50}$ for AE compound was 4.59 mg/L and 6.75 mg/L for the AES. Epithelial tissue, particularly in the gills, were the most sensitive tissues; ultrastructural evaluation indicated alterations to the mitochondria while evaluation of oxygen consumption indicated collapse of the electrochemical gradient in mitochondria (Cardellini and Ometto 2001).

#### 7.11.2.3.3 Degradation/Treatment

Ethoxylated alcohols are readily biodegradeable within 28 days (ECHA 2020l).

### 7.11.3 Isoparaffinic Hydrocarbon/Distillates Petroleum, Hydrotreated, Light (CAS 64742-47-8)

#### 7.11.3.1 General Information

Hydrotreated light petroleum distillates are obtained by treated fractionated petroleum with hydrogen in the presence of a catalyst (Distillates (petroleum), hydrotreated light. In general, these distillates are complex mixtures with carbon chains between 6 and 16. The distillation range depends on the distribution of the carbon chain lengths and complexity of the mixture (aliphatic and cyclic). Factors such as the source of the crude oil and the specific refinery can contribute to the composition of the distillates. Synonyms include naphtha (petroleum) hydrotreated, light; hydrocarbons, C11-C12 isoalkanes, <2% aromatics (ExxonMobil 2002); distillates (petroleum), hydrotreated light, isoparafinnic hydrocarbon and Hydrotreated light distillates (petroleum), and several commercial names. The product is flammable and potentially explosive in air.

Figure 16 shows the chemical structure of the component.

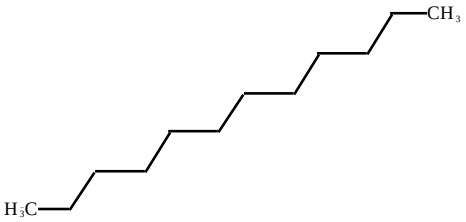


**Figure 16. Isoparaffinic Hydrocarbon/Distillates Petroleum, Hydrotreated, Light**

### 7.11.3.2 Toxicology Data

Toxicological data for hydrocarbon distillates (C8-C16) are available; however, not every possible product has toxicity data. For most endpoints, there are data for a selection of compounds, such as kerosene, jet fuels, and other distillates. Reviews of the data by regulatory agencies have resulted in the use of read-across analysis for estimating toxicity of untested distillates based on structural and compositional similarity to products with toxicity data. The product used here is described as hydrocarbons C10-C13, N-alkanes, isoalkanes, cyclics and <2% aromatics. This product (CAS RN 64742-47-8) is within the size range for read across and data for this product may include data from other similar distillates. Hydrocarbon solvents and distillates can produce acute, reversible CNS depression at high exposure levels, chemical pneumonitis if aspirated, and irritation to the skin, eyes, and respiratory tract. Animal data in general suggest that hydrocarbon solvents produce only acute effects and normally only at relatively high levels of exposure. There are claims that exposure under occupational conditions has produced chronic neurological effects and renal disease (Hotz 1994; Lauwerys et al. 1985). Due to the chemical complexity of distillates, the evidence for chronic toxicity from occupational exposure is controversial.

#### 7.11.3.2.1 Oral

The reported acute oral $LD_{50}$ in rats is greater than 5,000 mg/kg (860 ppm). 5,000 mg/kg was the limit dose and no animals died; sedation, dyspnea, ruffled fur, and hunched posture were observed but resolved by day 5 (the last day of the study) (ECHA 2020q). Aspiration after ingestion may be fatal (GHS Category 1 aspiration hazard) (ExxonMobil 2002).

#### 7.11.3.2.2 Inhalation

Overexposure to fumes may cause headaches, dizziness, anesthesia, drowsiness, unconsciousness, and other CNS effects, including death. Small amounts of liquid aspirated into the lungs during ingestion or from vomiting may cause chemical pneumonitis or pulmonary edema (ExxonMobil 2002).

#### 7.11.3.2.3 Dermal

The dermal $LD_{50}$ is greater than 3,160 mg/kg; hydrocarbons are mildly irritating to skin. Prolonged or repeated exposure may cause drying of the skin (ExxonMobil 2002).

#### 7.11.3.2.4 Ocular

Exposure may cause mild, short-lasting discomfort to eyes (ExxonMobil 2002).

#### 7.11.3.2.5 Development and Reproduction

Based on testing of similar distillates, this mixture is not expected to be a reproductive toxicant (ExxonMobil 2002).

#### 7.11.3.2.6 Genotoxicity

Based on testing of similar distillates, this mixture is not expected to be genotoxic (ExxonMobil 2002).

#### 7.11.3.2.7 Carcinogenicity

C10-C13 hydrocarbons are not held to be carcinogenic by OSHA, IARC, NTP, or ACGIH (ExxonMobil 2002; MilliporeSigma 2018).

#### 7.11.3.2.8 Neurotoxicity

Inhalation of high concentrations may cause headaches, dizziness, anesthesia, drowsiness, unconsciousness, and other CNS effects, including death (Haz-Map 2018b).

#### 7.11.3.2.9 Mechanism/Mode of Action

No data were found.

### 7.11.3.3 Ecological Data

#### 7.11.3.3.1 Fate and Transport

Light distillates are slightly soluble in water and are expected to volatilize rapidly from rivers (half-life 1.3 hours) and lakes (half-life 5.2 days) (EPA 2015b). In soil, light distillates will bind to soil (and sediment), and in air, light distillates will exist in the vapor phase.

#### 7.11.3.3.2 Ecotoxicity

In rainbow trout (*O. mykiss*) the 24-, 48-, and 96-hour $LC_{50}$ were 3.2, 3.05, and 2.6 mg/L, respectively, and in bluegill (*Lepomis macrochirus*) the 4-day $LC_{50}$ was 5.9 mg/L (EPA 2019e).

#### 7.11.3.3.3 Degradation/Treatment

Atmospheric degradation due to action of photochemically produced hydroxyl radicals is expected to be rapid (ECHA 2020q). Light distillates are expected to biodegrade readily; half-life is 11.8 days (EPA 2015a). In WWTPs, 99.94% of light distillates will be removed–59% by

sludge absorption and 40.76% to the atmosphere. The biodegradation of light distillates in sediments is poor with a half-life of 3–4 months.

### 7.11.4 Dimethyl Adipate [DMA] (CAS 627-93-0)

#### 7.11.4.1 General Information

Dimethyl adipate (DMA) is a slightly soluble solid with a mild nutty taste. It is used as a solvent, flavoring agent or plasticizer. It has been designated as an EPA Safer Choice based on experimental and modeled data. Synonyms for this compound are dimethyl hexanedioate, dimethyladipate, adipic acid dimethyl ester, 1,6-dimethyl hexanedioate and more. Figure 17 shows the chemical structure of the component.

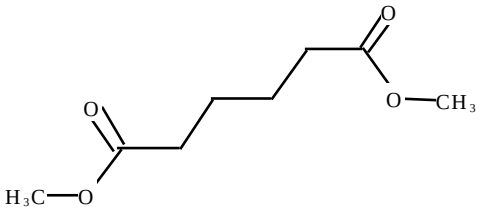


**Figure 17. Dimethyl Adipate**

#### 7.11.4.2 Toxicology Data

##### 7.11.4.2.1 Oral

The rat intraperitoneal $LD_{50}$ is 1.9 mg/kg (PubChem 2020n). The oral NOEL is 980 mg/kg-day and $LD_{50}$ is >5000 mg/kg in rats (ECHA 2020o).

##### 7.11.4.2.2 Inhalation

Dibasic esters can cause degeneration of the rat olfactory epithelium following a 90-day inhalation study (Trela et al. 1992). No effect on respiratory epithelium was noted. The effect is mitigated *in vitro* by pretreatment with carboxylesterase inhibitors, suggesting a carboxylesterase-mediated cytotoxic mechanism. In a reproductive study in rats, weight loss was observed at the highest dose (1 mg/L as an aerosol) in the parental generation, with some squamous metaplasia noted in the olfactory epithelium (Kelly et al. 1998). The $LC_{50}$ is >11 mg/L, with a subchronic NOEC of 50 mg/m$^3$ (ECHA 2020o).

##### 7.11.4.2.3 Dermal

The dermal $LD_{50}$ for DMA is >1,000 mg/kg as assayed with the rabbit (ECHA 2020o). No dermal irritation was noted in the acute study or in other studies. According to the LLNA and the guinea pig maximization test; DMA is not a sensitizer.

#### 7.11.4.2.4 Ocular

DMA may be an ocular irritant, but it does resolve upon removal of the chemical (ECHA 2020o).

#### 7.11.4.2.5 Development and Reproduction

No effects on reproduction were noted in a rat inhalation study where subjects were exposed to dibasic esters for a 14-week premating period, and for 8 weeks through breeding, gestation, and lactation (Kelly et al. 1998).

#### 7.11.4.2.6 Genotoxicity

No genotoxicity is expected. DMA was negative in the Ames assay with and without metabolic activation, and is presumed negative in the chromosomal aberration assay (ECHA 2020o). There was an increase in abnormal cells in the S9-treated assay; however, that was attributed to a change in pH due to the metabolism of the dibasic ester blend decreasing pH. The mouse micronucleus assay was also negative.

#### 7.11.4.2.7 Carcinogenicity

DMA is not expected to be a carcinogen.

#### 7.11.4.2.8 Neurotoxicity

No data were found.

#### 7.11.4.2.9 Mechanism/Mode of Action

Treatment of rat nasal explants with DMA results in microscopic and ultrastructural change that are mitigated by pretreatment with carboxylesterase inhibitors (Trela et al. 1992). This suggests that some effects of DMA are mediated by carboxylesterase. In the EPA's toxicity forecaster ToxCast, it was active as an agonist to Nrf2 binding, so may also be involved in cell cycle and redox regulation (Williams et al. 2017).

### 7.11.4.3 Ecological Data

#### 7.11.4.3.1 Fate and Transport

DMA is highly soluble and is not expected to adsorb onto soil, thus it is expected to readily transport in ground water. It will exist in the vapor phase in air. It is not expected to readily volatilize from wet soil or surfaces, with a half-life in river water of 13.9 days and lake water of 157 days. It is not expected to bioaccumulate.

#### 7.11.4.3.2 Ecotoxicity

The $EC_{50}$ in *Daphnia* is 72 mg/L and >85 mg/L in algae. In carp, an $EC_{50}$ between 89–122 mg/kg was described (ECOTOX 2009). In fathead minnow, the $EC_{50}$ was 24 mg/L.

#### 7.11.4.3.3 Degradation/Treatment

DMA is moderately persistent, with a biodegradation half-life of days to weeks. It will be slow to photodegrade, with a half-life for atmospheric oxidation of 2.69 days. Treatment at WWTPs will not be overly effective, with a total removal of 2.02%.

### 7.11.5 Diethylhexyl Sodium Sulfosuccinate [DOSS] (CAS 577-11-7)

#### 7.11.5.1 General Information

Diethylhexyl sodium sulfosuccinate (DOSS) is an odorless, colorless to white waxy solid. DOSS is the sodium salt of docusate, which is used as a laxative to soften stool and as a surfactant or emulsifying agent in adhesives, sealants, anti-adhesives, paints, pigments, etc. Synonyms include docusate sodium, dioctyl sodium sulfosuccinate, dioctyl sulfosuccinate sodium salt, Aerosol OT, Constonate, and others. Figure 18 shows the chemical structure of the component.

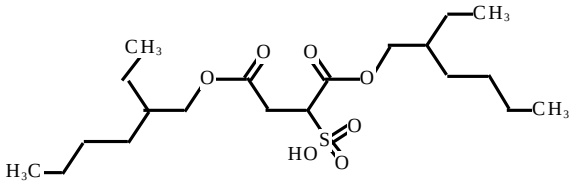


**Figure 18. Diethylhexyl Sodium Sulfosuccinate**

#### 7.11.5.2 Toxicology Data

The primary route of exposure for DOSS is oral, with limited uptake via inhalation or dermal routes (ECHA 2020p). It is not expected to accumulate, and it is metabolized via hydrolysis of the ester group. It is eliminated primarily through urine, with some excretion via bile.

#### 7.11.5.2.1 Oral

Ingestion of DOSS causes diarrhea and intestinal bloating, the properties of which are used pharmaceutically to help soften stool and as a laxative. The rat oral $LD_{50}$ is 1,900 mg/kg, with a mouse oral $LD_{50}$ of 2,600 mg/kg (PubChem 2020q). DOSS may also enhance absorption of other orally administered drugs. No negative effects were seen in a 1-year dog study (Case et al. 1977). The NOAEL in rats is 1,000 mg/kg-day derived from a subchronic study (ECHA 2020p). A NOAEL of 500 mg/kg-day for toxicity was described after a 2-year rat bioassay.

#### 7.11.5.2.2 Inhalation

The rat $LC_{50}$ is 20 mg/L (20,000 mg/m$^3$) DOSS (ECHA 2020p).

#### 7.11.5.2.3 Dermal

DOSS may irritate skin due to its surfactant properties; DOSS as a component of cosmetic formulations is not considered an irritant or sensitizer in isolation, but it is considered a cumulative irritant (Andersen 1998). However, ECHA lists DOSS as an irritant following an application of a 70% DOSS solution to rabbits (ECHA 2020p). It is not a sensitizer according to a modified Draize test. The dermal $LD_{50}$ in rabbits is > 10,000 mg/kg-bw.

#### 7.11.5.2.4 Ocular

DOSS is a strong ocular irritant, with a 10% formulation resulting in severe eye damage (PubChem 2020q; ECHA 2020p). Formulations containing 0.5% and 2% DOSS cause mild, reversible eye damage in the rabbit.

#### 7.11.5.2.5 Development and Reproduction

Male offspring of mice administered DOSS during pregnancy showed significantly increased body mass, overall and visceral fat masses, and decreased bone area (Temkin et al. 2019). Plasma adiponectin was decreased, with increased leptin, glucose intolerance, and hyperinsulinemia. In a three-generation reproductive study, males fed 1% DOSS during the premating period were lower than controls in all three generations, while in the F1 and F2 generations, both males and females had lower bodyweights in the 0.5% dose group (MacKenzie et al. 1990). Pup weights were also affected in the mid- and high-dose groups; however, no effect on reproduction was evident.

#### 7.11.5.2.6 Genotoxicity

The Ames test was negative both with and without metabolic activation up to concentrations close to the toxic range, with a top dose of 2,500 µg/mL(ECHA 2020p). Similarly, the rat *in vivo* micronucleus assay was negative at doses up to 2,000 mg/kg-day.

#### 7.11.5.2.7 Carcinogenicity

A standard 2-year rat cancer bioassay and a colorectal carcinogenesis rat model assay were both negative for increase tumor incidence following DOSS treatment (ECHA 2020p). The NOAEL for toxicity for these studies was 0.5%, or 250 mg/kg-day, and for tumorigenicity was 500 mg/kg-day (1%).

#### 7.11.5.2.8 Neurotoxicity

Acute whole-body inhalation exposure of male rats to a commercial product comprised primarily of DOSS resulted in partial loss of olfactory signal transduction, and altered axonal function, and synaptic vesicle fusion (Sriram et al. 2011).

#### 7.11.5.2.9 Mechanism/Mode of Action

In peroxisome proliferator-activated receptor gamma (PPARγ) transactivation assays, application of DOSS to the cells resulted in PPAR response element binding as noted by luciferase induction as a component of the assay (Temkin et al. 2016). This could indicate that DOSS is an obesogen. DOSS also increased reactive oxygen species (ROS), glutathione peroxidase, glutathione-s-transferase levels, and lipid peroxidation in sheepshead minnow larvae, indicating a potential to affect antioxidant response capabilities (Dasgupta et al. 2018).

### 7.11.5.3 Ecological Data

#### 7.11.5.3.1 Fate and Transport

DOSS is not expected to transport in groundwater due to low solubility and moderate likelihood to adsorb to soil. In the atmosphere, it will exist as a particulate. It is also nonvolatile, and will not readily volatilize from moist surfaces or water. It also is not expected to bioaccumulate.

