EDGEWOOD CHEMICAL BIOLOGICAL CENTER
U.S. ARMY RESEARCH, DEVELOPMENT AND ENGINEERING COMMAND
Aberdeen Proving Ground, MD 21010-5424

ECBC-TR-1427

COMPARATIVE HUMAN HEALTH AND ENVIRONMENTAL TOXICOLOGY REVIEW OF SEVEN CANDIDATE OBSCURANT SMOKES FOR REPLACEMENT OF M83 GRENADE

Michael Simini
Ronald T. Checkai
Nancy A. Chester
Robert Kristovich
Mark V. Haley

RESEARCH AND TECHNOLOGY DIRECTORATE

March 2017

Disclaimer

The findings in this report are not to be construed as an official Department of the Army position unless so designated by other authorizing documents.

REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-0188

1. REPORT DATE *(DD-MM-YYYY)*	**2. REPORT TYPE**	**3. DATES COVERED** *(From - To)*
XX-03-2017	Final	Nov 2013 – Aug 2015

4. TITLE AND SUBTITLE
Comparative Human Health and Environmental Toxicology Review of Seven Candidate Obscurant Smokes for Replacement of M83 Grenade

5a. CONTRACT NUMBER

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER

6. AUTHOR(S)
Simini, Michael; Checkai, Ronald T.; Chester, Nancy, A.; Kristovich, Robert; and Haley, Mark V.

5d. PROJECT NUMBER
WBS R.0010255.28.2

5e. TASK NUMBER

5f. WORK UNIT NUMBER

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Director, ECBC, ATTN: RDCB-DRT-M, APG, MD 21010-5424

8. PERFORMING ORGANIZATION REPORT NUMBER
ECBC-TR-1427

9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)
Obscuration and Nonlethal Engineering Branch, Engineering Support Division, Engineering Directorate, ECBC, ATTN: RDCB-DEJ-S, APG, MD 21010-5424

10. SPONSOR/MONITOR'S ACRONYM(S)

11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION / AVAILABILITY STATEMENT
Approved for public release; distribution unlimited.

13. SUPPLEMENTARY NOTES

14. ABSTRACT-LIMIT 200 WORDS

The goal of this effort was to review available information regarding human health toxicology, ecotoxicology, and environmental fate and transport data for the component materials and reaction products from seven candidate smokes for replacement of the M83 grenade. A literature search and data survey were performed to initially assess candidate materials. Potential human health risks were estimated, including reported toxicity effects of oral, inhalation, dermal, and ocular exposures; developmental or reproductive effects such as the potential for mutagenesis and carcinogenesis; and mode(s) and mechanisms of toxicity. Estimated potential ecotoxicological risks included effects caused by exposures of aquatic and terrestrial species of different trophic levels to component compounds and reaction products. The primary component compounds of concern to human health were hexachloroethane and the aluminum/magnesium alloy. The primary reaction product of concern for human health risk was inhalation of titanium tetrachloride. The greatest risk to ecological receptors and environmental habitats was from excessive levels of soluble aluminum salts under acidic poorly buffered conditions in soil and water (pH < 4.9) and hydrochloric acid. Study results provide information that serves the decision-making process toward selection of a less-toxic obscurant and should not be considered a definitive analysis of candidate smoke toxicity.

15. SUBJECT TERMS

Toxicology	BC smoke	HX-10 smoke	HXLCO-10 smoke
DC-20 smoke	DCLCO-20 smoke	MGALLCO-15 smoke	LP smoke
M83 grenade			

16. SECURITY CLASSIFICATION OF:			**17. LIMITATION OF ABSTRACT**	**18. NUMBER OF PAGES**	**19a. NAME OF RESPONSIBLE PERSON**
a. REPORT	**b. ABSTRACT**	**c. THIS PAGE**			Renu B. Rastogi
U	U	U	UU	114	**19b. TELEPHONE NUMBER** *(include area code)* (410) 436-7545

Standard Form 298 (Rev. 8-98)
Prescribed by ANSI Std. Z39.18

Blank

EXECUTIVE SUMMARY

The goal of this effort was to review available human health, ecotoxicological, and environmental fate and transport data for the component materials and the reaction products from seven candidate smokes for replacement of the M83 grenade. A literature search and data survey were performed to initially assess candidate materials. The reaction products and ratios of compounds within each candidate formulation are approximations that were based on chemical properties and molecular masses of the component compounds. The human health and ecological toxicity assessments presented herein are provided as a tool to screen for the potential toxicity of candidate materials in several candidate smoke formulations. The assessment is based on toxicology data in the available literature for the individual chemicals that are components of the candidate materials. In some cases, models were based on the chemical and physical properties of the material and its expected behavior when present in living systems and the environment. For some chemical components and reaction products, there are significant data gaps, which increases the uncertainty of the results. Furthermore, none of the disseminated products of the candidate materials have been collected, analyzed, or evaluated for fate, bioavailability, or toxicological effects to human, animal, or ecological receptors under operational environments. Dissemination of these compounds simultaneously may result in cumulative effects that may not have been predicted, given the current information. Conditions unique to smoke dissemination from hand grenades may alter the toxicity designation that is presented in this report. This assessment is not intended to be used in a regulatory context. Study results are intended to provide information that will serve the decision-making process toward selection of a less-toxic obscurant and should not be considered a definitive analysis of candidate smoke toxicity.

The U.S. Army develops smoke- and obscurant-generation systems to mask movements of troops and mechanized equipment in support of Warfighter readiness and battlefield protection. Current warfare conditions and tactics require that land, sea, and air forces be trained with and have at their disposal a wide variety of offensive and defensive systems. The Army–Navy (AN)-8 and M83 grenades are used for deployment of white smoke in the visible range. Currently, formulations used for deployment of white smoke include HC smoke, with hexachloroethane (HCE) and zinc oxide that produces zinc chloride as the primary obscurant (AN-8 grenade), or terephthalic acid (TA) smoke, with TA as the primary obscurant (M83 grenade). HC smoke is an effective obscurant, but both HCE and zinc chloride have immediate toxic effects when inhaled and are also toxic to wildlife, plants, and aquatic organisms. TA smoke, which was developed to decrease human and ecological effects, has been found to be less toxic than HC smoke, but it lacks sufficient operational-screening characteristics and dissipates quickly. A formulation was needed for a replacement grenade that had greater screening capability than the M83 TA grenade and minimized toxicological impacts to the Warfighter and to ecological receptors.

Toxicological information that was reviewed to estimate potential human health risks includes reported toxicity effects of oral, inhalation, dermal, and ocular exposures; possible developmental or reproductive effects such as the potential for mutagenesis and carcinogenesis; and mode(s) and mechanisms of toxicity. Estimates of potential ecotoxicological risks include the effects caused by exposures of aquatic and terrestrial species of different trophic levels to

component compounds and predicted reaction products from the candidate smoke formulations. Toxicological information was derived directly from primary sources when available. Sources used in this search included publications from international journals and national, international, and Department of Defense databases.

Based on the available literature, the primary component compounds of concern in the production of the candidate smoke formulations are HCE and the aluminum/magnesium (Al/Mg) alloy. HCE is listed as a carcinogen for laboratory animals and a possible carcinogen for humans. The Al/Mg alloy is an inhalation hazard and possible explosive hazard in an occupational setting during production of the munitions. HCE and Dechlorane Plus additives (Occidental Oil and Gas Corporation, Houston, TX) can potentially biomagnify in ecosystems, although field surveys have shown that biomagnification in the field is highly variable and species-dependent. Based on data collected during this review and on potential exposure scenarios, smoke formulations HX-10 and HXLCO-10, which contain HCE and Al/Mg alloys, pose the greatest risk to production workers.

The primary reaction product of concern for human inhalation health risk during operational activities with the HX-10 and HXLCO-10 smoke formulations is titanium tetrachloride, which readily transforms to titanium dioxide and to hydrochloric acid under moist conditions in the environment and within body tissues. Human exposure to these chemicals can be mitigated by the use of personal protective equipment (PPE), and these formulations should not be used without PPE; they are not recommended for indoor use because of inhalation risk.

For the reasons stated above, the smoke formulations HX-10 and HXLCO-10 also pose a risk to ecological receptors. The greatest persistent risk to ecological receptors and environmental habitats is from excessive levels of soluble aluminum salts under acidic, poorly-buffered conditions in soil and water (pH < 4.9). All of the candidate formulations contained aluminum, except BC smoke.

Respective scores for relative toxicities of predicted reaction products from the candidate smoke formulations were calculated as the averages of the combined human health and toxicity scores. The resulting predicted toxicities of the candidate smokes, with respect to each other and those of HC smoke and TA are, in order from greatest to least toxicity:

HC smoke > HXLCO-10 = HX-10 > DCLCO-20 > DC-20 > LP = MGALLCO-15 > BC > TA.

PREFACE

The work described in this report was authorized under project no. WBS R.0010255.28.2. It was started in November 2013 and completed in August 2015.

The use of either trade or manufacturers' names in this report does not constitute an official endorsement of any commercial products. This report may not be cited for purposes of advertisement.

This report has been approved for public release.

Acknowledgements

We acknowledge the Obscuration and Nonlethal Engineering Branch, Engineering Support Division, Engineering Directorate, U.S. Army Edgewood Chemical Biological Center (ECBC; Aberdeen Proving Ground, MD), especially Mr. Tim Lyons and Ms. Christine Pan for supporting this project. We also thank Dr. Joseph Domanico, Ammunition Control Officer at ECBC and Dr. Anthony Shaw, Research Chemist at Armament Research, Development and Engineering Center (Picatinny Arsenal, NJ) for providing modeled balanced equations for the candidate smokes.

Blank

CONTENTS

TABLES

Blank

COMPARATIVE HUMAN HEALTH AND ENVIRONMENTAL TOXICOLOGY REVIEW OF SEVEN CANDIDATE OBSCURANT SMOKES FOR REPLACEMENT OF M83 GRENADE

1. PURPOSE AND NEED

The U.S. Army develops smoke and obscurant generation systems to mask movements of troops and mechanized equipment in support of Warfighter readiness and battlefield protection. Current warfare conditions and tactics require that land, sea, and air forces be trained with and have at their disposal a wide variety of offensive and defensive systems. Among these systems, smokes and obscurants have long been employed to mask movements of troops and mechanized equipment. Smoke formulations, which have been employed on the battlefield since World War I (AMC, 1967), are widely used to visually mask the movements of ground and sea forces (Driver et al., 1993).

The AN-8 and M83 grenades are used for deployment of white smoke in the visible range (U.S. Army, 2000). Currently, formulations used for deployment of white smoke include HC smoke with hexachloroethane (HCE) and zinc oxide that produces zinc chloride as the primary obscurant (AN-8 grenade) or terephthalic acid (TA) smoke with TA as the primary obscurant (M83 grenade). HC smoke is an effective obscurant, but both HCE and zinc chloride have immediate toxic effects when inhaled and are also toxic to wildlife, plants, and aquatic organisms (ATSDR, 1997a; Cichowicz, 1983; Eaton et al., 1994; Fisher et al., 1990; IARC, 1999; Sadusky et al., 1993; Shinn et al., 1985), and therefore, is no longer in production. TA smoke, which was developed to decrease human and ecological effects, has been found to be less toxic than HC smoke, but it lacks sufficient operational-screening characteristics and dissipates quickly (Haley et al., 1995; Sadusky et al., 1995; Thomson et al., 1988). Therefore, a formulation was needed for a replacement grenade that had greater screening capability than the M83 TA grenade and minimized toxicological impacts to the Warfighter and to ecological receptors.

Recently, research has focused on developing less-toxic compositions that disseminate benign salts and oxides. Webb and colleagues (2012) examined several formulations that used synthetic sea salt. Transmissions through the sea salt compositions were much higher as compared with HC smoke, but lower as compared with TA smoke. The smoke formulations from this study were less toxic than the HC and TA smokes. The authors recommended searching for component materials that efficiently absorb atmospheric moisture and have a low toxicity profile. Studies with compositions of boron carbide (B_4C) and various salts produced moderate obscuration with relatively low toxicity (Shaw et al., 2013, 2015). As a result of the encouraging findings from recent studies, an effort is underway to optimize obscuration qualities and minimize human health and environmental effects. Several candidate formulations are under consideration for the replacement of the current HC smoke formulation. As part of item development, analyses of the potential human health and environmental effects of both the grenade components and disseminated reaction products from the candidate obscurants are warranted.

Statement of Work

The goal of this effort was to provide human health, ecotoxicological, and environmental fate and transport data for the component materials and reaction products from the candidate items. This was accomplished through a literature search and data survey to provide an initial assessment of the candidate materials. This review is based on toxicology data for individual chemicals that are components of the candidate materials. None of the disseminated products of the candidate materials have been collected, analyzed, or evaluated for fate, bioavailability, or toxicological effects to human, animal, or ecological receptors. The reaction products and ratios of compounds within each candidate formulation are approximations based on chemical properties of the component compounds. Furthermore, dissemination of these compounds simultaneously may result in cumulative effects that may not have been predicted, given the current information. This assessment is not intended to be used in a regulatory context. Study results provide information that will serve the decision-making process toward selection of a less-toxic obscurant and should not be considered a definitive analysis of candidate smoke toxicity.

2. METHODS

A literature review was conducted on the candidate materials, formulations, and predicted reaction products for which the following required information has been made available to the extent possible. This literature review focused on information regarding human health and terrestrial and aquatic toxicity and fate and is presented in report form. The format for comparative analysis of toxicological data was adapted from Adams and Eck (2013).

Required information included the following:

1. Full candidate material fill and formulation composition, including coatings, additives, percentages, and any disclosed proprietary information;
2. Description of how the item functions, including chemistry, pyrotechnics, detonation, aerosolization, etc.;
3. Proposed canister and payload configuration, if known;
4. Description of the material that will ultimately be deposited to soil and/or water surfaces during use, if known. If unknown, provide best estimate of predicted results; and
5. Airborne and deposition concentrations measured, modeled, or otherwise estimated, if known. If unknown, provide best estimate of predicted results.

Physical and toxicological properties for the study compounds were sought through identification by Chemical Abstracts Service Registry Numbers (CAS RNs), and where applicable, by correlations between synonyms and trade names.

Basic physical and chemical properties were determined by consulting authoritative primary and secondary sources when such information was available. The properties necessary to assess fate and transport in the environment included the following:

- molecular weight;
- Henry's law constant;
- logarithm of octanol–water partition coefficient (log K_{OW});
- water solubility;
- boiling point;
- logarithm of organic carbon partition coefficient (log K_{OC}); and
- vapor pressure (VP).

Seven white smoke formulations were considered in this review: BC, HX-10, HXLCO-10, DC-20, DCLCO-20, MGALLCO-15, and LP. Individual chemicals and representative proportions of components and combustion products for the white smoke formulations are shown in Table 1.

The balanced equation for BC smoke including primary grenade reactants (primary components) and corresponding reaction products predicted by chemical reaction modeling (FactSage thermodynamic modeling software, FactSage 6.3; Center for Research in Computational Thermochemistry-ThermFact, Inc. [Montréal, Canada] and GTT-Technologies [Herzogenrath, Germany]) are shown in eq 1 (Shaw et al., 2015). The products potassium metaborate and boron trioxide are initially produced in the BC smoke gas phase, but rapidly condense upon exiting the reaction zone. Potassium chloride is also volatilized and subsequently condensed unchanged. The small amounts of calcium stearate (secondary component) are expected to decompose or oxidize to water and carbon–calcium oxides. Air oxidation of carbon monoxide produces carbon dioxide, and calcium oxide can react with moisture and carbon dioxide to ultimately form calcium carbonate.

The balanced equations for the HX-10, HXLCO-10, DC-20, DCLCO-20, MGALLCO-15, and LP formulations, including grenade reactants (components) and corresponding reaction products predicted by chemical reaction modeling (ICT thermodynamic code, Windows Frontend, version 1.00; Fraunhofer-Institut für Chemische Technologie [ICT; Pfinztal, Germany]), are shown in eqs 2 through 7.

BC:

$$5B_4C + 14KNO_3 + 8KCl \rightarrow 14KBO_2 + 3B_2O_3 + 8KCl + 7N_2 + 5CO \quad (1)$$
$$5CO + 5/2O_2 \text{ (atmospheric)} \rightarrow 5CO_2$$

HX-10:

$$4Mg_{(s)} + 4Al_{(s)} + 6C_2Cl_6 + 6TiO_2 \rightarrow \quad (2)$$
$$3MgCl_2 + 2AlCl_3 + 6TiCl_4 + Al_2O_3 + MgO + 4CO_2 + 8C$$

HXLCO-10:

$$4Mg_{(s)} + 4Al_{(s)} + 7C_2Cl_6 + 6TiO_2 + 4Li_2CO_3 \rightarrow \quad (3)$$
$$3MgCl_2 + 2AlCl_3 + 6TiCl_4 + 6LiCl + Al_2O_3 + MgO + Li_2O + 5CO_2 + 9CO + 4C$$

DC-20:

$$3Mg + 3Al_{(s)} + C_{18}H_{12}Cl_{12} + NH4Cl \rightarrow 3MgC_{l2} + 2AlCl_3 + NH_4Cl + 18C + 6H_2 + Al_{(l)} \quad (4)$$

DCLCO-20:

$$Mg_{(s)} + 2Al_{(s)} + C_{18}H_{12}Cl_{12} + NH_4Cl + 2Li_2CO_3 \rightarrow MgCl_2 + 2AlCl_3 + NH_4Cl + 4LiCl + 20C + 6H_2O \quad (5)$$

MGALLCO-15:

$$8Mg_{(s)} + 8Al_{(s)} + 6KNO_3 + KCl + 3Li_2CO_3 + 2C_2H_4O_2N_4 \rightarrow 6MgO + 4Al_2O_3 + KCl + 3K_2CO_3 + 3Li_2O + 2Mg_{(l)} + 7N_2 + 4H_2 + 3C + CO \quad (6)$$

LP:

$$3LiClO_4 + 8B \rightarrow 3LiCl + 4B_2O_3 \quad (7)$$

Table 1. Individual Chemicals, Chemical Symbols, CAS RNs, and Representative Proportions of Components and Predicted Combustion Products for Candidate White Smoke Formulations*

BC Smoke				
Formulation	**Name**	**Chemical Symbol**	**CAS RN**	**Percentage**
Component	Potassium nitrate	KNO_3	7757-79-1	60
	Potassium chloride	KCl	7447-40-7	25
	Boron carbide	B_4C	12069-32-8	13
	Calcium stearate	$[CH_3(CH_2)_{16}COO]_2Ca$	1592-23-0	2
Reaction product	Potassium metaborate	KBO_2	13709-94-9	50.5
	Potassium chloride	KCl	7447-40-7	24.1
	Nitrogen gas	N_2	7727-37-9	7.9
	Boron trioxide	B_2O_3	1303-86-2	6.9
	Carbon dioxide	CO_2	124-38-9	6.6
	Calcium carbonate	$CaCO_3$	471-34-1	Trace amount
HX-10 Smoke				
Formulation	**Name**	**Chemical Symbol**	**CAS RN**	**Percentage**
Component	Hexachloroethane	C_2Cl_6	67-72-1	67.5
	Titanium dioxide	TiO_2	13463-67-7	22.8
	Aluminum metal	Al	7429-90-5	5.1
	Magnesium metal	Mg	7439-95-4	4.6
Reaction product	Titanium tetrachloride	$TICl_4$	7550-45-0	54.1
	Magnesium chloride	MgCl2	7786-30-3	13.6
	Aluminum chloride	$AlCl_2$	7446-70-0	12.7
	Carbon dioxide	CO_2	124-38-9	8.4
	Aluminum oxide	Al_2O_3	1344-28-1	8.4
	Carbon	C	7440-44-0	4.6
	Magnesium oxide	MgO	1309-48-4	1.9

table continued

Table 1. Individual Chemicals, Chemical Symbols, CAS RNs, and Representative Proportions of Components and Predicted Combustion Products for Candidate White Smoke Formulations* (continued)

HXLCO-10 Smoke				
Formulation	**Name**	**Chemical Symbol**	**CAS RN**	**Percentage**
Component	Hexachloroethane	C_2Cl_6	67-72	62.8
	Titanium dioxide	TiO_2	13463-67-7	18.2
	Lithium carbonate	Li_2CO_3	554-13-2	11.2
	Aluminum metal	Al	7429-90-5	4.1
	Magnesium metal	Mg	7439-95-4	3.7
Reaction product	Titanium tetrachloride	$TiCl_4$	7550-45-0	43.2
	Magnesium chloride	$MgCl_2$	7786-30-3	10.8
	Aluminum chloride	$AlCl_2$	7446-70-0	10.1
	Lithium chloride	LiCl	7447-41-8	9.6
	Carbon monoxide	CO	630-08-0	9.6
	Carbon dioxide	CO_2	124-38-9	9.1
	Aluminum oxide	Al_2O_3	1344-28-1	3.9
	Carbon	C	7440-44-0	1.8
	Magnesium oxide	MgO	1309-48-4	1.5
	Lithium oxide	Li_2O	12057-24-8	1.1
MGALLCO-15 Smoke				
Formulation	**Name**	**Chemical Symbol**	**CAS RN**	**Percentage**
Component	Potassium nitrate	KNO_3	7757-79-1	39.3
	Lithium carbonate	Li_2CO_3	554-13-2	14.4
	Aluminum metal	Al	7429-90-5	14.0
	Magnesium metal	Mg	7439-95-4	12.6
	Azodicarbonamide	$C_2H_4O_2N_4$	123-77-3	15.0
	Potassium chloride	KCl	7447-40-7	4.8
Reaction product	Potassium carbonate	K_2CO_3	584-08-7	27.7
	Aluminum oxide	Al_2O_3	1344-28-1	27.2
	Magnesium oxide	MgO	1309-48-4	16.2
	Nitrogen	N_2	7727-37-9	9.3
	Lithium oxide	Li_2O	12057-24-8	6.0
	Potassium chloride	KCl	7447-40-7	5.0
	Magnesium liquid	Mg	7439-95-4	3.2
	Carbon	C	7440-44-0	2.4
	Carbon monoxide	CO	630-08-0	1.9
	Hydrogen	H_2	1333-74-0	1.1
LP Smoke				
Formulation	**Name**	**Chemical Symbol**	**CAS RN**	**Percentage**
Component	Lithium perchlorate	$LiClO_4$	7791-03-9	78.7
	Boron	B	7440-42-8	21.3
Reaction product	Boron trioxide	B_2O_3	1303-86-2	68.6
	Lithium chloride	LiCl	7447-41-8	31.4

*Percentages of components were estimated as follows: molecular weight of compound multiplied by the number of moles divided by total mass of formulation, then multiplied by 100. Percentages of reaction products were estimated based on the balanced equations for each formulation (eqs 1–7), which were derived from FactSage thermodynamic modeling software for BC smoke; and from ICT thermodynamic code, Windows Frontend, version 1.00 for HX-10, HXLCO-10, DC-20, DCLCO-20, MGALLCO-15, and LP smokes.

