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Toxicology Assessment for Safer Alternatives for Readiness (SAFR) Work Unit TMR 15-01 Chromium Free Coating Tartaric Sulfuric Acid Anodizing (TSAA) March 2016–December 2019

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14. ABSTRACT Anodizing is an electrolytic process that increases the natural surface layer of oxide on the surface of metal parts, thereby increasing resistance to corrosion and wear. Chromic acid anodizing (CAA) has traditionally been used by the U.S. Army but the solutions utilized not only pose a significant human health hazard, but also require special handling for disposal that is technically complex and has high associated costs. In this work unit, the replacement of chromic acid (hexavalent chromium), a known carcinogenic metal, with a less toxic formulation for anodizing aluminum parts will reduce risks to occupational health and as well as environmental impact. While the tartaric sulfuric acid anodizing (TSAA) process includes chromium in trivalent form (Cr(III)), this form of chromium is considered non-carcinogenic, with the removal of hexavalent chromium (Cr(VI)) representing an important hazard reduction over legacy approaches to aluminum anodizing.

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COMMONLY USED TERMS

ACGIH
American Conference of Governmental Industrial Hygienists

AERTA
Army Environmental Requirement and Technology Assessment

AFC
Army Futures Command

Al
Aluminum

ALTBIB
Animal Testing Alternatives database

APHC
U.S. Army Public Health Center

AMCOM
Army Aviation and Missile Command

AR
Army Regulation

ARL
Army Research Laboratory

ATSDR
Agency for Toxic Substances and Disease Registry

BCF
Bioconcentration Factor

bp
Boiling point

CAS RN
Chemical Abstracts Service Registry Number

CAA
Chromic acid anodizing

CCAD
Corpus Christi Army Depot

CCDC
Combat Capabilities Development Command

CCRIS
Chemical Carcinogenesis Research Information System

CPDB
Carcinogenic Potency Database

Cr_2O_3
Chromium oxide

Cr(III)
Trivalent chromium

Cr(VI)
Hexavalent chromium

CTD
Comparative Toxicogenomics Database

DA
Department of the Army

DART
Developmental and Reproductive Toxicology

DNA
deoxyribonucleic acid

DTIC
Defense Technical Information Center

DWEL
Drinking water exposure limit

EC_{50}
Effective concentration to achieve 50% effect

ECOSAR
EPA's Ecological Structure Activity Relationships

ECOTOX
EPA ECOTOXicology Database System

EHS
Extremely hazardous substance

EPA
U.S. Environmental Protection Agency
EQT
Environmental Quality Technology

ESOH
Environment, Safety, and Occupational Health

FDA
U.S. Food and Drug Administration

GHS
Globally Harmonized System

GRAS
Generally recognized as safe

H_2SO_4
Sulfuric acid

IARC
International Agency for Research on Cancer

IRIS
Integrated Risk Information System

K_H
Henry's law constant

LC_{50}
Concentration resulting in 50% mortality

LD_{50}
Dose resulting in 50% mortality

LDLo
Lowest dosage causing death

LOAEL
Lowest observed adverse effect level

log K_{OC}
Organic carbon partition coefficient

log K_{OW}
Octanol-water partition coefficient

mg/kg
milligrams per kilogram

mg/kg-d
milligrams per kilogram per day

mg/L
milligrams per liter

MW
Molecular weight

mg/m^3
milligrams per cubic meter

mmHg
millimeters of mercury

NIH
National Institutes of Health

NIOSH
U.S. National Institute for Occupational Safety and Health

NLM
National Library of Medicine

NOAEL
No observed adverse effect level

NPDES
National Pollution Discharge Elimination System

NTP
National Toxicology Program

OSHA
Occupational Health and Safety Administration

PEL
Permissible exposure limit

pH
power of hydrogen

PHT
Potassium hydrogen tartrate

ppm
parts per million

QSAR
Quantitative Structure Activity Relationship

RCRA
Resource Conservation and Recovery Act

RDT&E
Research, Development, Testing, and Evaluation

RfD
Reference dose

SDS
Safety Data Sheet

TLV
threshold limit value

TOPKAT
Toxicity Prediction Komputer Assisted Technology

TOXNET
U.S. National Library of Medicine's Toxicology Data Network

TPQ
Threshold planning quantity

TSAA
Tartaric Sulfuric Acid Anodizing

TWA
Time weighted average

vp
Vapor pressure

WHO
World Health Organization

TOXICOLOGY REPORT NO. S.0052729.8-16
TOXICOLOGY ASSESSMENT FOR EQT WORK UNIT TMR 15-01: CHROMIUM FREE COATING (TARTARIC SULFURIC ACID ANODIZING) MARCH 2016–DECEMBER 2019

1. SUMMARY

1.1 Overview

Research, development, testing, training, and use of substances potentially less hazardous to human health and the environment is vital to the readiness of the U.S. Army. Safeguarding the health of Soldiers, Civilians, and the environment requires an assessment of proposed alternative chemicals/formulations before they are fielded. Continuous assessments begun early in the RDT&E process can save significant time and effort during RDT&E, as well as over the life cycle of development. Proactive assessment of proposed replacement materials and formulations early in the lifecycle will avoid these problems in the future.

1.2 Purpose

Anodizing is an electrolytic process that increases the natural surface layer of oxide on the surface of metal parts, thereby increasing resistance to corrosion and wear. Chromic acid anodizing (CAA) has traditionally been used by the U.S. Army; however, the solutions utilized pose a significant human health hazard and require special handling for disposal that is technically complex and has high associated costs. The purpose of this work unit is to demonstrate and assess Tartaric Sulfuric Acid Anodizing (TSAA) as a replacement for CAA.

1.3 Conclusions

While the use of certain metal ions is unavoidable in the electroplating industry, the continued use of electrolyte solutions containing (hexavalent) chromic acid represents a significant occupational and environmental hazard. Advancement of occupational and environmental regulations has promoted improvements in manufacturing processes, especially relating to the use of such toxic chemicals. Anodizing of aluminum parts has traditionally been carried out using CAA. In this work unit, the replacement of chromic acid (hexavalent chromium), a known carcinogenic metal, with a less toxic formulation for anodizing aluminum parts will reduce risks to occupational health and as well as environmental impact. The TSAA post-process may use trivalent chromium (Cr(III)) in the sealer; Cr(III) is considered non-carcinogenic, with the removal of Cr(VI) representing an important hazard reduction over legacy approaches to aluminum anodizing. While potential toxicological hazards associated with this new formulation remains (such as ocular, dermal, and inhalation effects), these hazards can be addressed with existing engineering and administrative controls while the overall effects associated with historical use of CAA are reduced by replacing with TSAA.

1.4 Recommendations

Although there are concerns associated with the acute effects of sulfuric acid (as mist) and tartaric acid, the formulations represent an improvement over CAA. Post-treatment sealers that contain Cr(III) are less toxic than Cr(VI) solutions; however, operators and the industrial l hygiene officers should carefully assess the occupational risks of exposure to hydrofluoric acid, if used. Appropriate PPE is necessary to protect workers from exposure to TSAA and all applicable local, state and federal guidelines for disposal/waste management should be followed.

2. REFERENCES

See Appendix A for list of references.

3. AUTHORITY

Funding for this work was provided under Military Interdepartmental Purchase Request 10756068. This Toxicology Assessment addresses, in part, the ESOH requirements outlined in the following DA Regulations and directives: AR 200-1, Environmental Protection and Enhancement, 2007; AR 40-5, Preventive Medicine, 2007; and AR 70-1, Army Acquisition Policy, 2018; Department of the Army Pamphlet 40-11, Preventive Medicine, 2005; Department of Defense Instruction (DoDI) 4715.4, Pollution Prevention, 1998; and AERTA requirement PP-2-02-04, *Toxic Metal Reduction in Surface Finishing Processes*, (AERTA 2012). The initial sponsor was the Environmental Technology Acquisition Program (ETAP), Army EQT Program, which has since transitioned to the CCDC Safer Alternatives for Readiness (SAFR) Program. The Principle Investigator is Mr. Scott Howison, Aviation and Missile Command.

4. BACKGROUND

This project will compare CAA, a traditionally used aluminum anodizing method at Corpus Christi Army Depot (CCAD) with TSAA, a process that replaces the chromic acid used in CAA with tartaric and sulfuric acid. Table 1 gives the composition of each formulation.

Current regulations require assessment of human health and environmental effects arising from exposure to substances in soil, surface water, and ground water, and in this case the occupational environment. When carried out after an item has been fielded, these post-hoc assessments can reveal the existence of adverse environmental and human health effects that must then be addressed, often at substantial additional cost. A more proactive approach would be to make an assessment of exposure, effects, and environmental transport of military-related compounds/substances early in the RDT&E process to avoid unnecessary costs, conserve physical resources, and sustain the health of our Forces and others potentially exposed.

To support this preventive approach, the APHC has created a phased approach to identify ESOH effects that would potentially impact readiness, training, and development

costs. This is an on-going effort, and this report represents the status of information available for this work unit as of the date of publication.