#### 7.11.5.3.2 Ecotoxicity

In the acute aquatic toxicity (Microtox™) assay, an $EC_{50}$ value of 43–75 mg/L was found, additionally, it inhibited algae growth in this same concentration range (Rosal et al. 2010). The NOEC for algae is 22 mg/L (ECHA 2020p). Similarly, in rainbow trout, coho, chinook, and chum, the $LD_{50}$ ranged from 35.3 to 59.8 mg/L for the parent product that is comprised primarily of DOSS (MacInnis et al. 2018). In *Daphnia*, the $LC_{50}$ was determined to be 15.2 mg/L (ECHA 2020p). The NOEC for freshwater invertebrates is 9 mg/L.

#### 7.11.5.3.3 Degradation/Treatment

Biodegradation is expected to take days to weeks, and it will readily photodegrade in the air, with a rapid atmospheric oxidation rate and half-life of 0.5 days. Removal from WWTPs will be moderately successful (28%) due to the likelihood that DOSS will adsorb to sludge (27%).

## 7.12 Eastman Omnia Solvent

### 7.12.1 Butyl-3-hydroxybutyrate [BHB] (CAS 53605-94-0)

#### 7.12.1.1 General Information

Butan-1-yl-3-hydroxybutanoate (BHB) is a solvent used in industrial settings and in cosmetic applications such as nail polish removers. Synonyms include Butyl 3-hydroxybutanoate, Butyl 3-

hydroxybutyrate, Butanoic acid, 3-hydroxy-, butyl ester, and more. Figure 19 shows the chemical structure of the component.

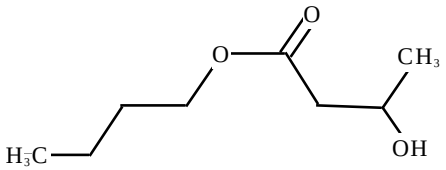


**Figure 19. Butyl-3-hydroxybutyrate**

#### 7.12.1.2 Toxicology Data

##### 7.12.1.2.1 Oral

The rat oral $LD_{50}$ was greater than 5,000 mg/kg, with a 4 week exposure NOAEL of 1,000 mg/kg-day, the critical effect being a transient failure to gain weight in males (ECHA 2020m).

##### 7.12.1.2.2 Inhalation

The rat $LC_{50}$ is greater than 5.11 mg/L (ECHA 2020m).

##### 7.12.1.2.3 Dermal

The dermal $LD_{50}$ in the rat is greater than 5,000 mg/kg, it was slightly irritating in the Draize rabbit assay, and was not a sensitizer in the LLNA (ECHA 2020m).

##### 7.12.1.2.4 Ocular

BHB is a moderate to several eye irritant (ECHA 2020m; PubChem 2020j).

##### 7.12.1.2.5 Development and Reproduction

BHB does not affect reproduction or development (ECHA 2020m).

##### 7.12.1.2.6 Genotoxicity

BHB was negative in the Ames assay with and without S9 activation (ECHA 2020m).

##### 7.12.1.2.7 Carcinogenicity

No data were found.

#### 7.12.1.2.8 Neurotoxicity

No data were found.

#### 7.12.1.2.9 Mechanism/Mode of Action

No data were found.

### 7.12.1.3 Ecological Data

#### 7.12.1.3.1 Fate and Transport

BHB is highly soluble and with a $K_{oc}$ of 1.16, it is not expected to readily adsorb to soils, so may be transported via ground water. With a vapor pressure of 0.015 mmHg, it will exist in the vapor phase in the atmosphere but is readily oxidized so will not be persistent. It will not volatilize from wet soil or water well, with a predicted half-life in model rivers of 2,573 days, and in lakes 28,000 days. It is not expected to bioaccumulate.

#### 7.12.1.3.2 Ecotoxicity

In the rainbow trout (*O. mykiss*) and *Daphnia* acute assays, the $LC_{50}$ was 100 mg/L (the limit of the test) for both assays (ECHA 2020m).

#### 7.12.1.3.3 Degradation/Treatment

Overall persistence of BHB is low, taking 3.4 days to biodegrade. Sewage treatment will not be effective removal, with a total expected removal of 1.93%, 1.83% of that to sludge.

## 7.12.2 Proprietary Chemical

No information on this compound was provided.

## 7.13 Ecolink 250-SS

This product contains decamethylcyclopentasiloxane, octamethylcyclotetrasiloxane, and dipropylene glycol dimethyl ether.

## 7.13.1 Decamethylcyclopentasiloxane [D5] (CAS 541-02-6)

### 7.13.1.1 General Information

D5 is a colorless liquid used in consumer products, cosmetics and to make silicone materials, inclusive of breast implants. Alternative nomenclature for decamethylcyclopentasiloxane includes cyclic dimethylsiloxane pentamer, cyclopentasiloxane, and dimethylsiloxane pentamer. Figure 20 shows the chemical structure of the component.

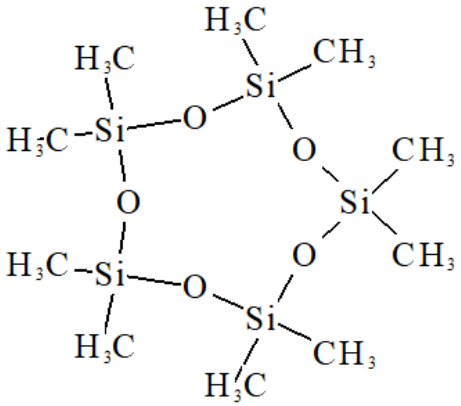

**Figure 20. Decamethylcyclopentasiloxane**

#### 7.13.1.2 Toxicology Data

##### 7.13.1.2.1 Oral

The acute oral $LD_{50}$ in rats has been reported as greater than 5,000 mg/kg (ECHA 2020n).

##### 7.13.1.2.2 Inhalation

The acute inhalation $LC_{50}$ is reported as 8,600 mg/m$^3$ in rat (ECHA 2020n). Female rats (F344) were exposed (whole body) to 160 ppm D5 for 6 h/day, 7 days/week for 28 days (PubChem 2020l). The endpoints included alterations in the inducible liver enzyme profile. Rat liver size was increased by 16% and a shift in enzyme activities were observed indicating that D5 is a weak "phenobarbital-like" inducer.

In a separate study, male and female F344 were exposed nose only to 26, 46, 86, and 244 ppm for 3 months (6 h/day, 5 days/week). A 4-week recovery group was included, as well. Exposure related liver and lung weights and changes in clinical chemistry parameters were observed. These changes did not resolve in the recovery groups; however, histopathological evaluation did not find concordant changes at the cellular level in the lungs or livers.

##### 7.13.1.2.3 Dermal

The acute dermal $LD_{50}$ is reported as greater than 2,000 mg/kg (>16mL/kg) in rabbit (ECHA 2020n). The dermal NOAEL is 960–1,000 mg/kg-day in rabbits (EPA 2019b). *In vitro* and *in vivo* tests indicated D5 is not dermally absorbed (2017). It is not a dermal irritant or a sensitizer (ECHA 2020n).

##### 7.13.1.2.4 Ocular

D5 was reported as minimal or negative in tests for eye irritation potential (2017).

#### 7.13.1.2.5 Development and Reproduction

Low molecular weight organosiloxane compounds, including D5, administered orally (0.01–100 mg/kg/d) to ovariectomized female rats increased uterine weight, produced uterine hyperemia, and changed uterine morphology (NCBI 2020a).

In a two-generation study, rats were exposed whole-body vapor inhalation to 30, 70, or 160 ppm, 6 h/day. Exposures began 70 days prior to mating and continued through postnatal day 21 in the offspring and parents (NCBI 2020a). Negligible reproductive or developmental effects were observed and the NOAEL was determined to be 160 ppm (the highest dose tested).

#### 7.13.1.2.6 Genotoxicity

D5 was tested in the Ames, *E.coli*, and mouse lymphoma tests and was found to be not mutagenic (NCBI 2020a). Silanes, silano, and siloxanes were investigated for clastogenic activity in male rats, and were found to be non-clastogenic (NCBI 2020a).

#### 7.13.1.2.7 Carcinogenicity

D5 has shown potential carcinogenicity in rats at an inhalation dose above 100 ppm (NCBI 2020a). However, two large human studies have not found an increase in cancer among patients with silicone breast implants. In repeated studies in rats with subacute, subchronic and chronic inhalation exposure a small increase in the incidence of uterine adenocarcinoma (uterine tumor) in female rats (2-year inhalation chronic bioassay) was observed.

#### 7.13.1.2.8 Neurotoxicity

No data were found for D5 neurotoxicity. Rat pups from the two-generation reproductive study were evaluated and found normal for neuropathology and neurobehavioral effects (2017). No signs of neurotoxicity have been reported for either the acute or chronic D5 exposures.

#### 7.13.1.2.9 Mechanism/Mode of Action

In female F344 rats, D5 induces hepatic microsomal cytochromes P450 (CYP1A, CYP2B, CYP3A, and CYP4A), epoxide hydrolase, and UDP-glucuronosyltransferase (UDPGT); liver size is also increased in these rats (McKim et al. 1999). The profile of induction is similar to that reported for phenobarbital.

Mechanistic studies to elucidate the mode-of-action for uterine tumor induction observed in female rats at the conclusion of the 2-year chronic inhalation bioassay suggest an interaction of D5 with dopamine signal transduction pathways altering the pituitary control of the estrus cycle. The resulting estrogen imbalance may cause the small increase in uterine tumor incidence at the highest D5-exposure concentration (160 ppm) over that seen in control rats. It is suggested that the mode of action for these uterine adenocarcinomas is rat specific and not relevant to human biological mechanisms (Klaunig et al. 2016).

#### 7.13.1.3 Ecological Data

##### 7.13.1.3.1 Fate and Transport

D5 has low water solubility and will strongly adsorb to organic matter and would not enter ground water (Mackay et al. 2015). D5 will bind strongly to sediments. D5 is volatile and is anticipated to travel long distances when released to the atmosphere. It will accumulate in tissues; however, the low water solubility may limit aqueous D5 availability (EPA 2014). In a biomagnification study using rainbow trout, the half-life of D5 was calculated at 69 days (Woodburn et al. 2013).

##### 7.13.1.3.2 Ecotoxicity

D5 has been tested for aquatic toxicity using the standard test approaches. Due to its low solubility and high volatility, most of the assays were conducted with no headspace to prevent loss of test compound. The experimentally derived rainbow trout chronic $LC_{50}$ is 0.017 mg/L and the short term NOEC in trout is 0.016 mg/L (EPA 2019b). The predicted toxicity using ECOSAR are as follows: green algae 96 hr $EC_{50}$ = 0.373 mg/L, daphnid 48 hr $LC_{50}$ = 0.309 mg/L, and fish 96 hr $LC_{50}$ = 0.407 mg/L (EPA 2014).

Toxicity to terrestrial plants is low, with the red clover growth half-maximal inhibitory concentration $IC_{50}$ of 209–2,051 mg/kg soil (EPA 2019b).

##### 7.13.1.3.3 Degradation and Treatment

D5 may hydrolyze in soil and water and is subject to biotransformation in the atmosphere (McLachlan et al. 2010). D5 is considered persistent in sediments.

### 7.13.2 Octamethylcyclotetrasiloxane [D4] (CAS 556-67-2)

#### 7.13.2.1 General Information

Octamethylcyclotetrasiloxane (D4) is an oily liquid with low water solubility. Its uses include silicone chemical production, as well as inclusion in cosmetics, hair care products and deodorants. Alternative nomenclature for D4 includes dimethylsiloxane tetramer, and cyclotetrasiloxane. Figure 21 shows the chemical structure of the component.

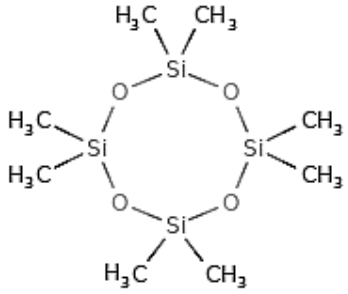

**Figure 21. Octamethylcyclotetrasiloxane**

### 7.13.2.2 Toxicology Data

Exposure to D4 may be via vapor, ingestion or direct skin contact. If silicone implants are present, there is the potential for internal exposure via the implant. Ingestion may result in nausea and vomiting.

#### 7.13.2.2.1 Oral

The acute oral $LD_{50}$ is reported to be >4,800 mg/kg in rat (ECHA 2020r). D4 is considered to have low oral toxicity.

#### 7.13.2.2.2 Inhalation

The acute inhalation $LC_{50}$ is reported to be 36,000 mg/m$^3$ for a 4-hour exposure in rat (PubChem 2020v). Inhalation exposure of rats to 700 ppm for up to 28 days resulted in transient hyperplasia of the liver, ultimately resulting in an increased liver weight in treated groups. The 90-day exposures to rats resulted in reversible histopathological changes to the female reproductive tract at 898 ppm D4. In humans, brief (1 hr) exposures to low-level (10 ppm) D4 revealed no immunologic or pro-inflammatory effects (NCBI 2020c).

#### 7.13.2.2.3 Dermal

The dermal $LD_{50}$ is 1,770 mg/kg in rats (PubChem).

Dermal application of radiolabeled D4 in men and women resulted in increased blood and plasma D4 up to 6 hr post application and also in exhaled air up to the final sampling time (24 hr) (NCBI 2020c).

Human volunteers did not develop allergic reactions after repeated skin contact with D4 (NCBI 2020c). Data from an absorption study in rats treated with D4 or D5 show that most of the compound volatilized from the skin and less than 1% of D4 was absorbed (NCBI 2020c). D4 is also considered a mild skin irritant.

#### 7.13.2.2.4 Ocular

D4 is considered a mild eye irritant (PubChem 2020v).

#### 7.13.2.2.5 Development and Reproduction

Inhalation of D4 at the several hundred ppm level or oral exposure to several hundred µg/kg of D4 have resulted in interference with the reproductive health of rats and impairment of their fertility (Quinn, et al. 2007; Quinn, et al. 2007). From the inhalation study, D4 attenuated the pre-ovulatory luteinizing hormone surge and significantly decreased the portion of female rats that ovulated.

In two different strains of female rat pups, D4 was examined for potential estrogenic and anti-estrogenic activities. D4 co-administered over a wide-range of ethinyl estradiol doses resulted in a significant reduction in uterine weight compared to ethinyl estradiol alone (NCBI 2020c).

#### 7.13.2.2.6 Genotoxicity

*In-vitro* Ames and mouse lymphoma tests were negative for mutagenicity and chromosomal aberration results (NCBI 2020c).

#### 7.13.2.2.7 Carcinogenicity

Female rats exposed to up to 700 ppm D4 for 2 years showed an increased incidence of endometrial adenomas and cystic endometrial epithelial hyperplasia (ECHA 2020r). The NOAEL in the study was 150 ppm for females and males was greater than 700 ppm (8,492 mg/m$^3$). In humans, the ECHA calculated NOAEC is > 8,492 mg/m$^3$ (700 ppm) due to physiological and hormonal differences between rats and humans.

#### 7.13.2.2.8 Neurotoxicity

No developmental neurotoxicity was observed in the F(2a) generation of rat pups from male and female rats exposed to D4 before and during mating, gestation, and lactation periods (NCBI 2020c).

#### 7.13.2.2.9 Mechanism/Modes of Action

D4 induces the expression of cytochrome P450 (CYP) enzymes CYP2B1, CYP2B2, CYP3A2, and CYP3A23/3A1 in rats. It also and effects binding and/or increases activity of the estrogen receptor (ESR1) and pregnane X receptor NR112 proteins in human-cell based assays (EPA 2019d). These two activities are found in adverse outcome pathways (AOP) 200 and 60, respectively.

### 7.13.2.3 Ecological Data

#### 7.13.2.3.1 Fate and Transport

D4 is expected to be highly persistent and will readily adsorb to soils. It has very low solubility, so it is not expected to transport in groundwater. It is highly volatile and will exist as vapor in the atmosphere. It will volatilize from a river within hours, and a lake within a week. It will persist in the atmosphere for approximately a week and is likely to bioaccumulate.

#### 7.13.2.3.2 Ecotoxicity

The algae 96-hour $EC_{50}$ is 22 µg/L, with a similar 48-hour $EC_{50}$ in *Daphnia* of 15 µg/L (ECHA 2020r). For fish, the 96-hour $LC_{50}$ is 22 µg/L, with a 14-day $LC_{50}$ of 10 µg/L in rainbow trout. In the sheepshead minnow, the 96-hour $LC_{50}$ was 6.3 µg/L.