Information on combustion, explosion, and thermal decomposition products was also collected, if it was available. Toxicological information needed to estimate potential human health risks includes reported toxicity effects of oral, inhalation, dermal, and ocular exposures; possible developmental or reproductive effects such as the potential for mutagenesis and carcinogenesis; and mode(s) and mechanisms of toxicity. Toxicological information was derived directly from primary sources when available. Estimates of potential ecotoxicological risks include the effects caused by exposures of aquatic species, soil invertebrates, plants, mammals, and birds to component compounds and reaction products from the candidate smoke formulations.

Sources used in this search include publications from the U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry (ATSDR); and Merck and Company, Inc. (O'Neil, 2006; O'Neil et al., 2001); the Defense Technical Information Center (DTIC); Food and Agriculture Organization of the United Nations (FAO); the World Health Organization (WHO); the International Agency for Research on Cancer (IARC); the International Chemical Safety Cards developed by the National Institute for Occupational Safety and Health (NIOSH); and the U.S. National Library of Medicine Toxicology Data Network (TOXNET), which provides access to information from the National Institutes of Health (NIH) and the U.S. Environmental Protection Agency (USEPA).

TOXNET is a suite of individual databases including ChemIDPlusLite (CIDPL) and ChemIDPlus Advanced, which are used to search for chemical and registration numbers and chemical identification and structure, respectively; Hazardous Substances Databank (HSDB); Chemical Carcinogenesis Research Information System (CCRIS); Developmental and Reproductive Toxicology; Directory of Information Resources Online (DIRLINE); Genetic Toxicology; Haz-Map (database linking chemicals, jobs, and diseases); Household Products Databank (potential health effects of chemicals in common household products); Integrated Risk Information System (IRIS); International Toxicity Estimates for Risk; Toxicology Information Online (TOXLINE); Toxic Release Inventory; and lactation database (LactMed; a database of drugs and other chemicals to which breastfeeding mothers may be exposed).

DTIC, the USEPA ECOTOXicology database system (ECOTOX), and the National Institute of Environmental Health Sciences National Toxicology Program (NTP) databases were used as document and information resources. Primary sources were identified and retrieved using the NIH PubMed database, and ISI Web of Science. (Web of Science is a registered trademark of Thomson Reuters; DTIC is a registered trademark of the Defense Technical Information Center; TOXNET, CIDPL, ChemIDPlus, DIRLINE, TOXLINE, and PubMed are registered trademarks of the U.S. National Library of Medicine; Ovid is a registered trademark of Ovid Technologies, Inc.; and EBSCOhost is a registered trademark of EBSCO Publishing.)

Persistence, bioaccumulation, human health toxicity, and ecotoxicity were evaluated using the general categories of hazard (i.e., low, moderate, and high) that were modified from Griffiths (2009). Table 2 describes the criteria used in this categorization. Relative proportions of each substance were also factored into final assessments.

Table 2. Categorization Criteria Used in the Development of ESOH Severity†

Criteria	Low	Moderate	High
Persistence	Readily biodegrades (<28 days)	Degradation half-life: water < 40 days soil < 120 days	Degradation half-life: water > 40 days soil > 120 days
Transport	Water soluble < 10 mg/L Log K_{OC} > 2.0	Water soluble 10–1000 mg/L Log K_{OC} 2.0–1.0	Water soluble > 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 or mutagenicity; subchronic oral LOAEL > 200 mg/kg/day; acute oral and dermal LD_{50} > 2000 mg/kg; inhalation LD_{50} > 20 mg/L	Mixed evidence for carcinogenicity and mutagenicity (B2, 2); subchronic oral LOAEL 5–200 mg/kg/day; acute oral 25 < LD_{50} ≤ 2000 mg/kg; dermal 50 < LD_{50} ≤ 2000 mg/kg; inhalation 0.5 < LD_{50} ≤ 20 mg/L	Positive corroborative evidence for carcinogenicity and mutagenicity; subchronic LOAEL < 5 mg/kg/day; acute oral LD_{50} ≤ 25 mg/kg; dermal LD_{50} ≤ 50 mg/kg; inhalation LD_{50} ≤ 0.5 mg/L
Ecotoxicity	Acute $LC(D)_{50}$ > 1 mg/L or 1500 mg/kg; subchronic EC_{50} > 100 µg/L or LOAEL > 100 mg/kg/day	Acute $LC(D)_{50}$ 1–0.1 mg/L or 1500–150 mg/kg; subchronic EC_{50} 100–10 µg/L or LOAEL 10–100 mg/kg/day	Acute $LC(D)_{50}$ < 100 µg/L or < 150 mg/kg; subchronic LOAEL < 10 mg/kg/day

†Notes: Information modified from Griffiths, 2009; O'Brien and Ross, 1988; and Swanson and Socha, 1997.
EC_{50}, 50% effective concentration.
ESOH, environment, safety, and occupational health.
LOAEL, lowest-observed-adverse-effect level.
LC_{50}, lethal concentration (concentration expected to result in 50% lethality to a population of test animals).
LD_{50}, lethal dose (dose expected to result in 50% lethality to a population of test animals).

Mode of Dissemination

A combustion reaction produces clouds of white smoke to screen the activities of small military units and for use in ground-to-air signaling.

3. RESULTS

3.1 Physical and Chemical Properties

Physical and chemical properties of component compounds and disseminated reaction product compounds are summarized in Tables 3 and 4, respectively. Chemical compounds found in Tables 3 and 4 correspond to the designated smoke candidates: (1) BC, (2) HX-10, (3) HXLCO-10, (4) DC-20, (5) DCLCO-20, (6) MGALLCO-15, and (7) LP. When data were not found, the indication "nd" (no data) was inserted. In some cases, the property named in

the table was not applicable to the substance being described. For example, if the substance is a nonvolatile solid or an inorganic salt, volatilization and bioconcentration will not be expected. In these cases, vapor pressure, log K_{OW}, log K_{OC}, and the Henry's Law constant are negligible and will not apply. Properties that were not applicable were noted as "n/a."

Table 3. Physical and Chemical Properties of Component Compounds

Compound	Smoke Formulation	Molar Mass (g/mol)	Melting Point (°C)	Boiling Point (°C)	Aqueous Solubility at 25 °C (mg/L)	Log K_{OW} at 25 °C	Log K_{OC} (soil)	Henry's Law Constant at 25 °C (atm-m^3/mol)	Vapor Pressure at 25 °C (mmHg)
Boron carbide	(1)	55.2[a]	2450[a]	3500	Insoluble[f]	n/a	n/a	n/a	n/a
Calcium stearate	(1)	607.02[a]	145–160	nd	2[a]	8.23[a]	8.4[a]	n/a	4.76×10^{-7} [a]
Potassium chloride	(1) (6)	74.55[a]	771	1500 (sublimes)	Soluble[f]	n/a	n/a	n/a	n/a
Potassium nitrate	(1) (6)	101.10[a]	633.2[a]	400[a]	357 g/L[a]	n/a	n/a	n/a	n/a
Hexachloroethane	(2) (3)	236.74[b]	186.8[a] (sublimes)	186.8[a] (sublimes)	Insoluble[a]	4.04[d]	2188[d]	3.89×10^{-3} [d]	0.4 at 20 °C[d]
Titanium dioxide	(2) (3)	79.90[f]	1855.0[a]	3000[f]	Insoluble[a]	n/a	n/a	n/a	n/a
Magnesium metal	(2) (3) (4) (5)	24.30[a]	651[a]	1100[a]	Insoluble[a]	n/a	n/a	n/a	n/a
Aluminum metal	(2) (3) (4) (5)	26.98[a]	660[a]	2327[a]	Insoluble[a]	n/a	n/a	n/a	n/a
Dechlorane Plus	(4) (5)	653.72[b]	350[e] (decomposes)	350[e] (decomposes)	Insoluble[a]	9.0[b] (est.)	7.7[e] (est.)	7.4×10^{-6} [b] (est.)	4.71×10^{-8} [b] (est.)
Ammonium chloride	(4) (5)	53.5[a]	338[a] (sublimes)	520[a]	28% (w/w)[a]	n/a	n/a	n/a	n/a
Lithium carbonate	(5) (6) (7)	73.89[a]	618[a]	1300[a] (decomposes)	Low[f]	n/a	–3.27[d]	n/a	n/a
Azodicarbonamide	(6)	116.08[a]	225[a] (decomposes)	n/a	Insoluble[a]	–1.7[d]	0.48[d]	8.2×10^{-13} [d]	1.9×10^{-10} [d]
Lithium perchlorate	(7)	106.39[a]	236[a]	430[a] (decomposes)[a]	37.5%, Hygroscopic[f]	n/a	n/a	n/a	n/a
Boron	(7)	10.81[a]	2075[a]	4000[a]	Insoluble[a]	n/a	n/a	n/a	n/a

Notes: [a]O'Neil, 2006; [b]Feo et al., 2012; [c]USEPA, 1988; [d]HSDB, 2013; [e]USEPA, 2011a; [f]Haynes, 2010.
Smoke formulations: (1) BC, (2) HX-10, (3) HXLCO-10, (4) DC-20, (5) DCLCO-20, (6) MGALLCO-15, and (7) LP.
K_{OC} (soil), soil organic carbon–water partition coefficient.
n/a, not applicable.
nd, data were not found (no data).
est, estimated.

Table 4. Physical and Chemical Properties of Reaction Products

Compound	Smoke Formulation	Molar Mass (g/mol)	Melting Point (°C)	Boiling Point (°C)	Aqueous Solubility at 25 °C (mg/L)	Log K_{OW} at 25 °C	Log K_{OC} (soil)	Henry's Law Constant at 25 °C (atm-m^3/mol)	Vapor Pressure at 25 °C (mmHg)
Potassium metaborate	(1)	81.91[a]	950[f]	nd	Soluble[f]	n/a	n/a	n/a	n/a
Potassium chloride	(1) (6)	74.55[a]	771[f]	1500 (sublimes)	Soluble[f]	n/a	n/a	n/a	n/a
Titanium tetrachloride	(2) (3)	189.73[a]	–24.1[f]	136.4[f]	Soluble[f]	n/a	n/a	n/a	n/a
Carbon dioxide	(1) (2) (3)	44.01[f]	–55.6[a]	–78.5[a]	Soluble[f]	n/a	n/a	n/a	830
Magnesium chloride	(2) (3) (4) (5)	95.23[a]	708[f]	1412[f]	Soluble[f]	n/a	n/a	n/a	n/a
Aluminum chloride	(2) (3) (4) (5)	133.34[a]	Volatilizes without melting[a]	n/a	Explosive when mixed with water[a]	n/a	n/a	n/a	n/a
Magnesium oxide	(2) (3) (6)	40.32[a]	2800[a]	3600[f]	Slight[g]	n/a	n/a	n/a	n/a
Aluminum oxide	(2) (3) (6)	74.55[a]	771[a]	1500[a] (sublimes)	Insoluble[a]	n/a	n/a	n/a	n/a
Carbon	(2) (3) (4) (5) (6)	12.01[a]	3550[f]	4827[f]	Insoluble[a]	n/a	n/a	n/a	n/a
Carbon monoxide	(3) (6)	28.01	–205.0[a]	–191.5[a]	Slight[f]	n/a	n/a	1.04	1.55×10^8
Lithium chloride	(3) (5) (7)	42.4[a]	613[a]	1360[a]	Soluble[f]	n/a	n/a	n/a	n/a
Lithium oxide	(3) (6)	29.88[a]	1570[a]	1200 at 600 atm[a]	Reacts to form Li_2OH[d]	n/a	n/a	n/a	n/a
Potassium carbonate	(6)	138.20[a]	891[a]	Decomposes[f]	Soluble[f]	n/a	n/a	n/a	n/a

table continued

Table 4. Physical and Chemical Properties of Reaction Products (continued)

Compound	Smoke Formulation	Molar Mass (g/mol)	Melting Point (°C)	Boiling Point (°C)	Aqueous solubility at 25 °C (mg/L)	Log K_{OW} at 25 °C	Log K_{OC} (soil)	Henry's Law Constant at 25 °C (atm-m^3/mol)	Vapor Pressure at 25 °C (mmHg)
Ammonium chloride	(4) (5)	53.5[a]	338[a] (sublimes)	520[a]	28% (w/w)[a]	n/a	n/a	n/a	n/a
Water	(5)	18.01[a]	0[a]	100[a]	n/a	n/a	n/a	n/a	n/a
Nitrogen gas	(1)(6)	28[a]	–210.01[f]	–195.79[f]	2.33[f]	n/a	n/a	n/a	n/a
Boron trioxide	(1) (7)	69.62[a]	450[a]	1860[a] (sublimes)	Soluble[a]	n/a	n/a	n/a	n/a

Notes: [a]O'Neil, 2006; [b]Feo et al., 2012; [c]USEPA, 1988; [d]HSDB, 2013; [e]USEPA, 2011; [f]Haynes, 2010; [g]McGraw-Hill, 2003.
Smoke formulations: (1) BC, (2) HX-10, (3) HXLCO-10, (4) DC-20, (5) DCLCO-20, (6) MGALLCO-15, and (7) LP.
K_{OC} (soil), soil organic carbon–water partition coefficient.
n/a, not applicable.
nd, data were not found (no data).

3.2 Estimated Airborne and Deposition Concentrations

No published data were found for airborne and deposition concentrations of the reaction products from the candidate formulations described herein. Estimated airborne and deposition concentrations cannot be reasonably predicted using the reaction products resulting from the use of models. This assessment will be used to select formulations that will minimize toxicity and maintain operational effectiveness. Testing will then need to be performed to determine airborne and deposition concentrations that result from the smoke cloud.

3.3 Toxicology of Component Compounds and Reaction Products from Candidate Smokes

The comparative human health and ecotoxicology information discussed herein was determined by analysis of data that were derived from literature searches relating to the chemical and physical properties, toxicology, transport, and environmental fate of component compounds and reaction products. These are summarized in Tables 5 through 32, which use criteria established in Table 2. Detailed characterization, toxicological data, and fate and transport data for component compounds and reaction products can be found in the appendix. These data were the best available at the time of this study. In many cases, data from empirical studies were lacking, and the toxicity assessment was based on chemical and physical properties of the chemical compound and its environment. Furthermore, toxicity testing was not performed on the component compounds or reaction products as functional grenades under operational conditions. At the time of this review, the nature and quantity of the disseminated products had not been analytically determined. The characterization and relative quantities of the disseminated reaction products were estimated. Therefore, the toxicity values presented in this report were best estimates that may change after data from empirical tests are collected and analyzed. Similar data were also summarized for the reaction products from HC smoke to compare with those of the candidate smokes (Tables 33 and 34).

3.3.1 BC Smoke

Toxicity from exposure to the component compounds of BC smoke during production was low (Table 5). Potassium chloride caused anemia in laboratory animals at extremely high levels (HSDB, 2013). Boron carbide is typically not toxic in the micron-size range, which is the size of the particles most likely to occur during smoke dissemination. Nano-sized particles can potentially cause increased toxicity; therefore, particle size should be monitored during smoke production (HSDB, 2013). Effects can be avoided by the use of personal protective equipment (PPE) by production workers. Carbon dioxide may be toxic at extremely high levels, which are not expected to be attained during dissemination. Ecotoxicity of component compounds of BC smoke (Table 6) is low, except in rare instances where excessive concentrations of potassium nitrate occur in water, which is usually due to high nutrient inputs from fertilizer runoff (HSDB, 2013). Proper disposal of component products can prevent contamination of ecosystems. Toxicity to humans from reaction products from BC smoke is expected to be low (Table 7).

Reaction products from BC smoke are also expected to be of low toxicity in the environment (Table 8). Many of the elements are essential plant nutrients (Barker and Pilbeam, 2006).

3.3.2 HX-10 Smoke

The HX-10 smoke component compound that is of primary concern for both human health and ecological toxicity is HCE (Tables 9 and 10). The USEPA lists HCE as a possible human carcinogen (Group C), and HCE has been shown to be carcinogenic to mice and rats (USEPA, 1988). HCE also has potential to bioconcentrate in the environment (HSDB, 2013). A detailed literature review, including data from studies that estimated the effects of HCE on human health and ecological receptors, is provided in the appendix. In addition, inhalation of excessive levels of magnesium metal has been shown to cause "metal fume fever", which is a condition that causes flu-like symptoms in production workers. The dust particles from metals are potentially explosive during production (HSDB, 2013).

Regarding a reaction product of HX-10 smoke, titanium tetrachloride by itself is not highly toxic to human health. However, it readily reacts with water to form hydrochloric acid in the air and in the human body (ATSDR, 1997b; Tables 11 and 12). Inhalation of titanium tetrachloride by unprotected individuals has been shown to produce severe burns caused by hydrolysis to hydrochloric acid after inhalation (ATSDR, 1997b). Inhalation studies with titanium tetrachloride in rats resulted in increased edema and death in the study animals (DuPont, 1980). Aluminum chloride was toxic to the aquatic invertebrate, *Daphnia magna* (LC_{50} = 39 mg/L). Contaminated sediments in acidified watersheds can provide a source of aluminum to the water column (Wong et al., 1989).

3.3.3 HXLCO-10 Smoke

The HXLCO-10 smoke formulation is the same as HX-10, except for the addition of lithium carbonate. Risks to human health and ecotoxicological effects are similar to those posed by HX-10 smoke, with the addition of toxicity to citrus plants (Bingham et al., 1964) upon exposure to excessive lithium in the soil (Tables 13–16).

3.3.4 DC-20 Smoke

Component compounds of the DC-20 smoke formulation have low human health toxicity, except for moderate toxicity of magnesium and aluminum metals through the inhalation route of exposure (Table 17). Metal dust particles may cause the respiratory disorder metal fume fever and may be an explosive hazard in an occupational setting (ATSDR, 2008a; HSDB, 2013). For ecotoxicological risk, the component compound Dechlorane Plus has the potential to be highly persistent and bioaccumulative and has a relatively high log K_{OW} (9.0 as calculated; Table 3); therefore, the ecotoxicology assessment for Dechlorane Plus in aquatic organisms is moderate (Table 18). Fish tissue in many parts of world have detectable levels of Dechlorane Plus; however, the biomagnifications factors (BMFs) are frequently <1.0. Detection of BMFs >1.0 are considered to be significant (HSDB, 2013). Sverko (2011) suggested that variability may be a result of species-specific differences in biotransformation, limited sample

size, or a violation of the steady-state assumption that is required for calculating BMFs (i.e., the chemical concentrations in the food web do not approximate steady-state conditions).

Reaction products from DC-20 smoke are expected to have little impact on human health (Table 19). Aluminum chloride is expected to have a moderate impact on ecological receptors based on toxicity to *Ceriodaphnia* and to plants in low pH soils (Tables 20 and A.7).

3.3.5 DCLCO-20 Smoke

The DCLCO-20 smoke formulation has the same component compounds as those of DC-20, except for the addition of lithium carbonate. Therefore, the primary human health risk in the component mixture is from metal dust (Table 21), and the primary ecotoxicological risk (Table 22) is from Dechlorane Plus (Sverko, 2011). In addition, available lithium is relatively toxic to citrus plants (Bingham et al., 1964).

Potential effects on human health from reaction products are low (Table 23). Ecosystem impacts are expected to be moderate for aquatic organisms and plants (Table 24). Aluminum salts, under certain conditions, have been shown to be toxic to aquatic and terrestrial invertebrates and plants (EC, 2008). Aluminum salts hydrolyze in water, and monomeric aluminum can be formed in the dissolved fraction (EC, 2008). It is the monomeric aluminum, and not the salts, that can adversely affect organisms (Driscoll et al. 1980; Parker et al., 1989; Baker et al., 1990). Under acidic conditions in soil (pH < 4.9), aluminum solubility increases, and this has been shown to cause mortality in earthworm (EC_{50} of 359 mg/kg) and to inhibit root growth in plants (EC, 2008). Lithium is relatively toxic to citrus plants (Bingham et al., 1964).

3.3.6 MGALLCO-15 Smoke

Component compounds of the MGALLCO-15 smoke formulation have low human health toxicity (Table 25), except for moderate toxicity from magnesium and aluminum metal through the inhalation route of exposure (Appendix Section A.2). Metal dust particles may cause the respiratory disorder metal fume fever and may be an explosive hazard in an occupational setting (ATSDR, 2008a; HSDB, 2013). The ecotoxicological risk from MGALLCO-15 smoke component compounds is low (Table 26 and Appendix Section A.2).

The reaction products from MGALLCO-15 smoke (Table 27) pose a low risk to human health (Appendix Section A.3). Aluminum chloride is moderately toxic to aquatic (e.g., *Daphnia*) and terrestrial (e.g., earthworm) invertebrates (Table 28 and Appendix Section A.3). Aluminum salts may be toxic to aquatic and terrestrial invertebrates and plants under acidic conditions (EC, 2008; Section 3.3.5). Lithium is relatively toxic to citrus plants (Bingham et al., 1964).

3.3.7 LP Smoke

Component compounds of LP smoke have low toxicity to human health (Table 29) and low ecotoxicity (Table 30 and Appendix Section A.2).

Lithium chloride, a reaction product of LP smoke (Table 31), has moderate human health toxicity (Appendix). It has LD_{50} values of 526 (rat), 850 (rabbit), and 1165 mg/kg (mouse). Ecological toxicity (Table 32 and Appendix) is moderate overall. Citrus plants show high toxicity (Table 32) to lithium chloride in soil (LOAEL of 2 mg/kg; Aldrich, et al., 1951). No toxicity value for lithium has been found for other plant species.