5. STATEMENT OF PROBLEM

Corrosion protection of metal parts is initially carried out by anodizing, where the metal part to be treated acts as an anode in an acidic solution and the chemical process increases the natural oxide layer on the surface of the metal. Traditional anodizing is a 16-step process with steps 12 and 14 consisting of both the anodize and the anodize-seal phases, respectively; both these steps use hexavalent chromium, as well as step 9, which is a deoxidizing step prior to anodizing. Follow-up treatment can include a sealant and painting as finishing steps. Chromic acid (hexavalent chromium)-based processes have traditionally been used in anodizing, but the toxicity and health effects associated with hexavalent chromium are an ongoing concern. There has been a general trend in the aerospace industry to move away from these processes. At the CCAD, metal parts for Army Aviation have traditionally been treated to prevent corrosion using CAA. The ARL and AMCOM, under the auspices of the EQTP program are now developing a replacement process called TSAA for use with Army aviation assets, which eliminates the use of hexavalent chromium. The TSAA process uses sulfuric acid/tartaric acid in the plating process, and a formulation with basic chromium (III) sulfate as a sealant.

The CAA process uses formulations that that may pose a human health hazard to workers, and must be disposed of as RCRA (Resource Conservation and Recovery Act) hazardous waste. Wastewater treatment must meet NPDES, EPA requirements, and must be reported to EPA as toxic releases (Feathers 2012).

6. METHODS

To determine the human health and environmental impact of compounds employed in these formulations, it is necessary to identify each compound correctly and its physical, chemical, and toxicological properties. The CAS RN is the primary way to identify each compound in this program (see Table 1). While all compounds do not necessarily have a single CAS RN, this number 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 non-systematic) names for chemical substances. In some cases, synonyms and trade names are also used to identify structures.

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

- Molecular weight (MW).
- Boiling point (bp).
- Octanol-water partition coefficient (log K_{OW}).
- Organic carbon partition coefficient (log K_{OC}).
- Water solubility.
- Henry’s law constant (K_H).
- Vapor pressure (vp).

Table 1. Formulation Components

Chemical Substance	CAS Number	Percentage Formulation
Sulfuric Acid (Puritan Products, Inc)		
Sulfuric Acid	7664-93-9	52-100
Water	7732-18-5	0-48
Tartaric Acid (ScienceLab.com, Inc)		
L- Tartaric Acid	87-69-4	100 (by weight)
Chemeon® TCP-HF		
Chromium sulfate basic solution	12336-95-7	<2[a]
Water	7732-18-5	>97
Prorietary mixture [a]	CBI	<1%

Note:
[a]Confidential Business Information; exact percentage of composition withheld.

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, mutagenesis and carcinogenesis; and mode(s) and mechanisms of toxicity. Toxicological information was derived directly from primary sources whenever possible.

Sources used in this search included *The Merck Index* (O'Neil 2006), the TOXNET® providing access to information from the NIH and the EPA, the U.S. Department of Health and Human Services' ATSDR, the EPA ECOTOX, and the DTIC®. Additional sources may include publications from the NIOSH, the WHO, and the IARC.

Primary references were identified and retrieved using PubMed® and the EBSCOhost® research databases. In December 2019, the NLM was reorganized and most of the toxicology information services available through TOXNET were integrated into other NLM products and services (https://www.nlm.nih.gov/toxnet/index.html). Prior to December 2019, TOXNET provided links to a suite of individual databases including ChemIDPlus® (chemical structures, registration numbers, and links to other sites providing physical chemical properties of the compound), the Hazardous Substances Data Bank (HSDB®), TOXLINE (references to literature on biochemical, pharmacological, physiological, and toxicological effects of drugs and other chemicals), the DART database, the CTD, the IRIS, and the ALTBIB database, as well as several others, including the archived databases for the Chemical Carcinogen CCRIS, the CPDB, and GENE-TOX genetic toxicity database. 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) using criteria modified from Howe et al. (2007). Table 2 describes the criteria used in the categorization, though the relative proportions of each substance were also factored into the final assessment. Where applicable, hazard is also characterized using the GHS Classification and Labeling of Chemicals (U.N. 2017) (see Appendix B, Tables B-1–B-4 and B-8). In some cases, toxicity values for substances are less than category 4, and do not meet the criteria for category 5; such compounds are not classified in the GHS.

If no experimental data can be located in the literature, toxicity values for the various parameters are predicted using QSAR software where possible. Modeling packages include EPA's Estimation Programs Interface Suite EPI Suite™ 4.0, EPA's ECOSAR™, and TOPKAT (BIOVIA Dassault Systèmes).

7. RESULTS

7.1 Physical and Chemical Properties

Table B-5 (Appendix B) summarizes physical and chemical properties. 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 Summaries

Table B-6 (Appendix B) summarizes the mammalian toxicity data. 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. Sections 7.3–7.5 provide the toxicology of each substance listed in Table 1.

7.3 Sulfuric Acid

7.3.1 General Information

Sulfuric acid (H_2SO_4 in H_2O) is a strong mineral acid, which is listed at 52–98% in this application (Puritan 2013). It is mixed with tartaric acid to form the electrolyte in the anodize phase of the treatment process. Electrical current drives the process as positive ions of hydroxide, sulfate, and oxide move towards the anode. The external electrical current causes Al^{3+} to move towards the cathode, but at the anode surface, the Al^{3+} reacts with the oxides to form a thin layer of Al-oxides. The replacement of chromic acid with sulfuric acid in this process removes the health concerns related to hexavalent chromium.

7.3.2 Toxicology Data

There is a significant body of literature available for sulfuric acid, as it has been used in industrial/chemical processes for some time. Pure sulfuric acid is not found naturally, but is manufactured for industrial applications and for use as a laboratory reagent; weak sulfuric acid can be formed from the reaction of air-polluting sulfur dioxide with rain. Occupational exposure to sulfuric acid, especially in fine mists, can occur during manufacturing processes. Sulfuric acid is a direct acting toxicant, so there are no reported effects on internal organs after oral, inhalation, or dermal exposure—no specific target organ has been identified. Fine mists of sulfuric acid are listed as Type 1 human carcinogens by the IARC.

7.3.2.1 Oral

Sulfuric acid is a strong acid when dissolved in water because H_2SO_4 liberates both hydrogen ions. It is corrosive to tissues, so ingestion will cause severe burns in the mouth and digestive tract, and circulatory shock is listed as the immediate cause of death (Gosselin 1984). Exposure can occur via ingestion, inhalation, or dermal routes. Ingestion of sulfuric acid is usually related to individual acute exposures (sometimes intentional), whereas dermal or inhalational occupational exposure can occur from exposure to mists. Sulfuric acid can also burn the eyes. Outside of acid rain from sulfur dioxide, oral exposure can occur from accidental or intentional exposure or occupational exposure (ATSDR, 1998).

Ingestion of sulfuric acid can burn the mouth and throat, and erode the stomach; death can occur in severe cases (HSDB 2019a). In India, where sulfuric acid is used as a cleaning agent, fatalities have occurred from gastric perforations and bronchopneumonia (Dilawari 1984).

An acute oral LD_{50} value of 2,140 mg/kg in rats at a concentration of 250 mg/mL (Smyth 1969) make sulfuric acid a Category 4 (Low Toxicity) for ingestion under the GHS system. However, this contrasts with the effects on the upper gastrointestinal system, which is the most likely target in humans (ATSDR 1998).

No EPA IRIS assessment of sulfuric acid has been carried out and consequently there is no chronic oral RfD for humans.

7.3.2.2 Inhalation

Inhalation exposure to sulfuric acid for humans occurs mainly through fine mists in occupational settings, though adequate engineering and administrative controls are expected to eliminate this pathway. In addition to concentration, the toxic effects of sulfuric acid aerosols are determined by particle size, which is in turn determined by humidity (ATSDR 1998).

Adult respiratory distress syndrome was reported in a man accidently exposed to high concentrations of sulfuric acid fumes, though no permanent outcomes were reported on follow-up (Knapp 1991); other cases have reported development of fibrosis and emphysema after accidental exposure to inhalation of 35% sulfuric acid mists and vapors (Goldman 1953). Acute-duration exposure studies of volunteers indicate that

adolescent asthmatics are generally more sensitive to inhalation of sulfuric acid aerosols than healthy older individuals (HSDB 2019a).

Inhalation produces respiratory track irritation upper respiratory tract and symptoms may include irritation of the nose and throat and labored breathing (ATSDR 1998). The manufacturer reports that sulfuric acid may cause lung edema; however, this effect has only reported in the literature for animal studies and not for humans (Puritan 2013). Acute exposure studies attempting to show that people with pre-existing respiratory conditions such as asthma may be more sensitive to sulfuric acid aerosols than healthy adults were equivocal (ATSDR 1998); Some suggest that asthmatics are more sensitive because the pH of their mucus is lower than non-asthmatics (Holma 1989). A 3-hour exposure of 30 asthmatics or 30 normal volunteers to sulfuric acid had no significant effects on measures of pulmonary function (Frampton 1995). One study in healthy asthmatics showed changes in pulmonary function at sulfuric acid concentration of 0.45 mg/m^3 but not at 0.1 mg/m^3 (Utell 1983).