#### 7.13.2.3.3 Degradation/Treatment

WWTPs will remove D4 from water due to likelihood of sludge adsorption and volatilization from the water (total removal of 99%, 60% to sludge and 40% to air).

### 7.13.3 Dipropylene Glycol Dimethyl Ether [DPGDME] (CAS 111109-77-4)

#### 7.13.3.1 General Information

DPGDME is an aprotic solvent used in polyurethane/isocyanate coating systems, as a solvent, and in cleaning/furnishing care products. It is a colorless liquid with a mild odor. DPGDME is manufactured by Dow Chemical Company under the trade name Proglyde™ DMM; other synonyms include 1-methoxy-3-(3-methoxypropoxy)propane, and propane, bis(methoxypropyl) ether. Figure 22 shows the chemical structure of the component.

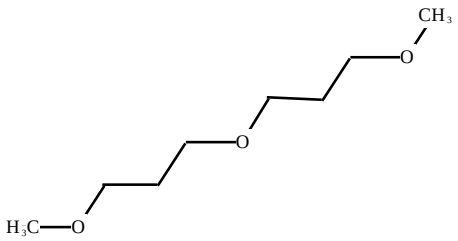


**Figure 22. Dipropylene Glycol Dimethyl Ether**

#### 7.13.3.2 Toxicology Data

Based upon the closely-related dipropylene glycol methyl ether, human exposure routes are anticipated to include inhalation, dermal absorption, ingestion, and skin or eye contact. Symptoms of overexposure include irritation to the eyes, nose, and throat, weakness or exhaustion, dizziness, and headache (NIOSH 2016).

##### 7.13.3.2.1 Oral

The oral $LD_{50}$ in the rat is reported to be 3300 mg/kg. Chronic exposure is reported to have unspecified effects on the adrenal gland, kidneys, and liver (Dow Chemical Company 2018). TOPKAT modeling predicts a chronic LOAEL of 24.0 mg/kg-day at high confidence.

##### 7.13.3.2.2 Inhalation

According to the SDS, the $LC_{50}$ in rats for a 4-hour exposure is greater than 5.25 mg/L; no deaths occurred at this concentration. Prolonged exposure is not expected to cause adverse effects. Based upon available data, narcotic effects and respiratory irritation were not observed (Dow Chemical Company 2018).

##### 7.13.3.2.3 Dermal

The acute dermal $LD_{50}$ in the rat is reported to be >2,000 mg/kg. Mild irritation may occur and skin contact may cause an allergic skin reaction (Dow Chemical Company 2018).

TOPKAT modeling predicts DPGDME is a probable irritant, but unlikely sensitizer.

##### 7.13.3.2.4 Ocular

DPGDME may cause slight eye irritation; corneal injury is unlikely (Dow Chemical Company 2018).

##### 7.13.3.2.5 Development and Reproduction

Birth defects were noted at doses causing toxicity to the mother. Birth defects were not noted in laboratory animals, and exposure did not interfere with reproduction (Dow Chemical Company 2018).

TOPKAT predicts DPGDME is not a developmental toxicant.

##### 7.13.3.2.6 Genotoxicity

Both *in vitro* and animal mutagenicity studies are reportedly negative (Dow Chemical Company 2018). TOPKAT predicts DPGDME is not mutagenic in the Ames test.

#### 7.13.3.2.7 Carcinogenicity

According to the SDS, no relevant data were found (Dow Chemical Company 2018). TOPKAT modeling of DPGDME was equivocal for carcinogenicity.

#### 7.13.3.2.8 Neurotoxicity

No narcotic symptoms were noted during inhalation studies (Dow Chemical Company 2018).

#### 7.13.3.2.9 Mechanism/Modes of Action

No data were found.

### 7.13.3.3 Ecological data

#### 7.13.3.3.1 Fate and Transport

With a predicted log $K_{oc}$ of 0.89 and a solubility of $5.3 \times 10^5$ mg/L, DPGDME is expected to be highly mobile in soil and will likely pose a hazard to surface and drinking water.

Based upon a Henry's Law constant of $3.67 \times 10^{-7}$ atm/$m^3$-mol, DPGDME is not expected to volatilize from wet surfaces; however, the vapor pressure of 0.55 mmHg indicates it will volatilize from dry surfaces and exist in the atmosphere exclusively as a vapor.

With a log $K_{ow}$ of 0.42, DPGDME is not expected to bioaccumulate; a bioconcentration factor of 4 was measured for a 43-day exposure of rainbow trout (*O. mykiss*) (Dow Chemical Company 2018).

#### 7.13.3.3.2 Ecotoxicity

No experimental data were found for toxicity toward green algae; EPA's ECOSAR program predicts a 96-hour $EC_{50}$ in green algae of 777 mg/L.

The 24-hour $LC_{50}$ in *Daphnia* was >1,000 mg/L. In a 21-day semi-static exposure test in *Daphnia*, the NOEC and LOEC were 10 mg/L and 32 mg/L, respectively on the basis of number of offspring (Dow Chemical Company 2018). The 96-hour $LC_{50}$ in guppies (*Poecilia reticulata*) was greater than 1,000 mg/L. The $LC_{50}$ for a 14-day exposure of earthworms (*Eisenia fetida*) was greater than 1,000 mg/kg.

#### 7.13.3.3.3 Degradation/Treatment

DPGDME has a high vapor pressure and it will volatilize from dry surfaces. It is not predicted to be readily biodegradable. In air, it is susceptible to oxidation with a half-life of 2.8 hours. It is poorly removed by waste water treatment processes, with only 1.76% expected to adsorb to sludge and less than 0.01% biodegraded.

### 7.14 Ecolink NAVSOLVE

This product contains decamethylcyclopentasiloxane, octamethylcyclotetrasiloxane, and dipropylene glycol, n-butyl ether.

#### 7.14.1 Decamethylcyclopentasiloxane [D5] (CAS 541-02-6)

See paragraph 7.13.1 for toxicity data on D5.

#### 7.14.2 Octamethylcyclotetrasiloxane [D4] (CAS 556-67-2)

See paragraph 7.13.2 for toxicity data on D4.

#### 7.14.3 Dipropylene Glycol, n-Butyl Ether [DGME] (CAS 29911-28-2)

##### 7.14.3.1 General Information

Dipropylene glycol monobutyl eather (DGME) is a colorless liquid that is used in cosmetics and as a fly repellent. Synonyms include Stabilene, 2-propanol, 1-(2-butoxy-1-methylethoxy)-, dipropylene glycol, n-butyl ether, PPG-14 Butyl ether, Crag fly repellent and more. Figure 23 shows the chemical structure of the component.

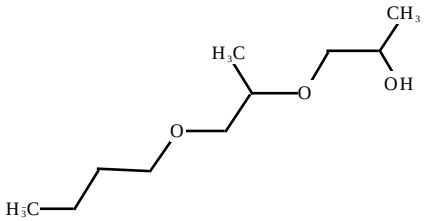


**Figure 23. Dipropylene Glycol Monobutyl Ether**

##### 7.14.3.2 Toxicology Data

###### 7.14.3.2.1 Oral

DGME is moderately toxic with a probable oral lethal dose (human) of 500–5,000 mg/kg (Gosselin et al. 1976). DGME has U.S. Food and Drug Administration approval as an indirect additive used in food-contact paper at a concentration of up to 0.0008% (21CFR175).

The doses of DGME reported to cause acute oral toxicity range widely. DGME has oral $LD_{50}$ values of 1,620 µL/kg (1,513 mg/kg, based on density of 0.934 mg/mL) to 9,100 mg/kg in rat (Lewis 2004, CDC 1997) and 23,900 mg/kg in rabbit (Lewis 2004). Rats were fed diets containing 0, 0.004, 0.016, 0.064, or 0.256% Crag Fly Repellant (DGME) for up to 2 years. Body weights in females at 0.256% of the diet were lower than other groups while males were

comparable. Transitory cloudy swelling of kidney convoluted tubules was seen for 0.064 and 0.256% at 6 months but not later. The NOAEL was determined to be 0.256% based on body weight (California Environmental Protection Agency (CalEPA) 2018a). Dogs given 0.8, 3.2, or 12.8 mg/kg DGME in capsules 5 days/week for 1 year resulted in no observable hematology, clinical chemistry, or histopathology effects (CalEPA 2018a).

#### 7.14.3.2.2 Inhalation

The acute inhalation $LC_{50}$ is reported as 2,040 mg/m$^3$ for a 4-hour exposure in rat (ECHA 2020a).

#### 7.14.3.2.3 Dermal

DGME has dermal $LD_{50}$ values ranging from 5,860 µL/kg (5,473 mg/kg, based on density of 0.934 mg/mL) to 21,000 mg/kg in rabbit (Lewis 2004, CDC 1997).

In a 90-day dermal toxicity study, rats were dermally exposed to 0, 0.5, 1, or 4 mL/kg/day Stabilene® Fly Repellent (equivalent to 500, 1,000 or 4,000 mg/kg/day) for 6 h/day for 3 days. Systemic toxicity endpoints noted in the 4,000 mg/kg/day group were significant reductions in body weight and body weight gain throughout the treatment period. The differences in body weight were 8–11% for males and 3–6% for females throughout the treatment. Dermal toxicity was noted in the mid and high dose groups as exfoliation (scaling) and/or excoriation (cracking) of the skin beginning at day 7 for scaling and at day 21 for cracking. The LOAEL for systemic toxicity was 4,000 mg/kg/day based on reduced body weight gain, reduced food efficiency, and changes in hematological parameters. The NOAEL for systemic toxicity was 1,000 mg/kg/day. The LOAEL for dermal toxicity was 1,000 mg/kg/day and the NOAEL for dermal irritation was 500 mg/kg/day based on changes in the skin (EPA 2007).

DGME is not a sensitizer according to the Buehler guinea pig test (ECHA 2020a).

#### 7.14.3.2.4 Ocular

DGME is not an ocular irritant (ECHA 2020a).

#### 7.14.3.2.5 Development and Reproduction

Pregnant rats exposed dermally to 400, 2,000, or 4,000 mg/kg/day DGME for 6 h/day from gestational days 6–15 showed maternal toxicity in the mid and high dose groups as reduced body weight gain and skin irritation. In the high dose group, an increase in total leukocytes and segmented neutrophils was also observed. No developmental toxicity was noted at any dose (EPA 2007).

#### 7.14.3.2.6 Genotoxicity

Stabilene fly repellent tested negative in a micronucleus assay in mice after intraperitoneal exposure. A sister chromatid exchange assay in CHO cells and an Ames assay in strains of *Salmonella typhimurium* both showed inconsistent results in which significant increases were observed, but these effects were subtle and not dose-dependent (EPA 2007). The conclusion is that DGME is not mutagenic.

#### 7.14.3.2.7 Carcinogenicity

Chronic oral exposures to DGME in rats and dogs showed no evidence of carcinogenicity (PubChem 2020o).

#### 7.14.3.2.8 Neurotoxicity

No neurotoxicity data are found for DGME.

#### 7.14.3.2.9 Mechanism/Modes of Action

Physiologically based pharmacokinetic data are available for DGME. The bioactivity of DGME was evaluated in the Comptox (400) and Pubchem (258) bioassays (EPA 2019c). Only a single receptor, a pregnane X receptor (NR1I2) scored as positive in the Pubchem suite of tests. None of the Comptox tests were scored as active.

### 7.14.3.3 Ecological Data

#### 7.14.3.3.1 Fate and Transport

DGME has high water solubility (45,000 mg/L) and low estimated log $K_{ow}$ (1.40), suggesting that it will readily dissolve in water, but pose a low risk of bioaccumulation. DGME vapor pressure ($3.6 \times 10^{-3}$ mmHg) suggests DGME would exist in the vapor phase if released to the atmosphere. It has low potential to volatilize from wet surfaces (Williams et al.).

#### 7.14.3.3.2 Ecotoxicity

DGME has an $LC_{50}$ value of 17 mg/L (96 h) in *Gammarus fasciatus* (scud, freshwater shrimp). DGME also has dietary $LC_{50}$ concentrations of >5,620 ppm in *Anas platyrhynchos* (mallard duck) and *Colinus virginianus* (northern bobwhite quail) (8 days) (PubChem 2020o, Williams et al. 2017)

#### 7.14.3.3.3 Degradation/Treatment

DGME is expected to be environmentally persistent; however, it will quickly photodegrade in the atmosphere. As it is unlikely to adsorb to sludge and is not biodegradable, treatment at WWTPs will not remove DGME.

### 7.14.4 Hexylene Glycol (CAS 107-41-5)

#### 7.14.4.1 General Information

Hexylene glycol is an oily, colorless liquid with a mild sweet odor. It is less dense than water and is slow to combine. It is used primarily as a solvent. Synonyms for this compound include 2-methylpentane-2,4-diol, Diolane, 4-methyl-2,4-pentanediol and others. Figure 24 shows the chemical structure of the component.

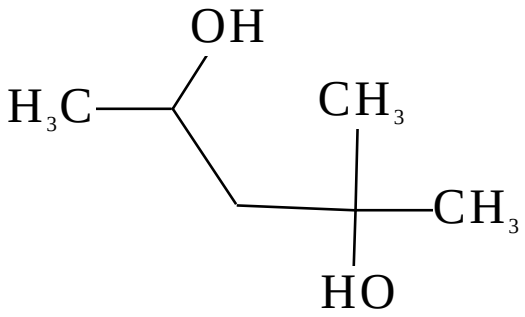


**Figure 24. Hexylene Glycol**

#### 7.14.4.2 Toxicology Data

Exposure to hexylene glycol can result in muscle weakness, irritation to nose and eyes, gastrointestinal disruption, depressed liver and kidney function, CNS depression, nausea, headache, and dermatitis (PubChem 2020f).

##### 7.14.4.2.1 Oral

The acute oral $LD_{50}$ is reported as 3,097 mg/kg in mouse, as 3,700 mg/kg in rat, as 2,800 mg/kg in guinea pig, and as 3,200 mg/kg in rabbit. Hexylene glycol is considered to have low oral toxicity (PubChem 2020f).

##### 7.14.4.2.2 Inhalation

The acute inhalation 1-hour exposure $LC_{50}$ is reported as greater than 310 mg/m$^3$ in rat and in a 4-hour exposure as ~77mg/m$^3$ in rat (PubChem 2020f). A 15-minute exposure TCLo (lowest concentration resulting in a toxic effect) is reported as 72.3 mg/m$^3$ (50 ppm) in human. Hexylene glycol is considered to have extreme inhalation toxicity in rat.

#### 7.14.4.2.3 Dermal

The dermal $LD_{50}$ is reported as 8,217 mg/kg (8.56 mL/kg) in rabbit (PubChem 2020f). The hexylene glycol density is reported as 0.96 $g/cm^3$. It is a dermal irritant and sensitizer. Hexylene glycol is considered to have moderate dermal toxicity.

#### 7.14.4.2.4 Ocular

Hexylene glycol is an eye irritant (PubChem 2020f).

#### 7.14.4.2.5 Development and Reproduction

Following daily oral administration in male and female rats pre-mating through to 4 days postpartum, there was an effect on pup mortality and decreased body weight gain at doses of 1,000 mg/kg-day (PubChem 2020f). In a separate study in male rats, there was no effect on fertility following exposure up to 190 mg/day for 190 days.

#### 7.14.4.2.6 Genotoxicity

No evidence of genotoxicity has been noted in the bacterial reverse mutation assay, a mammalian gene mutation assay, the yeast gene mutation assay, and the mammalian chromosome aberration test, both with and without metabolic activation (ECHA 2020g).

#### 7.14.4.2.7 Carcinogenicity

There is no evidence that hexylene glycol causes cancer.

#### 7.14.4.2.8 Neurotoxicity

Exposure to vapors can result in transient generalized dizziness and suppression of the CNS (PubChem 2020f).

#### 7.14.4.2.9 Mechanism/Mode of Action

Hexylene glycol can increase microtubule polymerization during mitosis (PubChem 2020f).