Table 5. Human Health Impact Assessment for Component Compounds of BC Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Potassium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Potassium nitrate	Low	Low	Low	Low	Low	Low	Causes anemia in rats at high concentrations
Boron carbide	Low	Low	Low	Low	Low	Low	Micro-sized particles inert; nano-sized particles may have increased toxicity; potential for mechanical injury to eyes
Calcium stearate	Low	Low	Low	Low	Low	Low	Reversible lipogranulomas in adipose tissue occurred in rats following 24 weeks exposure to 50 g/kg/day

Table 6. Ecotoxicology Assessment for Component Compounds of BC Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Potassium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Potassium Nitrate	Low (algae, fish spp.); moderate (*Daphnia*)	Low	Low	Low	Low	Moderately toxic to *Daphnia*; essential nutrients for animals and plants
Boron carbide	Low	Low	Low	Low	Low	Nonreactive at ambient temperatures; boron is essential element for plants and animals
Calcium stearate	Low	Low	Low	Low	Low	Relatively safe saturated fatty acid

Table 7. Human Health Impact Assessment for Reaction Products from BC Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Potassium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Potassium metaborate	Low	Low	Low	Low	Low	Low	Potassium is an essential nutrient; boron is an essential element
Carbon dioxide	Low	Low	Low	Low	Low	Low	May be toxic at extremely high levels when inhaled (>5% in air)
Nitrogen gas	Low	Low	Low	Low	Low	Low	Inert gas
Boron trioxide	Low	Low	Low	Low	Low	Low	Irritant to mucus membranes

Table 8. Ecotoxicology Assessment for Reaction Products from BC Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Potassium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Potassium metaborate	Low	Low	Low	Low	Low	Potassium is essential element for plants and animals; boron is essential element for animals and plants
Carbon dioxide	Low	Low	Low	Low	Low	Potential greenhouse gas; essential for carbohydrate production in plants
Nitrogen gas	Low	Low	Low	Low	Low	Inert gas

Table 9. Human Health Impact Assessment for Component Compounds of HX-10 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Hexachloroethane	Low	Moderate	Low	Moderate	Low	Moderate	Animal carcinogen (mice, rats); possible human carcinogen (USEPA Group C, IARC Class 2B)
Magnesium metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Aluminum metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Titanium dioxide	Low	Low	Low	Low	Low	Low	Chemically inert; insoluble in water

Table 10. Ecotoxicology Assessment for Component Compounds of HX-10 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Hexachloroethane	Moderate	Low	Low	Low	Low	Bioconcentration potential in some species (log K_{OW} = 4.0; BCF = 1 to 708).
Magnesium metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; In water reacts very slowly to form magnesium hydroxide and hydrogen gas
Aluminum metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; Insoluble in water
Titanium dioxide	Low	Low	Low	Low	Low	Chemically inert; insoluble in water

BCF, bioconcentration factor.

Table 11. Human Health Impact Assessment for Reaction Products from HX-10 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Aluminum chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 370 mg/kg (rat)
Titanium tetrachloride	Low	High	Moderate	Moderate	Low	Low	Corrosive; inhalation of the hydrolysis product hydrochloric acid caused edema and death in rats
Magnesium oxide	Low	Low	Low	Low	Low	Low	Used in some antacids; relatively nontoxic
Aluminum oxide	Low	Low	Low	Low	Low	Low	Insoluble in water
Carbon dioxide	Low	Low	Low	Low	Low	Low	May be toxic at extremely high levels when inhaled (>5%)

Table 12. Ecotoxicology Assessment for Reaction Products from HX-10 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Aluminum chloride	Moderate	Low at pH > 4.9; moderate at pH < 4.9	Low at pH > 4.9; moderate at pH < 4.9	Low	Low	Acute toxicity to *Ceriodaphnia*
Titanium tetrachloride	Low	Low	Low	Low	Low	Hydrolyzes in moist air to form hydrochloric acid
Magnesium oxide	Low	Low	Low	Low	Low	Magnesium is essential element for animals and plants
Aluminum oxide	Low	Low	Low	Low	Low	May contribute aluminum to water column from sediment under pH < 4.9 conditions
Carbon dioxide	Low	Low	Low	Low	Low	Potential greenhouse gas; essential for carbohydrate production in plants

Table 13. Human Health Impact Assessment for Component Compounds of HXLCO-10 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Hexachloroethane	Low	Moderate	Low	Moderate	Low	Moderate	Animal carcinogen (mice, rats); possible human carcinogen (USEPA Group C, IARC Group 2B)
Magnesium metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Aluminum metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Titanium dioxide	Low	Low	Low	Low	Low	Low	Chemically inert; insoluble in water
Lithium carbonate	Low	Low	Low	Low	Low	Low	Prescription drug

Table 14. Ecotoxicology Assessment for Component Compounds of HXLCO-10 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Hexachloroethane	Moderate	Low	Low	Low	Low	Moderate bioconcentration potential in some species (log K_{OW} = 4.0; BCF = 1 to 708)
Magnesium metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; in water reacts very slowly to form magnesium hydroxide oxide and hydrogen gas
Aluminum metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; insoluble in water
Titanium dioxide	Low	Low	Low	Low	Low	Chemically inert; insoluble in water
Lithium carbonate	Low	Low	Low	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil

Table 15. Human Health Impact Assessment for Reaction Products from HXLCO-10 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Aluminum chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 370 mg/kg (rat)
Titanium tetrachloride	Low	High	Moderate	Moderate	Low	Low	Corrosive; inhalation of hydrolysis product hydrochloric acid caused edema and death in rats
Magnesium oxide	Low	Low	Low	Low	Low	Low	Used in some antacids; relatively nontoxic
Aluminum oxide	Low	Low	Low	Low	Low	Low	Insoluble in water
Lithium chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 526 mg/kg (rat)
Lithium oxide	Low	Low	Low	Low	Low	Low	Irritant to mucus membranes
Carbon	Low	Low	Low	Low	Low	Low	Nonreactive
Carbon dioxide	Low	Low	Low	Low	Low	Low	May be toxic at extremely high levels when inhaled (>5%)
Carbon monoxide	Low	High	Low	Low	Low	Low	High levels can result in death

Table 16. Ecotoxicology Assessment for Reaction Products from HXLCO-10 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Aluminum chloride	Moderate	Low at pH > 4.9; moderate at pH < 4.9	Low at pH > 4.9; moderate at pH < 4.9	Low	Low	Toxicity is pH dependent; acute toxicity to *Ceriodaphnia*
Titanium tetrachloride	Low	Low	Low	Low	Low	Hydrolyzes in moist air to form hydrochloric acid
Magnesium oxide	Low	Low	Low	Low	Low	Magnesium is essential element for animals and plants
Aluminum oxide	Low	Low	Low	Low	Low	May contribute aluminum to water column from sediment under pH < 4.9 conditions
Lithium chloride	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Lithium oxide	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Carbon	Low	Low	Low	Low	Low	Nonreactive
Carbon dioxide	Low	Low	Low	Low	Low	Potential greenhouse gas; essential for carbohydrate production in plants
Carbon monoxide	Low	Low	Low	Low	Low	Reacts with oxygen in ambient air to form carbon dioxide

Table 17. Human Health Impact Assessment for Component Compounds of DC-20 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Dechlorane Plus	Low	Low	Low	Low	Low	Low	Potential for bioaccumulation, based on calculated log K_{OW}
Magnesium metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Aluminum metal	Low	Moderate	Low	Low	Low	Low	Dust particles are potentially explosive in occupational setting
Ammonium chloride	Low	Low	Low	Low	Low	Low	Nitrogen (in ammonium) and chloride are essential nutrients

Table 18. Ecotoxicology Assessment for Component Compounds of DC-20 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Dechlorane Plus	Moderate	Low	Low	Low	Low	Aquatic biomagnification potential is high (log K_{OW} = 9.0), but results of field surveys were highly variable and species-specific
Magnesium metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; in water reacts very slowly to form magnesium hydroxide and hydrogen gas
Aluminum metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; insoluble in water
Ammonium chloride	Low	Low	Low	Low	Low	Chloride and nitrogen in ammonium are essential nutrients for animals and plants

Table 19. Human Health Impact Assessment for Reaction Products from DC-20 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Aluminum chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 370 mg/kg (rat)
Ammonium chloride	Low	Low	Low	Low	Low	Low	Chloride and nitrogen (in ammonium) are essential nutrients
Carbon	Low	Low	Low	Low	Low	Low	Nonreactive
Water	Low	Low	Low	Low	Low	Low	Essential compound

Table 20. Ecotoxicology Assessment for Reaction Products from DC-20 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Aluminum chloride	Moderate	Low at pH > 4.9; moderate at pH < 4.9	Low at pH > 4.9; moderate at pH < 4.9	Low	Low	Acute toxicity to *Ceriodaphnia*
Ammonium chloride	Low	Low	Low	Low	Low	Nitrogen (in ammonium) and chloride are essential nutrients for animals and plants
Carbon	Low	Low	Low	Low	Low	Nonreactive
Water	Low	Low	Low	Low	Low	Essential compound for animals and plants

Table 21. Human Health Impact Assessment for Component Compounds of DCLCO-20 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Dechlorane Plus	Low	Low	Low	Low	Low	Low	Potential for bioaccumulation, based on calculated log K_{OW}
Magnesium metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Aluminum metal	Low	Low	Low	Low	Low	Low	Dust particles are potentially explosive in occupational setting
Ammonium chloride	Low	Low	Low	Low	Low	Low	Chloride and nitrogen (in ammonium) are essential nutrients
Lithium carbonate	Low	Low	Low	Low	Low	Low	Prescription drug

Table 22. Ecotoxicology Assessment for Component Compounds of DCLCO-20 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Dechlorane Plus	Moderate	Low	Low	Low	Low	Biomagnification potential is high (log K_{OW} = 9.0), but results of field surveys were highly variable and species-specific
Magnesium metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; in water reacts very slowly to form magnesium hydroxide and hydrogen gas
Aluminum metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; insoluble in water
Ammonium chloride	Low	Low	Low	Low	Low	Chloride and nitrogen (in ammonium) are essential nutrients for animals and plants
Lithium carbonate	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil

Table 23. Human Health Impact Assessment for Reaction Products from DCLCO-20 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Aluminum chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 370 mg/kg (rat)
Ammonium chloride	Low	Low	Low	Low	Low	Low	Chloride and nitrogen (in ammonium) are essential nutrients
Lithium chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 526 mg/kg (rat)
Carbon	Low	Low	Low	Low	Low	Low	Nonreactive
Water	Low	Low	Low	Low	Low	Low	Essential compound

Table 24. Ecotoxicology Assessment for Reaction Products from DCLCO-20 Smoke.

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Aluminum chloride	Moderate	Low at pH > 4.9; moderate at pH < 4.9	Low at pH > 4.9; moderate at pH < 4.9	Low	Low	Toxicity is pH dependent; acute toxicity to *Ceriodaphnia*
Ammonium chloride	Low	Low	Low	Low	Low	Chloride and nitrogen in ammonium are essential nutrients for animals and plants
Lithium chloride	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Carbon	Low	Low	Low	Low	Low	Nonreactive
Water	Low	Low	Low	Low	Low	Essential compound for animals and plants

Table 25. Human Health Impact Assessment for Component Compounds of MGALLCO-15 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Aluminum metal	Low	Moderate	Low	Low	Low	Low	Excessive inhalation may cause metal fume fever; dust particles are potentially explosive in occupational setting
Potassium nitrate	Low	Low	Low	Low	Low	Low	Causes anemia at very high concentrations
Potassium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Lithium carbonate	Low	Low	Low	Low	Low	Low	Prescription drug
Azodicarbonamide	Low	Low	Low	Low	Low	Low	Food additive (cereal flour); elimination via feces or rapid as biurea

Table 26. Ecotoxicology Assessment for Component Compounds of MGALLCO-15 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; In water reacts very slowly to form magnesium oxide and deuterium
Aluminum metal	Low	Low	Low	Low	Low	Tarnishes in air to form passive oxide layer; Insoluble in water
Potassium nitrate	Low (algae, fish spp.); moderate (*Daphnia*)	Low	Low	Low	Low	Moderately toxic to *Daphnia*; essential nutrients for animals and plants
Potassium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Lithium carbonate	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Azodicarbonamide	Low	Low	Low	Low	Low	Estimated based on calculated fate properties and unpublished industry report

Table 27. Human Health Impact Assessment for Reaction Products from MGALLCO-15 Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Magnesium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Aluminum chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 370 mg/kg (rat)
Lithium oxide	Low	Low	Low	Low	Low	Low	Irritant to mucous membranes
Potassium carbonate	Low	Low	Low	Low	Low	Low	Inhalation, skin, and ocular irritant
Potassium chloride	Low	Low	Low	Low	Low	Low	Essential nutrients
Carbon	Low	Low	Low	Low	Low	Low	Nonreactive
Carbon dioxide	Low	Low	Low	Low	Low	Low	May be toxic at extremely high levels when inhaled (>5% in air)
Carbon monoxide	Low	High	Low	Low	Low	Low	High levels can result in death
Nitrogen	Low	Low	Low	Low	Low	Low	Inert gas
Water	Low	Low	Low	Low	Low	Low	Essential compound

Table 28. Ecotoxicology Assessment for Reaction Products from MGALLCO-15 Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Magnesium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Aluminum chloride	Moderate	Low at pH > 4.9; moderate at pH < 4.9	Low at pH > 4.9; moderate at pH < 4.9	Low	Low	Toxicity is pH dependent; acute toxicity to *Ceriodaphnia*
Lithium oxide	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Potassium carbonate	Low	Low	Low	Low	Low	LC_{50} = 200–300 mg/L (*Daphnia*); LC_{50} = 300 mg/L (fish)
Potassium chloride	Low	Low	Low	Low	Low	Essential nutrients for animals and plants
Carbon	Low	Low	Low	Low	Low	Nonreactive
Carbon dioxide	Low	Low	Low	Low	Low	May contribute to global warming; essential for carbohydrate production in plants
Carbon monoxide	Low	Low	Low	Low	Low	Reacts with oxygen in ambient air to form carbon dioxide
Nitrogen gas	Low	Low	Low	Low	Low	Inert gas
Water	Low	Low	Low	Low	Low	Essential compound for animals and plants

Table 29. Human Health Impact Assessment for Component Compounds of LP Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Lithium perchlorate	Low	Low	Low	Low	Low	Low	Worker lifetime perchlorate inhalation up to 84 mg/kg caused no attributable health effects
Boron	Low	Low	Low	Low	Low	Low	Short-term irritant to mucous membranes

Table 30. Ecotoxicology Assessment for Component Compounds of LP Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Lithium perchlorate	Low	Low	Low	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil
Boron	Low	Low	Low	Low	Low	Elemental boron is insoluble in water; boron is an essential nutrient for plants and animals

Table 31. Human Health Impact Assessment for Reaction Products from LP Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Boron trioxide	Low	Low	Low	Low	Low	Low	Irritant to mucous membranes
Lithium chloride	Moderate	Low	Low	Low	Low	Low	Oral LD_{50} = 526–1165 mg/L in laboratory animals

Table 32. Ecotoxicology Assessment for Reaction Products from LP Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Boron trioxide	Low	Low	Low	Low	Low	Irritant to mucous membranes
Lithium chloride	Low	Low	Moderate	Low	Low	Lithium is toxic to citrus plants at ≥2 mg/kg in soil

Table 33. Human Health Impact Assessment for Reaction Products from HC Smoke

Compound	Route of Exposure				Mutagenicity	Carcinogenicity	Comments
	Oral	Inhalation	Dermal	Ocular			
Zinc chloride	Moderate	High	High	High	Low	Low	Oral LD_{50} = 200 mg/kg (guinea pig); inhalation LC_{50} = 7 mg/L (mouse)[a]
Aluminum oxide	Low	Low	Low	Low	Low	Low	Insoluble in water[b]
Lead	High	High	Low	Low	Low	High	Group 2A carcinogen[c]
Arsenic	High	High	High	Moderate	High	High	Oral LD_{50} = 15 mg/kg (rat);[d] confirmed Class A human carcinogen (skin; respiratory) and mutagen
Antimony	Moderate	High	Low	Low	High	Low	Chronic exposure causes respiratory diseases[e]; produces chromosomal aberrations or abnormal cell division in animal cells[f]

[a]NIOSH, 1994; [b]O'Neil, 2006; [c]IARC, 2006; [d]Hei et al., 1998; [e]Sundar et al., 2010; and [f]HSDB, 2013.

Table 34. Ecotoxicology Assessment for Reaction Products from HC Smoke

Compound	Aquatic Organisms	Soil Invertebrates	Plants	Mammals	Birds	Comments
Zinc chloride	Moderate	High	Moderate	High	Moderate	LC_{50} = 0.13 mg/L (*Daphnia*); LC_{50} = 147 mg/kg (earthworm); 108 mg/kg (Norway rat); LOAEL = 50 mg/L (domestic chicken embryos); LOAEL = 20 mg/kg (*Brassica*)[a]
Aluminum oxide	Low	Low	Low	Low	Low	Insoluble in water[b]
Lead	High	Low	Moderate	Moderate	Low	LC_{50} = 100 µg/L (sea bass); LOAEC = 46 mg/kg (grass); LOAEC = 20 mg/kg (Norway rat)[c]
Arsenic	Moderate	Moderate	Moderate	Moderate	High	LC_{50} = 0.3 mg/L (clam); LC_{50} = 400 mg/L (earthworm); MATC = 18 mg/kg (plants); LOAEL = 46 mg/kg (shrew); LC_{50} = 43 mg/kg (dove)[d]
Antimony	Low	High	Low	High	Low	EC_{20} = 78 mg/kg (soil invertebrates); LOAEC = 0.27 mg/kg (shrew)[c]

Notes: LOAEC, lowest-observed-adverse-effect concentration; MATC, maximum acceptable toxicant concentration.
[a]Lock and Janssen, 2001; [b]O'Neil, 2006; [c]USEPA, 2013a; and [d]USEPA, 1988.

4. COMPARATIVE TOXICITY OF CANDIDATE SMOKES

4.1 Comparative Toxicity of Candidate Smoke Formulation Component Compounds

Determination of the toxicity of component compounds of the candidate smoke formulations is relevant primarily for the occupational exposure of workers in production facilities. Exposure in production facilities can be mitigated by the use of engineering controls and PPE. Exposure of soldiers, workers, and ecological receptors may occur at training or testing facilities, if unexploded ordnance or dud munitions are left in the field. For formulations in hand grenades, the risk of exposure to component compounds at training and testing facilities is low.

Based on the available literature, the primary component compounds of concern in the production of the smoke formulations are HCE and the aluminum/magnesium (Al/Mg) alloy. HCE is listed as a carcinogen in laboratory animals and a possible carcinogen in humans (USEPA, 1988; IARC, 1999). The Al/Mg alloy is an inhalation hazard and possible explosive hazard in an occupational setting (HSDB, 2013). Based on data collected during this review and on potential exposure scenarios, formulations HX-10 and HXLCO-10, which contain HCE and Al/Mg alloys, pose the greatest risk to production workers.

HCE and Dechlorane Plus can potentially biomagnify in ecosystems, but field surveys have shown that biomagnification in the field is highly variable and species-dependent. HX-10, HXLCO-10, and DCLCO-20, which all contain lithium carbonate, pose an ecotoxicological risk to citrus plants upon exposure to excessive lithium in soil.

4.2 Comparative Toxicity of Candidate Smoke Reaction Products

Determination of the toxicity of candidate smoke reaction products that result from combustion of the production compounds is relevant primarily for the field crews on testing ranges and soldiers at training facilities. Exposure of personnel and biota at testing and training facilities results from dissemination of the reaction products from the grenade formulations. The reaction products will also be released to environmental media (i.e., air, water, soil, and sediment).

The primary reaction product of concern for human health risk during operational activities is the inhalation of titanium tetrachloride that results from the HX-10 and HXLCO-10 smoke formulations. Titanium tetrachloride readily transforms to titanium dioxide and hydrochloric acid under moist conditions in the environment and within body tissues, which poses a potentially high risk of inhalation toxicity. This exposure can be mitigated by using PPE, and these smoke formulations should not be used without PPE; they are not recommended for indoor use because of inhalation risk.

For the reasons stated here, the smoke formulations HX-10 and HXLCO-10 also pose a risk to ecological receptors. The greatest persistent risk to ecological receptors and environmental habitats is from excessive levels of soluble aluminum salts under acidic, poorly

buffered conditions in soil and water (pH < 4.9). All formulations of the candidate smokes contain aluminum, except for BC smoke.

5. TOXICITY OF CANDIDATE SMOKES RELATIVE TO HC AND TA SMOKES

The human and ecological toxicities to HC smoke reaction products are summarized in Tables 33 and 34, respectively. Relative to HC smoke, the toxicity of each of the candidate smokes (Tables 5–32) is low. However, HCE in the component mix of HX-10 and HXLCO-10 is a moderate risk to production workers if proper engineering controls are not maintained and utilized, or if PPE is not maintained and used properly (Tables 9, 13, and A.1). The primary obscurant in the reaction products from HX-10 and HXLCO-10 is titanium tetrachloride, and although it is less toxic than the zinc chloride in HC smoke, titanium tetrachloride hydrolyzes readily in moist air to form hydrochloric acid, which poses a high risk for inhalation toxicity (Tables 11, 15, and A.2). Detailed descriptions of results of human health and ecological toxicities to HC smoke reaction products have been well documented (ATSDR, 1997a; Cichowicz, 1983; Eaton et al., 1994; Fisher et al., 1990; Hei et al., 1998; HSDB, 2013; IARC, 1999; Lock and Janssen, 2001; NIOSH, 1994; NRC, 1997; Sadusky et al., 1993; Shinn et al., 1985; Sundar et al., 2010; USEPA, 1988, 2013a). The human and ecological toxicities to components and reaction products from TA smoke have been previously reviewed and documented, and the respective toxicities of TA smoke have been shown to be relatively low (Haley et al., 1995; Sadusky et al., 1995; Thomson et al., 1988).

We have developed an a priori scoring system to give perspective to the relative toxicities of the candidate smokes, with respect to HC and TA smokes. The scoring system (Table 35) was derived from the rankings of the reaction products from the candidate smokes and HC and TA smokes (Tables 5–34). The respective human health and ecological impacts of the reaction products from each formulation were categorized as low, medium, or high, based on criteria from fate and toxicological studies (Table 2). This scoring system was based on the information currently available and is presented only as a tool to compare the relative toxicities of the reaction products from the candidate materials to each other and to the HC and TA smokes. None of the candidate formulations were evaluated for human health and ecological toxicities that can result from dissemination of the reaction products. Therefore, we presented these comparisons with some uncertainty. The toxicological risks cannot be fully evaluated until the reaction products are identified and quantified under operational conditions and toxicological studies are completed. This scoring system was intended to provide information that can serve the decision-making process toward selection of a less-toxic obscurant and should not be considered as a definitive analysis of candidate smoke toxicity. The mean scores presented in Table 35 are the averages of the combined human health and ecotoxicity scores, which were calculated for the predicted reaction products from each candidate smoke.

6. CONCLUSIONS

The component and reaction products from the potential candidates for replacement of the smoke formulation in grenades, which are described in this report, are less toxic overall to humans and ecological receptors as compared with those of the HC smoke. However, several component compounds of the candidate smoke formulations warrant concern, and therefore, they merit further investigation regarding human health and environmental toxicological risk.