Long-term exposure to sulfuric acid mist or vapors may cause damage to teeth due to lowered pH of saliva, including teeth etching and erosion and the comparative effect was 4-fold greater in the highest exposures group. The estimated average exposure was 0.23 mg/m^3 of sulfuric acid (Gamble 1984).

A threshold concentration of < 1 mg/m^3 sulfuric acid could be detected by odor, taste, or irritation (HSDB 2019a).

In animal studies, depending on the duration of exposure, the LC_{50} values for sulfuric acid aerosol are low. The lowest reported values were 0.37, 0.6, and 1.47 mg/L in rats, mice, and rabbits, respectively (HSDB 2019a).

7.3.2.3 Dermal

The manufacturer (Puritan 2013) reports that sulfuric acid is corrosive to skin (Category 1A). Chemical burns and scarring on the skin have occurred after acid assaults using sulfuric acid from car batteries (Branday 1996). Circulatory collapse with clammy skin, weak and rapid pulse, shallow respirations, and scanty urine may follow skin contact or ingestion. Circulatory shock is often the immediate cause of death (Gosselin 1984).

Repeated contact with more diluted sulfuric acid (concentration not provided) is known to cause skin desiccation, ulceration, and chronic purulent inflammation around the nails (OECD SIDS 2009). Dilute solutions of sulfuric acid (10%) did not appear to be irritating to the skin of rabbits, guinea pigs and human volunteers (Nixon 1975). Persons with pre-existing skin disorders or eye disease may be more susceptible to the effects of sulfuric acid (Puritan 2013).

7.3.2.4 Ocular

The manufacturer reports this product is corrosive and can cause blurred vision, redness, pain, and severe tissue burns (including blindness). Thirty-three percent of acid burn patients (from assault) in a hospital in Uganda suffered partial or complete

blindness (Asaria 2004). Eye damage as a result of contact with sulfuric acid from car batteries has been reported (Holekamp 1977). Eye irritation has also been reported in volunteers exposed to sulfuric acid aerosols at 20.8 mg/m^3 (Sim 1957). The most common eye injuries are conjunctival and corneal chemical burns and iritis (Holekamp 1977).

Results of the Standard Draize (eye rabbit) test using 250 µg were listed as severe (Puritan 2013). Conflicting results (not irritating or severely irritating) are observed in eye irritation studies using 10% sulfuric acid, depending on the protocol used (Organization for Economic Cooperation and Development (OECD/EU or US)) (HSDB 2019a). Sulfuric acid is listed as Category 1 (causing serious eye damage) under the GHS system.

7.3.2.5 Development and Reproduction

Data on the potential for sulfuric acid to cause infertility, abortion, or birth defects in humans were not available (HSDB 2019a). Since sulfuric acid is a direct-acting corrosive toxicant (acts at the point of contact), it is unlikely to reach the reproductive organs and reproductive effects in mammals exposed to sulfuric acid are unlikely (HSDB 2019a). In a developmental toxicity/teratogenicity study conducted by whole body inhalation of sulfuric acid aerosol at concentrations of 0, 5, and 20 mg/m^3 during gestation, the NOAEL for maternal toxicity was 20 mg/m^3 in mice and rabbits; no evidence of fetotoxicity or teratogenicity was seen in either species (Murray 1979).

7.3.2.6 Genotoxicity

The manufacturer's SDS does not provide supporting data for mutagenicity of sulfuric acid (Puritan 2013). Sulfuric acid was shown to be negative, with and without S9 activation, in the Ames assay (HSDB 2019a). Low pH caused by sulfuric acid exposure to a human tracheal epithelial cell line for 10 minutes caused changes in intracellular pH, but no biomarkers of effect were assessed (Chen 1995); Morita et al. (1989) have shown increases in chromosomal aberrations in Chinese Hamster Ovary cells exposed to sulfuric acid (Shimada and Ingalls 1975). In *Salmonella typhimurium* testing, sulfuric acid tested negative with and without rat S9 activation (Cipollaro 1986). Sulfuric acid has been shown to cause chromosomal aberrations in CHO cells (pH 3.5 to 7.4), both with and without metabolic activation (Cipollaro 1986). There are no *in vivo* studies of genotoxicity, the observed number of chromosomal abnormalities in human lymphocytes *in vitro* is associated with internal pH levels (Shimada and Ingalls 1975).

7.3.2.7 Carcinogenicity

The IARC has classified strong inorganic acid mists containing sulfuric acid as a known human carcinogen (IARC category 1) with increased risks for the larynx and lung. This classification applies only to mists containing sulfuric acid and not to sulfuric acid or sulfuric acid solutions. A study by Steenland et al. (1988) showed workers in pickling operations had a 2.3-fold incidence rate increase in laryngeal cancer with a 10-year follow-up showing a consistent 2.2-fold increase overall. However, many cancer studies are confounded by smoking status, which makes it difficult to isolate sulfuric acid alone as carcinogenic. While sulfuric acid mists have been deemed carcinogenic, the lack of

robust dose-response data in epidemiological studies means there is no quantitative cancer risk value. While epidemiological studies have suggested a relationship between exposure to inorganic acid mists containing sulfuric acid and an increased incidence of respiratory tract cancers, there are a lack of animal studies using only sulfuric acid (ATSDR 1998). Nonetheless, it has been proposed that sulfuric acid may promote carcinogenesis via chronic tissue irritation or chronic inflammation, since both processes can release free radicals, which can be genotoxic (ATSDR 1998). It has also been proposed that significantly lowering intracellular pH may contribute to the carcinogenesis of sulfuric acid in the respiratory tract (Soskolne 1989).

7.3.2.8 Neurotoxicity

The manufacturer has no entry for neurotoxicity in the SDS and no evidence of neurotoxicity was reported in the literature (HSDB 2019a).

7.3.2.9 Ecological Data

7.3.2.9.1 Fate and Transport

Sulfuric acid is fully ionized in water and will be transported in ground and surface water, likely as the sulfate ion where it will react with calcium and magnesium to form sulfate salts. In air, sulfuric acid (vp 5.93 X 10-5 mm Hg) exists in vapor and particulate phases. Direct photolysis of sulfuric acid can occur. Sulfuric acid is removed from the air by both wet and dry deposition. Bioaccumulation is not a concern for sulfuric acid. However, consider the potential acidification of any surface or medium that comes in contact with sulfuric acid (HSDB 2019a). If released into water, sulfuric acid is not expected to adsorb to suspended solids and sediment (HSDB 2019a). Volatilization from water surfaces is not expected to be an important fate process based upon this compound's ionization and K_H (HSDB 2019a).

7.3.2.9.2 Ecotoxicity

In aquatic systems, the toxicity of sulfuric acid depends on the concentration of acid and buffering capacity of the water; a 0.1 N (0.2 M) solution of sulfuric acid has a pH of 1.2 (HSDB 2019a). The optimal pH for fish ranges from 6.5 to 8.5, depending on the species; values outside of this range can damage eggs and juveniles, as well as adults (Svobodova 1993). The 24-hour LC_{50} range in freshwater zebrafish (*Brachydanio rerio*) was reported as 82 mg/L while the 48-hour LC_{50} for bluegill (*Lepomis macrochirus*) was reported to range from 16 to 28 mg/L (HSDB 2019a). The no observed effect levels (NOEC) for embryo survival and time hatching of brook trout (*Salvelinus fontinalis*) was dependent on pH (i.e., 0.31 mg/L at pH 5.2 and 0.15 mg/L at pH 5.5) (HSDB 2019a). Another study found an NOEC for the weight of young brook trout to be 0.13 mg/L at pH of 5.56. In flagfish (*Jordanella floridae*), the lowest observed effect level (LOEC) for fry growth was 0.049 mg/L at pH 6.0 (HSDB 2019a). A toxicity study on an experimentally acidified lake focusing on the phytoplankton community structure identified a NOEC of 0.13 mg/L at a pH of 5.6. This NOEC integrates algae growth rate and consumption by invertebrates and fish (HSDB 2019a). A pH of 4.0 gave pronounced gill irritation and 3.5 caused death of sunfish, bass, and carp. The pH which caused 50% mortality of bluegill

(*Lepomis macrochirus*) (96-hour LC_{50}) was between 3.5 and 3.0 (HSDB 2019a). In fish, the major causes of toxicity appear to be related to the disruption of sodium balance and respiration (HSDB 2019a).

The manufacturer lists sulfuric acid (52–198%) as Category 3 (acute aquatic toxicity). The following toxicity values were reported by the manufacturer and confirmed in the literature (HSDB 2019a). LC_{50} for flounder (species not identified) was 100 to 330 mg/L/48 hr aerated water, but the conditions of bioassay were not specified.

7.3.2.9.3 Degradation and Treatment

The manufacturer states that this material is expected to be readily biodegradable (Puritan 2013). The two products are hydrogen ions and sulfate; subsequently, sulfate may be converted to hydrogen sulfide by sulfate reducing bacteria (D'Aoust 2018).