### 7.14.4.3 Ecological Data

#### 7.14.4.3.1 Fate and Transport

Hexylene glycol is very soluble in water and with a low log $K_{oc}$ it is not expected to adsorb to soils; therefore, there is a concern that hexylene glycol may be transported in groundwater. It will exist in the atmosphere as combination of vapor and particulates, and will have a half-life in the atmosphere of approximately 9 hours due to degradation following interaction with hydroxyl radical. It is not expected to be volatile, and will not readily volatilize from soil or wet surfaces. Hexylene glycol also will not bioaccumulate.

#### 7.14.4.3.2 Ecotoxicity

The $EC_{50}$ in *Daphnia* ranges from 3,200–8,700 mg/L for a 48-hour exposure (PubChem 2020f). The $LC_{50}$ in the bleak (*Alburnus alburnus*) was 8,000 mg/L for a 96-hour exposure and 9,450 mg/L in rainbow trout (*O. mykiss*). Similar values in the fathead minnow (*Pimephales promelas*; 8,690 mg/L) and the blue gill sunfish (*L. macochirus*; 12,800 mg/L) have been found. The brine shrimp (*Artemia salina*) and tadpole (*Rana catesbiana)* also had similarly high $LC_{50}$ values
(5,900 mg/L and 11,800 mg/L, respectively). In algae (*Selenastrum capriconutum*), the $EC_{50}$ was >429 mg/L for a 72-hour exposure.

#### 7.14.4.3.3 Degradation/Treatment

Hexylene glycol will be somewhat persistent in the environment, with an expected biodegradation half-life of weeks. Volatilization from a lake is expected to take more than 700 days, while volatilization from a river will take 65 days. Treatment at WWTPs will not remove hexylene glycol, with a total removal of 1.88%, mostly to sludge.

### 7.15 LPS A-151

### 7.15.1 Isoparaffinic Hydrocarbon/Distillates Petroleum, Hydrotreated, Light (CAS 64742-47-8)

See paragraph 7.11.3 for toxicity data on this compound.

### 7.15.2 Other

No information on this compound was provided.

### 7.16 Pantheon X-IT Wash

### 7.16.1 Diethylene Glycol Monobutyl Ether [DEGBE] (CAS 112-34-5)

#### 7.16.1.1 General Information - Diethylene Glycol Monobutyl Ether

DEGBE is a colorless liquid with a mild odor (ALS 2014). Synonyms include 2-(2-butoxyethoxy)ethanol, diethyleneglycol butyl ether (DEGBE), butyldiglycol, butyl carbitol, butyl digol, butoxydiglycol, Ektasolve DB, Jeffersol db, Dowanol DB, and several others (PubChem 2020c). Figure 25 shows the chemical structure of the component.

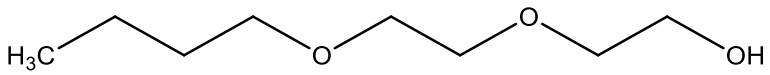


**Figure 25. Diethyleneglycol Monobutyl Ether**

### 7.16.1.2 Toxicology Data

#### 7.16.1.2.1 Oral

The acute oral $LD_{50}$ test has been conducted in several species: rat (range 3306-9623 mg/kg), guinea pig (2,000 mg/kg), mouse (4,500 mg/kg) and rabbit (2,200 mg/kg) (PubChem 2020c). The minimal lethal dose in humans is 0.5 mL/kg. These values place DEGBE in the low toxicity classification.

#### 7.16.1.2.2 Inhalation

No experimental data were found. TOPKAT modeling predicts an acute $LC_{50}$ in rats of more than 10 $g/m^3$-hour.

#### 7.16.1.2.3 Dermal

The dermal $LD_{50}$ in rabbit is 2,700 mg/kg (PubChem 2020c). Skin irritation from chronic exposure to DEGBE may occur due to defatting of the skin. DEGBE is capable of penetrating skin. The intraperitoneal $LD_{50}$ in mouse is 850 mg/kg suggesting DEGBE systemic toxicity via the dermal route may be higher than oral toxicity. An entry in the TOPKAT database indicates DEGBE is not a dermal sensitizer.

#### 7.16.1.2.4 Ocular

DEGBE is an ocular irritant (PubChem 2020c).

#### 7.16.1.2.5 Development and Reproduction

Several studies in rats have been conducted with no overt effects on development or reproduction (ECHA 2020e). The NOAEL for reproduction is >1,000 mg/kg-day.

TOPKAT modeling predicts DEGBE will not be a developmental or reproductive toxicant at high confidence.

#### 7.16.1.2.6 Neurotoxicity

Exposure to some ether derivatives of diethylene glycol may cause CNS depression (PubChem 2020c).

#### 7.16.1.2.7 Genotoxicity

There is no evidence that DEGBE is a genotoxicant (ECHA 2020e). It was negative with and without metabolic activation in the bacterial reverse mutation assay (Ames), the cytogenicity assay, the hypoxanthine phosphoribosyl transferase (HGPRT) gene mutation assay, the *in vivo* mouse micronucleus assay and the Drosophila sex-linked recessive lethal assay. The TOPKAT database indicates DEGBE is not mutagenic in the Ames assay.

#### 7.16.1.2.8 Carcinogenicity

No experimental data were found. TOPKAT modeling predicts DEGBE will not be carcinogenic.

#### 7.16.1.2.9 Mechanism/Mode of Action

No data were found

### 7.16.1.3 Ecological Data

#### 7.16.1.3.1 Fate and Transport

If released to soil, DEGBE is expected to be highly mobile in groundwater due to aqueous solubility and low affinity for organic carbon. DEGBE is unlikely to partition to the atmosphere from water or wet surfaces due to the very low $K_H$, but will readily evaporate from dry surfaces due to its vapor pressure. Any DEGBE present in the atmosphere is expected to be present as a vapor, where it will be subject to oxidation by photolytically produced hydroxide radicals with an estimated half-life of 342 hours. The tendency to bioaccumulate/bioconcentrate is low due to the very low log $K_{ow}$.

#### 7.16.1.3.2 Ecotoxicity

The EPA ECOTOX database reports a population LOEC for green algae (*S. quadricauda*) to be 1,000 mg/L (EPA 2019e). In *Daphnia*, DEGBE was not toxic at a 48-hour limit test of 1,000 mg/L (ECHA 2020e). The 96-hour $LC_{50}$ for DEGBE in bluegill *(L. macrochirus*) is reported to be 1,300 mg/L. These values indicate ecotoxicity is low according to both GHS and the APHC system.

#### 7.16.1.3.3 Degradation/Treatment

DEGBE is predicted to be readily biodegradable with environmental persistence of only days.

DEGBE will not be readily removed from waste streams by physical processes at WWTPs but should be readily treated by biodegradation.

## 7.17 Pantheon X-IT Carbon Remover and Cleaner

### 7.17.1 Diethylene Glycol Monobutyl Ether [DEGBE] (CAS 112-34-5)

See paragraph 7.16.1 for toxicity data on DEGBE.

## 7.18 Penair C-5572

### 7.18.1 Alcohols, $C_{9-11}$, Ethoxylated (CAS 68439-46-3)

See paragraph 7.10.2 for toxicity data on this compound.

### 7.18.2 Capramide DEA (CAS 136-26-5)

#### 7.18.2.1 General Information

Synonyms for this compound include Capric diethanolamide, Capric acid diethanolamide, and numerous others. Figure 26 shows the chemical structure of the component.

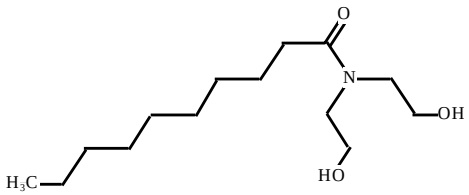


**Figure 26. Capramide DEA**

#### 7.18.2.2 Toxicology Data

There are no experimental toxicity data for capramide DEA. A similar compound, lauramide DEA has been used as a comparator in the absence of suitable data for capramide DEA.

##### 7.18.2.2.1 Oral

TOPKAT predicts capramide DEA to have low oral toxicity at high confidence, with a predicted $LD_{50}$ of >10,000 mg/kg. The chronic LOAEL for capramide DEA is predicted to be 218.6 mg/kg-day with low confidence. The $LD_{50}$ for lauramide DEA in rats is 2700 mg/kg (PubChem 2020u).

##### 7.18.2.2.2 Inhalation

TOPKAT predicts an $LC_{50}$ of 34.1 mg/m$^3$ for capramide DEA, with low confidence.

##### 7.18.2.2.3 Dermal

Capramide DEA is not predicted to be a dermal irritant or sensitizer with high confidence.

##### 7.18.2.2.4 Ocular

Capramide DEA is predicted to be at most mildly irritating to the ocular membranes with high confidence.

##### 7.18.2.2.5 Development and Reproduction

Capramide DEA is not predicted to be a developmental or reproductive toxicant with high confidence.

#### 7.18.2.2.6 Genotoxicity

Capramide DEA is not predicted to be positive in the Ames assay with high confidence.

#### 7.18.2.2.7 Carcinogenicity

Despite not being expected to be a genotoxicant, capramide DEA is still predicted to be positive in the rat and mouse cancer bioassays, and thus is predicted to be a carcinogen with moderate confidence.

#### 7.18.2.2.8 Neurotoxicity

No data were found.

#### 7.18.2.2.9 Mechanism/Mode of Action

No data were found.

### 7.18.2.3 Ecological Data

#### 7.18.2.3.1 Fate and Transport

Capramide DEA is anticipated to be moderately mobile in ground water, with moderate solubility and negligible likelihood of adsorption to soil. It will exist as a particulate in air and is susceptible to photoxidation with a half-life of 2.7 hours. It is nonvolatile, so will not volatilize from surface water or moist soils. It is not anticipated to bioaccumulate.

#### 7.18.2.3.2 Ecotoxicity

ECOSAR predicts that capramide DEA has an $EC_{50}$ of 2.8 mg/L for green algae. The $LC_{50}$ in *Daphnia* is predicted to be 104.7 mg/L and in fish to be 90.9 mg/L. TOPKAT predicts the $LC_{50}$ in fathead minnow will be 2,500 mg/L with low confidence. The $EC_{50}$ in *Daphnia* is predicted to be 1,700 mg/L with low confidence.

#### 7.18.2.3.3 Degradation/Treatment

Capramide DEA is not expected to be overly persistent in the environment with a biodegradation half-life of days to weeks; it is susceptible to atmospheric oxidation. Waste water treatment will be of limited utility as the compound does not readily bind sludge and does not volatilize.

## 7.19 Socomore DS-108

### 7.19.1 Ethyl Lactate [EL] (CAS 97-64-3)

#### 7.19.1.1 General Information

Ethyl lactate is a clear colorless liquid with a mild, buttery or fruity odor. It is more dense than water and vapors are heavier than air. Synonyms include Ethyl 2-hydroxypropanoate, Actylol, 2-hydroxypropanoic acid ethyl ester, lactic acid ethyl ester, and more. Figure 27 shows the chemical structure of the component.

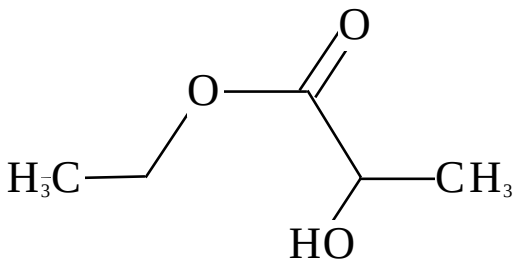


**Figure 27. Ethyl Lactate**

#### 7.19.1.2 Toxicology Data

##### 7.19.1.2.1 Oral

The $LD_{50}$ in rats was 8,200 mg/kg, while the mouse $LD_{50}$ was 2,500 mg/kg (PubChem 2020r). Oral administration causes CNS depression and can be lethal to animals in high concentrations due to respiratory paralysis.

##### 7.19.1.2.2 Inhalation

Toxicity can occur at doses above 1,800 mg/m$^3$ in rats, sensory irritation occurs at 75 mg/m$^3$ (Clary et al. 1998, PubChem 2020r).

##### 7.19.1.2.3 Dermal

The $LD_{50}$ was greater than 5,000 mg/kg in the rabbit (PubChem 2020r). It is not considered to be an irritant or sensitizer following assessment in cosmetics (Api et al. 2020).

##### 7.19.1.2.4 Ocular

Ethyl lactate was an ocular irritant in rabbits (PubChem 2020r).

#### 7.19.1.2.5 Development and Reproduction

No evidence of teratogenicity or maternal toxicity was noted for ethyl lactate (PubChem 2020r). Percutaneous application of doses up to 3,619 mg/kg on days 6 to 15 of gestation resulted in slight erythema and desquamation in treated animals, but no other signs were noted.

#### 7.19.1.2.6 Genotoxicity

Ethyl lactate was negative in the Ames assay for genotoxicity (Api et al. 2020, PubChem 2020r).

#### 7.19.1.2.7 Carcinogenicity

No data were found.

#### 7.19.1.2.8 Neurotoxicity

Ethyl lactate can cause CNS depression and can cause respiratory paralysis (PubChem 2020r).

#### 7.19.1.2.9 Mechanism/Mode of Action

Tox21/ToxCast found that ethyl lactate is an estrogen related receptor alpha antagonist (Williams et al. 2017). It was not active in any other assays.

### 7.19.1.3 Ecological Data

#### 7.19.1.3.1 Fate and Transport

The high solubility and low likelihood of adsorption to soil indicates that ethyl lactate is likely to transport in water. It will exist in the atmosphere as a vapor and will have moderate volatility from wet surfaces. It will have a half-life in the atmosphere of approximately 2.6 days due to degradation following interaction with hydroxyl radical. It will not bioaccumulate.

#### 7.19.1.3.2 Ecotoxicity

For algae, the $EC_{50}$ was found to be 2,300 mg/L, while in *Daphnia* it was 560 mg/L in a 48-hour exposure (EPA 2019e). The NOEC in *Daphnia* was 320 mg/L. The $LC_{50}$ in zebrafish was 320 mg/L in a 96-hour exposure, with a NOEC of 180 mg/L.

#### 7.19.1.3.3 Degradation/Treatment

Ethyl lactate will be somewhat persistent in the environment, with an expected biodegradation half-life of days to weeks. Volatilization from a lake is expected to take more than 500 days, while volatilization from a river will take 45 days. Treatment at WWTPs will not remove ethyl lactate, with a total removal of 1.88%, mostly to sludge.

### 7.19.2 1-propoxy-2-propanol [P2P] (CAS 1569-01-3)

#### 7.19.2.1 General Information

1-propoxy-2-propanol (P2P) is a liquid that is miscible in water. It is used as a solvent in coatings and inks. A synonym is propylene glycol-n-monomethyl ether (PubChem 2020b). Figure 28 shows the chemical structure of the component.

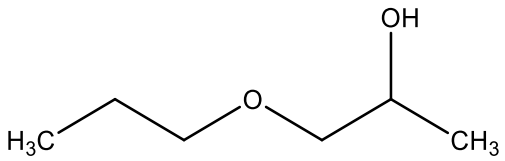


**Figure 28. 1-propoxy-2-propanol**

#### 7.19.2.2 Toxicology Data

Cases of human exposure have not been reported. Experimental animals have developed eye, skin, and mucous membrane irritation. Percutaneous absorption occurs. CNS depression with ataxia and prostration were noted following gavage, inhalation, or percutaneous exposure.

##### 7.19.2.2.1 Oral

The acute oral $LD_{50}$ in rats has been found to be 2,504 mg/kg (PubChem 2020b). At necropsy, animals demonstrated a reddened glandular stomach and dark red lungs.

Repeated dose toxicity testing TOPKAT modeling predicts a chronic LOAEL of 50.6 mg/kg-day.

##### 7.19.2.2.2 Inhalation

The $LC_{50}$ is >1,725 ppm (>8,300 mg/m$^3$), and exposure resulted in mild lethargy (PubChem 2020b). Subchronic inhalation exposure (whole body) 6 h/day, 5 days/week, for 14 weeks had a NOAEC of 300 ppm (1,474 mg/m$^3$), the highest dose tested.

##### 7.19.2.2.3 Dermal

Acute dermal toxicity has been observed in New Zealand white rabbits at 4.29 mL/kg in males and 4.92 mL/kg in females (ECHA 2020f). Local dermal effects included edema, necrosis, and desquamation.