Based on the current literature, the primary component compounds of concern in the production of the smoke formulations are HCE and the Al/Mg alloy, respectively. HCE is listed as a carcinogen in laboratory animals and as a possible carcinogen in humans. The Al/Mg alloy is an inhalation hazard and a possible explosion hazard in an occupational setting during the production of the munitions. Both HCE and Dechlorane Plus can potentially biomagnify in ecosystems, but field surveys have shown that biomagnification in the field is highly variable and species-dependent. Based on data collected during this review and potential exposure scenarios, formulations HX-10 and HXLCO-10, which contain HCE and Al/Mg alloys, pose the greatest risk to production workers. Exposure to workers can be mitigated by using PPE, and these formulations should not be used without PPE. Exposure to ecosystems from the production of the smoke formulations can be mitigated using engineering controls to prevent release into the environment.

The primary reaction product of concern for human health risk during operational activities is the inhalation of titanium tetrachloride from the HX-10 and HXLCO-10 smoke formulations, which readily transforms to titanium dioxide and hydrochloric acid under moist conditions in the environment and within body tissues. Exposure can be mitigated by using PPE, and these formulations should not be used without PPE; they are not recommended for indoor use because of inhalation risk. For the reasons stated here, the smoke formulations HX-10 and HXLCO-10 also pose a risk to ecological receptors. The greatest persistent risk of the reaction products to ecological receptors and environmental habitats results from excessive levels of soluble aluminum salts under acidic, poorly buffered conditions in soil and water ($pH < 4.9$). In contrast with the controlled conditions in production facilities, mitigation of exposure to the reaction products from the smoke formulations is difficult, if not impossible, under training and battlefield conditions, and therefore, environmental exposure to these chemicals is likely.

The respective scores (Table 35) for relative toxicities of predicted reaction products from the candidate smoke formulations were calculated as the averages of the combined human health and toxicity scores. The resulting predicted toxicities of the candidate smokes, with respect to each other and those of HC smoke and TA are as follows, in order from greatest to least toxicity:

HC smoke > HXLCO-10 = HX-10 > DCLCO-20 > DC-20 > LP = MGALLCO-15 > BC > TA.

Table 35. Scoring System for Reaction Products from M83 Replacement Smokes, HC Smoke, and TA Smoke

Smoke	Human Health Toxicity Score				Ecotoxicity Score				Mean Score
	Low Rankings[a] (%)	Moderate Ranking Points (A)[b]	High Ranking Points (B)[b]	Score[c] [Low – (A+B)]	Low Rankings[a] (%)	Moderate Ranking Points (A)[b]	High Ranking Points (B)[b]	Score[c] [Low – (A+B)]	
HC	50	3	24	23	40	9	12	19	21.0
BC	100	0	0	100	95	1	0	94	97.0
HX-10	89	3	2	84	87	4	0	83	83.5
HXLO-10	90	4	4	82	90	5	0	85	83.5
DC-20	97	1	0	96	88	3	0	85	90.5
DCLCO-20	97	1	0	96	87	4	0	83	89.5
MGALLCO-15	96	1	1	94	92	4	0	88	91.0
LP	92	1	0	91	92	1	0	91	91.0
TA	100	0	0	100	100	0	0	100	100.0

Notes: This a priori scoring system is intended to provide information that will serve the decision-making process toward selection of a less-toxic obscurant and should not be considered a definitive analysis of candidate smoke toxicity. Higher scores represent less toxicity from predicted reaction products. See tables pertaining to human health impact and ecotoxicological assessment for reaction products.

[a]Low rankings (%) = (number of low rankings divided by total number of rankings per smoke for reaction products from candidate formulations).

[b]For each reaction product: low ranking counts 0 points, moderate ranking counts 1 point, and high ranking counts 2 points.

[c]Score = {Low ranking (%) – [Moderate ranking points (A) + High ranking points (B)]}.

LITERATURE CITED

Adams, V.H.; Eck, W.S. *Toxicology Assessment for Work Unit PYRO 11-01 Environmentally Benign White Smoke Formulation*; Toxicology Report No. TA S.0002754b-13; U.S. Army Public Health Command, Army Institute of Public Health, Portfolio of Toxicology Health Effects Research Program: Aberdeen Proving Ground, MD, 2013; UNCLASSIFIED Report.

Agency for Toxic Substances and Disease Registry (ATSDR). Aluminum ToxFAQs. ATSDR, Division of Toxicology and Human Health Sciences: Atlanta, GA, 2008a. http://www.atsdr.cdc.gov/toxfaqs/tfacts22.pdf (accessed March 2015).

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Hexachloroethane. Public Health Service, U.S. Department of Health and Human Services, ATSDR: Atlanta, GA, 1997a. http://www.atsdr.cdc.gov/toxprofiles/tp97.pdf (accessed March 2015).

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Perchlorates. Public Health Service, U.S. Department of Health and Human Services, ATSDR: Atlanta, GA, 2008b. http://www.atsdr.cdc.gov/toxprofiles/tp162.pdf (accessed March 2015).

Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Titanium Tetrachloride. Public Health Service, U.S. Department of Health and Human Services, ATSDR: Atlanta, GA, 1997b. http://www.atsdr.cdc.gov/ToxProfiles/tp101.pdf (accessed March 2015).

Aldrich, D.G., Jr.; Vanselow, A.P.; Bradford G.R. Lithium Toxicity in Citrus. University of California College of Agriculture, Riverside, CA. *California Agriculture* **1951,** 6. http://ucce.ucdavis.edu/files/repositoryfiles/ca510p6-71656.pdf (accessed March 2015).

Anand, M.; Hobbs, P.; Kaziunas, A.; Campion, B.; DeLacy, B. *Storage Device/Package for Titanium Dioxide Particles*. *Final Report*; ECBC-TR-971; U.S. Army Edgewood Chemical Biological Center: Aberdeen Proving Ground, MD, 2012; UNCLASSIFIED Report (ADA566068).

Andreev, G.; Simeonov, V. Distribution and Correlation of Elements in Waters, Suspensions, Sediments and Marine Organisms from the Black Sea. *Toxicol. Environ. Chem*. **1990,** *28* (1), 1–9.

Aral, H.; Vecchio-Sadus, A. Toxicity of Lithium to Humans and the Environment—A Literature Review. *Ecotoxicol. Environ. Saf.* **2008,** *70* (3), 349–356.

Ash, M.; Ash, I. *Handbook of Green Chemicals*, 2nd ed. Synapse Information Resources, Inc.: Endicott, NY, 2004; p 1348.

Baker, J.P.; Bernard, D.P.; Christensen, S.W.; Sale, M.J.; Freda, J.; Heltcher, K.; Marmorek, D.; Rowe, L.; Scanlon, P.; Suter, G.; Warren-Hicks, W.; Welbourn, P. Biological Effects of Changes in Surface Water Acid–Base Chemistry. NAPAP Report 13. In *National Acid Precipitation Assessment Program, Acidic Deposition: State of Science and Technology*, Vol. II; Oak Ridge National Laboratory, Environmental Sciences Division: Oak Ridge, TN, 1990; pp 13.47–13.61 (Publication No. 3609).

Barker, A.V.; Pilbeam, D.J., Eds.; *Handbook of Plant Nutrition*; CRC Press: Boca Raton, FL, 2006; p 632.

Beer, C.; Reichstein, M.; Tomelleri, E.; Ciais, P.; Jung, M.; Carvalhais, N.; Rödenbeck, C.; Altaf Arain, M.; Baldocchi, D.; Bonan, G.; Bondeau, A.; Cescatti, A.; Lasslop, G.; Lindroth, A.; Lomas, M.; Luyssaert, S.; Margolis, H.; Oleason, K.W.; Roupsard, O.; Veenendaal, E.; Viovy, N.; Williams, C.; Woodward, F.I.; Papale, D. Terrestrial Gross Carbon Dioxide Uptake: Global Distribution and Covariation with Climate. *Science* **2010,** *329* (5993), 834–838.

Beisinger, K.E.; Christensen, G.M. Effects of Various Metals on Survival, Growth, Reproduction and Metabolism of *Daphnia magna*. *J. Fish Res. Bd. Canada* **1972,** *29* (12), 1691–1700.

Bidleman, T.F. Atmospheric Processes—Wet and Dry Deposition of Organic Compounds Are Controlled by Their Vapor Particle Partitioning. *Environ. Sci. Technol*. **1988,** *22* (4), 361–367.

Bingham, F.T.; Bradley, G.R.; Page, A.L. Toxicity of Lithium to Plants. *Calif. Agric.* **1964,** 6–7.

Bouchard, D.C.; Williams, M.K.; Surampalli, R.Y. Nitrate Contamination of Groundwater: Sources and Potential Health Effects. *J. Am. Water Works Assoc.* **1992,** *84* (9), 85–90.

Brezonick, P.L.; Mach, C.E.; Downing, G.; Richardson, N.; Brigham, M. Effects of Acidification on Minor and Trace-Metal Chemistry in Little Rock Lake, Wisconsin. *Environ. Toxicol. Chem.* **1990,** *9* (7), 871–885.

Carter, N.W.; Seldin, D.W.; Teng, H.C. Tissue and Renal Response to Chronic Respiratory Acidosis. *J. Clin. Invest*. **1959,** *38* (6), 949–960.

Centers for Disease Control (CDC). *Occupational Exposure to Titanium Dioxide. Current Intelligence Bulletin 63*, Department of Health and Human Services, CDC and National Institute for Occupational Safety and Health: Cincinnati, OH, 2011.

Chemical Carcinogenesis Research Information System (CCRIS). TOXNET Databases, U.S. National Library of Medicine: Bethesda, MD, 2013. http://toxnet.nlm.nih.gov/ (accessed March 2015).

ChemIDplus Lite (CIDPL). *Full Record: Stearic Acid.* TOXNET Database, U.S. National Library of Medicine: Bethesda, MD, 2009. http://chem.sis.nlm.nih.gov/chemidplus/chemidlite.jsp (accessed June 2015).

Churg, A.; Stevens, B. Calcium-Containing Particles as a Marker of Cigarette Smoke Exposure in Autopsy Lungs. *Exp. Lung Res.* **1992,** *18* (1), 21–27.

Cichowicz, J.J. *Programmatic Life Cycle Environmental Assessment for Smoke/Obscurants (Vol 4), HC Smoke*; ARCSL-EA-83007; Chemical Research and Development Center, U.S. Army Armament, Munitions and Chemical Command: Aberdeen Proving Ground, MD, 1983; UNCLASSIFIED Report (ADA141972).

Cox, P.M.; Betts, R.A.; Jones, C.D.; Spall, S.A.; Totterdell, I.J. Acceleration of Global Warming Due to Carbon-Cycle Feedbacks in a Coupled Climate Model. *Nature* **2000,** *408* (6813), 184–187.

Davey, B.G.; Wheeler, R.C. Some Aspects of the Chemistry of Lithium in Soils. *Plant and Soil* **1980,** *57* (1), 49–60.

Davy, J., Ed.; *The Collected Works of Sir Humphry Davy, Bart.* Vol. 5: Bakerian Lectures and Miscellaneous Papers from 1806 to 1815. Smith, Elder, and Company Cornhill: London, 1840.

Davis, F.R. Micronutrients and Plant Nutrition. *J. American Rhododendron Society* **1983,** *37,* 1.

Dillon, P.J.; Evans, H.E.; Scholer, P.J. The Effects of Acidification on Metal Budgets of Lakes and Catchments. *Biogeochemistry* **1988,** *5* (2), 201–220.

Driscoll, C.T.; Baker, J.P.; Bisogni, J.J.; Schofield, C.L. Effect of Aluminum Speciation on Fish in Dilute Acidified Waters. *Nature* **1980,** *284* (5752), 161–164.

Driver, C.J.; Ligotke, M.W.; Downs, J.L.; Tiller, B.L.; Poston, T.M.; Moore, E.B., Jr.; Cataldo, D.A. *Environmental and Health Effects Review for Obscurant Fog Oil*; ERDEC-CR-071; Edgewood Research Development and Engineering Center, U.S. Army Chemical and Biological Defense Agency: Aberdeen Proving Ground, MD, 1993; UNCLASSIFIED Report (ADA271244).

DuPont Company. *Acute Inhalation Studies with Titanium Tetrachloride*; Haskell Laboratory Report No. 658-680; E.I. du Pont de Nemours and Company: Wilmington, DE, 1980.

DuPont Company. *Four-Week Inhalation Study with Titanium Tetrachloride*; Haskell Laboratory Report No. 459-479; E.I. du Pont de Nemours and Company: Wilmington, DE, 1979.

Eaton, J.C.; Lopinto, R.J.; Palmer, W.G. *Health Effects of Hexachloroethane (HC) Smoke;* Technical Report 9402; U.S. Army Medical Research and Development Command: Fort Detrick, Frederick, MD, 1994; UNCLASSIFIED Report (ADA277838).

Ellington, J.J.; Wolfe, N.L.; Garrison, A.W.; Evans, J.J.; Avants, J.K.; Teng, Q. Determination of Perchlorate in Tobacco Plants and Tobacco Products. *Environ.Sci. Technol.* **2001,** *35* (15), 3213–3218.

Environment Canada (EC). *Aluminum Chloride. Aluminum Nitrate, Aluminum Sulphate. Priority Substances List Assessment Report. Follow-Up to the State of Science Report, 2000*. Environment Canada, Health Canada: Ontario, Canada, 2008. http://www.ec.gc.ca/lcpe-cepa/documents/substances/sa-as/assessment_rpt-eng.pdf (accessed March 2015).

European Chemicals Agency (ECHA). Carbon Monoxide, European Chemicals Agency Annankatu 18: Helsinki, Finland, 2009. http://echa.europa.eu/ (accessed May 2015).

European Chemicals Agency (ECHA). Lithium Chloride, European Chemicals Agency Annankatu 18: Helsinki, Finland, 2014a. http://echa.europa.eu/ (accessed March 2015).

European Chemicals Agency (ECHA). Titanium Tetrachloride, European Chemicals Agency Annankatu 18: Helsinki, Finland, 2014b. http://echa.europa.eu/ (accessed March 2015).

Feo, M.L.; Barón, E.; Eljarrat, E.; Barceló, D. Dechlorane Plus and Related Compounds in Aquatic and Terrestrial Biota: A Review. *Anal. Bioanal. Chem.*, **2012,** *404*, 2625–2637.

Fernandez-Turiel, J.L.; López-Soler, A.; Llorens, J.F.; Querol, X.; Aceñolaza, P.; Durand, F.; López, J.P.; Medina, M.E.; Rossi, J.N.; Toselli, A.J.; Saavedra, J. Environmental Monitoring Using Surface Water, River Sediments, and Vegetation: A Case Study in the Famatina Range, La Rioja, NW Argentina. *Environ. Int.* **1995,** *21*, 807–820.

Fischer, E.; Molnar, L. Growth and Reproduction of *Eisenia fetida* (Oligochaeta, Lumbricidae) in Semi-Natural Soil Containing Various Metal Chlorides. *Soil Bio. Biochem.* **1997,** *29*, 667–670.

Fisher, D.J.; Burton, D.T.; Paulson, R.L. Acute Toxicity of a Complex Mixture of Synthetic Hexachloroethane (HC) Smoke Combustion Products: II. Determination of Component Toxicity. *Environ. Toxicol. Chem*. **1990,** *9*, 755–760.

Food and Agriculture Organization of the United Nations (FAO). *The Nutrition and Feeding of Farmed Fish and Shrimp; a Training Manual. Essential Nutrients-Minerals.* Fisheries and Aquaculture Department, FAO: Brasilia, Brazil, 2014. http://www.fao.org/docrep/field/003/ab470e/AB470E06.htm (accessed March 2015).

Frick K.G.; Herrmann J. Aluminum Accumulation in a Lotic Mayfly at Low pH—A Laboratory Study. *Ecotoxicol. Environ. Saf.* **1990,** *19*, 81–88.

Garrett, D. *Handbook of Lithium and Natural Calcium*. Elsevier Academic Press: Waltham, MA, 2004; pp 1–235.

Gensemer, R.W.; Playle, R.C. The Bioavailability and Toxicity of Aluminum in Aquatic Environments. *Crit. Rev. Environ. Sci. Technol.* **1999,** *29*, 315–450.

Gibbs, J.P.; Ahmad, R.; Crump, K.S.; Houck, D.P.; Leveille, T.S.; Findley, J.E.; Francis, M. Evaluation of a Population with Occupational Exposure to Airborne Ammonium Perchlorate for Possible Acute or Chronic Effects on Thyroid Function. *J. Occup. Environ. Med.* **1998,** *40* (12), 1072–1082.

Goodrich, A.E. Changes in Roadside Soils and Vegetation Through the Use of Magnesium Chloride Dust Suppression Products. M.Sc. Thesis, Colorado State University, Fort Collins, CO, 2008, p 191.

Gosselin, R.E.; Smith, R.P.; Hodge, H.C. *Clinical Toxicology of Commercial Products*, 5th ed.; Williams and Wilkins: Baltimore, MD, 1984; p 2012.

Great Lakes Water Quality Board (GLWQB). *An Inventory of Chemical Substances Identified in the Great Lakes Ecosystem, Volume 1—Summary.* Report to the Great Lakes Water Quality Board: Windsor, Ontario, Canada, 1983.

Great Lakes Water Quality Board (GLWQB). *1987 Report on the Great Lakes Water Quality. Appendix B—Great Lakes Surveillance, Volume I.* Report to the International Joint Commission: Windsor, Ontario, Canada, 1989.

Greenspan, B.J.; Allen, M.D.; Rebar, A.H.; Boecker, B.B. Inhalation Toxicity of Lithium Combustion Aerosols in Rats. *J. Toxicol. Environ. Health* **1986,** *18* (4), 627–637.

Greenwood, N.N.; Earnshaw, A. *Chemistry of the Elements*, 1st ed.; Butterworth-Heinemann, Elsevier: Oxford, U.K., 1984; p 1542.

Griffiths, T.T. *Alternative for Perchlorates in Incendiary and Pyrotechnic Formulations for Projectiles. Final Report*; SERDP Project WP-1424; Strategic Environmental Research and Development Program: Arlington, VA, 2009; UNCLASSIFIED Report (ADA520701).

Guais, A.; Brand, G.; Jacquot, L.; Karrer, M.; Dukan, S.; Grevillot, G.; Molina, T.J.; Bonte J.; Regnier, M.; Schwartz, L. Toxicity of Carbon Dioxide: A Review. *Chem. Res. Toxicol*. **2011,** *24* (12), 2061–2070.

Habib, N.A.; Wood, C.B.; Apostolov, K.; Barker, W.; Hershman, M.J.; Aslam, M.; Heinemann, D.; Fermor, B.; Williamson, R.C.N.; Jenkins, W.E. Stearic Acid and Carcinogenesis. *Br. J. Cancer* **1987,** *56* (4), 455–458.

Haley, M.V.; Chester, N.A.; Kurnas, C.W.; Muse, W.T.; Phillips, C.T. *Toxicity of Terephthalic Acid (TPA) Smoke Mix to Algae, Daphnia, Fathead Minnows, and Earthworms*; ERDEC-TR-275; U.S. Army Edgewood Research, Development and Engineering Center: Aberdeen Proving Ground, MD, 1995; UNCLASSIFIED Report.

Hausenbuiller, R.L. *Soil Science: Principles and Practices*. William C. Brown Company Publishers: Dubuque, IA, 1972; p 450.

Havas, M. Aluminum Bioaccumulation and Toxicity to *Daphnia magna* in Soft Water at Low pH. *Can. J. Fish. Aquat. Sci.* **1985,** *42* (11), 1741–1748.

Havas, M.; Likens, G.E. Changes in ^{22}Na Influx and Outflux in *Daphnia magna* (Straus) as a Function of Elevated Al Concentrations in Soft Water at Low pH. *Proc. Natl. Acad. Sci. U.S.A.* **1985,** *82* (21), 7345–7349.

Haynes, W.M., Ed.; *CRC Handbook of Chemistry and Physics*, 91st ed. CRC Press Inc.: Boca Raton, FL, 2010; pp 2610.

Hazardous Substances Data Bank (HSDB): Peer-Reviewed Toxicology Database for about 5,000 Chemicals. TOXNET, U.S. National Library of Medicine: Bethesda, MD, 2013. http://toxnet.nlm.nih.gov/ (accessed March 2015).

Hei, T.K.; Liu, S.X.; Waldren, C. Mutagenicity of Arsenic in Mammalian Cells: Role of Reactive Oxygen Species. *Proc. Natl. Acad. Sci.U.S.A.* **1998,** *95* (14), 8103–8107.

Hemond, H.F.; Fechner, E.J. *Chemical Fate and Transport in the Environment*, 3rd ed.; Elsevier: Waltham, MA, 2015; p 486.

Hilaski, R.J.; Bergmann, J.D.; Carpin, J.C.; Muse, W.T., Jr.; Thomson, S.A. *Acute Inhalation Toxicity Effects of Explosively Disseminated—XM82 Grenade—Titanium Dioxide*; CRDEC-TR-363; U.S. Army Chemical Research, Development and Engineering Center, U.S. Army Armament, Munitions and Chemical Command: Aberdeen Proving Ground, Edgewood, MD, 1992.

Hochreiter, J.J., Jr. *Chemical-Quality Reconnaissance of the Water and Surficial Bed Material in the Delaware River Estuary and Adjacent New Jersey Tributaries*; USGS/WRI/NTIS 82-36; U.S. Geological Survey: West Trenton, NJ, 1982. http://pubs.usgs.gov/wri/wri82-36/ (accessed June 2015).

Human and Environmental Risk Assessment (HERA). *Human and Environmental Risk Assessment on Ingredients of Household Cleaning Products. Sodium Carbonate, CAS Number 497-19-8*, Edition 2.0. HERA Project: Brussels, Belgium, 2005. http://www.heraproject.com/RiskAssessment.cfm (accessed March 2015).

International Agency for Research on Cancer (IARC). *Monographs on the Evaluation of Carcinogenic Risks to Humans*. *Volume 73: Some Chemicals that Cause Tumours of the Kidney or Urinary Bladder in Rodents and Some Other Substances*. World Health Organization, IARC: Lyon, France, 1999. (Multivolume work.) http://monographs.iarc.fr/ENG/Monographs/vol73/mono73.pdf (accessed June 2015).

International Agency for Research on Cancer (IARC). *Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 87: Inorganic and Organic Lead Compounds*. World Health Organization, IARC, Lyon, France, 2006. (Multivolume work.) http://www.inchem.org/documents/iarc/vol87/volume87.pdf (accessed June 2015).