7.4 L-Tartaric Acid

7.4.1 General Information

L-tartaric acid (synonyms natural tartaric acid or fruit acid) is widespread in nature, has a strong acid taste associated particularly with grapes and tamarinds, and is one of the main acids found in wine. It is a strong organic acid, with a pKa_1 of 2.98 and pKa_2 of 4.34; a 0.1N solution has a pH of 2.2 (O'Neil 2006). Note that the terms "tartarate" and "tartrate" are interchangeable. It is used as a food additive in processing as an acidifier or antioxidant. The naturally occurring form of the acid is a racemic mixture of L-(+)-tartaric acid or dextrotartaric acid (NCBI 2017). L-Tartaric acid is listed by the manufacturer as being used at 5–20% in the TSAA processes. It is added to sulfuric acid solutions in plating processes. This is to control and improve the anodic deposition of aluminum oxides during the anodizing process.

7.4.2 Toxicology Data

As stated above, TSAA solutions contain about 5–20%. Since it is widely present in nature and plant substances that are ingested, natural levels of L-tartaric acid are not thought to be toxic. One record of a fatal dose of 30 grams (g) tartaric acid following accidental ingestion has been reported (Robertson 1968). Potential acute health effects include skin contact (irritant), eye contact (irritant), ingestion, or inhalation (lung irritant). There are no available long-term effects. The manufacturer lists ingestion and inhalation as the main routes of entry.

7.4.2.1 Oral

Sodium tartrate and other salts up to 10 g have been used as laxatives (Gold 1943), while accidental ingestion of 30 g of tartaric acid was reported to result in human fatality due to acute nephropathy (Robertson 1968). Patients treated with salts of tartrate typically experienced transient nausea and stomach pain (Gold 1943); while the acute effects of ingesting large amounts of tartaric acid include gastrointestinal tract irritation with nausea, vomiting, and diarrhea (Gold 1943). Conversely, tartrate is used in foods or

found naturally in fruits without reported side effects. A study showed ingestion of 280 mL of grape juice (which naturally contains tartaric acid) by volunteers increased creatinine-corrected urinary tartrate 40-fold, indicating that dietary intake of tartrate is most likely the biggest single contributor to internal tartrate concentration (Lord 2005). Acute injury from tartaric acid was reported in 4 out of 184 cases at a poisoning center but the doses were not specified (Naqvi 2017). The FDA has deemed that food grade tartaric acid (L form) is listed as GRAS (generally recognized as safe; CFR 2019), indicating no historically reported toxicity. There were no long-term oral studies of the effects of L-tartrate on humans.

Animal toxicity studies on L-tartaric acid alone are infrequent and are mostly in older literature. L-tartaric acid oral LD_{Lo} (lowest dosage causing death) is listed as 5 g/kg in dogs and rabbits and 7.5 g/kg in rats (Sourkes 1950). In the mouse, the oral LD_{50} of sodium tartrate was 4360 mg/kg (Locke 1942). Other studies have used doses of 400 mg/kg and 600 mg/kg in rats and dogs, respectively, without reports of mortality. Many values stem from older studies, where GLP was not used and purity of the test article could not be verified. However, the overall consistency of the values would place L-tartaric acid in the GHS category 5, making it the lowest in terms of oral toxicity.

Repeated doses of ^{14}C labelled 2.73 g/kg of monosodium DL-tartarate in rats caused increased kidney body weight ratios compared with controls, but radiolabeled monosodium L-tartarate showed no effect (Down 1977). Rats rapidly excreted oral doses of L-tartaric acid with a proportion metabolized to carbon dioxide (Chasseaud 1977). Histological studies have shown an increasing degree of kidney damage in rabbits with increasing doses from 50 to 300 mg/kg, with a parallel of decreasing urinary excretion. In a 13-week oral repeated dose study of DL PHT, rats fed a diet containing up to 2.0% PHT had microscopic findings consistent with obstructive nephropathy in doses at and above 0.5% (Inoue 2015). This finding is consistent with that reported by Robertson (above) for a human case fatality.

In a chronic study of rats fed tartaric acid for 2 years in diets containing 0.1, 0.5, 0.8, and 1.2% tartaric acid, no significant effects were observed with mortality, growth rate, gross, or microscopic findings (Sourkes 1950). The peak value of 1.2% is roughly equivalent to a dose of 600 mg/kg-day.

In a separate chronic study where rats were fed monosodium tartrate for 2 years, concentrations as high as 76,800 ppm (2,460 mg/kg-day) were not toxic (ECHA 2020).

7.4.2.2 Inhalation

The manufacturer-provided SDS states that L-tartaric acid is a lung irritant and causes respiratory tract irritation; however, supporting data were not provided (ScienceLab 2010). No data were found for inhalation of L-tartaric acid in the peer-reviewed literature. In the European Chemical Agency REACH dossier, the data requirement for inhalation toxicity was waived (ECHA 2020). However, the bronchodilator, aformoterol tartrate, which is used for obstructive lung diseases contains free tartrate in the formulation, has been shown to be effective and non-toxic when used long-term for relief of pulmonary disease. This indicates that the tartrate component is not itself an irritant with long-term

low-dose use in humans (Baumgartner 2007). However, tartaric acid occupational exposures during the TSAA process may be higher than concentrations of tartrate used in bronchodilator medication.

7.4.2.3 Dermal

The manufacturer reports this material to be hazardous with skin contact (irritant), which is consistent with its designation as a strong acid. Tartaric acid has been used in cosmetics as a superficial peeling agent for the skin, but adverse reactions can occur such as swelling, burning, and pruritus (Tang 2018). Skin contact has also been reported to cause redness, pain, and blisters (NCBI 2017).

7.4.2.4 Ocular

Because it is considered a strong acid, tartaric acid can cause serious eye irritation. Tartaric acid has been listed as causing redness, pain, and severe deep burns (NCBI 2017).

7.4.2.5 Development and Reproduction

Tartaric acid was negative for prenatal developmental toxicity in four species (mouse, rat, hamster and rabbit) and the NOAEL, based on the rat exposures, was 181 mg/kg-day (ECHA 2020).

7.4.2.6 Genotoxicity

Tartaric acid was negative in the Ames assay both with and without S9 activation; the identity of the compound, either L-tartaric acid or D-tartaric acid, was not clearly established due to conflicting name/CAS identity in the original report (ECHA 2020). In yeast (*Saccharomyces cerevisiae*), the highest no effect level for genetic toxicity was 2% tartaric acid (USEPA 2020). A dominant lethal chromosome aberration assay using rats was conducted with doses as high as125 mg/kg-day (ECHA 2020). Briefly, male rats were dosed prior to a mating period and then offspring from the females bred by treated male rats were evaluated. Chemicals that generate germ cell DNA damage will affect the number/quality/survival of offspring. The findings from the assay indicated there were some notable differences between treated and untreated animals; however, the effects were too inconsistent to provide a definitive interpretation (ECHA 2020).

7.4.2.7 Carcinogenicity

Monosodium L(+) tartrate was tested in a 2 year rat dietary up to 76800 ppm; no evidence of tumors were found (ECHA 2020). Compared to the control rats, weight reduction due to decreased food intake was observed in the treated rats.

7.4.2.7 Neurotoxicity

No comments were made by the manufacturer regarding neurotoxic effects L-tartaric acid. No data were found regarding neurotoxic effects of L-tartaric acid (HSDB 2019c).

7.4.2.8 Ecological Data

7.4.2.8.1 Fate and Transport

Due to its high solubility in water, L-tartaric acid will be highly mobile in groundwater, and may contaminate surface and drinking water. Bioaccumulation of L-tartaric acid is unlikely, but microbial degradation is highly likely.

7.4.2.8.2 Ecotoxicity

The tartaric acid LC50 in zebrafish was 100 mg/L and in Daphnia the LC50 was >538 mg/L (USEPA 2020). The NOEC and EC_{50} for cyanobacteria was 3.1 mg/L and 51.4 mg/L, respectively (USEPA 2020). Using ECOSAR, tartaric acid was modeled as a neutral organic, which is the baseline toxicity potential model. The predicted concentrations for all endpoints, with the exception of earthworm, are > 8 g/L. The predicted 14-day earthworm LC50 is 5.3 g/L.

7.4.2.8.3 Degradation and Treatment

The manufacturer states that hazardous short-term degradation products are not likely (ScienceLab 2010). As a naturally occurring or organic substance, degradation by microorganisms is rapid with a predicted ½ life of 5 days (USEPA 2020).

7.5 CHEMEON TCP-HF

CHEMEON TCP-HF is described as a conversion coating and anodizing sealant; prior to June 2015, the listed name was Metalast TCP-HF (Chemetall 2016). The CHEMEON TCP-HF product is an aqueous solution containing <2% chromium hydroxide sulfate ($Cr(OH)(SO_4)$; CAS RN 12336-95-7) and <1% of pH modifiers (either sulfuric acid or sodium/potassium hydroxide) and trace levels of fluoride (verbal communication with vendor representative Sept 3, 2020). The manufacturer lists the eye, skin, and lungs as target organs (Chemetall 2016). For CHEMEON TCP-HF, the listed hazards are skin irritation and serious eye irritation. Note, the “HF” in the name stands for hexachromium free and it is a coincidence that HF is the chemical abbreviation for hydrogen fluoride.