P2P was tested for skin irritation and corrosion in rabbits and was negative (ECHA 2020f).

#### 7.19.2.2.4 Ocular

Studies in rabbits have found P2P to cause moderate to marked irritation and moderate corneal injury and slight iritis that healed within a week (ECHA 2020f).

#### 7.19.2.2.5 Development and Reproduction

TOPKAT modeling predicts P2P is unlikely to be a developmental or reproductive toxicant. A timed developmental study by inhalation exposure 6 h/day on gestation days 6–15 was conducted in rats (ECHA 2020f). Mild maternal toxicity (decreased body weight gain and reduced food consumption) was observed at the highest concentration 1,500 ppm (7,251 $mg/m^3$). One dam in this treatment group developed corneal opacity later described as ulceration with keratitis. At 1,500 ppm, poorly ossified hind limb phalanges were observed in fetuses; the developmental toxicity NOAEC was 750 ppm (3,625.77 $mg/m^3$).

#### 7.19.2.2.6 Genotoxicity

TOPKAT modeling predicts P2P is unlikely to be mutagenic in the Ames assay. P2P was negative in the Ames assay both with and without metabolic activation up to 5000 µg/plate, the highest dose tested (ECHA 2020f).

#### 7.19.2.2.7 Carcinogenicity

No data were found. TOPKAT does not predict that P2P will be a carcinogen.

#### 7.19.2.2.8 Neurotoxicity

Experimental animals exposed to high doses develop CNS depression or narcosis (PubChem 2020b).

#### 7.19.2.2.9 Mechanism/Mode of Action

No data were found.

### 7.19.2.3 Ecological Data

#### 7.19.2.3.1 Fate and Transport

If released to soil or water, P2P is expected to demonstrate very high mobility due to its reported miscibility with water, and will therefore pose a hazard to surface and drinking water. Evaporation from water or wet surfaces is not favored; the half-life for evaporation from a river is 100 days and it is over 1000 days for a lake. In the atmosphere, P2P is expected to exist primarily in particulate form. P2P is not expected to bioaccumulate.

#### 7.19.2.3.2 Ecotoxicity

P2P aquatic toxicity is low. The 96-hour acute toxicity to rainbow trout (*O. mykiss*) was > 100 mg/L; the 48-hour $LC_{50}$ in *D. magna* was > 100 mg/L and the 72-hour green algae (*P. subcapitata*) $LC_{50}$ was 3,440 mg/L (ECHA 2020f).

Using QSAR, TOPKAT predicts an $EC_{50}$ in *Daphnia* of 13.2 mg/L and an $LC_{50}$ in fathead minnow of 279.3 mg/L, both at low confidence. The ECOSAR models P2P as a neutral organic. The predicted 96-hour $EC_{50}$ in green algae is 453.4 mg/L, the 48-hour $LC_{50}$ in *Daphnia* is 1,082 mg/L, and the 96-hour $LC_{50}$ in fish is 2,190 mg/L.

#### 7.19.2.3.3 Degradation/Treatment

P2P rapidly biodegrades. Degradation via hydroxyl radicals in the atmosphere is rapid (half-life of 4.9 hours), but accessibility by hydroxyl radicals is limited by adsorption to atmospheric particulates.

P2P is predicted to be poorly removed by wastewater treatment processes (<2%), primarily via sludge adsorption.

## 8. DISCUSSION

### 8.1 Compound Summaries

For all of the formulations, an individual assessment of the toxicities of all of the components has been completed where possible. Some components could not be analyzed. A summary for each of the components is found below. See the appendix for more detailed summary tables for each of the components, including physical-chemical properties, summary toxicity data, and toxicity assessments.

#### 8.1.1 MPK

MPK is not overtly toxic, although it is an eye irritant. Appropriate PPE precautions should be taken when using MPK. It is expected to be mobile in groundwater, so appropriate environmental controls are recommended.

#### 8.1.2 MIBK

MIBK appears to have fairly low toxicity, although there are some concerns with effects on reproduction and carcinogenicity. It has not been proven to be a human carcinogen, but ample evidence indicates that it will be an animal carcinogen. There is evidence this is a mild ocular irritant. It will be mobile in groundwater but will volatilize from the water fairly quickly once exposed to air. Standard precautions for handling and environmental releases should be taken with MIBK.

### 8.1.3 2-BE

2BE is moderately toxic for oral, inhalation, and dermal exposures. It is a dermal and ocular irritant. It may have reproductive effects and is a mutagen, although it may not be a human carcinogen and does not appear to be a direct acting carcinogen in animals. It is of low acute toxicity in the environment and is not overly persistent. Appropriate PPE should be worn when contact is anticipated and appropriate engineering controls will limit its release into the environment, although release into the environment is not a concern.

### 8.1.4 Sodium Disilicate

Overall acute toxicity via oral or inhalation routes of exposure is moderate. Sodium disilicate presents a hazard to the skin and eyes, and requires use of appropriate PPE. There are no indications of developmental or reproductive toxicity, or genotoxicity. Sodium disilicate is not suspected to be a human carcinogen.

Ecotoxicity is low, although data have not been collected for green algae.

### 8.1.5 Ammonium Hydroxide

Toxic effects of ammonia are dependent upon the concentration of the solution involved. When ingested or inhaled, irritation of the relevant route of entry is to be expected. Only extremely high exposures are expected to be fatal, and the inhalation threshold concentration is sufficiently low to alert the individual to potential exposure. The greatest hazard appears to be occupational exposure to skin or eyes.

Environmental hazard is also dependent upon the concentration. As a base, ammonia will raise the pH of aquatic systems posing a hazard to aquatic species. Terrestrial release is expected to be relatively short-lived, as vaporization will result in the reduction of concentration. Plants may take up limited amounts of ammonia as a nutrient.

### 8.1.6 DEA

DEA is of moderate toxicity except for dermal exposures, where it is an irritant and corrosive. Appropriate PPE is recommended when working with DEA. It is likely to transport via groundwater and is not likely to biodegrade; therefore, engineering controls to prevent release are recommended.

### 8.1.7 DPGME

DPGME is of low concern for oral, inhalation, or dermal toxicity, except where CNS depression or narcosis occur. It has low ecotoxicity concerns as well. It is likely to transport via groundwater but is susceptible to both biodegradation and photodegradation.

### 8.1.8 C8-18-Alkyldimethylbenzyl Ammonium Chlorides

ABAC has few data available on its toxicity. It does appear to have a low oral $LD_{50}$, causing concern for acute oral exposures. There may be concern for endocrine disruption, as it binds to both the estrogen α and retinoid x receptors; however, no confirmatory data are available beyond ToxCast. It is predicted to be highly toxic to aquatic species, and is not readily biodegradable. Therefore, caution in handling and minimizing environmental releases is highly recommended.

### 8.1.9 Solvent Naphtha (Petroleum), Heavy Aromatic

Heavy aromatic naphtha is a mixture of primarily aromatic hydrocarbons and is not toxic except as an aspiration hazard. Oral (except for aspiration), inhalation, and dermal exposure are not considered to be a significant hazard. Occupational exposure is likely to result in irritation to skin and eyes, but sensitization is not a hazard. Neurotoxicity and resulting CNS symptoms are likely with prolonged exposure to vapor. Heavy aromatic naphtha is not known to be genotoxic, but presence of naphthalene in the mixture may be a carcinogenicity hazard.

Few data exist for ecotoxicity, likely due to lack of solubility in aqueous systems. Manufacturer literature caution against environmental release, warning of chronic hazard to fish and wildlife.

### 8.1.10 Nonylphenol, Ethoxylated

NPEO shows some evidence that it may be an endocrine disruptor with respect to aquatic species. It is moderately toxic to a variety of animals and poses some ESOH concerns as it is an eye and skin irritant with the potential to cause severe eye damage following prolonged contact. It will not transport in groundwater, but does not readily biodegrade either, so may be environmentally persistent. Caution in handling and minimizing release to the environment is recommended.

### 8.1.11 Propylene Glycol n-Butyl Ether

Acute toxicity to PGBE via oral, inhalation or dermal exposure does not appear to be a hazard. Occupational exposure hazards to skin and eye appear to be moderate. Due to the level of persistence in water, it is not recommended to release PGBE into streams. Waste can be reduced by biological treatment at WWTPs.

### 8.1.12 Alcohols, $C_{9-11}$, Ethoxylated

Ethoxylated alcohols are of moderate toxicity via oral and inhalation routes of exposure, and they are considered nontoxic via dermal exposure. Exposure via oral ingestion or inhalation is expected to cause ataxia and somnolence, as well as gastrointestinal disturbances. Occupationally, they are expected to be both dermal and ocular irritants but are not dermal sensitizers. Developmental/reproductive effects, mutagenicity, and carcinogenicity have not been observed.

Ecotoxicity is predicted to be low to both aquatic species and terrestrial invertebrates (earthworms). As with the hydrocarbon mixture above, the GHS classification is Acute Category II. Mobility in soil is expected to be moderate, and environmental persistence limited due to environmental degradation.

### 8.1.13 IDPA Ester Salts

IDPA ester salts are dermal and ocular irritants, and appropriate PPE is recommended when using this compound. Additional toxicity data are required to determine the acute and chronic hazards of this compound.

### 8.1.14 Methanol

Methanol is a relatively non-toxic substance. Its primary effects are on the eye and at higher doses, the nervous system. Ecotoxicity is also low.

### 8.1.15 Orange Terpenes

Orange terpenes is not overtly toxic to humans; however, ESOH exposures are a concern as it is a dermal irritant and sensitizer. There are no concerns regarding carcinogenicity or other chronic exposures. Release into the environment is a concern, as orange terpenes is acutely toxic to aquatic life. It will not transport in groundwater.

### 8.1.16 Alcohols, $C_{12\text{-}15}$, Ethoxylated

Ethoxylated alcohols are of low toxicity via oral and inhalation routes of exposure; they are considered nontoxic via dermal exposure. Exposure via oral ingestion or inhalation may cause ataxia and somnolence, as well as gastrointestinal disturbances. Developmental/ reproductive effects, mutagenicity, and carcinogenicity have not been observed.

Ecotoxicity is predicted to be high to both aquatic species. Mobility in soil is expected to be moderate, and environmental persistence limited.

### 8.1.17 Isoparaffinic Hydrocarbon

C10 hydrocarbons have a low acute toxicity by oral, inhalation, or dermal routes of exposure. There is a hazard of pneumonitis if orally ingested and aspirated into the lungs. Occupational hazards are minimal, being primarily drying of skin and mild irritation to the eyes. C10 hydrocarbons are not genotoxic or carcinogenic; however, inhalation of vapors may cause CNS depression. These mixtures are not expected to be developmental or reproductive toxicants.

Aquatic toxicity is categorized in GHS acute toxicity category 2 (United Nations Economic Commission for Europe (UNECE) 2015). C10 hydrocarbons are not readily biodegradable in aqueous environments but are subject to hydroxyl radical degradation in the atmosphere.

### 8.1.18 Dimethyl Adipate

Dimethyl adipate is not overtly toxic in any category. There are few concerns with respect to acute or chronic exposures, although weight loss has been observed with long-term exposure. It will transport in groundwater, so care with discharge should be taken.

### 8.1.19 Diethylhexyl Sodium Sulfosuccinate

DOSS is not overtly toxic via oral routes, as it is used for several medical purposes. It is an ESOH hazard due to its irritant properties for both eye and skin, so appropriate PPE is necessary when working with the material at higher concentrations. It is not expected to transport in groundwater, although release should still be monitored due to some toxicity in aquatic species.

### 8.1.20 Butyl-3-hydroxybutyrate

BHB does not appear to be overly toxic, except as an eye irritant. Appropriate PPE is recommended to reduce exposure. Additional testing is not necessary. Groundwater release does not appear to be a concern, although engineering controls to limit release are still recommended.

### 8.1.21 Decamethylcyclopentasiloxane

D5 is not overtly toxic from oral or inhalation exposure; however, it is extremely toxic to aquatic life. Release into the environment will not likely result in transport in groundwater, and with its high volatility, it will dissipate quickly. However, release should still be controlled due to its aquatic toxicity concerns.

### 8.1.22 Octamethylcyclotetrasiloxane

D4 is a particular concern for its purported reproductive toxicity and high persistence and likelihood to bioaccumulate. It is also highly toxic to aquatic species and invertebrates. Care should be taken to limit exposures of workers and release into the environment to mitigate these risks.

### 8.1.23 Dipropylene Glycol Dimethyl Ether

Toxicity of DPGDME by common routes of exposure appears to be low. There is a small hazard from occupational exposure to skin and eyes—indicating use of appropriate PPE. There appears to be no long-term hazard of mutagenicity, carcinogenicity, or developmental/ reproductive effects.

Environmental toxicity is low to aquatic and terrestrial species, but there is some groundwater transport hazard.

### 8.1.24 Dipropylene Glycol Monobutyl Ether

DGME toxicity data suggest low toxicity overall. Of note, DGME is approved as an indirect additive used in food-contact substances and is used as a fly repellant commercially. Oral and dermal exposure demonstrates low toxicity. A fly repellant containing DGME did not trigger genotoxic effects *in vitro* and oral exposures did not induce carcinogenic effects.

Environmental fate and transport of DGME will be a function of its high aqueous solubility and low vapor pressure. A low estimated log $K_{ow}$ suggests a low likelihood of bioaccumulation. Ecotoxicity data suggest low toxicity in aquatic invertebrates and low toxicity in terrestrial vertebrates (birds).

### 8.1.25 Hexylene glycol

Inhalation exposure to hexylene glycol is a concern and should be limited through engineering controls or adequate PPE. It is also a dermal irritant and sensitizer, so caution should be made in handling the material. Oral and aquatic toxicity are not considered to be concerns; however, due to its environmental persistence and high solubility, releases into the environment should be limited.

### 8.1.26 Diethyleneglycol Monobutyl Ether (DEGBE)

DEGBE is of low toxicity by all routes of exposure with the exception of the eyes, so appropriate use of PPE is recommended. It is not expected to be a developmental or reproductive toxicant, genotoxic, or carcinogenic.

Ecotoxicity is low, and DEGBE will be readily degraded in the environment with a persistence of only days.

### 8.1.27 Capramide DEA

Capramide DEA is not expected to be overtly toxic except by inhalation, but it may be a carcinogen. It is not expected to be a development or reproductive toxicant. It is not expected to be a major ecotoxicant. It is expected to be moderately mobile in groundwater.

### 8.1.28 Ethyl Lactate

Ethyl lactate is not expected to cause overt toxicity through any routes of exposure. It is anticipated to be an ocular irritant, so appropriate PPE is recommended. Its high solubility indicates that transport in groundwater is a concern; therefore, releases should be controlled or limited.

### 8.1.29 1-propoxy-2-propanol

P2P demonstrates toxicity effects comparable to other nonspecific solvents, with the primary mode of action likely to be degradation of exposed membranes, causing irritation and neurological effects. Workers should avoid inhalation of vapors and skin contact. Overt oral,

inhalation, and dermal toxicity are low; however, P2P is damaging to the eyes. P2P developmental toxicity occurs at high concentrations and only at concentrations that also caused maternal toxicity. P2P has not been extensively tested for mutagenicity but it was negative in the Ames assay and it does not appear on the ACGIH, NTP or IARC lists of carcinogens.

Ecotoxicity is low; however, environmental persistence is long, with half-lives exceeding 2 years in the best-case predictions. Bioaccumulation is not expected.

## 8.2 Regulations and Standards

### 8.2.1 Methyl Propyl Ketone

MPK is predicted to be a GHS category 4 for acute oral toxicity (UNECE 2015). The OSHA permissible exposure limit (PEL) is 200 ppm (700 mg/m$^3$), while the NIOSH relative exposure limit (REL) is 150 ppm (530 mg/m$^3$) (OSHA 2011; NIOSH 2016). ACGIH has derived a threshold limit value (TLV)-short-term exposure limit (STEL) of 150 ppm (530 mg/m$^3$) (ACGIH 2018). The California OSHA PEL is 200 ppm (700 mg/m$^3$), with a PEL-STEL of 250 ppm (875 mg/m$^3$).