Interstate Technology and Regulatory Council (ITRC). *Perchlorate: Overview of Issues, Status, and Remedial Options*. PERCHLORATE-1; ITRC, Perchlorate Team: Washington, DC, 2005. http://www.itrcweb.org/GuidanceDocuments/PERC-1.pdf (accessed March 2015).

Jasinski, S.M. *U.S. Geological Survey (USGS) 2008 Minerals Yearbook: Potash*. U.S. Department of the Interior, USGS: Reston, VA, 2010. http://minerals.usgs.gov/minerals/pubs/commodity/potash/myb1-2008-potas.pdf (accessed June 2015).

Johansson, A.; Camner, P.; Curstedt, T.; Jarstrand, C.; Robertson, B.; Urban, T. Rabbit Lung after Inhalation of Lithium Chloride. *J. Appl. Toxicol.* **1988,** *8* (5), 373–375.

Kirk-Othmer. *Kirk-Othmer Encyclopedia of Chemical Technology*, 5th ed.; Seidel, A., Bickford, M., Eds.; John Wiley and Sons, Inc.: New York, 2007; p 22950.

Knobeloch, L.; Salna, B.; Hogan, A.; Postle, J.; Anderson, H. Blue Babies and Nitrate-Contaminated Well Water. *Environ. Health Perspect*. **2000,** *108* (7), 675–678.

Krewski, D.; Yokel, R.A.; Nieboer, E.; Borchelt, D.; Cohen, J.; Harry, J.; Kacew, S.; Lindsay, J.; Mahfouz, A.M.; Rondeau, V. Human Health Risk Assessment for Aluminium, Aluminium Oxide, and Aluminium Hydroxide. *J. Toxicol. Environ. Health B. Crit. Rev*. **2007,** *10* (1), 1–269.

Kszos, L.A.; Beauchamp, J.J.; Stewart, A.J. Toxicity of Lithium to Three Freshwater Organisms and the Antagonistic Effect of Sodium. *Ecotoxicology* **2003,** *12* (5), 427–437.

Lal, R. World Cropland Soils as a Source or Sink for Atmospheric Carbon. *Adv. Agron.* **2001,** *71*, 145–191.

Leach, J.M.; Thakore, A.N. Components Toxic to Fish in Pulp Mill Waste Streams. *Progress in Water Technology* **1977,** *9*, 787–798.

Lee, K.P.; Trochimowicz, H.J.; Reinhardt, C.F. Pulmonary Response of Rats Exposed to Titanium Dioxide (TiO_2) by Inhalation for Two Years. *Toxicol. Appl. Pharmacol*. **1985,** *79* (2), 179–192.

Lewis, R.J., Sr., Ed.; *Sax's Dangerous Properties of Industrial Materials*, 11th ed. Wiley-Interscience, John Wiley and Sons, Inc.: Hoboken, NJ, 2004; pp 1076, 4860.

Lock, K.; Janssen, C.R. Modeling Zinc Toxicity for Terrestrial Invertebrates. *Environ. Toxicol. Chem.* **2001,** *20* (9), 1901–1908.

Loeser, E. Acute Oral Toxicity: Porofor ADC/K. Institute of Toxicology, Bayer AG: Wuppertal-Elberfeld, Germany, 1976, Unpublished report.
Lyman, W.J.; Reehl, W.J.; Rosenblatt, D.H. *Handbook of Chemical Property Estimation Methods*. American Chemical Society: Washington, DC, 1990; pp 4–9, 15-1 to 15-29, 960.

McGraw-Hill. *McGraw-Hill Dictionary of Scientific and Technical Terms*, 6th ed.; McGraw-Hill: New York, 2003; p 2380.

Meacham, S.; Karakas, S.; Wallace, A.; Altun, F. Boron in Human Health: Evidence for Dietary Recommendations and Public Policies. *The Open Mineral Processing Journal* **2010,** *3*, 36–53.

Moore, J.A. An Assessment of Lithium Using the IEHR Evaluative Process for Assessing Human Developmental and Reproductive Toxicity of Agents. *Reproductive Toxicology* **1995,** *9* (2), 175–210.

Mount, D.R.; Gulley, D.D.; Hockett, J.R.; Garrison, T.D.; Evans, J.M. Statistical Models to Predict the Toxicity of Major Ions to *Ceriodaphnia dubia*, *Daphnia magna* and *Pimephales promelas* (Fathead Minnows). *Environ. Toxicol. Chem*. **1997,** *16*, 2009–2019.

Mount, D.I.; Norberg, T.J. A 7-Day Life-Cycle Cladoceran Toxicity Test. *Environ. Toxicol. Chem*. **1984,** *3* (3), 425–434.

National Institute for Occupational Safety and Health (NIOSH). *Immediately Dangerous to Life or Health Concentrations (IDLHs): Zinc Chloride Fume.* Centers for Disease Control and Prevention: Atlanta, GA, 1994.

National Institute for Occupational Safety and Health (NIOSH). *International Safety Cards. Lithium Carbonate*. CAS No. 554-13-2; Centers for Disease Control and Prevention: Atlanta, GA, 2014. http://www.cdc.gov/niosh/ipcsneng/neng1109.html (accessed October 2015).

National Institutes of Health (NIH). *Magnesium Fact Sheet for Health Professionals*. NIH Office of Dietary Supplements: Washington, DC, 2013. http://ods.od.nih.gov/factsheets/Magnesium-HealthProfessional/ (accessed March 2015).

National Research Council (NRC). *Toxicity of Military Smokes and Obscurants*, Vol. 1. National Academies Press: Washington, DC, 1997; p 178.

National Research Council (NRC). *Toxicity of Military Smokes and Obscurants*, Vol. 2. National Academies Press: Washington, DC, 1999; p 132.

National Toxicology Program (NTP). *Report on Carcinogens, Twelfth Edition: Hexachloroethane* (67-72-1). Department of Health and Human Services: Washington, DC, 2011; pp 227–229.

Neuhauser, E.F.; Loehr, R.C.; Malecki, M.R.; Milligan, D.L.; Durkin, P.R. The Toxicity of Selected Organic Chemicals to the Earthworm *Eisenia fetida*. *J. Environ. Qual*. **1985,** *14* (3), 383–388.

O'Brien, T.R.; Ross, R.H. Chemical Scoring System for Hazard and Exposure Identification. *J. Tox. Environ. Health* **1988,** *25* (1), 119–134.

O'Neil, M.J., Ed.; *The Merck Index: An Encyclopedia of Chemicals, Drugs and Biologicals,* 14th ed.; Merck and Company, Inc.: Kenilworth, NJ, 2006.

O'Neil, M.J.; Smith, A.; Heckelman, P.E.; Budavi, S., Eds.; *The Merck Index,* 13th ed.; Merck and Company, Inc.: Kenilworth, NJ, 2001; p 2564.

Opresko, D.M. *Toxicity Profiles, Formal Toxicity Summary for Lithium.* The Risk Assessment Information System (RAIS), Environmental Sciences Division, Oak Ridge National Laboratory: Oak Ridge, TN, 1995. https://rais.ornl.gov/tox/profiles/lith.html (accessed September 2016).

Organization for Economic Cooperation and Development (OECD). *Small Island Developing States (SIDS) Initial Assessment Report for 13th SIAM, Potassium Chloride*. Bern, Germany, 6–9 Nov. 2001. OECD, United Nations Environmental Programme (UNEP) Publications: Nairobi, Kenya, 2001. http://www.inchem.org/documents/sids/sids/KCHLORIDE.pdf (accessed December 2016).

Organization for Economic Cooperation and Development (OECD). *Small Island Developing States (SIDS) Initial Assessment Report for SIAM 17, Ammonium Chloride*. Arona, Italy, 11–14 Nov. 2003. OECD, United Nations Environment Programme (UNEP) Publications: Nairobi, Kenya, 2004.

Organization for Economic Cooperation and Development (OECD). *Small Island Developing States (SIDS) Initial Assessment Report for SIAM 32, Magnesium Chloride.* Arona, Italy, 19–21 April 2011. OECD, United Nations Environment Programme (UNEP) Publications: SIAM 32, 2011. http://webnet.oecd.org/hpv/UI/handler.axd?id=A70BB73C-12F8-447D-90D3-24535BDE3819 (accessed March 2015).

Othman, S.H. Bio-Nanocomposite Materials for Food Packaging Applications: Types of Biopolymer and Nano-Sized Filler. In *Agriculture and Agricultural Science Procedia*, Volume 2; Chin, N.L., Man, H.C., Talib, R.A., Eds.; Elsevier Science BV: Amsterdam, Netherlands, 2014; pp 296–303.

Otto, C.; Svensson, B.S. Properties of Acid Brown Water Streams in South Sweden. *Arch. Hydrobiol.* **1983,** *99* (1), 15–36.

Parker, D.R.; Zelazny, L.W.; Kinraide, T.B. Chemical Speciation and Plant Toxicity of Aqueous Aluminum. In *Environmental Chemistry and Toxicology of Aluminum*, Lewis, T.E., Ed.; CRC Press: Boca Raton. FL, 1989; pp 117–145, 350.

Peterson, K.P. Effect of Age and Route of Administration on LD_{50} of Lithium Chloride in the Rat. *Acta Pharmacol. Toxicol.* **1980,** *47* (5), 351–354.

Pohanish, R.P. *Sittig's Handbook of Toxic and Hazardous Chemicals and Carcinogens,* 6th ed.; Elsevier: London, 2011; p 3100.

Puckett, L.J.; Tesoriero, A.J.; Dubrovsky, N.M. Nitrogen Contamination of Surficial Aquifers—A Growing Legacy. *Environ. Sci. Technol.* **2011,** *45* (3), 839–844.

Registry for Toxic Effects of Chemical Substances (RTECS). A compendium of data extracted from the open scientific literature with six types of toxicity data included in the file. National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention: Washington, DC, 2013. http://www.cdc.gov/niosh/rtecs (accessed March 2015).

Robidoux, P.Y.; Hawari, J.; Thiboutot, S.; Ampleman, G.; Sunahara, G.I. Acute Toxicity of 2,4,6-Trinitrotoluene in Earthworm (*Eisenia andrei*). *Ecotoxicol. Environ. Saf.* **1999,** *44* (3), 311–321.

Ross, A.C.; Caballero, B.; Cousins, R.J.; Tucker, K.L.; Ziegler, T.R. *Modern Nutrition in Health and Disease,* 11th ed.; Lippincott Williams and Wilkins: Philadelphia, 2012; p 1648.

Royal Society of Chemistry (RSC). *Periodic Table: Lithium*, 2015. http://www.rsc.org/periodic-table/element/3/lithium (accessed June 2015).

Sadler K.; Lynam S. *The Mineral Content of Some Freshwater Invertebrates in Relation to Stream pH and Calcium Concentration.* Central Electric Research Laboratory: Leatherland, Surrey, UK, 1985.

Sadusky, M.C.; Simini, M.; Skelly, J.M.; Checkai, R.T.; Wentsel, R.S. *Relationships between Hexachloroethane Obscurant, Zinc Deposition, and Foliar Injury of Several Forest Tree Species*; ERDEC-CR-169; U.S. Army Edgewood Research, Development and Engineering Center: Aberdeen Proving Ground, MD, 1995. http://www.dtic.mil/dtic/tr/fulltext/u2/a291489.pdf (accessed June 2015).

Sadusky, M.C.; Skelly, J.M.; Simini, M.; Checkai, R.T.; Wentsel, R.S. Hexachloroethane Obscurant: Assessing Tree Foliage Injury. *Environ. Toxicol. Chem.* **1993,** *12* (4), 685–694.

Sax, N.I.; Lewis, R.J. *SAX's Dangerous Properties of Industrial Materials*, 7th ed.; Van Nostrand Reinhold: New York, 1989; p 2494.

Saxena, K. Clinical Features and Management of Poisoning Due To Potassium Chloride. *Med. Toxicol. Adverse Drug Exp.* **1989,** *4* (6), 429–443.

Schrauzer, G.N. Lithium: Occurrence, Dietary Intakes, Nutritional Essentiality. *J. Am. Coll. Nutr.* **2002,** *21* (1), 14–21.

Servos, M.R.; Rooke, J.B.; Mackie, G.L. Reproduction of Selected Mollusca in Some Low Alkalinity Lakes in South-Central Ontario. *Can. J. Zool.* **1985,** *63* (3), 511–515.

Shand, M.A. *The Chemistry and Technology of Magnesia*. John Wiley and Sons: Hoboken, NJ, 2006; p 266.

Shaw, A.P.; Diviacchi, G.; Black, E.L.; Moretti, J.D.; Sadangi, R.K.; Grau, H.A., Jr.; Gilbert, R.A., Jr. Versatile Boron Carbide-Based Visual Obscurant Compositions for Smoke Munitions. *ACS Sustainable Chem. Eng.* **2015,** *3* (6), 1248–1254.

Shaw, A.P.; Poret, J.C.; Gilbert, R.A.; Domanico, J.A.; Black, E.L. Development and Performance of Boron Carbide-Based Smoke Compositions. *Propellants Explos. Pyrotech*. **2013,** *38* (5), 622–628.

Shinn, J.H.; Martins, S.A.; Cederwall, P.L.; Gratt, L.B. *Smokes and Obscurants: A Health and Environmental Effects Data Base Assessment*. Lawrence Livermore National Laboratory: Livermore, CA, 1985; UNCLASSIFIED Report (ADA185377).

Sleight, S.D.; Atallah, O.A. Reproduction in the Guinea Pig as Affected by Chronic Administration of Potassium Nitrate and Potassium Nitrite. *Toxicol. Appl. Pharmacol*. **1968,** *12* (2), 179–185.

Sorensen, M.A.; Jensen, P.D.; Walton, W.E.; Trumble, J.T. Acute and Chronic Activity of Perchlorate and Hexavalent Chromium Contamination on the Survival and Development of *Culex quinquefasciatus Say* (Diptera: Culicidae). *Environ. Pollut*. **2006,** *144* (3), 759–764.

Sparling, D.W.; Lowe, T.P. Environmental Hazards of Aluminum to Plants, Invertebrates, Fish and Wildlife. In *Reviews of Environmental Contamination and Toxicology*, Vol. 145; Springer-Verlag, Inc.: New York, 1996; pp 1–127.

Storer, N.L.; Nelson, T.S. Effect of Various Aluminum Compounds on Chick Performance. *Poultry Sci*. **1968,** *47* (1), 244–247.

Sullivan, J.B. *Hazardous Materials Toxicology: Clinical Principles of Environmental Health*; Krieger, G.R., Ed.; Williams and Wilkins: Baltimore, MD, 1992.

Sundar, S.; Chakravaty, J. Antimony Toxicity. *J. Environ. Res. Public Health* **2010,** *7* (12), 4267–4277.

Sverko, E. Determination of Dechlorane Plus and Related Compounds in the Environment; Paper 6330, McMaster University Open Access Dissertations and Theses, 2011. http://digitalcommons.mcmaster.ca/cgi/viewcontent.cgi?article=7388&context=opendissertations (accessed March 2015).

Swanson, M.B.; Socha, A.C., Eds.; *Chemical Ranking and Scoring: Guidelines for Relative Assessments of Chemicals*. Society of Environmental Toxicology and Chemistry (SETAC) Press: Pensacola, FL; 1997, p 154.

Thomson, S.A.; Burnett, D.C.; Crouse, C.L.; Hilaksi, R.J.; Muse, W.T., Jr. *Acute Inhalation Toxicity of Pyrotechnically Disseminated Terephthalic Acid*; CRDEC-TR-88134; U.S. Army Chemical Research, Development and Engineering Center: Aberdeen Proving Ground, MD, 1988.

Thurston, R.V.; Gilfoil, T.A.; Meyn, E.L.; Zajdel, R.K.; Aoki, T.L.; Veith, G.D. Comparative Toxicity of 10 Organic-Chemicals to 10 Common Aquatic Species. *Water Res*. **1985,** *19* (9), 1145–1155.

Til, H.P.; Falke, H.E.; Kuper, C.F.; Willems, M.I. Evaluation of the Oral Toxicity of Potassium Nitrite in a 13-Week Drinking-Water Study in Rats. *Food Chem. Toxicol*. **1988,** *26* (10), 851–859.

Trochimowicz, H.J.; Lee, K.P.; Reinhardt, C.F. Chronic Inhalation Exposure of Rats to Titanium Dioxide Dust. *J. Appl. Toxicol*. **1988,** *8* (6), 383–385.

U.S. Army. *Grenades and Pyrotechnic Signals*; FM 3-23-30. Headquarters, Department of the Army: Washington, DC, 2000; UNCLASSIFIED Manual.

U.S. Army Materiel Command (AMC). *Engineering Design Handbook: Military Pyrotechnics Series, Part One—Theory and Application*. AMC: Alexandria, VA, 1967; UNCLASSIFIED Report (AD817071).

U.S. Environmental Protection Agency (USEPA). *Ambient Water Quality Data for Chlorinated Ethanes*; EPA 440/5-80-029; Office of Water Regulations and Standards, Criteria and Standards Division: Washington, DC, 1980. http://water.epa.gov/scitech/swguidance/standards/criteria/upload/2001_10_12_criteria_ambientwqc_chlorietha80.pdf (accessed March 2015).

U.S. Environmental Protection Agency (USEPA). *Ecological Soil Screening Level for Aluminum*; OSWER Directive 9285.7–60; ECOTOX Knowledgebase, USEPA, Office of Solid Waste and Emergency Response (OSWER): Washington, DC, 2003. http://www.epa.gov/ecotox/ecossl/pdf/eco-ssl_aluminum.pdf (accessed June 2015).

U.S. Environmental Protection Agency (USEPA). *ECOTOX User Guide: ECOTOXicology Database System*. Version 4.0. USEPA: Washington, DC, 2013a. http:/www.epa.gov/ecotox/ (accessed March 2015).

U.S. Environmental Protection Agency (USEPA). *Health Effects Assessment for Hexachloroethane*; EPA/600/8-88/043; Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development: Cincinnati, OH, 1988.

U.S. Environmental Protection Agency (USEPA). *Nitrates and Nitrites: TEACH Chemical Summary.* USEPA Toxicity and Exposure Assessment for Children's Health (TEACH), last revised 5/22/07; USEPA: Washington, DC, 1992.

U.S. Environmental Protection Agency (USEPA). *Screening-Level Hazard Characterization, Dechlorane Plus.* Hazard Characterization Document, High Production Volume (HPV) Challenge Program, USEPA: Washington, DC, 2011a.

U.S. Environmental Protection Agency (USEPA). *Titanium Tetrachloride Hazard Summary*. USEPA: Washington, DC, 2000. http://www.epa.gov/ttn/atw/hlthef/titanium.html (accessed June 2015).

U.S. Environmental Protection Agency (USEPA). *Toxicological Review of Hexachloroethane (CASRN 67-72-1)*; EPA/635/R-09/007F; IRIS Database, USEPA: Washington, DC, 2011b.

U.S. Food and Drug Administration (USFDA). *U.S. Food and Drug Administration's Total Diet Study: Dietary Intake of Perchlorate and Iodine*. USFDA: Washington, DC, 2008. http://www.nature.com/jes/journal/v18/n6/pdf/7500648a.pdf (accessed June 2015).

Van Gestel, C.A.M.; Hoogerwerf, G. Influence of Soil pH on the Toxicity of Aluminium for *Eisenia andrei* (Oligochaeta: Lumbricidae) in an Artificial Soil Substrate. *Pedobiologia* **2001,** *45* (5), 385–395.

Vaughan, G.L.; Trently, S.A.; Wilson, R.B. Pulmonary Response, in Vivo, to Silicon Carbide Whiskers. *Environ. Res*. **1993,** *63* (2), 191–201.

Webb, R.; Ramlal, D.R.; Langenberg, J.P.; Albas, M.J. *Technical Feasibility Study of an Effective Low-Toxicity Obscurant Material, Final Report*; SERDP Project WP-2148; Strategic Environmental Research and Development Program (SERDP): Alexandria, VA, 2012; UNCLASSIFIED Report.

Weeks, M.H.; Angerhofer R.A.; Bishop R.; Thomasino, J.; Pope, C.R. The Toxicity of Hexachloroethane in Laboratory Animals. *Am. Ind. Hyg. Assoc. J.* **1979,** *40* (3), 187–199.

White, M.A.; Cerqueira, A.B.; Whitman, C.A.; Johnson, M.B; Ogitsu, T. Determination of Phase Stability of Elemental Boron. *Angew. Chem. Int. Ed*. **2015,** *54* (12), 3626–3629.

Wold, A. Photocatalytic Properties of TiO_2. *Chem. Mater.* **1993,** *5* (3), 280–283.

Wold, L.A.; Moore, B.C.; Dasgupta, N. Life-History Responses of *Daphnia pulex* with Exposure to Aluminum Sulfate. *Lake Reserv. Manag.* **2005,** *21* (4), 383–390.

Wong H.K.T.; Nriagu J.O.; McCabe K.J. Aluminum Species in Porewaters of Kejimkujik and Mountain Lakes, Nova Scotia. *Water Air Soil Pollut*. **1989,** *46* (1–4), 155–164.

World Health Organization (WHO). *Azodicarbonamide. Concise International Chemical Assessment Document 16*; WHO: Geneva, Switzerland, 1999.

World Health Organization (WHO). *International Programme on Chemical Safety. Environmental Health Criteria 194. Aluminium*; WHO: Geneva, Switzerland, 1997, pp 1–13.

World Health Organization (WHO). *International Programme on Chemical Safety. Environmental Health Criteria 204. Boron*; WHO: Geneva, Switzerland, 1998. http://www.inchem.org/documents/ehc/ehc/ehc204.htm (accessed June 2015).

World Health Organization (WHO). *WHO Model List of Essential Medicines*, 18th ed.; WHO: Geneva, Switzerland, 2013. http://www.who.int/medicines/publications/essentialmedicines/en/ (accessed June 2015).

Yalamanchali, R.C. How Mobile Is Lithium in the Soil-Plant System? M.S. Thesis, Lincoln University: Christchurch, New Zealand, 2012. https://researcharchive.lincoln.ac.nz/bitstream/handle/10182/5231/Yalamanchali_MApplSc.pdf;jsessionid=E3F4A3825FBC974BE464FB108BAEC7D5?sequence=3 (accessed June 2015).

Yu, L.; Canas, J.E.; Cobb, G.P.; Jackson, W.A.; Anderson, T.A. Uptake of Perchlorate in Terrestrial Plants. *Ecotoxicol. Environ. Saf.* **2004,** *58* (1), 44–49.