Sulfuric acid is discussed in section 7.3. Sodium hydroxide does not impact the toxicity of this product and is not evaluated here. The trace level of fluoride ion and the toxicity evaluation of Cr(III) is evaluated in the following sections.

7.5.1 Chromium hydroxide sulfate (CAS RN 12336-95-7)

Synonyms for chromium hydroxide sulfate include basic chromium sulfate, chromosulfuric acid, monobasic chromium sulfate, Peachrome and Chromedol (PubChem 2020). The chromium ion valence state is Cr(III), which is considered significantly less toxic than Cr(VI) due to reduced bioavailability. Most of the toxicity data associated with chromium sulfate are from inhalation and dermal studies (HSDB 2019a).

7.5.1.1 Toxicology Data

Basic chromium sulfate has a wide range of industrial uses ranging from solubilizing gelatin to paint manufacture (HSDB 2019a). Chronic occupational exposure can induce contact dermatitis and asthma. Occupational exposures can often include mixtures of Cr(VI) and Cr(III) (HSDB 2019b). There is inadequate information to determine if Cr(III) alone is carcinogenic; however, Cr(III) is poorly absorbed, which reduces the internal dose. For this assessment, the literature review included data from a variety of chromium (III) salts to include Cr(III) oxide. As these inorganic salts will all dissociate to yield the Cr(III) cation, it is scientifically justified to use a read-across approach to assess Cr(III) toxicity.

7.5.1.1.1 Oral

A fatal dose of chromium sulfate in humans was reported; ingestion of leather tanning solution containing 48 g of basic chromium sulfate by a woman resulted in cardiac shock, complicated by pancreatitis and gut hemorrhage (van Heerden 1994).

Short-term animal studies on oral exposure to basic chromium sulfate have not been reported. While there are no available LD_{50} values for basic chromium sulfate, the LD_{50}'s for other forms of trivalent chromium (e.g., chromium acetate) can be up to 10-fold less than hexavalent chromium, though this could be complicated by differences in solubility (ATSDR 2012). The Cr(III) oxide LD_{50} was higher than 15,000 mg/kg in Wistar rats (ECHA 2020). At 15,000 mg/kg, no animals died and ruffled fur was the only noted clinical sign of toxicity. Generally compared to Cr(VI), Cr(III) is poorly absorbed with only 0.5–2.8% of dietary Cr(III) absorbed via the gastrointestinal tract of humans (ATSDR 2012).

No sub-chronic or chronic oral studies using basic chromium sulfate alone were found. However, in a study to assess the oral carcinogenicity of trivalent chromium oxide (Cr_2O_3), groups of 60 male and female rats were fed Cr_2O_3 baked in bread at dietary levels of 0, 1, 2, and 5% for 840 days. Average ingested doses were 360, 720, and 1,800 mg/kg body weight. The only observed treatment-related effects were reductions in absolute weights of the livers and spleens of animals in the high dose group, but these were not normalized to body weight (Ivankovic 1975). Based on Ivankovic (1975) study, the EPA used a NOAEL of 1,468 mg/kg-day and a composite uncertainty factor of 1,000 to derive an IRIS determined oral RfD of 1.5 mg/kg-day (EPA 1998).

There were no long-term studies for basic chromium sulfate, but long-term feeding studies in animals have demonstrated that other trivalent chromium salts are relatively nontoxic. Rats fed chromium oxide at 2,040 mg/kg-day (5 days per week) for 2 years showed no signs of adverse toxicity (Ivanovic 1975), while chromium chloride in drinking water at 2.7 mg/kg-day for 1 year showed no adverse effects (MacKenzie et al. 1958). In an ACGIH revision of TLVs for chromium compounds, a literature review concluded that trivalent chromium was relatively non-toxic when ingested (ACGIH 2018).

7.5.1.1.2 Inhalation

No short-term studies were found for humans; case reports indicate that inhalation of basic chromium sulfate results in acute asthma but there are few clear studies on Cr(III)

that are not confounded by the presence of other metals (HSDB 2019b). Three-hundred workers exposed for 20–25 years to Cr(III) as chromic oxide and chromic sulfate were examined for respiratory effects. Preliminary results showed no differences from controls with respect to respiratory illness, x-ray, clinical, or blood studies (Korallus 1974). For inhalation effects, the manufacturer's SDS for chromium sulfate exposure indicates no (inhalation exposure) is expected and chromium sulfate may exacerbate lung disorders (Chemetall 2016). Those with preexisting respiratory conditions (sensitive subpopulations) may be exacerbated by inhalation exposure of Cr(III). Workers exposed to Cr(III) in a factory that produced Cr(III) compounds reported an increase in respiratory conditions (phlegm production, shortness of breath on exertion) compared to controls, but there were no differences in spirometry or chest x-ray (Huvinen 2002).

No acute animal inhalation toxicity data on chromium sulfate were available.

In a subchronic rat study, nose-only exposures to insoluble chromic oxide dust at 4.4, 15, or 44 mg/m^3 or soluble basic chromium sulfate dust at 17, 54, or 168 mg/m^3 (trivalent chromium equivalent concentrations of 3, 10, and 30 mg/m^3) were carried out for 6 hr/day, 5 days/week for 13 weeks. General toxic effects, only observed with high-exposure levels of basic chromium sulfate, included sporadic signs of labored breathing and depressed body weights. No compound-related mortality occurred; however, there were sporadic differences in respiratory toxicity between the oxide and sulfate, which may have been due to acidity and water-solubility. Inflammation of lung, nasal tissues, and larynx were also observed. A NOAEL was not established for chromium sulfate, but 4.4 mg/m^3 was suggested to be near the NOAEL for the chromium oxide. There was no effect on internal organs (Derelanko 1999).

7.5.1.1.3 Dermal

Basic chromium sulfate has been designated Category 2 for skin irritation under GHS (Chemetall 2016). The manufacturer states that prolonged and/or repeated contact may cause fluoride-type irritation and/or dermatitis (Chemetall 2016). The manufacturer SDS did not have information available on the mixture, however none of the components in the mixture have been classified for skin sensitization or are below the concentration threshold for classification (Chemetall 2016). According to the manufacturer, there are no data available for acute dermal toxicity (Chemetall 2016).

Eczematous dermatitis due to Cr(III) compounds has been reported (Lewis 2004). Although Cr(VI) is most frequently associated with allergic skin reactions, Cr(III) provokes an immune response as well. In humans, the 48-hour minimum elicitation threshold (MET) for Cr(III) is 0.18 ug/cm^2; this value is approximately 6 times higher than the MET for Cr(VI) (Hansen 2003). Tests show that provocation challenges for dermal (or respiratory) sensitivity will cause a reaction to Cr(III) (ACGIH 2018). Aqueous chromium solutions are more likely to cause reactions and only water-soluble trivalent chromium compounds are listed as respiratory and dermal sensitizers (ACGIH 2018).

7.5.1.1.4 Ocular

This chemical has been designated as Category 2A for eye irritation under GHS. The manufacturer reports that this mixture may cause irritation to the eye (Chemetall 2016). No studies were found for ocular studies in humans.

7.5.1.1.5 Development and Reproduction

The manufacturer SDS does not provide information for the mixture; however, none of the components have been classified for reproductive toxicity (or are below the concentration threshold for classification) (Chemetall 2016).

No studies were located regarding reproductive effects in humans after exposure to Cr(III) compounds (ATSDR 2012).

In a feeding study, Ivanovic (1975) saw no male reproductive effect in rats that were administered 1,806 mg of chromium oxide per day for 60 days. No effects on sperm motility, morphology, or concentration were observed in a 13-week nose-only inhalation study (doses listed on page 15) of basic chromium sulfate and chromium oxide (Derelanko 1999). A NOAEL 30 mg/m^3 was recorded for this study (Derelanko 1999).

7.5.1.1.6 Genotoxicity

A review of 700 reports representing 32 Cr(III) compounds was organized based on solubility (ECHA 2020). Soluble Cr(III) compounds did not illicit genotoxicity in a variety of assays and endpoints. Poorly soluble or insoluble Cr(III) compounds were genotoxic; however, further analysis demonstrated contamination of these Cr(III) compounds with Cr(VI) (ECHA 2020). Cr(III) ions enter cells very slowly (days) by simple diffusion, compared to the rapid facilitated diffusion (minutes) of Cr(VI). By extension, Cr(III) is less likely to enter the nucleus and generate DNA damage (ATSDR, 2012).

In general, *in vivo* studies of micronuclei for Cr(III) have been shown to be negative (ATSDR 2012). Tannery workers exposed to Cr(III) have increased protein-DNA crosslinks, micronuclei and DNA damage in peripheral lymphocytes (Medeiros et al. 2003, Zhang 2008).