### 8.2.2 Methyl Isobutyl Ketone

MIBK is predicted to be a GHS category 4 for acute toxicity and category 3 for aquatic toxicity, indicating that it is not overtly toxic (UNECE 2015). An OSHA PEL of 100 ppm (410 mg/m$^3$; 8-hour) has been derived (OSHA 2011). NIOSH has derived a REL of 50 ppm (205 mg/m$^3$, 10-hour), with a STEL of 75 ppm (300 mg/m$^3$) (NIOSH 2016). ACGIH has developed an 8-hour TLV of 20 ppm (81.9 mg/m$^3$), with a STEL of 75 ppm (300 mg/m$^3$)(ACGIH 2018). The California OSHA PEL is 50 ppm (205 mg/m$^3$) with a STEL of 75 ppm (300 mg/m$^3$) (CalEPA 2018b).

### 8.2.3 2-Butoxyethanol

2BE is a GHS category 4 for acute toxicity and a category 2 for eye and skin irritation (UNECE 2015). Appropriate PPE is recommended due to its irritating properties. There is an OSHA PEL (8-hour TWA) of 240 mg/m$^3$ for dermal exposure, with a NIOHS REL (10-hour TWA, skin) of 25 mg/m$^3$ (NIOSH 2007; OSHA 2011). The ACGIH TLV (8-hour TWA) is 96 mg/m$^3$ (20 ppm) (ACGIH 2018). The ATSDR minimal risk level (MRL) for acute inhalation is 29 mg/m$^3$ (6 ppm) and for chronic exposures is 5.7 mg/m$^3$ (0.2 ppm) (ATSDR 1998). The acute oral MRL is 0.4 mg/kg/day.

### 8.2.4 Sodium Disilicate

No regulations or standards pertaining to sodium disilicate were found.

### 8.2.5 Ammonium Hydroxide

Ammonia has a short-term exposure limit of 35 ppm (27 mg/m$^3$) (NIOSH 2018). The immediately dangerous to life or health (IDLH) level is 300 ppm (HSDB 1999). It is a GHS acute oral category 4, a category 3 for acute inhalation and respiratory irritation, category 1 for skin

corrosion/irritation and ocular effects (UNECE 2015). It is also a GHS acute aquatic toxicity category 1.

Ammonia/ammonium hydroxide is designated as a hazardous substance under the Federal Water Pollution Control Act and the Clean Water Act Amendments of 1977 and 1978. Releases of 1,000 lb or 454 kg are reportable under CERCLA (HSDB 1999).

### 8.2.6 DEA

DEA is predicted to be in GHS acute category 4, a category 2 for skin toxicity, and a category 1 for eye damage (UNECE 2015). NIOSH has established a REL (10-hour TWA) of 15 mg/m$^3$, while the California PEL is 2 mg/m$^3$ (8-hour TWA), and the ACGIH TLV is 1 mg/m$^3$ (8-hour TWA) (NIOSH 2007; ACGIH 2018; CalEPA 2018b). ACGIH has labelled DEA as an A3 carcinogen, confirmed in animals, but of unknown relevance in humans. It is listed as a hazardous air pollutant by EPA. The Texas Commission on Environmental Quality developed a reference concentration (RfC) of 25 µg/mL (Haney et al. 2018).

### 8.2.7 DPGME

There is very limited information on the occupation exposure limitations for this compound. However, the European Union (ECHA 2020c), has provided long-term exposure (LTEL) values (with skin designation) of 308 mg/m$^3$ or 50 ppm for this compound. It is predicted to be GHS Category 5 for all acute toxicity categories and is not categorized for ecotoxicity (UNECE 2015).

### 8.2.8 C8-18 Alkyldimethylbenzyl Ammonium Chlorides

ABAC is predicted to be GHS category 2 for acute oral toxicity, and for acute aquatic toxicity, it is predicted to be a category 1 (UNECE 2015). The National Sanitation Foundation Drinking Water Standards indicate a short-term exposure limit of 5 mg/L, a single product allowable concentration of 0.3 mg/L, and a total allowable concentration of 3 mg/L (NSF International 2019).

### 8.2.9 Solvent Naphtha (Petroleum), Heavy Aromatic [CAS 64742-94-5]

No regulations directly relevant to this formulation were found. A related mixture is assigned an OSHA TWA PEL of 500 ppm (2,000 mg/m$^3$) and a NIOSH REL of 350 mg/m$^3$ (NIOSH 2018b).

Several of the components of HAN are listed under Pennsylvania Right-to-Know requirements (Chevron-Phillips 2018).

### 8.2.10 Nonylphenol, Ethoxylated

NPEO is expected to be a GHS category 4 for acute toxicity. It is a category 2 for skin and eye irritation, as it can cause eye damage after prolonged exposure; therefore, is also considered a category 1 (UNECE 2015). It is a category 2 for aquatic chronic and for acute aquatic, can vary from category 1 to category 2. No other regulations or standards were found.

### 8.2.11 Propylene Glycol n-Butyl Ether

PGBE is predicted to be a GHS category 2 for skin and eye irritation, and a category 2B for respiratory sensitization (UNECE 2015). It is predicted to be a GHS category 5 for acute oral toxicity, and category 3 for acute aquatic toxicity.

### 8.2.12 Alcohols, $C_{9-11}$, Ethoxylated

No regulations or standards were found for CAS 68439-46-3.

### 8.2.13 IDPA Ester Salts

No regulations or standards were found.

### 8.2.14 Methanol

NIOSH has established a REL of 200 ppm as a TWA; the OSHA PEL is also 200 ppm. The IDLH concentration is 6,000 ppm (NIOSH 2016).

The ACGIH TLV is also 200 ppm as a TWA (HSDB 2012).

There is no Federal standard for methanol in drinking water, but the following states have enacted the indicated limits: Minnesota, 3,000 µg/L; New Hampshire, 4,000 µg/L; Wisconsin and Florida, 5,000 µg/L (HSDB 2012).

### 8.2.15 Orange Terpenes

Orange terpenes is a GHS category 5/not-categorized for acute oral toxicity. It is a category 2 for skin irritation and a category 1B for skin sensitization (UNECE 2015). It is a category 1 for acute and chronic aquatic toxicity.

### 8.2.16 Alcohols, $C_{12-15}$, Ethoxylated

This mixture is categorized as an acute hazard to the aquatic environment, GHS Category 1, it is not categorized according to GHS for acute oral toxicity (UNECE 2015).

### 8.2.17 Isoparaffinic Hydrocarbon

A TWA exposure limit has been established by a manufacturer for internal use at a vapor concentration 177 ppm (ExxonMobil 2010).

### 8.2.18 Dimethyl Adipate

DMA is uncategorized for acute toxicity according to GHS, but for aquatic toxicity it is a category 3 chemical (UNECE 2015).

### 8.2.19 Diethylhexyl Sodium Sulfosuccinate

DOSS is GHS category 4 for acute toxicity, category 2 for skin irritation, category 1 for eye damage, and category 3 for acute aquatic toxicity (UNECE 2015).

### 8.2.20 Butyl-3-Hydroxybutyrate

BHB is not categorized by GHS for any category, except for eye irritation, where it is a category 2 (UNECE 2015). No other regulations or standards were found.

### 8.2.21 Decamethylcyclopentasiloxane

D5 is predicted to be a GHS category 5 or uncategorized for acute oral toxicity. It is a category 1 chemical for acute aquatic toxicity. No other regulations or standards were found (UNECE 2015).

### 8.2.22 Octamethylcyclotetrasiloxane

D4 is predicted to be a GHS category 5 for acute oral toxicity. It is a category 1 chemical for acute aquatic toxicity. No other regulations or standards were found (UNECE 2015).

### 8.2.23 Dipropylene Glycol Dimethyl Ether

DPGMDE is a GHS Category 2 for skin and eye irritation (UNECE 2015). The SDS for Proglyde DMM indicates a company exposure limit of 20 ppm (TWA) (Dow Chemical Company 2018). NIOSH exposure standards were not found for DPGDME; however, the closely related dipropylene glycol monomethyl ether has a NIOSH TWA REL of 100 ppm (600 mg/m3), which also corresponds to the OSHA PEL (NIOSH 2016).

### 8.2.24 Dipropylene glycol monobutyl ether

DGME is predicted to be a GHS category 4 for acute toxicity. For skin and eye irritation, it is a category 2 chemical (UNECE 2015).

### 8.2.25 Hexylene glycol

Hexylene glycol is predicted to be a GHS category 5 for acute oral, category 1 for acute inhalation, and not categorized for aquatic toxicity (UNECE 2015). The NIOSH REL-C is 25 ppm (125 mg/m$^3$), the ACGIH TLV-TWA is 25 ppm for the vapor fraction while the STEL is 50 ppm for the vapor fraction and 10 mg/m$^3$ for the inhalable particulate matter, Cal/OSHA has a PEL-C of 25 ppm (125 mg/m$^3$) (NIOSH 2016; ACGIH 2018; CalEPA 2018b).

### 8.2.26 Diethyleneglycol Monobutyl Ether (DEGBE)

No regulations or standards relevant to DEGBE were found. It is predicted to be a GHS category 5 for acute oral and inhalation and a category 3 for acute aquatic toxicity (UNECE 2015).

### 8.2.27 Capramide DEA

No regulations or standards were found. It is predicted to be a GHS category 1 for inhalation toxicity and a GHS category 2 for aquatic algae (UNECE 2015).

### 8.2.28 Ethyl Lactate

Ethyl lactate is predicted to be a GHS acute oral toxicity category 5, and for acute aquatic toxicity, it is not categorized (UNECE 2015).No regulatory limits were found.

### 8.2.29 1-propoxy-2-propanol

No regulations or standards pertaining to P2P were found. It is anticipated to be a GHS category 5 for acute oral toxicity and not categorized for aquatic toxicity (UNECE 2015).

## 8.3 Formulation Summaries

### 8.3.1 Eastman MPK

Both components of Eastman MPK have fairly low toxicity concerns, although MPK is listed on the EPA HAP list. The formulation may cause both ocular and dermal irritation. There is concern that the MIBK may be a carcinogen, although it is present in the formulation at less than 10%.

### 8.3.2 Aerogreen 4015/Aerogreen 4065

The primary component for both formulations is 2-butoxyethanol. They are skin and eye irritants and have long-term aquatic toxicity concerns. It is not expected that these formulations will be carcinogenic, despite being mutagenic with some concerns for reproductive toxicity.

### 8.3.3 Ardrox JC-5

Ardrox JC-5 is primarily made up of a proprietary component, so full analysis of the formulation's toxicity is not possible. The other listed components of the formulation are sodium disilicate and ammonium hydroxide. The formulation is considered to be GHS category 1 for ocular and dermal irritation (UNECE 2015).

### 8.3.4 Bonderite C-AK 6871

The two primary components of this formulation have no toxicity data available, so a full analysis is not possible. For the remaining two components (DEA and DPGME), DEA is a severe ocular and dermal irritant. The relatively low percent DEA (<5%) may reduce the irritation severity; however, the hazard warning for the product does include skin and eye irritation as well as skin sensitization, as well as targeted organ toxicities and the potential to be a carcinogen.

### 8.3.5 Calla 804

The primary component of this formulation does not appear to be a major hazard, although the secondary component (present at < 1%) does have a high acute oral toxicity concern. The total formulation does cause a concern for dermal and ocular irritation, so appropriate handling and PPE is recommended.

### 8.3.6 CeeBee R-681 Wipes

CeeBee R-681 wipes contain a mixture of solvent naptha, 2-BE, and nonylphenol ethoxylated. According to the product SDS, the product may be carcinogenic and that there are concerns related to negative impacts on fertility or damage to an unborn child. Additionally, the formulation may be hazardous to aquatic life over a chronic exposure, indicating a need to control releases to the environment, even in small quantities. These concerns are primarily related to the presence of the nonylphenol, ethoxylated compound.

### 8.3.7 CeeBee Super Bee 210

This formulation is a major concern regarding dermal and ocular exposures, with a high concern for dermal irritation/corrosion. The primary component of the formulation (PGBE) is the driver for this concern, as it is present at 6–10%; however, all of the components pose moderate dermal and ocular hazards.

### 8.3.8 Chemsol Wipes

Chemsol wipes are made up of orange terpenes, alcohols ($C_{12-15}$) ethoxylated, isoparaffinic hydrocarbons, dimethyl adipate, and diethylhexyl sodium sulfosuccinate. Individual components may cause narcotic effects; however, none are present at levels high enough in the final formulation to cause concern. Additionally, there may be some concern with exposure causing mild skin irritation. Environmental releases should be controlled due to moderate aquatic toxicity.

### 8.3.9 Eastman Omnia Solvent

A full assessment of the Eastman Omnia solvent is not possible due to the presence of a proprietary component. However, it is listed as less than 1% of the final formulation, which is made up primarily of butyl-3-hydroxybutyrate. The primary concern for this compound is eye irritation.

### 8.3.10 Ecolink 250-SS

Ecolink 250-SS is comprised of multiple siloxanes and dipropylene glycol dimethyl ether. The primary concerns around this formulation are that it is an ocular irritant and that the octamethylcyclotetrasiloxane present in the formulation may cause damage to fertility or an unborn child. No data were available on aquatic toxicity for the formulation according to the SDS; however, both siloxanes present have high aquatic toxicity and are both present at high concentrations in the formulation; therefore, environmental releases are a concern due to the high aquatic toxicity.

### 8.3.11 Ecolink NAVSOLVE

Ecolink NAVSOLVE is, like Ecolink 250-SS, primarily composed of two siloxanes, additionally containing dipropylene glycol, n-butyl ether, and hexylene glycol. This formulation poses a dermal and ocular irritation hazard, as well as an aquatic toxicity hazard. The full formulation is listed as a potential chronic aquatic toxicant, but the two siloxanes in the formulation are also acutely toxic in an aquatic environment. The octamethylcyclotetrasiloxane present in the formulation may cause damage to fertility or an unborn child.

### 8.3.12 LPS A-151

LPS A-151 is comprised of petroleum distillates and a proprietary component. A full evaluation is not possible. Based on the SDS provided, there is concern for dermal and ocular irritation. It is a severe aspiration hazard that may lead to chemical pneumonia and there may be additional issues with narcosis following exposure.

### 8.3.13 Pantheon Formula 223

The SDS for this formulation provides some information on its toxicity, where it may be a skin irritant. However, no additional data are available and the individual components are listed as proprietary. No analysis may be made on this formulation.

### 8.3.14 Pantheon X-IT Aircraft Wash/ Pantheon X-IT Carbon Remover and Cleaner

These Pantheon formulations are primarily comprised of water, with the remainder made up of diethylene glycol monobutyl ether. The primary concerns for these formulations are dermal and ocular irritation. There may be some concerns for low chronic aquatic toxicity as well.

### 8.3.15 Penair C-5572

Penair C-5572 is comprised of Alcohol, $C_{9\text{-}11}$, ethoxylated, capramide DEA, and water. The primary concerns for this mixture are dermal and ocular irritation and aquatic toxicity. Capramide DEA may also pose a cancer concern; however, it is present at no more than 10% of the formulation, so exposure is relatively low. The alcohols are the primary concern, but as they are not more than 20% of the formulation, their relative oral toxicity risk is somewhat mitigated.

### 8.3.16 Socomore DS-108

Socomore DS-108 is comprised of ethyl lactate and 1-propoxy-2-propanol. It is a severe ocular irritant, with both components contributing to this endpoint. According to the SDS, it is also an aspiration hazard, although the actual acute toxicity via inhalation is likely low. The 1-propoxy-2-propanol may cause developmental toxicity; however, exposure would have to be extremely high.

### 8.3.17 TDA Research SSDX-12

No data are available on the components of this formulation, and the SDS does not provide any additional information on the toxicity of the full formulation. No analysis may be made on this formulation.