Zuurdeeg, B.W.; van der Weiden, M.J.J. Geochemical Aspects of European Bottled Water. In *Geothermics, Thermal-Mineral Waters and Hydrogeology*; Theophrastus Publications: Athens, Greece, 1985; p 264.

ACRONYMS AND ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
AMC	U.S. Army Materiel Command
AN	Army–Navy
ATSDR	Agency for Toxic Substances and Disease Registry
B_4C	boron carbide
BCF	bioconcentration factor
BMF	biomagnification factor
bw	body weight
CAS RN	Chemical Abstracts Service Registry Number
CCRIS	Chemical Carcinogenesis Research Information System
CDC	Centers for Disease Control
CIDPL	ChemIDplus Lite database
DIRLINE	Directory of Information Resources Online
DTIC	Defense Technical Information Center
EC	Environment Canada
EC_{50}	50% effective concentration
ECHA	European Chemicals Agency
ESOH	Environment, safety, and occupational health
ECOTOX	ECOTOXicology database system
FAO	Food and Agriculture Organization of the United Nations
GLWQB	Great Lakes Water Quality Board
HCE	hexachloroethane
HERA	Human and Environmental Risk Assessment
HSDB	Hazardous Substances Data Bank
IARC	International Agency for Research on Cancer
IDLH	immediately dangerous to life or health concentrations
IRIS	Integrated Risk Information System
ITRC	Interstate Technology and Regulatory Council
IV	intravenous
K_{OC}	soil organic carbon–water partition coefficient (usually given as a logarithm value)
K_{OW}	octanol–water partition coefficient (usually given as a logarithm value)
LC_{50}	concentration required to kill 50% of the population
LD_{10}	lethal dose for 10% of the population
LD_{50}	lethal dose for 50% of the population
LOAEC	lowest-observed-adverse-effect concentration
LOAEL	lowest-observed-adverse-effect level
LOEC	lowest-observed-effect concentration
LOEL	lowest-observed-effect level
log K_{OC}	logarithm of organic carbon partition coefficient
log K_{OW}	logarithm of octanol–water partition coefficient
LTD	lowest toxic dose
MATC	maximum acceptable toxicant concentration

NIH	National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
NOAEL	no-observed-adverse-effect level
NOEC	no-observed-effect concentration
NOEL	no-observed-effect level
NRC	National Research Council
NS	not statistically significant
NTP	National Toxicology Program
OECD	Organization for Economic Cooperation and Development
OSHA	Occupational Safety and Health Administration
$Pa(CO_2)$	partial pressure of carbon dioxide
PPE	personal protective equipment
RSC	Royal Society of Chemistry
RTECS	Registry for Toxic Effects of Chemical Substances
SIDS	Small Island Developing States
TA	terephthalic acid
TMF	trophic magnification factor
TOXLINE	Toxicology Information Online
TOXNET	U.S. National Library of Medicine's Toxicology Data Network
USEPA	U.S. Environmental Protection Agency
USFDA	U.S. Food and Drug Administration
VP	vapor pressure
WHO	World Health Organization

APPENDIX

CHARACTERIZATION, TOXICOLOGICAL DATA, AND FATE AND TRANSPORT DATA FOR COMPONENT COMPOUNDS AND REACTION PRODUCTS FROM CANDIDATE SMOKES

A.1 GENERAL INFORMATION

Summaries of human health and ecological toxicity data are presented in alphabetical order in Sections A.2 and A.3. Section A.2 contains summaries of human health and ecological toxicity data for the component compounds of the candidate materials. Section A.3 contains summaries of human health and ecological toxicity data for the reaction products from the candidate smokes. The summaries were derived from literature searches of peer-reviewed articles, technical reports, and databases, as described in Section 2, Methods. The literature citations included in this appendix are found in the Literature Cited section in the body of the report.

A.2 CHARACTERIZATION, TOXICOLOGICAL SUMMARIES, AND FATE AND TRANSPORT SUMMARIES FOR COMPONENT COMPOUNDS OF CANDIDATE SMOKES

A.2.1 Ammonium Chloride

Ammonium chloride is an odorless, colorless or white, granular powder that is somewhat hydroscopic, with a tendency to cake. It is soluble in water and sublimes without melting (O'Neil, 2006). In addition to its use as a flame suppressant in explosives, it is used as a flux in zinc and tin plating. Ammonium chloride is also used in electroplating, electrolytic refining of zinc, in etching solutions during the manufacture of printed circuit boards, in dry and Leclanche batteries, as a nitrogen source for fertilization of rice and wheat and in the manufacturing of explosives, as a hardener for formaldehyde-based adhesives, and as a mordant for dyes and printing (O'Neil, 2006). Ammonium chloride occurs naturally in crevices in the vicinity of volcanoes, but it is not considered a serious industrial hazard.

A.2.1.1 Ammonium Chloride Fate and Transport

Ammonium chloride is highly soluble in water and is dissociated to its respective ions, NH_4^+ and Cl^-, in water. The ion Cl^- is not expected to be adsorbed in most soils; however, NH_4^+ is subjected to ion exchange and can form inorganic or organic salts with other counter ions in soil and water (OECD, 2004). It is known that ammonia, which reacts with soil moisture to become NH_4^+, can be easily mineralized to nitrite by numerous bacteria species and is not expected to undergo photolysis. The fugacity models cannot be applied to estimate the distribution of ammonium chloride in the environment because it is beyond the application limit of these models. Based on the physical and chemical properties of ammonium chloride, it can be

assumed that water is the preferred environmental compartment of the substance. Considering its properties, ammonium chloride is not likely to accumulate in living organisms (OECD, 2004).

A.2.1.2 Human Health Toxicology of Ammonium Chloride

Cell membranes are relatively impermeable to ionized ammonia, whereas un-ionized ammonia passes through tissue barriers with ease. Toxicity of ammonium chloride depends on the ammonia that enters the living organism and then the cell. After oral administration, ammonium chloride is rapidly absorbed from the gastrointestinal tract and used in the liver to form amino acids and proteins. Wistar rats (10 animals per group per sex) were administered ammonium chloride by gavage at 681, 1000, 1470, 1780, and 2150 mg/kg of body weight (bw). Dead male and female animals were observed in those given a dose of 1470 mg/kg of bw or greater and 1000 mg/kg of bw or greater, respectively, within 1 day after ammonium chloride administration. Dyspnea, apathy, abnormal position, and staggering were observed at a dose of 1000 mg/kg of bw or greater. In necropsy findings, no abnormalities were detected in surviving animals. The lethal dose for 50% of the population (LD_{50}) values for males, females, and both combined were 1630, 1220, and 1410 mg/kg of bw, respectively. The Ames test for mutagenicity was negative for ammonium chloride, and carcinogenicity tests were also negative (OECD, 2004).

A.2.1.3 Ecotoxicology of Ammonium Chloride

An acute growth inhibition test for algae was performed using *Chlorella vulgaris*. The 50% effective concentration (EC_{50}; biomass; 0–5 days) was 1300 mg/L. An acute toxicity test with daphnids (*Daphnia magna*) and bivalves (*Mulinia lateralis*) reported results of 101 mg/L (survival; 48 h concentration required to kill 50% of the population [LC_{50}]) and 42.0 mg/L (growth weight; 10 day EC_{50}), respectively. The 96 h LC_{50} values using various kinds of fish ranged from 96.2 mg/L (lowest was for fathead minnows [*Pimephales promelas*]) to 218 mg/L. The lowest acute toxicity value for this substance was 42.0 mg/L (10 day EC_{50} for bivalves). A chronic toxicity test for algae was performed using *Navicula* sp. The no-observed-effect concentration (NOEC; growth rate; 0–10 days) was 26.8 mg/L. A chronic reproduction toxicity test was performed using daphnids (*D. magna*). The 21 day NOEC was 14.6 mg/L, and NOECs from 28 and 44 day chronic tests for fish were in a range of 8.0 to 23.9 mg/L. The lowest chronic toxicity value of ammonium chloride was reported for a marine fish, inland silverside (*Menidia beryllina*) to be 8.0 mg/L for a 28 day NOEC. In soil organisms, an LC_{50} value for earthworms (*Eisenia fetida*) was reported to be 163 mg/kg. The authors concluded that the effects from ammonium chloride were rather mild and transient (USEPA, 2013a).

A.2.2 Azodicarbonamide

Azodicarbonamide is a synthetic chemical that exists at ambient temperatures as a yellow-orange crystalline solid. It is poorly soluble in water. In addition to its use in pyrotechnics, azodicarbonamide is used in the expansion of a wide range of polymers, including polyvinyl chloride, polyolefins, and natural and synthetic rubbers. It is also used as an aging and bleaching ingredient in cereal flour (HSDB, 2013).

A.2.2.1 Azodicarbonamide Environmental Fate and Transport

If released to air, a vapor pressure of 1.9×10^{-10} mmHg at 20 °C indicates that azodicarbonamide will exist solely in the particulate phase in the ambient atmosphere. The particulate phase will be removed from the atmosphere by wet and dry deposition. If released to soil, it is expected to have very high mobility, based on an estimated soil organic carbon–water partitioning coefficient (log K_{OC}) of 3. Volatilization from moist soil surfaces is not expected to be an important fate process, based on an estimated Henry's Law constant of 8.2×10^{-13} atm-m^3/mol. If released into water, azodicarbonamide is not expected to adsorb to suspended solids or sediment, based on the estimated log K_{OC}. Volatilization from water surfaces is not expected to be an important fate process, based on azodicarbonamide's estimated Henry's Law constant. An estimated bioconcentration factor (BCF) of 3 suggests that the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process because this compound lacks functional groups that hydrolyze under environmental conditions. Occupational exposure may occur by dermal contact with this compound at workplaces where azodicarbonamide is produced or used. The general population may be exposed through dermal contact with plastic products or by ingestion as a result of its use as a polymer additive in food packaging (HSDB, 2013).

A.2.2.2 Human Health Toxicology of Azodicarbonamide

Toxicokinetic data on azodicarbonamide are limited. The chemical appears to be well-absorbed by the inhalation and oral routes in rodents (WHO, 1999). Substantial quantities of the substance remain unabsorbed within the gastrointestinal tract and are passed out in the feces. Azodicarbonamide is readily converted to biurea, which was the only breakdown product identified, and it is likely that systemic exposure is principally to this derivative rather than to the parent compound. Elimination of absorbed azodicarbonamide or biurea is rapid and occurs predominantly through the urine; there is very little systemic retention of biurea. Azodicarbonamide is of low acute toxicity by all relevant routes of exposure. The LC_{50} value was greater than 6100 mg/m^3 in rats and mice that were exposed to a dry aerosol (median mass aerodynamic diameter of 5.8 ± 2.25 m; geometric standard deviation) of azodicarbonamide for 4 h (WHO, 1999). No mortality was observed in rats given oral doses of up to 5000 mg/kg of bw (Loeser, 1976). The dermal LD_{50} was >2000 mg/kg of bw in rabbits following the application of azodicarbonamide under an occlusive dressing for 24 h (HSDB, 2013). Few specific toxic effects were observed in any single exposure study. Reproductive, mammalian cell genotoxicity, and carcinogenicity tests with azodicarbonamide were negative.

A.2.2.3 Environmental Toxicology of Azodicarbonamide

Azodicarbonamide was tested under OECD guideline protocols, which resulted in a 96 h mortality NOEC value of 50 mg/L in the fathead minnow (*P. promelas*), a 48 h NOEC of 4800 µg/L (mortality), and a 48 h EC_{50} of 11,000 µg/L (immobilization) in the water flea (*D. magna*). These results are in an unpublished industry report that has not been peer-reviewed. A second study that was not conducted to a protocol or under Good Laboratory Practices, showed no effect on the zebra fish (*Brachydanio rerio*) exposed to an azodicarbonamide solution that was analyzed to have a concentration of 8 mg/L. There was no effect on oxygen consumption of

sewage sludge organisms that were exposed over 3 h to azodicarbonamide at >10,000 mg/L (this concentration was substantially greater than the solubility of the compound, and no information is available on how this concentration was achieved). Overall, it was not possible to draw firm conclusions from these azodicarbonamide studies (WHO, 1999).

A.2.3 Boron

Elemental boron exists as a solid at room temperature. In powder form, elemental boron appears either as black monoclinic crystals or as a yellow or brown amorphous powder when impure. Boron is a naturally occurring element that is found in the form of borates in the oceans, sedimentary rocks, coal, shale, and soil. Boron enters the environment mainly through the weathering of rocks, boric acid volatilization from seawater, and volcanic activity. Boron is also released from anthropogenic sources to a lesser extent. These anthropogenic sources include the burning of agricultural waste, refuse, and fuel wood; generation of power using coal and oil; manufacture of glass products; use of borates or perborates in the home and industry; mining and processing of borate; leaching of treated wood or paper; and disposal of sewage and sludge (HSDB, 2013).

A.2.3.1 Boron Fate and Transport

Boron compounds are ubiquitous in the environment. Elemental boron has never been found occurring naturally in the free state on earth (White et al., 2015). It occurs principally as boric acid in volcanic steam and as metallic borates, which most commonly include sodium borate (borax), boracite, and boranatrocalcite. Boron is an essential element in humans, animals, and plants (Barker and Pilbeam, 2006; Meacham et al., 2010).

A.2.3.2 Human Health Toxicology of Boron

The greatest exposure to boron for the general population comes from food (WHO, 2013). Sodium borate and boric acid are used in various cosmetic products including makeup, skin and hair-care preparations, deodorants, moisturizing creams, breath fresheners, and shaving creams; concentrations may be up to 5% (WHO, 1998). Occupational exposure to boron compounds may occur through inhalation and dermal contact with this compound at workplaces where boron or boron compounds are produced or used. Industries where workers may be occupationally exposed to boron compounds include borax mining and refining plants or plants that produce fiberglass and other glass products, cleaning and laundry products, fertilizers, pesticides, and cosmetics (WHO, 1998). Concentrations of borax dust ranging from 1.1 to 14.6 mg/m^3 have been reported in large borax mining and refining plants (WHO, 2013). A mean boric acid/boric oxide dust concentration of 4.1 mg/m^3 has been reported in a boric acid manufacturing plant (WHO, 1998). However, elemental boron is insoluble, whereas boric acid is soluble in water (O'Neil, 2006). The LD_{50} value for boric acid is 5.14 g/kg of bw for oral doses given to rats, and ingested boric acid doses ranging from 5 to 20 g/kg of bw have produced death in adult humans. In comparison, the reported LD_{50} value for sodium chloride (table salt) by ingestion is 3.75 g/kg of bw in rats (O'Neil, 2001). Developmental and reproductive toxicity studies for boric acid showed that lower fetal body weight in rats is a critical effect of exposure. As the dose level of boric acid increases, negative effects increase, including rib effects in the

rat, decreased fetal body weight and increased fetal cardiovascular malformations in the rabbit, severe testicular pathology in the rat including testicular atrophy and sterility, and reduced fetal body weight in the mouse. Animal studies on mice and rats showed no evidence of carcinogenicity with boric acid (HSDB, 2013). Only a few human studies have been conducted to assess health effects associated with exposure to boron compounds. The available data show that exposure is associated with short-term irritant effects in the upper respiratory tract, nasopharynx, and eye. These effects, however, appear to be reversible. The sole long-term study (7 years) failed to identify any long-term health effects. No studies were identified that assessed reproductive effects (WHO, 1998). Based on the lack of human data and the limited animal data, boron was not classifiable regarding its human carcinogenicity (HSDB, 2013).

A.2.3.3 Ecotoxicology of Boron

There was no published data on the ecotoxicology of elemental boron at the time of this report. Elemental boron has never been found occurring naturally in the free state on earth (White et al., 2015). Boron in its elemental form is expected to have low environmental toxicity due to its insolubility in water (O'Neil, 2006). Although boron is an essential element for plant and animal nutrition, boron fertilizer is typically applied as soluble boron salts to boron-deficient soils (Hausenbuiller, 1972).

A.2.4 Boron Carbide

Boron carbide is a high-performance abrasive material, with chemical and physical properties that are similar to those of diamond. Standard uses for boron carbide include industrial grinding and cutting applications and use as a neutron absorber for nuclear reactor shielding. The ability of nanoparticle-sized boron carbide to adsorb neutrons has led to a potential cancer treatment (boron neutron capture therapy) using immune cells loaded with boron carbide particles (HSDB, 2013). In the white smoke formulation, boron carbide particles are in the micro, not nano-size range (manufacturer specification sheet), and boron carbide is used as pyrotechnic fuel. Boron carbide is not subject to the reporting requirements of the Superfund Amendments and Reauthorization Act or SARA, Title III, Section 302. Few reports on the toxicity of boron carbide were found. Under ambient or physiological conditions, boron carbide is essentially inert.

A.2.4.1 Boron Carbide Fate and Transport

No fate or transport data were found for boron carbide. Due to its unreactive nature, boron carbide is not expected to be mobile or to transform in the environment. Boron carbide released to the atmosphere is expected to be subject to dry and possibly wet deposition. Boron carbide is expected to be stable in the environment at ambient temperatures and is not expected to bioaccumulate due to its unreactive and insoluble nature. The chemical and physical properties of boron carbide indicate that it is inert. As a particulate, boron carbide is not expected to remain suspended in aqueous environments.

A.2.4.2 Human Health Toxicology of Boron Carbide

No experimental data were found for boron carbide orally applied to humans. Boron carbide whiskers 6.1 µm in diameter by 81 µm in length were instilled into the trachea of anesthetized rats as part of a study to investigate the toxicity of silicon carbide whiskers (Vaughan et al., 1993). Histological findings from animals exposed to the silicon fibers showed multiple lesions, whereas animals exposed to the boron carbide whiskers produced no significant histological findings during the 18 month study. Due to its particle size and abrasive properties, boron carbide is expected to be an ocular irritant, and introduction to the eye may result in corneal abrasion. No published toxicological studies were found for boron carbide related to dermal application, reproduction and development, carcinogenicity, or mutagenicity.

A.2.4.3 Ecotoxicology of Boron Carbide

No ecotoxicological data were found for boron carbide. Due to its refractory and insoluble nature, boron carbide is not expected to be a toxic hazard in the ambient environment, although data gaps exist for possible combustion or pyrolysis products resulting from its application in a smoke formulation. Large quantities of micro-sized particles of boron carbide may create transient turbid conditions that might be physically stressful to the gill structures of aquatic organisms.

A.2.5 Calcium Stearate

The calcium ion associated with calcium stearate is not anticipated to be of concern. Stearate is the anionic form of stearic acid (*n*-octadecanoic acid). Stearic acid is a naturally occurring fatty acid of animal and plant origin (e.g., cotton, coconut, and palm kernel). It is used in the manufacturing of soaps, crayons, candles, pharmaceuticals, food packaging, and cosmetics; as a waterproofing agent; and as a dispersing agent and softener in rubber compounds. Stearic acid exists as an amorphous solid or leaflet. When stearic acid is combined with a base (e.g., calcium hydroxide), a salt is produced (e.g., calcium stearate). Stearates in general are expected to be released into the environment from several sources because of the production and use patterns. Stearic acid is toxic by intravenous (IV) route (mouse IV LD_{50} of 25 mg/kg); however, it is only slightly toxic in rats when administered orally (CIDPL, 2009). In humans, it is practically nontoxic and is generally regarded as safe by the U.S. Food and Drug Administration (USFDA) when food-grade quality stearic acid is used according to Good Manufacturing Practices (Adams and Eck, 2013).

A.2.5.1 Calcium Stearate Fate and Transport

The solubility of calcium stearate in cold water is 40 mg/L (Haynes, 2010); the solubility of stearic acid in water is also very low (O'Neal, 2006), and its high logarithm octanol–water partition coefficient (log K_{OW}) indicates that it will bind to soils and sediments. Stearic acid may be in surface waters as lipid droplets, but under normal ambient conditions it will not enter subsurface water. Stearic acid can be degraded via acid hydrolysis and by biodegradable bacteria (CIDPL, 2009); therefore, calcium stearate is expected to biodegrade to water and calcium oxides or carbonates.

A.2.5.2 Human Health Toxicology of Calcium Stearate

No published studies were found that were related to calcium stearate and inhalation toxicology or reproduction and development effects. Stearic acid is a dermal irritant (Lewis, 2004). The lethal dose for 10% of the population (LD_{10} [oral]) for stearic acid administered to rats was determined to be 4600 mg/kg (CIDPL, 2009). Due to its surfactant properties, stearic acid may cause irritation to the eyes (HSDB, 2013). Results of Ames tests performed with a wide variety of *Salmonella* strains have been negative for stearic acid (CCRIS, 2013). No sarcomas at the injection site were noted in mice given injections of 0.05 mg stearic acid once weekly for 6 months and observed for 21 months (Sullivan, 1992). Neither the National Toxicology Program (NTP) nor the International Agency for Research on Cancer (IARC) has a human cancer classification for stearic acid. Preliminary data indicate that stearic acid may inhibit tumor development in rats (Habib et al., 1987). Stearic acid is not expected to be a neurotoxicant.

A.2.5.3 Ecotoxicology of Calcium Stearate

The calcium ion associated with calcium stearate is not anticipated to be of concern because calcium is an essential nutrient for plants (Hausenbuiller, 1972) and mammals, including humans (Ross, et al. 2012). Stearate is the anionic form of stearic acid. The 96 h LC_{50} value for Coho salmon (*Oncorhynchus kisutch*) exposure to stearic acid was found to be 12 mg/L (Leach and Thakore, 1977). The U.S. Environmental Protection Agency (USEPA) Ecological Structure Activity Relationships program, which estimates the toxicity of industrial chemicals to aquatic organisms, provided unreliable results for stearic acid (Adams and Eck, 2013). Stearic acid can be degraded via acid hydrolysis and by biodegradable bacteria (CIDPL, 2009); therefore, calcium stearate is expected to biodegrade to water and calcium oxides or carbonates.

A.2.6 Dechlorane Plus

Dechlorane Plus is a white, crystalline, chlorinated flame-retardant powder used in wire coatings, furniture, and hard plastic connectors that are found in computer monitors. It is manufactured in high volumes, with production facilities located in the United States and China (Sverko, 2011). The commercial formulation of Dechlorane Plus contains two stereoisomers, *syn*-Dechlorane Plus and *anti*-Dechlorane Plus, in a ratio of about 1:3 (Feo et al., 2012).