7.5.1.1.7 Carcinogenicity

Basic chromium sulfate or Cr(III) has not been listed in the NTP Report on Carcinogens (2016) and is considered not classifiable as to carcinogenicity in the IARC Monograph No. 49 (WHO 1990). The ACGIH has determined that trivalent chromium is not classifiable as a human carcinogen.

The EPA IRIS database states that Cr(III) is not classifiable as to human carcinogenicity based in insufficient evidence (EPA 1998).

No cancer studies on chromium sulfate were found; NTP carried out carcinogenesis studies on chromium picolinate (NTP 2010). Male and female F344/N rats and B6C3F1 mice were exposed to chromium picolinate monohydrate (95% to 96% pure) in the diet at levels of 0, 2,000, 10,000, or 50,000 ppm (equivalent to approximately 250, 1200, and

6200 mg/kg-day) for 2 years. Food consumption and mean body weights were similar to the control groups. Male F344/N rats in the 10,000 ppm group had statistically significant increases in the incidence of preputial gland adenomas. For mice, no neoplasms or nonneoplastic lesions were attributed to exposure to chromium picolinate monohydrate. The NTP concluded that incidence of adenomas in male rats only at 10,000 ppm (middle dose) was equivocal evidence of carcinogenesis of chromium picolinate due to a lack of dose-response at 50,000 ppm (highest dose).

7.5.1.1.8 Neurotoxicity

No studies were found that examined the neurotoxicity of basic chromium sulfate or other trivalent chromium compounds; note that one study reports that exposure to airborne hexavalent chromium in occupational settings produced dizziness, headache, and weakness (Lieberman 1941). There is some evidence that Cr(VI) is toxic to isolated neurons (Dashti 2016).

7.5.1.2 Ecological Data

7.5.1.2.1 Fate and Transport

The manufacturer states that there are no data available for persistence and degradability, bioaccumulation, or mobility in soil (Chemetall 2016).

In most soils, chromium will be present predominantly in the Cr(III) state. This form has very low solubility and low reactivity, which results in low mobility in the environment and low toxicity in living organisms (ATSDR 2012).

7.5.1.2.2 Ecotoxicity

The manufacturer states that no data were available for fish, invertebrates, or algae (Chemetall 2016). The bioavailability of Cr(III) to freshwater invertebrates decreased due its complexation with humic acid (ATSDR 2012). The effect of chromic sulfate on the common carp and bighead (*Aristichthys nobilis*) were studied. The carp was more susceptible to chromic sulfate than *A. nobilis* at the lowest concentrations tested (100 mg/L), whereas results obtained at other concentrations were similar for both species. Damage to gills, liver, and intestines were observed by histopathological examination (HSDB 2019b). In *Coturnix* (Japanese quail), dietary administration of $Cr_2(SO_4)_3$ resulted in an LC_{50} of >5000 ppm over 5 days (HSDB 2019b).

7.5.1.2.3 Degradation and Treatment

The Cr(III) forms insoluble precipitates and it's formation is considered to be useful for remediating Cr(VI) contamination. Releases of more than 1000 lbs are reportable (HSDB 2019b).

7.5.2 Hydrogen Fluoride

7.5.2.1 General Information

The concentration of fluoride ion in Chemeon TCP-HF is low- likely less than 2% based on the scant information provided by the vendor (Chemetall 2016). The review of HF includes toxicity from high HF concentrations. Hydrogen fluoride is a gaseous substance that is readily soluble in water. In the gas phase, the compound is hydrogen fluoride, and when in solution, it is called hydrofluoric acid. In either phase, HF is extremely corrosive and reactive with other materials. Glass containers are not suitable, as the HF will react with and etch the glass. HF is also used for cleaning in the manufacture of glass, semiconductors, computer chips and ceramics and industrial applications. Deaths due to HF exposure have been reported (HSDB 2017).

7.5.2.2 Toxicology Data

The HF functions as a toxicant via two modes of action. Firstly, the compound is extremely corrosive and damaging to lungs, skin, eyes and systemically. The primary mode of systemic action is to bind calcium and magnesium ions, resulting in hypocalcemia and cardiac dysrhythmia and arrest. Tetany may also result from hypocalcemia following exposure (HSDB 2017).

7.5.2.2.1 Oral

After ingestion, the oropharynx and esophagus are the primary sites of injury. Ingestion of an estimated 1.5 g of hydrofluoric acid produced sudden death without pathologic damage. Repeated ingestion of a smaller amount of hydrofluoric acid resulted in moderately advanced osteosclerosis (HSDB 2017).

7.5.2.2.2 Inhalation

After inhalation, severe pulmonary injury may occur with pulmonary edema and bronchopneumonia. The odor of HF can be recognized at a concentration of 0.03 mg/m3, with a sour taste and smarting eyes. Concentrations of 50-250 ppm can be dangerous even for brief exposures (HSDB 2017).

The LC50 for HF is 1278 ppm in rats, 500 ppm in mice, and 1780 ppm in monkeys, for a 1-hour exposure; the LC50 for a 15-minute exposure in rats is 2689 ppm and 3540 mg/m3 in guinea pigs (HSDB 2017).

7.5.2.2.3 Dermal

HF is highly corrosive to skin, producing burns, necrosis, and possibly affecting the underlying bone (HSDB 2017).

7.5.2.2.4 Ocular

Contact with eyes result in burns. Concentrations as low as 5 mg/L (5 ppm) may produce ocular irritation. Lacrymation, pain, and conjunctival injection are early symptoms of HF exposure. Corneal and conjunctival epithelium may be denuded, leading to edema and ischemia. Corneal vascularization and scarring may result. Toxicity may be delayed by up to 4 days after dilute exposures (HSDB 2017).

7.5.2.2.5 Development and Reproduction

No data on HF effects on development or reproduction were found. In studies testing NaF, there was no evidence of developmental or reproductive toxicity following drinking water exposure up to 13 mg/kg-d (rats) or 14 mg/kg-d (rabbits) (ECHA 2019). Some maternal toxicity in rats was observed at 13 mg/kg-d.

7.5.2.2.6 Genotoxicity

For in vitro genotoxicity assays, HF is negative in the Ames test and in the HPRT mammalian cell mutation assay utilizing NaF (ECHA 2019).

The HF was negative for dominant lethal mutations following inhalation exposure in C57Bl mice (HSDB 2017).

Increases in the occurrence of chromosome aberrations were found in the bone marrow cells of rats exposed by inhalation to 1.0 mg/m3 HF 6 hours/day, 6 days/week for 1 month (HSDB 2017).\

7.5.2.2.7 Carcinogenicity

No data relating to carcinogenicity were found.

7.5.2.2.8 Neurotoxicity

No data were found for HF neurotoxicity. Some studies with NaF have been conducted but the results were inconsistent and the experimental design did not include measuring the background concentration of fluoride ions in the food or drinking water (ECHA 2019).

7.5.2.3 Ecological data

7.5.2.3.1 Fate and Transport

HF is freely miscible in water and will migrate at will through aqueous systems, it will rapidly dissociate in aqueous conditions to form hydrogen and fluoride ions (ECHA 2019). Reaction with calcium and magnesium ions in soil and water may mitigate effects of any release. HF will react with living tissue and not bioaccumulate as HF, but upon dissociation, fluoride does accumulate in the skeleton of both vertebrates and invertebrates, weakening the structure. Accumulation in the skeleton of prey species can also be a cause biomagnification.

7.5.2.3.2 Ecotoxicity

No ecotoxicity data for HF on animal species were found, however, due to the dissociation of HF to fluoride and hydrogen ions, toxicity data on fluoride is available. In fish, the 96-h LC50 for NaF ranges from 51 mg/L (*Onchorynkus mykiss*) to 340 mg/L (Gasterosteus aculeatus) (ECHA 2019). The 21-d NOEC in *O. mykiss* is 4 mg/L.

For *Daphnia magna*, the EC50 values range from 97-352 mg/L for NaF, while the NOEC values range from 3.7-14.1 mg/L (ECHA 2019). Algae EC50 values range from 43-122 mg/L for acute exposure, with NOEC values ranging from 50-249 mg/L.

Some plants are sensitive to fumigation by HF, including tulip, gladiolus, fruit crops, conifers and grasses, which are affected by concentrations from 0.4 to 1.0 µg/m3 after exposure for several days (HSDB 2017). Fluoride in the soil will not be readily available for plant uptake (ECHA 2019).

7.3.2.3.3 Degradation/Treatment

No data were found for degradation of HF. Reaction with calcium or magnesium ions, or neutralization by alkaline materials (including limestone) will remove HF from the environment.

8. DISCUSSION

8.1 Compound Summaries

Table 2 presents toxicity hazard assessments for human health and environmental toxicity for each of the formula components. Each characterization is generally based on the criteria set forth in Appendix B, Table B-7. The GHS hazard codes from the respective SDS and PubChem (2020) are also included. For comparison, the GHS hazard codes for chromic acid (Cr(VI) are also provided. 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.