## 9. CONCLUSIONS

These formulations are primarily comprised of organic solvents, formulated to clean aircraft and ground vehicles, and were selected for their decrease in the use of hazardous air pollutants, as compared to the currently used formulation, Eastman MPK. The bulk of the formulations were able to be adequately assessed due to transparency in their formulations; however, a few were not due to listing of proprietary components. The majority of these formulations are dermal and/or ocular irritants; therefore, it is recommended that in the use of any of these formulations appropriate PPE be employed (such as coveralls, face shields/goggles, chemical protective globes, and respirators) when necessary. Additional engineering controls should also be in place to limit potential contact with the formulations, and environmental releases should be tightly controlled. Unused portions of the material should be recycled, returned to the manufacturer or supplier, or disposed of as hazardous waste. Ultimate disposal of the chemical must consider the material's impact on air quality; potential migration in air, soil, or water; effects on animal, aquatic, and plant life; and conformance with environmental and public health regulations.

Table 4 includes a matrix indicating degree of hazard for all the formulations. Where available, the SDS was utilized to make these determinations; however, some professional judgement was utilized where data were not provided based on the toxicity of individual components of the formulations.

**Table 4. Systemic Toxicity Results for Formulations [a]**

| | Skin Irritation | Eye Irritation | Skin Sensitization | Carcinogenicity | Spec. Organ Tox. Single | Spec. Organ Tox. Repeated | Germ Cell Mutagenicity | Acute Oral Toxicity | Acute Inhal. Toxicity | Respiratory Sensitization | Narcotic Effects | Aquatic toxicity | # of GHS Codes | State Right to Know |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Eastman MPK, UHP | NC | 2B | NC | 2B | ND | ND | NC | 4 | ND | NC | ND | NC | 2 | CA |
| Aerogreen 4015 | 3 | 2B | ND | 3[b] | ND | ND | ND | 5 | ND | ND | ND | 4 | 0 | NL |
| Aerogreen 4065 | 3 | 2B | ND | 3[b] | ND | ND | ND | 5 | ND | ND | ND | 4 | 0 | NL |
| Ardrox JC-5 | 1A | 1 | ND | ND | ND | ND | ND | 5 | ND | ND | ND | ND | 1 | MA, PA, NJ |
| Bonderite C-AK 6871 | 2 | 1 | 1 | 2B | Y | Y | ND | ND | ND | ND | ND | ND | 2 | CA |
| Calla 804 | 1 | 2 | ND | ND | ND | ND | ND | ND | ND | ND | ND | 1 | 1 | NL |
| CeeBee R-681 Wipes | U | U | U | 1 | Y | Y | 2 | ND | ND | U | ND | 3 | 1 | NL |
| CeeBee Super Bee 210 | 1A | 1 | ND | NL | ND | ND | ND | ND | ND | ND | ND | ND | 1 | NL |
| Chemsol Wipes | 3 | N | NL | 3 | U | U | U | 4 | ND | ND | ND | 2 | 0 | NJ, IL |
| Eastman Omnia Solvent | NC | 2A | NC | NL | ND | ND | NC | 5 | 3 | ND | ND | 4 | 1 | NL |
| Ecolink 250-SS | NC | 2A | ND | NL | 2 | Y | 2 | ND | ND | ND | ND | 1[c] | 1 | PA, NJ |
| Ecolink NAVSOLVE | 2 | 2 | ND | NL | 2 | Y | 2 | ND | ND | ND | ND | 1[c] | 1 | FL, MA, NJ, WI, RI, PA |
| LPS A-151 | 2 | 2 | NC | NC | Y | Y | NC | 4 | 1 | NC | Y | U | 2 | MA, NJ, PA, RI, CA |
| Pantheon Formula 223 | 3 | U | U | NL | U | U | U | 5 | U | U | U | ND | 0 | FL, MA, NJ, PA |

**Table 4. Systemic Toxicity Results for Formulations (continued)**

| | Skin Irritation | Eye Irritation | Skin Sensitization | Carcinogenicity | Spec. Organ Tox. Single | Spec. Organ Tox. Repeated | Germ Cell Mutagenicity | Acute Oral Toxicity | Acute Inhal. Toxicity | Respiratory Sensitization | Narcotic Effects | Aquatic toxicity | # of GHS Codes | State Right to Know |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pantheon X-IT Aircraft Wash | 2 | 2 | ND | NL | ND | ND | ND | 5 | ND | ND | ND | 4 | 1 | FL, MA, PA, NJ |
| Pantheon X-IT Carbon Remover and Cleaner | 2 | 2 | ND | NL | ND | ND | ND | 5 | ND | ND | ND | 4 | 1 | FL, MA, PA, NJ |
| Penair C-5572 | 2 | 2A | NC | NC | ND | ND | NC | 5 | ND | NC | ND | 2 | 1 | NJ |
| Socomore DS-108 | ND | 1 | ND | NL | Y | ND | ND | ND | 1 | ND | ND | ND | 4 | CA, MA, NJ, PA |
| TDA Research SSDX-12 | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | ND | 0 | MA, PA, NJ, CA |

Notes:
[a] Current as of publication date
[b] SDS states that primary component is not listed as a carcinogen, IARC classifies it as Group 3 (IARC 2006).
[c] SDS indicates that no acute toxicity data are available, but primary components are both highly acutely toxic (GHS Category 1).
GHS Categories 1–5 (acute toxicity); skin (1A–3); eye (1–2B); aquatic [Ecological] toxicity (1–5), See Appendix B
Green fill – classified as low hazard
Yellow fill – classified as moderate hazard
Orange fill – classified as moderate/high hazard
Red fill – classified as high hazard
Magenta fill – classified as extreme hazard
Blue fill – classified as unknown hazard
Legend:
Y = positive without categorization
NC = no category
ND = no data for mixture
U = insufficient data for mixture; evidence for constituents
NL = Not listed by any states

## 10. RECOMMENDATIONS

While these formulations are all commercially available, comprehensive information on some of the components of these formulations was not accessible. Product manufacturers' are often unwilling to share critical test data and there are not current regulations that require them to provide these data. Further, many chemicals in commerce do not have full data packages and there is an over reliance on "presumed safe" and regulatory bypass (grandfathered) status. Based on the available data, Pantheon Formula 223 has the most comprehensive data and appears to have lower toxicity concerns than the reference formulation Eastman MPK. However, a fully comprehensive assessment of all of these formulations should be undertaken and that approval for use should not be granted until more quantitative information on the hazards of all the formulations is provided. Samples of the leading candidate(s) should be submitted for toxicity analysis.

## 11. POINT OF CONTACT

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# APPENDIX A

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# APPENDIX B

# ENVIRONMENTAL SAFETY AND OCCUPATIONAL HEALTH SEVERITY CATEGORIZATION

## B.1 APHC CATEGORIZATION CRITERIA

**Table B-1. Categorization Criteria used in the Development of Environmental Safety and Occupational Health Severity[a]**

| | Low | Moderate | High | Unknown |
|---|---|---|---|---|
| PERSISTENCE | Readily biodegrades (<28 days) | Degradation ½ life: water <40 days, soil <120 days | Degradation ½ life: water >40 days soil > 120 days | Data are unavailable, insufficient, or unreliable. |
| TRANSPORT | Water sol. < 10 mg/L log $K_{OC}$ > 2.0 | Water sol. 10–1000 mg/L log $K_{OC}$ 2.0–1.0 | Water sol. > 1000 mg/L log $K_{OC}$ <1.0 | |
| BIOACCUMULATION | log $K_{OW}$ <3.0 | log $K_{OW}$ 3.0–4.5 | log $K_{OW}$ >4.5 | |
| TOXICITY | No evidence of carcinogenicity/ Mutagenicity (IARC group 3 & 4); Subchronic LOAEL > 200 mg/kg-d | Mixed evidence for carcinogenicity/mutagenicity (IARC group 2B) Subchronic LOAEL 5–200 mg/kg-d | Positive corroborative evidence for carcinogenicity (IARC group 1 & 2A)/ mutagenicity; LOAEL < 5 mg/kg-d | |
| ECOTOXICITY | Acute $LC_{50}/LD_{50}$ >1 mg/L or 1,500 mg/kg; Subchronic $EC_{50}$ >100 µg/L or LOAEL >100 mg/kg-d | Acute $LC_{50}/LD_{50}$ 1-0.1 mg/L or 1,500–150 mg/kg; Subchronic $EC_{50}$ 100-10 µg/L or LOAEL – 10–100 mg/kg-d | Acute $LC_{50}/LD_{50}$<100 µg/L or <150 mg/kg; Subchronic LOAEL <10 mg/kg-d | |

Legend:
mg/L = milligrams per liter
$K_{OC}$ = soil organic carbon-water partitioning coefficient
$K_{OW}$ = octanol-water partition coefficient
IARC = International Agency for Research on Cancer
mg/kg-d = milligrams per kilogram per day
LOAEL = lowest-observed adverse effect level
$LC_{50}$ = median lethal concentration; concentration expected to result in 50% mortality to a population of test animals
$LD_{50}$ = median lethal dose; dose resulting in 50% mortality
$EC_{50}$ = half maximal effective concentration
µg/L = micrograms per liter
Note:
[a]Modified from Howe, et al. (Howe 2006)

## GLOBALLY HARMONIZED SYSTEM

GHS is the acronym for the Globally Harmonized System of Classification and Labeling of Chemicals. The GHS attempts to establish international consensus for defining health, physical, and environmental hazards of chemicals; creating a classification process for comparison with defined hazard criteria; and communicating hazard information and protective measures on labels and Safety Data Sheets (SDS), (formerly known as Material Safety Data Sheets (MSDS)). The GHS attempts to reduce differences among levels of protection for workers established by the different countries and reduce regulatory burden and barriers to commerce while establishing consistent standards for classification.

The GHS is the result of an international mandate adopted in the 1992 United Conference on Environment and Development, often called the "Earth Summit." The harmonization and classification of chemicals was one of six program areas endorsed by the United Nations General Assembly to strengthen international efforts in the environmentally sound management of chemicals.

While there are several aspects of the GHS, the most important area for our purposes is classification of chemicals into various hazard categories based upon their effects and the route of exposure. Tabular extracts of the criteria for acute toxicity (both oral and inhalation), dermal, and ocular effects are included below. More information can be found in the original source (OSHA 2012).

**Table B-2. GHS Acute Toxicity**

| | Category 1 | Category 2 | Category 3 | Category 4 | Category 5 |
|---|---|---|---|---|---|
| Oral (mg/kg) | ≤5 | >5 ≤50 | >50 ≤300 | >300 ≤2000 | Criteria:<br>-Anticipated $LD_{50}$ between 2000 and 5000 mg/kg<br>-Indication of significant effects in humans.<br>-Any mortality in Category 4<br>-Significant clinical signs in Category 4<br>-Indications from other studies.<br><br>*If assignment to a more hazardous class is not warranted. |
| Dermal (mg/kg) | ≤50 | >50 ≤200 | >200 ≤1000 | >1000 ≤2000 | |
| Gases (ppm) | ≤100 | >100 ≤500 | >500 ≤2500 | >2500 ≤5000 | |
| Vapors (mg/L) | ≤0.5 | >0.5 ≤2.0 | >2.0 ≤10 | >10 ≤20 | |
| Dusts & Mists (mg/L or $g/m^3$) | ≤0.05 | >0.05 ≤0.5 | >0.5 ≤1.0 | >1.0 ≤5 | |

Legend:
mg/kg = milligrams per kilograms
ppm = parts per million
mg/L = milligrams per liter
**$LD_{50}$ = dose resulting in 50% mortality**

**Table B-3. GHS Skin Corrosion/Irritation/Sensitization**

| Category 1A | Category 1B | Category 1C | Category 2 | Category 3 | Not Categorized |
|---|---|---|---|---|---|
| **Corrosion**<br>< 3 minutes Observation < 1 hour | **Corrosion**<br>< 1 hour Observation < 14 days | **Corrosion**<br>< 4 hours Observation < 14 days | **Irritation**<br>Reversible adverse effects in dermal tissue<br>Draize score: ≥ 2.3, <4.0, or persistent inflammation | **Mild Irritation**<br>Reversible adverse effects in dermal tissue<br>Draize score: ≥ 1.5, <2.3 | Corrosion and irritation not observed |
| Destruction of dermal tissue; visible necrosis in at least one animal.<br>**Sensitization: Category 1 if present** | | | | | |

**Table B-4. GHS Eye Effects**

| Category 1 | Category 2A | Category 2B | Not categorized |
|---|---|---|---|
| **Irreversible** damage 21 days after exposure | **Irritant** Reversible in 21 days | **Mild irritant** Reversible in 7 days | **Non-irritating** |

**Table B-5. GHS Acute and Chronic Aquatic Toxicity**

| **Acute Aquatic Toxicity** | | | | |
|---|---|---|---|---|
| **Category I** | **Category II** | **Category III** | | **Not Categorized** |
| Acute toxicity ≤ 1.00 mg/L | Acute toxicity > 1.00 but ≤10.0 mg/L | Acute toxicity > 10.0 but < 100 mg/L | | Acute toxicity > 100 mg/L |
| **Chronic Aquatic Toxicity when biodegradation ½ life is > 7 days** | | | | |
| **Category I** | **Category II** | **Category III** | **Category IV** | **Not Categorized** |
| Acute Cat I and log $K_{ow}$ ≥ 4, unless BCF < 500; Or chronic toxicity ≤ 0.01 mg/L | Acute Cat II and log $K_{ow}$ ≥ 4, unless BCF < 500; Or chronic toxicity 0.01-0.1 mg/L . | Acute Cat III and log $K_{ow}$ ≥ 4, unless BCF < 500; Or chronic toxicity 0.1-1.0 mg/L | Acute toxicity > 100.0 mg/L, biodegradation ½ life >7 days, and log $K_{ow}$ ≥ 4, unless BCF < 500; Or chronic toxicity > 1.0 mg/L | Acute toxicity >100 mg/L, Log $K_{ow}$ < 4, BCF< 500 and chronic toxicity > 1.0 mg/L |

Legend:
mg/L = milligrams per liter
BCF = Bioconcentration factor

**Table B-6. GHS Carcinogenicity**

| Category 1A | Category 1B | Category 2 | Not categorized |
|---|---|---|---|
| Known to have carcinogenetic potential for humans (human evidence) | Presumed human carcinogens (animal evidence) | Suspected human carcinogen (human or animal evidence but not sufficiently convincing to place in category 1) | Non-carcinogenic |