A.2.6.1 Dechlorane Plus Fate and Transport

Dechlorane Plus has negligible vapor pressure and water solubility (Table 3). It is expected to exhibit low mobility in soil. Volatilization is considered moderate, based on its Henry's Law constant. The rate of hydrolysis is negligible. The rate of atmospheric photo-oxidation is moderate. In a USEPA rating system, where numbers 1 to 3 represent low to high values for persistence in the environment (P), accumulation in biological organisms (B; bioaccumulation), and toxicity (T), compounds with high values for all three parameters present potential risks to humans and ecosystems and may become designated as priority pollutants. In this USEPA rating system, Dechlorane Plus is expected to have high persistence (P3) and high bioaccumulation potential (B3) (USEPA, 2011a).

Detection of Dechlorane Plus in air that was sampled along an oceanic transect from Greenland to Antarctica indicated that Dechlorane Plus is a global pollutant and is susceptible to long-range atmospheric transport. Although the physical and chemical properties of Dechlorane Plus indicated that it has the potential to be highly bioaccumulative and to biomagnify in the environment, data collected in field studies were variable. Biomagnification factors (BMFs) >1 indicate chemical biomagnification, and BMFs <1 indicate no biomagnification. BMFs adjusted for trophic level ranged from <0.1 to 0.6 (*syn*-Dechlorane Plus) and from 0.8 to 11 (*anti*-Dechlorane Plus) for predator–prey relationships in Lake Winnipeg. BMFs ranged from 0.1 to 12 (*syn*-Dechlorane Plus) and 0.1 to 11 (*anti*-Dechlorane Plus) for predator–prey relationships in Lake Ontario.

Trophic magnification factors (TMFs) reflect the "average" degree of biomagnification (increasing lipid normalized concentrations with increasing trophic position) or biodilution (decreasing lipid normalized concentrations with increasing trophic position) for all species sampled in the food web. Lake Winnipeg's aquatic food web TMFs for *syn*-Dechlorane Plus and *anti*-Dechlorane Plus were 0.45 and 2.5, respectively. Lake Ontario's aquatic food web TMFs for *syn*-Dechlorane Plus and *anti*-Dechlorane Plus were 0.44 and 0.34, respectively (Sverko, 2011). The author suggested that this variability between TMFs may be a result of species-specific differences in biotransformation, limited sample size, or a violation of the steady-state assumption that is required for calculating valid BMFs (i.e., the chemical concentrations in the food web were not approximating steady-state conditions). Detectable levels of Dechlorane Plus have been found in terrestrial wildlife and in humans near production plants (Feo et al., 2012). Concentrations were highly variable and ranged from 10 to 810 ng/g of lipid masses in the muscle and liver tissues of six terrestrial raptor species that were collected during the period of 2004–2006 from urban sites in Beijing, China. These results suggested that the Dechlorane Plus burdens could be substantially driven by the accumulation of the *anti*-Dechlorane Plus isomer in terrestrial birds. The tissue-specific accumulation of Dechlorane Plus further suggested that factors (e.g., hepatic-binding enzymes) other than lipid solubility could be important in determining the tissue deposition of Dechlorane Plus. However, because elevated levels of Dechlorane Plus have only been found in tissues from birds that lived very close to production or waste sites, there is no conclusive evidence to suggest that Dechlorane Plus is biomagnifying in terrestrial food webs.

A.2.6.2 Human Health Toxicology of Dechlorane Plus

Sherman-Wistar rats (three males and two females per dose) were administered Dechlorane Plus (~99% pure) via gavage. There were no mortalities at concentrations up to and including 25,000 mg/kg (Lewis, 2004). In a separate study, Sprague–Dawley rats showed no mortality at concentrations up to and including 3160 mg/kg (USEPA, 2011a). In a study to determine the reproductive effects of Dechlorane Plus in rats, no treatment-related effects were observed using concentrations of up to and including 5000 mg/kg/day of Dechlorane Plus for up to 64 days. The Ames mutagenicity test for Dechlorane Plus was negative (USEPA, 2011a). Dechlorane Plus is not listed as a carcinogen by the American Cancer Society, IARC, or Occupational Safety and Health Administration (OSHA).

A.2.6.3 Ecotoxicology of Dechlorane Plus

Dechlorane Plus was tested for toxicity to freshwater fish in two studies, one static and one flow through, for a period of 4 days. Because of the low water solubility, Dechlorane Plus remained suspended as particulates, sank to the bottom of the test vessels, or floated on the surface. There were no mortality or other adverse effects observed during either test. For both studies, the median tolerance limit was >100 ppm, which was the highest concentration tested (USEPA, 2011a). No adverse toxicological effects were found for Dechlorane Plus in terrestrial or aquatic ecosystems.

A.2.7 Hexachloroethane (HCE)

HCE is a chlorinated alkane that exists at room temperature as a colorless, crystalline solid with a camphor-like odor. It is practically insoluble in water; soluble in ethanol, benzene, chloroform, and oils; and very soluble in diethyl ether and tetrachloroethylene (HSDB, 2013). HCE is used by the military in smoke-producing devices, in metal and alloy production, and as an ingredient in insecticides. HCE acts primarily as a central nervous system depressant in humans acutely (short term) exposed to it (Pohanish, 2011). HCE is also moderately irritating to the skin, mucous membranes, and liver in humans. Neurological, liver, and kidney effects have been observed in animals exposed to HCE. No information is available on the chronic (long-term), reproductive, developmental, or carcinogenic effects of HCE in humans. Hepatocellular carcinomas (liver tumors) were observed in mice after oral exposure to HCE. The USEPA has classified HCE as a Group C, possible human carcinogen (USEPA, 2011b).

A.2.7.1 HCE Fate and Transport

HCE in the environment is solely from anthropogenic production; it does not occur naturally. If released into air, its vapor pressure of 0.4 mmHg at 20 °C indicates that HCE will exist solely as a vapor in the atmosphere (Bidleman, 1988). Vapor-phase HCE does not contain functional groups that react with atmospheric oxidants such as hydroxyl radicals, nitrate radicals, and ozone (HSDB, 2013). HCE does not contain chromophores that absorb at wavelengths >290 nm; therefore, it is not expected to be susceptible to direct photolysis by sunlight (Lyman et al., 1990). If released into soil, HCE is expected to have low-to-slight mobility, based on the K_{OC} values that range from 1380 to 2360. Volatilization from moist soil surfaces is expected to be an important fate process, based on a Henry's Law constant of 3.89×10^{-3} atm-m^3/mol. HCE is not expected to volatilize from dry soil surfaces, based on its vapor pressure (Lyman et al., 1990). If released into water, HCE is expected to adsorb to suspended solids and sediment, based on the range of K_{OC} values. HCE injected into a shallow sand aquifer degraded with a half-life of 40 days in 10 °C groundwater under anaerobic conditions. Volatilization from water surfaces is expected to be an important fate process, based on the Henry's Law constant. Estimated volatilization half-lives for a model river and a model lake are 2 h and 6 days, respectively. BCF values ranging from 1.0 to 708 suggest that bioconcentration in aquatic organisms is species-dependent. BCF values of 139, 708, and 510 were measured for HCE in bluegill sunfish, fathead minnows, and rainbow trout, respectively; whereas, BCF values were 1.0 to 8.5 in carp (HSDB, 2013). HCE was not hydrolyzed at pH 3, 7, or 11 at 85 °C, which indicates that hydrolysis will be a slow process under environmental

conditions. The biodegradation half-life of HCE in a nonadapted aerobic sandy soil was reported to be 25 to 48 days (HSDB, 2013).

A.2.7.2 Human Health Toxicology of HCE

Occupational exposure to HCE may occur through inhalation and dermal contact at workplaces where HCE is produced or used. In this case, the general population is exposed to HCE through inhalation of ambient air. With respect to exposure from white smoke in the development of new smoke formulation that is related to the present study, exposure would be primarily during the production of HCE. Pulmonary function tests (vital capacity, forced expiratory volume at 1 s) were in the normal range for 11 workers occupationally exposed to HCE at 0.5 to 2.1 ppm, while wearing protective equipment including compressed-air-fed visors or full-facepiece masks with combination filters (ATSDR, 1997a). The testing was completed 5 weeks after production resumed at a smoke munitions plant after a prior 5week break. Plasma HCE levels averaged 0.08 ± 0.14 µg/L before production resumed and 7.30 ± 6.04 µg/L at 5 weeks after production resumed, thereby indicating that despite protective equipment, low-level exposure occurred. Mild skin and mucous membrane irritation were reported in the exposed group, which suggested that exposure may have been through either the inhalation or dermal routes of exposure (ATSDR, 1997a).

Acute oral toxicity with exposure to HCE was moderate in female rats, with oral LD_{50} values of 4460 mg/kg when HCE was given in corn oil and 7080 mg/kg when it was given in methylcellulose. In guinea pigs, when HCE was administered in corn oil, the oral LD_{50} value was 4970 mg/kg. In a subchronic study, a group of male rats was given HCE in doses of 62 mg/kg/day for 8 weeks to estimate tissue clearance. Clearance of HCE from fat, liver, kidney, and blood occurred in an apparent first-order manner, with a half-life of approximately 2.5 days. The apparent first-order elimination suggests that HCE metabolism and excretion were not saturated in rats that were given up to 62 mg/kg/day and suggests that, in the range of doses given, toxicity should be proportional to exposure concentration. The no-observed-effect level (NOEL) value for HCE toxicity was 1 mg/kg/day for male and female rats (HSDB, 2013). In a 6 week inhalation study, rats, dogs, and guinea pigs were exposed to HCE at a rate of 6 h/day, 5 days/week, for 6 weeks, at concentrations of 0, 145, 465, or 2520 mg/m^3. At the greatest exposure level (2520 mg/m^3), neurobehavioral effects occurred in rats and dogs, and guinea pigs had reduced body weights and increased relative liver weights and some experienced death. No effects were observed at 465 mg/m^3. Based on this inhalation, no-observed-adverse-effect level (NOAEL) value for rats, a reference dose of 0.03 mg/kg/day could be calculated using an uncertainty factor of 1000. HCE was found to be neither mutagenic (Ames test) nor a reproductive toxin (HSDB, 2013). For humans, there is inadequate evidence for the carcinogenicity of HCE. There is sufficient evidence in experimental animals for the carcinogenicity of HCE, and it is classified as a Class C carcinogen by the USEPA (USEPA, 2011b), and a Class 2B carcinogen by IARC (1999). Administration of HCE by stomach tube caused liver cancer (hepatocellular carcinoma) in mice of both sexes and caused benign and malignant kidney tumors (renal-tubular adenoma and carcinoma) in male rats (NTP, 2011).

A.2.7.3 Ecotoxicology of HCE

The acute 48 h LC_{50} values for HCE were 1360 and 3300 µg/L for *Daphnia* (Thurston et al., 1985) and *Ceriodaphnia* (Mount and Norberg, 1984), respectively. Experimental results for HCE yielded LC_{50} values for amphibians (bullfrog, *Rana catesbeiana*) of 2440 µg/L and 856 µg/L for freshwater fish (bluegill, *Lepomis macrochirus*). Saltwater fish (Sheepshead minnow, *Cyprinidon vaiegatus*) exposed to HCE responded with an LC_{50} value of 2400 µg/L (USEPA, 1980). The HCE 48 h LC_{50} value for earthworms (*E. fetida*) was 19 µg/cm^2 using the filter paper assay (Neuhauser et al., 1985). No published data were found for the ecotoxicology of HCE with respect to plants, mammals, or birds.

A.2.8 Lithium Carbonate

Lithium compounds are used in polymerization catalysts for the polyolefin plastics industry, in the manufacturing of high-strength glass and glass-ceramics, as an anode in electrochemical cells and batteries, and as chemical intermediates in organic syntheses (HSDB, 2013). Lithium carbonate is a widely prescribed drug used for the treatment of bipolar affective illness (WHO, 2013; HSDB, 2013).

A.2.8.1 Lithium Carbonate Environmental Fate and Transport

The solubility in water of lithium carbonate is low, 1.31 wt % at 20 °C (Kirk-Othmer, 2007), especially relative to other lithium salts. Ocean water has a lithium concentration of 0.18 ppm (HSDB, 2013). Lithium concentrations ranged from 73 to 95 µg/L and from 0.01 to 0.04 µg/L in water and suspension matter samples, respectively, at varying distances from an anthropogenic source on the Black Sea (Andreev et al., 1990). In a study that was started in 1976, lithium was found to be present in unknown concentrations in the Lake Erie and Green River, OH region and in the Lake Erie and Rocky River region (GLWQB, 1983). In the Great Lakes Basin ecosystem, average concentrations of 0.63 and 2.2 µg/L of lithium carbonate were reported in Lake Superior and Lake Michigan, respectively (GLWQB, 1989). Chemical analysis of 13 selected surface water sites in the Delaware River estuary detected lithium concentrations ranging from <4 to 12 µg/L (Hochreiter, 1982). Twenty-seven sites around the Famatina range in La Rioja, North West Argentina contained lithium concentrations ranging from 6.0 to 2313.6 µg/L in streams, and rivers of the Patquia-De La Cuesta formation had concentrations of lithium ranging from 130 to 440 µg/L (Fernandez-Turiel et al., 1995).

The name of the element lithium is derived from the Greek “lithos” meaning stone (RSC, 2015). Lithium naturally occurs in trace amounts in most soils within clay minerals and soil organic matter, with typical concentrations in soil ranging from 7 to 200 µg/g (Aral and Vecchio-Sadus, 2008; Schrauzer, 2002). Cation exchangeable (using 0.5 M ammonium cation with chloride anion) concentrations of lithium in five Australian soils ranged from 0.032 to 0.830 µg/g; however, no correlation was found between total and exchangeable lithium concentrations (Davey and Wheeler, 1980). Lithium concentrations found in 65 New Zealand soils ranged from 0.08 to 92 mg/kg, with the greatest lithium concentrations in soils having high clay content and the lowest concentrations in sandy soils. Most naturally occurring lithium in soil is insoluble and therefore, highly unavailable for uptake by plants. However, when soluble salts of lithium (e.g., lithium nitrate or lithium chloride) are amended into soil at levels ranging from

1 to 100 mg/kg, limited sorption of the added lithium typically occurs, and sorption increases with increasing soil pH (Yalamanchali, 2012).

A.2.8.2 Human Health Toxicology of Lithium Carbonate

Occupational exposure to lithium may occur through inhalation and dermal contact at workplaces where lithium compounds are produced or used. Because lithium is found in various environmental media, the general public is commonly exposed to small amounts of lithium through inhalation of ambient air and ingestion of food and drinking water. Industrial exposures to lithium may occur during extraction of lithium from its ores; preparation of various lithium compounds; processes involving welding, brazing, or enameling; and from the use of lithium hydrides (HSDB, 2013).

The National Institute for Occupational Safety and Health (NIOSH; National Occupational Exposure Survey of 1981–1983) statistically estimated that 198 workers (151 of these were female; Hazard Code X6906) and 39,147 workers (17,568 of these were female; Hazard Code 84414) were potentially exposed to lithium carbonate in the United States (NIOSH, 2014). The oral LD_{50} value for the mouse was 531 mg/kg of lithium carbonate, which showed symptoms of flaccid paralysis and cardiac arrhythmias. The oral LD_{50} value for the rat was 525 mg/kg of lithium carbonate, which showed symptoms of general depressed activity, respiratory depression, and diarrhea. The lowest toxic dose (LTD) of lithium carbonate was 1980 mg/kg for 30 days continuous exposure, which showed evidence of thyroid hypofunction and changes in blood serum composition (NIOSH, 2014). In an inhalation study using lithium combustion aerosols (80% lithium carbonate and 20% lithium hydroxide), calculated 14 day LC_{50} values for lithium carbonate (with 95% confidence limits) were 1700 (1300–2000) mg/m^3 for the male rats and 2000 (1700–2400) mg/m^3 for the female rats. Clinical signs of anorexia, dehydration, respiratory difficulty, and perioral and perinasal encrustation were observed. Body weights were decreased the first day after exposure, in direct relationship to the exposure concentration. In animals that were observed for an additional 2 weeks, body weights, organ weights, and clinical signs began to return to pre-exposure values (Greenspan et al., 1986).

A.2.8.3 Ecotoxicology of Lithium Carbonate

Kszos and colleagues (2003) evaluated the toxicity of lithium to fathead minnows (*P. promelas*), water fleas (*Ceriodaphnia dubia*), and freshwater snails (*Elimia clavaeformis*). They found that for most natural waters, the presence of sodium is sufficient to prevent lithium toxicity. However, in areas of historical disposal or heavy processing or use, an evaluation of lithium from a water quality perspective would be warranted. The acute environmental effect of lithium concentration (measured as EC_{50}) on *D. magna* was determined to be 33–197 mg/L, which is at least 1000 times greater than the level typically found in freshwater (GLWQB, 1989). No lithium compounds were classified for adverse environmental effects. The solubility in water of lithium carbonate is low, 1.31 wt % at 20 °C (Kirk-Othmer, 1995), especially relative to the more-soluble lithium salts. Most naturally occurring lithium in soil is insoluble, and therefore highly unavailable for uptake by plants. However, when soluble salts of lithium (e.g., lithium nitrate or lithium chloride) are amended into soil at levels ranging from 1 to 100 mg/kg, limited sorption of the added lithium typically occurs, and sorption increases with increasing soil pH

(Yalamanchali, 2012). Yalamanchali further found that in soil without added sources of lithium, monocot grass (*Lolium perenne*) and the salt-tolerant dicot beetroot (*Beta vulgaris*) accumulated the most lithium of the monocots and dicots tested, whereas seed and fruit crops had the least lithium concentrations. Grass (*L. perenne*) accumulated the greatest concentration of lithium and therefore, had the greatest bioaccumulation factor for lithium (0.082) of the 13 various monocot and dicot plant species tested, yet even this value was well below unity. With minimal data available, Aral and colleagues (2008) predicted that, based on its low affinity to particles, lithium was not expected to bioaccumulate, and this was confirmed by the greenhouse studies of Yalamanchali (2012). Bioavailable lithium is taken up by all plants and causes stimulation of plant growth in some species, although it has not been established as an essential element (Aral et al., 2008). The amount of lithium in plants usually lies between 0.2 and 30 ppm due to preferential uptake or rejection across species. Plants such as *Cirsium arvense* and *Solanum dulcamera* accumulate lithium in concentrations of three- to sixfold over most other plants. Nightshade species may reach lithium concentrations of up to 1 mg/g. Salt-tolerant plants such as *Carduus arvense* and *Holoschoenus vulgaris* may reach lithium contents of 99.6–226.4 mg/g. Lithium is relatively toxic to citrus plants (Bingham et al., 1964). Foliar symptoms of lithium toxicity included chlorosis (yellowing), necrosis, and leaf abscission at foliar concentrations of 100 mg/kg in soybeans, and sour orange at 2500 mg/kg in red beets. Tomato plants showed no toxic effects at up to 1700 mg/kg (Bingham et al., 1964).

A.2.9 Lithium Perchlorate

Perchlorate is both a naturally occurring and man-made anion that consists of chlorine bonded to four oxygen atoms (ClO^{4-}). It is typically found in the form of perchloric acid and salts such as ammonium perchlorate, potassium perchlorate, and sodium perchlorate. In the United States, approximately 90% by weight of industrial perchlorate production is dedicated to making ammonium perchlorate for use as an oxidizing agent for solid propellant rockets and missiles (ITRC, 2005). The majority of the remaining U.S. production capacity consists of perchloric acid, sodium and potassium perchlorate, and a variety of other perchlorate salts. Lithium perchlorate is a manufactured white or colorless crystalline salt that is used as a source of oxygen in some chemical oxygen generators. It is highly soluble in organic solvents and is used to accelerate several organic reactions and as an electrolyte in voltaic cells (ITRC, 2005). Very little information exists specifically for the fate and effects of lithium perchlorate. Information related to lithium compounds is covered in Section A.2.8. A review of the fate and effects of perchlorate compounds is presented in this section.

A.2.9.1 Perchlorate Fate and Transport

Perchlorate can occur naturally in the environment, or it may be released into the environment in the form of a number of highly soluble salts in solution as concentrated brine or as perchloric acid. Perchlorate does not appreciably bind to soil particles, and the movement of perchlorate in soil is largely a function of the amount of water present. Perchlorate salts that are released into the soil in solid form will readily dissolve in soil moisture. If sufficient infiltration occurs, the perchlorate will be completely leached from the soil and potentially contaminate groundwater (ITRC, 2005). Soil moisture that contains perchlorate in solution can be taken up by plants through their roots, and several ecological studies have demonstrated the tendency of

some plants to concentrate perchlorate in plant tissues (Ellington et al., 2001; Yu et al., 2004). Some perchlorate may be held in solution in the soil vadose zone by capillary forces. In arid regions, crystallized perchlorate salts may accumulate at various horizons in soil due to evaporation of infiltrating rainfall that had leached perchlorate from shallower depths.

A.2.9.2 Human Health Toxicology of Perchlorate

Primary pathways for human exposure to perchlorate occur through ingestion of contaminated food and drinking water. Perchlorate is readily adsorbed after oral exposure and can migrate from the stomach and intestines to the bloodstream. The thyroid gland is the primary target of perchlorate toxicity in humans. Thyroid hormones play an important role in regulating metabolism and are critical for normal growth and development in fetuses, infants, and young children. Perchlorate can interfere with iodide uptake into the thyroid gland at high enough exposures, and disrupt the functions of the thyroid, which can lead to a reduction in the production of thyroid hormones (ATSDR, 2008b).

Studies conducted on rodents showed that perchlorate concentrations below that required to alter thyroid hormone equilibrium are unlikely to cause thyroid cancer in humans (ATSDR, 2008b). Studies have also shown that perchlorate accumulates in some food crop leaves, tobacco plants, and in broad-leaf plants (ATSDR, 2008b). Surveys conducted by the U.S. Food and Drug Administration have detected perchlorate in food crops and milk (USFDA, 2008). Oral LD_{50} values for ammonium perchlorate are 4200 mg/kg (rat), 3310 mg/kg (guinea pig), and 1900 mg/kg (rabbit) (HSDB, 2013). In an occupational study at an ammonium perchlorate production facility, the calculated acute doses for a single-shift worker ranged from 0.2 to 436 µg/kg of perchlorate, and working-lifetime chronic inhalation exposures ranged from 8,000 to 84,000 µg/kg of perchlorate. No perchlorate-attributable effects on thyroid, bone marrow, kidney, or liver function were detected (Gibbs et al., 1998). Perchlorate has not been found to be carcinogenic or mutagenic in human and animal studies. Studies with rats have also shown no effects on reproductive function (ATSDR, 2008b).