8.1.1 Sulfuric Acid

The TSAA process uses sulfuric acid at concentrations from 52 to 98%. Sulfuric acid is a strong acid with potential corrosive effects on the skin, lungs, and digestive tract, depending on the concentration; toxic effects occur directly at the point of contact and there is no specific internal target organ of toxicity. The toxic effects are exclusively due to the hydrogen ions, while the sulfate anion is not considered to have a toxic impact. Strong sulfuric acid mists in occupational exposures have been identified as carcinogenic to humans, and high concentrations of sulfuric acid can harm aquatic life.

Table 2. Toxicity Hazard Assessment and GHS categorization for TSAA

	Oral	Inhalation	Dermal	Ocular	Carcinogenicity	Aquatic	Invertebrates	Plants	Mammals	Birds	Fate/transport	Persistence	Bioaccumulation	**GHS codes** (see Appendix B Table B8)

Sulfuric acid			1A	1	A2	3								H303, H314, H318, H330, H370, H402
L- Tartaric Acid				2A										H302, H315, H318, H319
Chemeon TCP-HF $Cr(III)SO_4$			1A	1										H314, H315, H317, H318, H319
Baseline Cr(VI)														H300, H310, H314, H317, H318, H330, H334, H340, H350, H361, H372, H400, H410
Low Hazard						Moderate Hazard						High Hazard		

due to low pH effects. The health effects are relevant only at high concentrations and are considered risk factors where significant protection for the worker is lacking. OSHA PELs provide a basis for regulatory protection in occupational settings. Regarding environmental impacts, the pH can locally affect aquatic life, especially with high hydrogen ion concentrations. Potential effects due to exposures in occupational settings are expected to be contained by following recommended exposure controls and personal protection. For waste disposal, this material will be treated as a hazardous waste.

8.1.2 Tartaric Acid

Tartaric acid is a naturally occurring acid found in plants and wine. The TSAA process uses 5–10% L-tartaric acid in combination with sulfuric acid. Solid tartaric acid can be irritating to the eyes, lung, and respiratory system, but effects are not expected in solution. There are no OSHA PELs available for tartaric acid. Since it is combined with sulfuric acid in process solutions, tartaric acid would be treated as a hazardous waste.

8.1.3 CHEMEON TCP-HF (basic chromium sulfate and hydrogen fluoride)

The primary active constituent in CHEMEON TCP-HF is basic chromium(III) sulfate. Trivalent chromium has low toxicity compared to hexavalent chromium compounds. Chromium sulfate is GHS Category 4 for oral toxicity system based on an LD_{50} of 1,350 mg/kg; but it is GHS-listed as a skin and eye irritant. HF is GHS acute category 2 for acute exposures, eye category 1, and skin corrosion category 1A (UNECE 2015). The CHEMEON product is a concentrate and is diluted 1:2 to 1:10 for use. This ready to use dilution is maintained at an acidic pH (~3.5) (CHEMEON 2019). Additionally, the vendor communicated that there are trace amounts of fluoride present in the product.

8.2 Regulations and Standards

8.2.1 Sulfuric Acid

The OSHA 8-hour TWA PEL is 1 mg/m^3. ACGIH recommends an 8-hour TLV-TWA of 0.2 mg/m^3. Sulfuric acid in strong inorganic mists is considered carcinogenic by IARC. Sulfuric acid is designated as a hazardous substance under section 311(b)(2)(A) of the Federal Water Pollution Control Act and further regulated by the Clean Water Act Amendments of 1977 and 1978. Sulfuric acid is an EHS subject to reporting requirements when stored in amounts in excess of its TPQ of 1,000 pounds (HSDB 2019a).

8.2.2 Tartaric Acid

The FDA has deemed tartaric acid to be GRAS in the absence of any toxicity data related to its use in food. There are no other reported governmental regulations or nongovernmental guidelines for tartaric acid exposure for humans.

8.2.3. CHEMEON TCP-HF (basic chromium sulfate and hydrogen fluoride)

The OSHA 8-hour TWA PEL for Cr(III) is 0.5 mg/m^3. The ACGIH TLV-TWA for trivalent chromium compounds is 0.003 mg/m^3 for inhalable particulate matter. ACGIH has given trivalent chromium (including chromium sulfate) a respiratory sensitization and a dermal sensitization warning for water-soluble compounds. EPA has established a DWEL for total chromium of 0.1 mg/L.

Regulations for hydrogen fluoride are based on HF vapor, which is difficult to translate to more diluted HF solutions. The OSHA PEL-TWA is 3 ppm, the ACGIH TLV-ceiling is 2 ppm and the TLV-TWA is 2.5 mg/m^3; all of these limits are stipulated for fluorine (‘as F’) (Ricca 2020). Immediate and potentially life threatening burns occur at aqueous HF concentrations > 20% (Schwerin 2020).

8.3 Summary and Conclusions

The TSAA process includes a mixture of sulfuric acid and tartaric acid as a replacement for the hexavalent chromic acid process in electroplating. The removal of hexavalent chromium is an important improvement in the potential exposure/cancer risk associated with this process. Trivalent chromium used in the sealing process after anodizing, in the form of basic chromium sulfate, has a lower toxic profile than hexavalent chromium. Some ambiguity exists regarding the chemistry of Chemeon TCP-HF; however, the relative low concentration <2%, lack of high hazard notations in the SDS, and further dilution of the product prior to use all suggest Chemeon TCH-HF toxicity is moderate to low.

The formulations assessed in this report advance the safety of the process that used Cr(VI) based formulations. Since these new formulations are already in use outside of the DOD (particularly in the commercial aerospace industry), this effort provides further confirmation that they represent an improvement over traditional hexavalent chromium-based methods for occupational exposures, toxicology, and environmental impact.

9. RECOMMENDATIONS

Although there are concerns associated with the acute effects of sulfuric acid (as mist) and tartaric acid, the formulations represent an improvement over CAA. The toxic effects of sulfuric acid, tartaric acid, basic chromium(III) sulfate and dihydrogen fluoride were each assessed separately. The assumption is that there would be no significant synergistic or non-additive effects with these compounds as a mixture. APHC-TOX recommends evaluation of the mixture for skin corrosion and irritation using *in vitro* test systems. The toxicity hazard review did not evaluate downstream processes, such as polishing or grinding of surfaces treated with TSAA, where there is a potential for the release of Cr(III) particulates. Use of proper PPE, engineering controls and adherence to local, state and federal guidelines for occupational exposure and hazardous waste disposal are necessary.

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APPENDIX A

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APPENDIX B

TABLES

THE GLOBALLY HARMONIZED SYSTEM (GHS)

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 U.N. 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. Table B-1 includes tabular extracts of the criteria for acute toxicity (both oral and inhalation), dermal, and ocular effects. More information can be found in the original source (OSHA 2012).

Table B-1. GHS Acute Toxicity

	Category 1	Category 2	Category 3	Category 4	Category 5
Oral (mg/kg)	≤ 5	> 5 ≤ 50	> 50 ≤ 300	> 300 ≤ 2000	Criteria: -Anticipated LD_{50} between 2000 and 5000 mg/kg -Indication of significant effects in humans* -Any mortality in category 4* -Significant clinical signs in category 4* -Indications from other studies* *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)	≤ 0.05	> 0.05 ≤ 0.5	> 0.5 ≤ 1.0	>1.0 ≤ 5	

Table B-2. GHS Skin Corrosion/Irritation

<table>
<tr><td colspan="3">Skin Corrosion
Category 1</td><td>Skin Irritation
Category 2</td><td>Mild Skin Irritation
Category 3</td></tr>
<tr><td colspan="3">Destruction of dermal tissue; visible necrosis in at least one animal.</td><td rowspan="2">Reversible adverse effects in dermal tissue
Draize score: ≥ 2.3, ≤ 4.0, or persistent inflammation</td><td rowspan="2">Reversible adverse effects in dermal tissue.