## Appendix C

## TOXICITY DATA TABLES

**Table C-1. Physical Properties (Sources: EPA 2015b, BIOVIA2015, PubChem 2020w)**

| Chemical Name | Molecular Weight (gms) | Melting Point (°C) | Boiling Point (°C) | Water Solubility (mg/L) | logP octanol/water $K_{OW}$ | Log octanol/carbon $K_{OC}$ | Henry's Law Constant atm-$m^3$/mole | Vapor Pressure (mmHg) |
|---|---|---|---|---|---|---|---|---|
| Methyl propyl ketone | 86.13 | -76.9 | 102.2 | 4.30E+04 | 0.915 | 1.88 | 8.36 E-05 | 35.4 |
| Methyl isobutyl ketone | 100.16 | -66.73 | 104.57 | 8888 | 1.31 | 1.1 | 1.38E-4 | 19.9 |
| 2-butoxyethanol | 118.18 | -74.8 | 168.4 | 6.45E4 | 0.83 | 0.882 | 1.6E-6 | 0.475 |
| Sodium disilicate | 122.08 | 1089 | ND | 350,000 at 20°C | n/a | n/a | n/a | 0.0077 at 1175°C |
| Ammonium Hydroxide | 35.046 | -58 | 28 | Miscible | ND | ND | ND | 2160 |
| Diethanolamine | 105.14 | 28.77 | 231.58 | 1E6 | -1.43 | -0.732 | 3.92E-11 | 5.4E-4 |
| DPGME | 148.2 | -83 | 190.0 | Miscible | ND | ND | ND | 0.4 @ 26°C |
| dipropylene glycol, n-butyl ether | 190.29 | -75 | 248.4 | 92955 | 0.957 | | 5.303 E-03 | 0.00361 |
| diethylene glycol monobutyl ether (DGBE) | 162.23 | -68 | 230 | 3.384 E+05 | 0.642 | | 3.235 E-08 | 2.19 E-02 |
| C8-18-alkyldimethylbenzyl ammonium chloride | 311.94 | 219.33 | 514.42 | 227.6 | 1.95 | 4.913 | 4.32E-12 | 9.95E-11 |
| HAN | Varies | ND | 165-290 | Negligible | ND | ND | ND | 0.05 |
| Nonylphenol, ethoxylated | 308.47 | 140.16 | 404.90 | 1.051 | 5.3 | 3.394 | 2.56E-9 | 9.14E-9 |
| Propylene glycol n-butyl ether | 132.2 | -21.73 | 171.5 | 4.21E4 | 0.980 | 0.965 | 4.8E-8 | 0.397 |
| Alcohols, $C_{9-11}$, ethoxylated | Mixture | -20 | 180 | 1540 | 2.42 | 1.4 | 3.13E-14 | 0.242 |
| IDPA Ester Salts | ND | ND | ND | ND | ND | ND | ND | ND |
| Methanol | 32.04 | -97.8 | 64.7 | Miscible | -0.77 | 2.75 | 4.55E-6 | 127 |
| Orange Terpenes | 136.24 | -40.76 | 178 | 13.8 | 4.57 | 3.049 | 3.8E-1 | 1.44 |
| Alcohols, $C_{12-15}$, ethoxylated | Varies | 2-26 | 260 | 1E6, may form gel | 3 | ND | ND | <10 |
| Distillates, petroleum hydrotreated, light | Varies | <-45.5 | 251-320 | 0.041 | 5.72 | | 9.34E-4 | 2.45E-4 |
| Dimethyl Adipate | 174.2 | -71.54 | 186.96 | 6000 | 1.03 | 1 | 9.77E-7 | 0.687 |
| Diethylhexyl sodium sulfosuccinate | 444.56 | 298.5 | 683.89 | 0.061 | 2.881 | 2.881 | 5.2E-12 | 1.22E-14 |
| Butan-1-yl-3-hydroxybutanoate (butyl-3-hydroxybutyrate) | 160.21 | 6.69 | 226.3 | 1.75E4 | 1.29 | 1.16 | 1.2E-8 | 0.015 |
| decamethylcyclopentasiloxane | 370.77 | -5.19 | 196.78 | 0.013 | 8.06 | 1.25E5 | 4.2E1 | 0.2 |
| octamethylcyclotetrasiloxane | 296.62 | 1.78 | 159.41 | 0.06 | 6.74 | 4.21 | 1.34 | 1.05 |
| dipropylene glycol dimethyl ether | 162.23 | <-71 | 175 | 5.3E5 | 0.42 | 0.87 | 3.67E-7 | 0.55 |
| Dipropylne glycol monomethyl ether | 190.28 | 17.98 | 248.4 | 45000 | 1.13 | 1 | 2.6E-9 | 0.00361 |
| hexylene glycol | 118.18 | -50 | 198 | Miscible | 0.58 | 0.423 | 4.06E-7 | 7E-2 |
| DEGBE | 162.23 | -68.4 | 231 | Miscible | 0.56 | 0.642 | 7.2E-9 | 0.02 @ 20°C |
| Capramide DEA | 259.39 | 145.77 | 407.43 | 497.3 | 1.9 | 1.076 | 1.89E-12 | 2.76E-9 |
| Ethyl lactate | 118.13 | -27.76 | 166.19 | 4.7E5 | -0.18 | 0.348 | 4.82E-5 | 1.08 |
| 1-propoxy-2-propanol | 118.2 | -80 | 150 | Miscible | 0.621<br>0.49 | 0.694 | 2.64E-7 | 1.7[a] at 20°C |

**Legend:**
**g/mol = grams per mole**
**mg/L = milligrams per liter**
atm-$m^3$/mol = air to mol per cubic meter

**Table C-2. Toxicity Data Summary**

| Compound (Chemical formula) | Acute Oral $LD_{50}$ (mg/kg) | Chronic Oral LOAEL (mg/kg-d) | Inhalation $LC_{50}$ ($mg/m^3$-h) | Dermal (Corrosion; Irritation; Sensitization) | Eye Effects (Corrosion; irritation) | Genotoxicity | Carcinogenicity |
|---|---|---|---|---|---|---|---|
| MPK | 1,600 (rat/mouse)[a] | ND | 22000[a] | Negative | Irritant | Negative | Unlikely |
| MIBK | >2,000 (rat)[a] 1900 (mice)[a] | ND | 100 (rat)[a] | Negative | Mild | Negative | Positive in animals |
| 2-BE | 250-1,500(rat)[a] 1,230 (mice)[a] 320 (rabbit)[a] | ND | >1000[a] | Irritant | Irritant | Positive[c] | Equivocal |
| Sodium disilicate | 1,100-1,600[a] | ND | ND | Corrosive and Irritant | Irritant | Negative[d] | Negative[e] |
| Ammonium Hydroxide | 350[a] | ND | 408 ppm[a] | Corrosive and Irritant[a] | Severe Irritant[a] | Positive[a] | Negative[a] |
| DEA | 1,600 (rat)[b] | ND | 150 (Rat, LOAEL sub-chronic)[f] | Moderate irritation, some evidence of senstization | Severe Irritant[a] | Negative | Evidence in animals, not in humans |
| DPGME | 5,000 (rat)[b] | 1000 (rat)[b] | >275 (rat)[b] | Negative | Negative | Negative | Negative |
| ABAC | 150 (rat)[a] | ND | ND | ND | ND | ND | ND |
| HAN | 7,050 (rat)[g] | 300 (rat)[g] | 590[a] | Negative | Irritant | Negative | Possibly |
| NPEO | 1,310 (rat)[h] >4000 (mouse)[b] | NOEL = 1000 (rat)[i] | ND | Irritant | Irritant | Negative | ND |
| PBGE | 3,300[j] | 1,000[a] | >5.83 | Irritant | Irritant | Negative | ND |
| Alcohol, $C_{9-11}$, Ethoxylated | 1,378[k] | ND | >1.600[l] | Irritant[11] | Probable irritant | Negative[m] | Unlikely |
| IDPA Ester Salts | ND | ND | ND | ND | ND | ND | ND |
| Methanol | 5,628 (rat)[a] 1,000 (human)[a] | ND | 0.109[a] | Irritant | Irritant | Negative | Negative |
| Orange Terpenes | >5,000 (rodent)[a] | 1,000 (dog)[b] | ND | Irritant Sensitizer | Irritant | Negative | Negative |
| Alcohols, $C_{12-15}$, ethoxylated | 5,000 (rodent)[b] | ND | ND | Negative | Negative | Negative | Negative |
| Distillates, petroleum hydrotreated, light | >5000[b] | ND | >1820[b] | Mild irritant | Mild irritant | Negative | Negative |
| DMA | >5,000[b] (rat) | 980 (rat)[b] | 11000 (rat)[b] | Negative | Negative | Negative | Negative |
| DOSS | 1,900 (rat)[a] 2,600 (mouse)[b] | NOAEL = 500 (rat)[b] | 20000 (rat)[b] | Irritant | Severe Irritant | Negative | Negative |
| BHB | >5,000 (rat)[b] | >1000 (rat)[b] | >5000 (rat)[a] | Negative | Irritant | Negative | ND |

**Table C-3. Toxicity Data Summary (continued)**

| Compound (Chemical formula) | Acute Oral $LD_{50}$ (mg/kg) | Chronic Oral LOAEL (mg/kg-d) | Inhalation $LC_{50}$ ($mg/m^3$-h) | Dermal | Ocular | Genotoxicity | Carcinogenicity |
|---|---|---|---|---|---|---|---|
| D5 | >5000 (rat)[b] | ND | 8,600 (rat)[b] | Negative | Mild irritant | Negative | Equivocal |
| D4 | 4,800 (rat)[b] | ND | 36000[a] | Mild Irritant | Mild Irritant | Reproductive Toxicant | Positive (rat)[a] |
| DPGDME | 3300[n] | 24.0[o] | >5250[n] | Mild Irritant[n] | Mild Irritant | Negative | Negative |
| DGME | 1513-9,100[a] | ND | 4,500[b] | Irritant | Negative | Negative | Negative |
| Hexylene glycol | 3,700 (rat)[a] 2,800 (guinea pig)[a] 3,097 (mouse)[a] | ND | 310 (rat)[a] | Irritant Sensitizer | Irritant | Negative | Negative |
| DEGBE | 3,306[a] | 1,500[o] | >10000[o] | Negative | Irritant | Negative | Negative |
| Capramide DEA | >10,000 (rat)[n] | 218.6[o] | 34[o] | Negative[o] | Mild[o] | Negative[o] | Positive[o] |
| Ethyl lactate | 8,200 (rat)[a] 2,500 (mouse)[a] | ND | 1,800 (rat)[p] | Negative | Irritant | Negative | Negative |
| P2P | 2,840[a] | 50[n] | >10000[n] | Mild irritant[a] | Moderate irritant | Negative[b] | Negative[a] |

Legend:
ND = No data
Notes:
[a] PubChem 2020w
[b] ECHA 2020i
[c] Elliott and Ashby 1997
[d] Sigma-Aldrich 2014
[e] Fisher 2018
[f] Gamer et al. 2008
[g] Cisco 2015
[h] Lewis 1999
[i] EPA 2019e
[j] UNEP 2003
[k] ChemIDPlus 2018
[l] Williams et al. 2017
[m] Gingell and Lu 1991
[n] Dow Chemical Company 2018
[o] BIOVIA 2015
[p] Clary 1998

**Table C-3. Toxicity Assessment and GHS Categorization**
**(See Appendix B for GHS Classification and Coloring Scheme)**

| Compound | Oral | Inhalation | Dermal | Ocular | Carcinogenicity | Aquatic | Invertebrates | Plants | Birds |
|---|---|---|---|---|---|---|---|---|---|
| MPK | 4 | 5 | NC | 2A | | NC | NC | | |
| MIBK | 5 | 5 | NC | 2B | | NC | NC | | |
| 2-BE | 4 | 5 | 3 | 2B | | NC | NC | | |
| Sodium disilicate | 4 | 3 | 1B | 1 | | NC | | | |
| Ammonium hydroxide | 4 | 2 | 1B | 1 | | 1 | | | |
| Alcohol $C_{12-15}$ poly(1-6) ethoxylates | | | | | | | | | |
| Coconut diethanolamide | | | | | | | | | |
| DEA | 4 | 5 | 1 | 1 | | NC | NC | | |
| DPGME | 5 | 5 | NC | NC | | NC | | | |
| ABAC | 3 | | | | | 1 | | | |
| Naphtha | 5 | 5 | NC | 1 | 2 | 2 | | | |
| NPEO | 4 | | 3 | 2B | | 2 | 2 | | |
| PGBE | 5 | 3 | 2 | 2B | | NC | NC | | |
| Alcohols, $C_{9-11}$, ethoxylated | 4 | | 2 | 2B | | 2 | 2 | | |
| IDPA Ester Salts | | | | | | | | | |
| Methanol | 4 | 4 | 2 | 2B | | NC | NC | | |
| Orange terpenes | 5 | | 1 | 2B | | 1 | 1 | | |
| Alcohols, $C_{12-15}$, ethoxylated | 5 | | NC | NC | | 1 | 1 | | |
| Distillates, petro.hydrotreated, light | 5 | 4 | 2 | 2B | | 2 | | | |
| Dimethyl adipate | 5 | 5 | NC | NC | | 3 | 3 | | |
| DOSS | 4 | 5 | 2 | 1 | | 3 | 3 | | |
| BHB | 5 | 5 | NC | 2A | | NC | NC | | |
| D5 | 5 | 5 | NC | 2B | | 1 | 1 | | |
| D4 | 5 | 5 | 2 | 2B | 2 | 1 | 1 | | |
| DPGDME | 5 | 5 | 3 | 2B | | NC | NC | | |
| DGME | 4 | 4 | 3 | NC | | 3 | | | |
| Hexylene glycol | 5 | 2 | 1 | 2B | | NC | NC | | |
| DEGBE | 5 | 5 | NC | 2B | | NC | NC | | |
| Capramide DEA | 5 | 2 | NC | 2B | 2 | NC | 2 | | |
| Ethyl Lactate | 5 | 5 | NC | 2B | | NC | NC | | |
| P2P | 5 | 5 | 3 | 2A | | NC | NC | | |

## GLOSSARY

| | |
|---|---|
| ABAC | C8-18-Alkyldimethylbenzyl ammonium chlorides |
| ACGIH | American Conference of Governmental Industrial Hygienists |
| AE | Alcohol ethoxylate |
| AERTA | Army Environmental Research and Technology Assessment |
| AES | Alcohol ethoxy sulfate |
| APHC | U.S. Army Public Health Center |
| AR | Department of the Army Regulation |
| 2-BE | 2-butoxyethanol/2-butyl cellusolve |
| BCF | Bioconcentration Factor |
| BHB | Butyl-3-hydroxybutryrate |
| $^{o}C$ | Degrees Celsius |
| CAS RN | Chemical Abstract Service Registry Number (CAS #) |
| CFR | Code of Federal Regulations |
| CNS | Central Nervous System |
| D4 | Octamethylcyclotetrasiloxane |
| D5 | Decamethylcyclopentanesiloxane |
| DA | Department of the Army |
| DEA | Diethanolamine |
| DEGBE | Diethylene glycol monobutyl ether |
| DGME | Dipropylene glycol, n-butyl ether |
| DMA | Dimethyl adipate |
| DOD | Department of Defense |
| DODI | Department of Defense Instruction |
| DOSS | Diethylhexyl sodium sulfonsuccinate |
| DPGDME | Dipropylene glycol dimethyl ether |
| DPGME | Dipropylene glycol monomethyl ether |
| $EC_{50}$ | Effective concentration to achieve 50 percent effect |
| ECHA | European Chemical Agency |
| ECOSAR | Ecological Structure Activity Relationships |
| EPA | U.S. Environmental Protection Agency |
| ESOH | Environmental Safety and Occupational Health |
| g | grams |
| GHS | Globally Harmonized System |
| $g/m^3$-h | grams per cubic meter per hour |
| HAN | Solvent naphtha (petroleum), heavy aromatics/ heavy aromatic naphtha |
| HSDB | Hazardous Substances Data Bank |
| IARC | International Agency for Research on Cancer |
| $IC_{50}$ | Concentration causing 50 percent inhibition |
| IDLH | Immediately Dangerous to Life or Health |
| IDPA | 1H-imidazoledipropanoic acid |
| IRIS | Integrated Risk Information System |
| kg | Kilogram |
| $K_H$ | Henry's law constant |
| $K_{oc}$ | organic carbon-water partition coefficient |
| $K_{OW}$ | octanol-water partition coefficient |

| | |
|---|---|
| L | Liter |
| $LC_{50}$ | Concentration resulting in 50 percent mortality |
| $LC_{LO}$ | Lowest lethal concentration |
| $LD_{50}$ | Dose resulting in 50 percent mortality |
| LOAEL | Lowest observed adverse effect level |
| mg | milligram |
| MIBK | Methyl isobutyl ketone |
| mL | milliliter |
| mmHG | millimeters of mercury |
| mM | millimolar |
| Min | minutes |
| MP | Melting point |
| MPK | Methyl propyl ketone |
| MW | molecular weight |
| NIOSH | United States National Institute for Occupational Safety and Health |
| nm | nanometers |
| NOAEL | No observed adverse effect level |
| NOEL | No observed effect level |
| NPEO | Nonylphenol, ethoxylated |
| NTP | National Toxicology Program |
| OECD | Organisation for Economic Co-operation and Development |
| OSHA | Occupational Safety and Health Administration |
| P2P | 1-propoxy-2-propanol |
| PEL | Permissible exposure limit |
| PGBE | Propylene glycol n-butyl ether |
| PND | Post-natal day |
| PPE | Personal Protective Equipment |
| QSAR | Quantitative Structure-Activity Relationship |
| REL | Relative exposure limit |
| RfD | Reference dose |
| ROS | Reactive oxygen species |
| SDS | Safety Data Sheet |
| STEL | Short-term exposure limit |
| TLV | Threshold Limit Value |
| TOPKAT | Toxicity Prediction by Komputer Assisted Technology |
| TOX | Toxicology Directorate |
| TWA | Time-weighted average |
| µg | micrograms |
| µL | microliter |
| UNECE | United Nations Economic Commission for Europe |
| Unk | Unknown |
| USFDA | U.S. Food and Drug Administration |
| w/v | Weight per volume |