Draize score: ≥ 1.5, ≤ 2.3</td></tr>
<tr><td>Subcategory 1A
Exposure < 3 minutes
Observation < 1 hour</td><td>Subcategory 1B
Exposure < 1 hour
Observation < 14 days</td><td>Subcategory 1C
Exposure < 4 hours
Observation < 14 days</td></tr>
</table>

Table B-3. GHS Eye Effects

<table>
<tr><td>Category 1
Serious Eye Damage</td><td colspan="2">Category 2
Eye Irritation</td></tr>
<tr><td rowspan="2">Irreversible damage 21 days after exposure

Draize score:
Corneal opacity ≥ 3
Iritis > 1.5</td><td colspan="2">Reversible adverse effects on cornea, iris, conjunctiva

Draize score:
Corneal opacity ≥ 1
Iritis > 1
Redness ≥ 2
Chemosis ≥ 2</td></tr>
<tr><td>Irritant
Subcategory 2A
Reversible in 21 days</td><td>Mild Irritant
Subcategory 2B
Reversible in 7 days</td></tr>
</table>

Table B-4. GHS Acute and Chronic Aquatic Toxicity

<table>
<tr><td>Acute Category I
Acute toxicity ≤ 1.00 mg/L</td><td>Acute Category II
Acute toxicity > 1.00 but ≤ 10.0 mg/L</td><td colspan="2">Acute Category III
Acute toxicity > 10.0 but < 100 mg/L</td></tr>
<tr><td>Chronic Category I
Acute toxicity ≤ 1.00 mg/L and lack of rapid biodegradability and log K_{OW} ≥ 4 unless BCF < 500.</td><td>Chronic Category II
Acute toxicity > 1.0 but ≤ 10.00 mg/L and lack of rapid biodegradability and log K_{OW} ≥ 4 unless BCF < 500 and unless chronic toxicity > 1 mg/L.</td><td>Chronic Category III
Acute toxicity > 10.0 but ≤ 100.00 mg/L and lack of rapid biodegradability and log K_{OW} ≥ 4 unless BCF < 500 and unless chronic toxicity > 1 mg/L.</td><td>Chronic Category IV
Acute toxicity > 100.0 mg/L and lack of rapid biodegradability and log K_{OW} ≥ 4 unless BCF < 500 and unless chronic toxicity > 1 mg/L.</td></tr>
</table>

Table B-5. List of Physical and Chemical Properties

	Molar Mass (g/mol)	**Melting Point (ºC)**	**Boiling Point (ºC)**	**Aqueous solubility (mg/L) @ 25ºC**	**log K_{ow}**	**log K_{oc}**	**Henry's Law Constant (atm-m^3/mol) @ 25ºC**	**Vapor Pressure mmHg @ 25°C**
Sulfuric acid (in H_2O)	98.08[a]	None, solution[a]	ca. 290[a]	1.77E+6[a]	-2.20[a] (est)	ND	$9.9X10^{-15}$ atm cu m/mol[e]	$5.93X10^{-5}$ mm Hg a[e]
L-Tartaric acid	150.09[b]	168 – 172[b]	None, solid	Soluble	ND	ND	na	na
Chromium sulfate, basic solution	392.17[c]	None, solution[c]	na	Soluble	na[c]	na[c]	na	na

Legend:
ND = no data;
na = not applicable.
Notes:
[a]O'Neil 2006 (p8973); Puritan Products. SDS. Sulfuric acid
[b]O'Neil 2006 (p9069); ScienceLab.com. MSDS. L-Tartaric acid
[c]O'Neil 2006 (p2223); ScienceLab.com. MSDS. Chromium sulfate
[d]Chemeon Surface Technology, Inc. CHEMEON TCP-HF, SDS.
[e]HSDB 2019a

Table B-6. Toxicity Data

Compound	Acute Oral LD_{50} (mg/kg)	Chronic Oral LOAEL (mg/kg-d)	Inhalation LC_{50} (g/m^3-h)	Dermal	Ocular	Mutagenicity	Carcinogenicity
Sulfuric acid H_2SO_4 in H_2O	2140 [a] (rat)	ND	0.255 [b] (rat) 0.160 [c] (mouse)	Corrosive [a]	corrosive[a]	ND	IARC 1 (only as mist)[a]
Basic Cr(III)Sulfate	1350 [d] (rat)	>1,468	ND	Irritant, sensitizer[d]	Irritant[d]	Negative	Negative (weight of evidence)[c]
L- Tartaric Acid	4360[e] (mouse) 7500[f] (rat) 5000[f] (dog)	600 (rat) NOAEL[f]	ND	Irritant[f]	Irritant[f]	Negative[g]	Negative[g]

Legend:
ND = no data
Notes:
[a]Smyth 1969; Puritan products.
[b]SDS inhalation rat LC_{50} given as 510/mg/m^3/2H; divided by 2 to get value per hour).
[c]HSDB, 2019a.
[d]ScienceLab.com. MSDS. Chromium sulfate.
[e]Locke 1942
[f]Sourkes 1950; ScienceLab.com. MSDS. L-Tartaric acid
[g]ECHA 2020

Table B-7. Categorization Criteria used in the Development of Environmental Safety and Occupational Health Severity (modified from Howe et al. 2006)

	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 Koc <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:
µg/L = micrograms per liter

Table B-8. GHS Health Hazard Statements (from https://pubchem.ncbi.nlm.nih.gov/ghs/#_haz; accessed Aug 3 2020)

Code	Hazard Statements	Hazard Class	Category
H300	Fatal if swallowed	Acute toxicity, oral	Category 1, 2
H301	Toxic if swallowed	Acute toxicity, oral	Category 3
H302	Harmful if swallowed	Acute toxicity, oral	Category 4
H303	May be harmful if swallowed	Acute toxicity, oral	Category 5
H304	May be fatal if swallowed and enters airways	Aspiration hazard	Category 1
H305	May be fatal if swallowed and enters airways	Aspiration hazard	Category 2
H310	Fatal in contact with skin	Acute toxicity, dermal	Category 1, 2
H311	Toxic in contact with skin	Acute toxicity, dermal	Category 3
H312	Harmful in contact with skin	Acute toxicity, dermal	Category 4
H313	May be harmful in contact with skin	Acute toxicity, dermal	Category 5
H314	Causes severe skin burns and eye damage	Skin corrosion/irritation	Category 1A, 1B, 1C
H315	Causes skin irritation	Skin corrosion/irritation	Category 2
H316	Causes mild skin irritation	Skin corrosion/irritation	Category 3
H317	May cause an allergic skin reaction	Sensitization, Skin	Category 1, 1A, 1B
H318	Causes serious eye damage	Serious eye damage/eye irritation	Category 1
H319	Causes serious eye irritation	Serious eye damage/eye irritation	Category 2A

Code	Hazard Statements	Hazard Class	Category
H320	Causes eye irritation	Serious eye damage/eye irritation	Category 2B
H330	Fatal if inhaled	Acute toxicity, inhalation	Category 1, 2
H331	Toxic if inhaled	Acute toxicity, inhalation	Category 3
H332	Harmful if inhaled	Acute toxicity, inhalation	Category 4
H333	May be harmful if inhaled	Acute toxicity, inhalation	Category 5
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled	Sensitization, respiratory	Category 1, 1A, 1B
H335	May cause respiratory irritation	Specific target organ toxicity, single exposure; Respiratory tract irritation	Category 3
H336	May cause drowsiness or dizziness	Specific target organ toxicity, single exposure; Narcotic effects	Category 3
H340	May cause genetic defects	Germ cell mutagenicity	Category 1A, 1B
H341	Suspected of causing genetic defects	Germ cell mutagenicity	Category 2
H350	May cause cancer	Carcinogenicity	Category 1A, 1B
H350i	May cause cancer by inhalation	Carcinogenicity	Category 1A, 1B
H351	Suspected of causing cancer	Carcinogenicity	Category 2
H360	May damage fertility or the unborn child	Reproductive toxicity	Category 1A, 1B
H360F	May damage fertility	Reproductive toxicity	Category 1A, 1B

Code	Hazard Statements	Hazard Class	Category
H360D	May damage the unborn child	Reproductive toxicity	Category 1A, 1B
H360FD	May damage fertility; May damage the unborn child	Reproductive toxicity	Category 1A, 1B
H360Fd	May damage fertility; Suspected of damaging the unborn child	Reproductive toxicity	Category 1A, 1B
H360Df	May damage the unborn child; Suspected of damaging fertility	Reproductive toxicity	Category 1A, 1B
H361	Suspected of damaging fertility or the unborn child	Reproductive toxicity	Category 2
H361f	Suspected of damaging fertility	Reproductive toxicity	Category 2
H361d	Suspected of damaging the unborn child	Reproductive toxicity	Category 2
H361fd	Suspected of damaging fertility; Suspected of damaging the unborn child	Reproductive toxicity	Category 2
H362	May cause harm to breast-fed children	Reproductive toxicity, effects on or via lactation	Additional category
H370	Causes damage to organs	Specific target organ toxicity, single exposure	Category 1
H371	May cause damage to organs	Specific target organ toxicity, single exposure	Category 2
H372	Causes damage to organs through prolonged or repeated exposure	Specific target organ toxicity, repeated exposure	Category 1

Code	Hazard Statements	Hazard Class	Category
H373	Causes damage to organs through prolonged or repeated exposure	Specific target organ toxicity, repeated exposure	Category 2
H400	Very toxic to aquatic life	Hazardous to the aquatic environment, acute hazard	Category 1
H401	Toxic to aquatic life	Hazardous to the aquatic environment, acute hazard	Category 2
H402	Harmful to aquatic life	Hazardous to the aquatic environment, acute hazard	Category 3
H410	Very toxic to aquatic life with long lasting effects	Hazardous to the aquatic environment, long-term hazard	Category 1
H411	Toxic to aquatic life with long lasting effects	Hazardous to the aquatic environment, long-term hazard	Category 2
H412	Harmful to aquatic life with long lasting effects	Hazardous to the aquatic environment, long-term hazard	Category 3
H413	May cause long lasting harmful effects to aquatic life	Hazardous to the aquatic environment, long-term hazard	Category 4
H420	Harms public health and the environment by destroying ozone in the upper atmosphere	Hazardous to the ozone layer	Category 1