A.2.9.3 Ecotoxicology of Perchlorate

The acute lowest-observed-effect concentration (LOEC) values for ammonium perchlorate in *D. magna* (24 h) and *Zebra danio* (5 days) were 150 mg/L and 677, respectively (USEPA, 2013a). The chronic (10 days) LC_{50} value for perchlorate toxicity in the mosquito *Culex quinquefasciatus* was 74 mg/L for water that was contaminated with ammonium perchlorate. The Relative Growth Index, which is a measure of growth and mortality rates in a population, was significantly reduced within 5 days for levels of perchlorate exposure as low as 25 mg/L in the southern house mosquito, *C. quinquefasciatus* Say (Sorensen et al., 2006).

The uptake and accumulation of perchlorates has been documented in some plant species (ASTDR, 2008b); however, there is no evidence of biomagnification between trophic levels nor are there studies showing that perchlorates are toxic to plants that are exposed to concentrations frequently found in field soils. With respect to lithium, Yalamanchali (2012) found that in soil without added sources of lithium, monocot grass (*L. perenne*) and the salt-tolerant dicot beetroot (*B. vulgaris*) accumulated the most lithium of the 13 plant species tested,

whereas seed and fruit crops had the least lithium concentrations. Grass (*L. perenne*) accumulated the greatest concentration of lithium and therefore, had the greatest bioaccumulation factor for lithium (0.082) of the various plant species tested; yet even this value was well below unity, which indicated relatively low levels of bioaccumulation (Yalamanchali, 2012). Bioavailable lithium is taken up by all plants and causes stimulation of plant growth in some species, although it has not been established as an essential element (Aral et al., 2008). The amount of lithium in plants usually lies between 0.2 and 30 ppm due to preferential uptake or rejection across species; however, soluble salts of lithium produced foliar injury symptoms in sweet orange plants that were exposed to ≥2 mg/kg of lithium salts in soil (Aldrich et al., 1951). Other citrus species have also shown foliar injury symptoms from soluble forms of lithium (Aldrich et al., 1951; Bingham et al., 1964).

A.2.10 Aluminum/Magnesium (Al/Mg) Alloy

There is very little published information regarding the Al/Mg alloy. These metal powders, when used in pyrotechnic production and dissemination, present primary safety concerns regarding their violent reactions with excess moisture and their health hazard as respirable dusts. Results of studies with the individual aluminum and magnesium metals alone (i.e., nonalloyed) are discussed in the following paragraphs.

Aluminum is the most-abundant metal in the earth's crust, comprising about 8% of its mass. Aluminum, in its elemental form, is very reactive and is never found as a free metal in nature (HSDB, 2013). Aluminum as a free metal is obtained by refining aluminum-containing minerals, primarily bauxite (ATSDR, 2008a). Aluminum is a silvery-white, ductile and malleable metal. It is released to the environment both by natural processes and from anthropogenic sources. It is highly concentrated in soil-derived dusts from such activities as mining and agriculture and in particulate matter from coal combustion. Aluminum occurs ubiquitously in the environment in the form of silicates, oxides, and hydroxides; combined with other elements such as sodium and fluorine; and as complexes with organic matter. It is not found as a free metal because of its reactivity (HSDB, 2013). Most industrial aluminum metal is used in single- and double-alloy systems, such as when it is alloyed with silicon or manganese or as a double alloy with silicon, manganese, copper, magnesium, nickel, lead, bismuth, lithium, or zinc (ATSDR, 2008a).

Magnesium is also a common element in earth's crust, averaging 27,640 mg/kg. It occurs naturally only in combination with other elements and is commonly found in rocks, soils, and minerals (e.g., magnesite, carnallite, dolomite, brucite, talc, olivine, epsomite, kieserite, and many other minerals). It is the second-most abundant cation in seawater and can also be found in underground natural brines and salt deposits. Magnesium is an essential nutrient for most plant and animal life (Barker and Pilbeam, 2006; FAO, 2014).

A.2.10.1 Al/Mg Alloy Fate and Transport

Free aluminum tends to tarnish in air to form a passive oxide layer; free aluminum is essentially insoluble in water (O'Neil, 2006). Aluminum cannot be destroyed in the environment; it can only change its form or become attached or separated from particles. Most of the aluminum in the air is in the form of small, suspended particles of soil (dust). Aluminum particles in the air settle to the ground or are washed out of the air by rain. However, very small aluminum particles can stay in the air for many days. Most aluminum-containing compounds do not dissolve to any large extent in water unless the water is very acidic or very alkaline (ATSDR, 2008a).

Free magnesium also tends to tarnish in air to form a passive oxide layer; free magnesium is essentially insoluble in water (O'Neil, 2006) but reacts with water very slowly to form magnesium oxide and hydrogen gas. Magnesium occurs ubiquitously in the natural environment but only in combination with other elements and is commonly found in rocks, soils, and many minerals. Magnesium cannot be destroyed in the environment; it can only change its form, become attached or separated from particles, and become a dissolved constituent of water. Coarse, naturally occurring airborne particulate matter (dust) frequently contains magnesium. Hard water often has relatively high concentrations of both calcium and magnesium ions, which are dissolved into water that comes into direct contact with dolomitic limestone. Such hard water tends toward pH 8. Soft water typically has much lower levels of calcium and magnesium ions, a correspondingly lower pH, and contains relatively higher levels of sodium ion. Freshwater typically contains much lower levels of dissolved magnesium than seawater (Hemond and Fechner, 2015). Sources of naturally occurring mineral waters occur worldwide and contain substantial levels of magnesium and other cations; these waters are commonly used in health spas and as drinking water. The chemical composition of mineral water is determined by the composition of the rock to which the water is naturally exposed, the availability of mineralizing agents (e.g., temperature, carbon dioxide concentration, and redox conditions), and other environmental conditions (Zuurdeeg and van der Weiden, 1985).

A.2.10.2 Human Health Toxicology of Al/Mg Alloy

Very little published literature was found for the toxicity of Al/Mg alloys, which are considered to have low toxicity as a nuisance dust.

Non-occupational human exposure to aluminum in the environment is primarily through ingestion of food and water (WHO, 1997). No acute pathogenic effects in the general population have been described after exposure to aluminum. Although it has been hypothesized that aluminum is a risk factor for Alzheimer's disease, present epidemiological evidence does not support a causal association between Alzheimer's disease and aluminum in drinking water. Neurological syndromes, including impairment of cognitive function, motor dysfunction, and peripheral neuropathy, have been reported in limited studies of workers exposed to aluminum fume. Iatrogenic exposure (i.e., from medical treatment), which occurs in patients with chronic renal failure who were exposed to aluminum-containing dialysis fluids and pharmaceutical products, may cause encephalopathy, vitamin D resistant osteomalacia, and microcytic anemia. Premature infants may develop increased tissue-loading of aluminum, particularly in bone, when

exposed to iatrogenic sources of aluminum. Although human exposure to aluminum is widespread, hypersensitivity has been reported in only a few cases after exposure to some aluminum compounds through dermal application or parenteral administration. There is insufficient information to allow for classification of the cancer risk from human exposures to aluminum and its compounds. Aluminum and its compounds appear to be poorly absorbed in humans. The mechanism of gastrointestinal absorption of aluminum has not yet been fully elucidated. The highest levels of aluminum may be found in the lungs, where it may be present as inhaled insoluble particles. Animal studies have shown that acute toxicity of metallic aluminum and aluminum compounds is low. In short-term studies using rats, mice, or dogs, exposing them to various aluminum compounds in the diet or drinking water caused only minimal effects, even at the highest administered doses. No adequate inhalation studies that evaluated aluminum toxicity were identified. Following intratracheal administration of aluminum oxide, particle-associated fibrosis was observed. No overt fetotoxicity was noted, nor were general reproductive parameters noted after gavage treatment of rats with aluminum oxide. There is no indication that aluminum is carcinogenic. It can form complexes with DNA and cross-link chromosomal proteins and DNA, but it has not been shown to be mutagenic in bacteria or induce mutation or transformation in mammalian cells in vitro (HSDB, 2013).

Magnesium is an essential element for humans and is not expected to cause adverse effects through oral ingestion (HSDB, 2013). Fine particles of magnesium that are dispersed in air during trimming, filing, or buffing of castings may cause irritation to mucous membranes. The greatest hazard from magnesium is in an occupational setting, such as in magnesium metal production plants, where explosive mixtures may inadvertently become present in air. Molten magnesium metal burns explosively in the presence of excessive moisture, as in molding sand. Fire and explosion are outstanding hazards during the manufacture of magnesium alloys (HSDB, 2013).

A.2.10.3 Ecotoxicology of Al/Mg Alloy

Al/Mg alloy is not toxic to ecological receptors until it reacts with moisture in the environment to form salts. Magnesium and magnesium salts are relatively nontoxic to living organisms, and effects are observed only at high doses. The chloride salts tend to be slightly more toxic than the sulfate salts. The 48 h LC_{50} values for magnesium chloride were 1330 and 2840 mg/kg for *Daphnia* and fathead minnow (*P. promelas*), respectively, whereas the 48 h LC_{50} values for magnesium sulfate were 1820 and 3510 mg/kg for *Daphnia* and fathead minnow, respectively (Mount et al., 1997).

The toxicity of aluminum has been studied extensively for fish and to a lesser extent for invertebrates, amphibians, and birds; but it was not studied at all for reptiles and free-ranging mammals (HSDB, 2013). For aquatic organisms, aluminum bioavailability and toxicity are intimately related to ambient pH. Changes in ambient acidity may affect aluminum solubility, dissolved aluminum speciation, and organism sensitivity to aluminum. At moderate acidity (pH 5.5 to 7.0), fish and invertebrates may be stressed due to aluminum adsorption onto gill surfaces and subsequent asphyxiation. At pH 4.5 to 5.5, aluminum can impair ion regulation and augment the toxicity of hydrogen ion (H^+). At lower pH, elevated levels of aluminum can temporarily ameliorate the toxic effects of acidity by competing for binding sites with H^+.

Aluminum toxicity in aquatic environments is further affected by the concentration of ligands such as dissolved organic matter, fluoride, or sulfate and by that of other cations such as calcium and magnesium, which compete for cellular binding sites. Although the risk of aluminum toxicity is often based on a model of free-ion (Al_3^+) activity, recent evidence suggests that factors determining aluminum toxicity are more complex. In general, aquatic invertebrates are less sensitive to aluminum toxicity and acidity than are fish; therefore, acidified, aluminum-rich waters may actually reduce predation pressure. Fish may be affected by asphyxiation at moderately acidic conditions or electrolyte imbalances at a lower pH. In amphibians, embryos and young larvae are typically more sensitive than are older larvae. Early breeding amphibians that lay eggs in ephemeral ponds and streams that are subject to spring runoff are most at risk from aluminum and acidification; those that breed later in the year in lakes or rivers are the least vulnerable. Birds and mammals are most likely exposed through dietary ingestion of soil or aluminum-contaminated foods. Concentrations of aluminum >1000 mg/kg in food may be toxic to young birds and mammals. Clinical signs in these animals are consistent with rickets because aluminum precipitates with phosphate in the gut. Terrestrial plants are typically not subject to aluminum toxicity, except under acidic soil conditions with pH <4.9 (USEPA, 2003).

A.2.11 Potassium Chloride

Potassium chloride is a white, granular powder with a saline taste. It occurs naturally as the minerals syline or sylvite and makes up 4% of the salts in the Great Salt Lake, UT (HSDB, 2013). In seawater, the concentration of K^+ is 380 mg/L and Cl^- is 19,000 mg/L. Potassium chloride is used medicinally as a prevention and treatment for potassium deficiency. Potassium is an essential element of animal life and is the main cellular cation (FAO, 2014). Potassium ions are an essential component of the sodium–potassium–chloride symporter in kidneys and help maintain blood levels of potassium. Potassium ions are also critical for proper cell membrane function and polarity. Other uses for potassium chloride include laboratory reagent, photographic chemical, and fertilizer.

A.2.11.1 Potassium Chloride Fate and Transport

Potassium chloride is water soluble and dissociates into the potassium ion (K^+) and chloride ion (Cl^-) in soil. The clay mineral type and content, pH, and organic material content will all affect the transport and leaching of the dissociated ions (OECD, 2001). As an inorganic salt, potassium chloride is expected to exist in the atmosphere in particulate form or dissolved in rain and is not subject to degradation by either hydroxyl radicals or sunlight. Due to its high aqueous solubility, potassium chloride will not bioaccumulate, except in plants.

A.2.11.2 Human Health Toxicology of Potassium Chloride

The oral LD_{50} values for potassium chloride in the guinea pig, mouse, and rat are 2500, 1500 and 2500 mg/kg of bw, respectively (HSDB, 2013). In humans, the LTD for potassium chloride is 20 to 60 mg/kg of bw (HSDB, 2013). The estimated fatal dose for potassium chloride in humans ranges from 500 to 5000 mg/kg of bw (Gosselin et al., 1984). Potassium chloride is generally recognized as safe by the USFDA as a substance that is added directly to food and is used as a nutrient or dietary supplement (HSDB, 2013). No data are

available on the acute inhalation or acute dermal toxicity for potassium chloride. Due to the low vapor pressure and the low octanol–water partition coefficient of the substance, the potential for toxicity because of either inhalation or dermal absorption is considered to be low (OECD, 2001). It is recommended that the occupational exposure limit for potassium chloride should be 10 mg/m^3 in accordance with the time-weighted average value for "Particulates Not Otherwise Classified" as proposed by the American Conference of Governmental Industrial Hygienists (ACGIH). Prolonged inhalation of large quantities of potassium chloride dust is likely to be irritating to the respiratory tract. On unbroken skin, the threshold for irritancy was 60% potassium chloride (aqueous); on broken skin, irritancy occurred at 5%. Potassium chloride dust is expected to be irritating to the eye, but it does not negatively affect reproduction or development of the fetus (OECD, 2001). Potassium chloride is not mutagenic in the Ames assay (HSDB, 2013), and it is not carcinogenic to rats consuming up to 4% of the compound in their diet (CCRIS, 2013). Potassium chloride overdoses can affect cardiac muscle (HSDB, 2013); large doses of potassium chloride may cause cardiac issues or neurotoxicity by disruption of the concentration gradient across the cell membrane, which results in depolarization. Clinical signs of potassium overdose include cardiovascular changes with electrocardiogram abnormalities and neuromuscular manifestations (Saxena, 1989).

A.2.11.3 Ecotoxicology of Potassium Chloride

Potassium chloride is readily taken up by both terrestrial and aquatic plants as essential nutrients (Barker and Pilbeam, 2006), and it will also be consumed by bacteria (HSDB, 2013). Generally, potassium chloride is not hazardous to freshwater organisms. In short-term acute toxicity tests with fish, *Daphnia*, and algae (OECD, 2001) the following results were found: 48 h LC_{50} values of 720 mg/L (fish, *Ictalurus punctulus*) and 177 mg/L (*D. magna)*; and 120 h EC_{50} value of 1337 mg/L (algae, *Nitzschia linearis*). A chronic reproductive test with the invertebrate *D. magna* gave an LOEC value of 101 mg/L (OECD, 2001). An acute (14 day) earthworm assay yielded an LC_{50} value of 5595 mg/kg for potassium chloride in soil (Robidoux et al., 1999).

A.2.12 Potassium Nitrate

For most nitrate salts, it is the nitrate anion and not the associated cation (e.g., sodium, potassium, or calcium) that is considered to be the toxicant. Sodium and potassium nitrate are often considered together or viewed as interchangeable in toxicity assessments, although there may be exceptions when large quantities are involved, and large quantities of potassium can cause neurological excursions.

A.2.12.1 Potassium Nitrate Fate and Transport

Potassium nitrate is readily soluble in water and will dissociate into the potassium ion (K^+) and nitrate ion (NO_3^-) in water or soil. The anion is an essential nutrient for plants (Barker and Pilbeam, 2006) and therefore, will be readily removed from soil when plants are present. Small discharges of ions to surface water are also expected to be taken up by plants, but larger ion discharges can result in toxicity to aquatic species or to ecological upset caused by rapid algal growth and subsequent death. For areas with excessive amounts of nitrates in soil,

leaching of nitrates into groundwater may contaminate wells with levels that can pose a health risk (Knobeloch et al., 2000; Puckett et al., 2011). Nitrates can also be metabolized by bacteria, especially in anaerobic environments (HSDB, 2013). Potassium is an essential plant nutrient, and its cationic form can undergo exchange with other cations in soil (Hausenbuiller, 1972).

A.2.12.2 Human Health Toxicology of Potassium Nitrate

In humans, death and other severe effects of nitrate ingestion are generally associated with doses in excess of 10 g of nitrate, which corresponds to nitrate concentrations that range from 33 to 150 mg/kg. Clinical signs of acute toxicity include violent gastroenteritis, anemia, methemoglobinemia, nephritis, vomiting, vertigo, muscular weakness, irregular pulse, convulsions, and collapse. Methemoglobinemia is the most common symptom of potassium nitrate exposure in humans. Sensitive human subpopulations include infants, especially those younger than 3 months of age, and individuals with a deficiency of the enzyme glucose-6-phosphate dehydrogenase or with hereditary deficiencies in methemoglobin reductase (HSDB, 2013). Two cases of infant methemoglobinemia, known as “blue baby syndrome”, involved infants that were fed formula that was prepared with nitrate-contaminated wellwater. Water collected from two of those wells contained nitrate concentrations of 22.9 and 27.4 mg/L (Knobeloch, et al., 2000).

The oral nitrate LD_{50} values for rats and rabbits have been determined to be 3750 and 1901 mg/kg, respectively, and the estimated minimum lethal dose of nitrate for cattle and sheep is 1000 mg/kg (Sax and Lewis, 1989). No systematic toxicology studies were found at the subacute level; however, there are considerable data on the toxicity to livestock of feed containing high nitrate levels (>1% by weight) (HSDB 2013). Potassium nitrate is irritating to the respiratory tract, skin, and eyes. However, no reproductive effects were found in female CD-1 mice administered potassium nitrate for 10 days at doses up to and including 400 mg/kg (HSDB, 2013). Female guinea pigs were given 0; 300; 2500; 10,000; or 30,000 ppm of potassium nitrate in drinking water for 204 days (Sleight and Atallah, 1968); decreases in the number of litters and the number of live births were observed for animals in the 30,000 ppm dose group, which were estimated to have received a nitrate dose equivalent to 1130 mg/kg. One female in that dose group died with four mummified fetuses in utero. The fetal deaths were attributed to hypoxia caused by maternal methemoglobinemia. An Ames assay with potassium nitrate was negative under aerobic conditions, either with or without metabolic activation. A Chinese hamster fibroblast assay with potassium nitrate was also found to be negative (HSDB, 2013). In a study by Til and co-authors (1988), 6 week old Wistar rats were given potassium nitrite, which is a less-oxidized compound that is related to nitrate, at 100, 300, 1000, and 3000 mg/L in drinking water for 90 days. Methemoglobin was significantly increased and plasma alkaline phosphatase activity was decreased in both sexes; plasma urea was increased in males at the 300 mg/kg treatment level. Nitrates can be transformed to nitrites by certain microorganisms in the soil and by microorganisms found in the mouth and stomach, followed by nitrosation of secondary amines and amides in the diet. The resulting nitrosamines are mutagenic, but humans metabolize or excrete most of the nitrite, unless levels are excessive. The average Western diet contains 1–2 mmol nitrate/person/day (HSDB, 2013). According to the USEPA, available information on the carcinogenic potential of nitrates is equivocal. The results of some carcinogenicity studies suggest that nitrates may cause tumors in laboratory animals, while

others do not (USEPA, 1992). The possible carcinogenicity of nitrate depends on the conversion of nitrate to nitrite and the subsequent reaction of nitrite with secondary amines, amides, and carbamates to form N-nitroso compounds that are carcinogenic (Bouchard et al., 1992). No experimental data were found for neurotoxicity of potassium nitrate.

A.2.12.3 Ecotoxicology of Potassium Nitrate

The LC_{50} value for a 28 day exposure of annelid worms to potassium nitrate averaged 2.23 mg/L. The 24, 48, and 96 h LC_{50} values for adult female mosquitofish (*Gambusia affinis*) exposed to potassium nitrate were 58.5, 31.1, and 22.5 mg/L, respectively (USEPA, 2013a). Reproduction rates for *Hydra attenuata* were evaluated at nitrate exposure levels of 50, 150, and 250 mg/L, which yielded growth rates for the two lower dose rates that were comparable to controls (i.e., no nitrate added); however, growth rates were retarded at a nitrate exposure level of 250 mg/L. When concurrent tests were run with sodium, ammonium, and potassium nitrates, the potassium salt was found to be the least toxic of the three nitrates, and the cation had a considerable effect on the toxicities of the respective solutions (HSDB, 2013). For potassium nitrate, the 96 h LC_{50} value for *D. magna* was 39 mg/L, and the LC_{50} value for a 24 h exposure in bluegill (*L. macrochirus*) was 5500 mg/L (HSDB, 2013). At concentrations of potassium nitrate up to 1035 mg/L, the development of loach (*Misgurnus fossilis*) embryos through resorption of the yolk sac was similar to those of the control treatments (i.e., no added potassium nitrate). At potassium nitrate concentrations from 2068 to 2586 mg/L, prolarvae hatched but then died; and a concentration of 5171 mg/L stopped all development of the embryos (HSDB, 2013).

A.2.13 Titanium Dioxide

Titanium dioxide is found in nature as the primary component of the minerals rutile, anatase (also known as octahedrite), and brookite, and it is also found in ilmenite ($FeTiO_3$) and perovskite ($CaTiO_3$). Titanium dioxide is the most-important commercially produced white pigment throughout the world. Rutile is the most-common natural form of this compound and is mined in the greatest volume. Titanium dioxide is insoluble in water, hydrochloric acid, nitric acid, and ethanol, but it is soluble in hot concentrated sulfuric acid, hydrogen fluoride, and alkali (NRC, 1999). Titanium dioxide is used to increase the whiteness or opacity of many consumer products such as paints, coatings, plastics, paper, printing inks, roofing granules, food, medicine, toothpaste, cosmetics, and skin care products including topical sunscreens. The pigment is extensively used because it efficiently scatters visible light, thereby providing whiteness, brightness, and opacity when incorporated into paint, plastic, or paper products. Titanium dioxide is proposed to replace zinc oxide in smoke formulations containing HCE because of the very efficient light-scattering properties of titanium dioxide and its relatively low toxicity compared with the toxicities of the reaction products from zinc oxide.

A.2.13.1 Titanium Dioxide Fate and Transport

Recent analyses of titanium dioxide particle sizes produced by smoke grenades showed that the majority of particles are considered to be in the fine range (150–600 nm) and are not in the ultrafine range (i.e., nanoparticles; <100 nm) (Anand et al., 2012). Titanium dioxide is chemically inert and insoluble in water (NRC, 1999), and fine particles will remain suspended or

settle into sediments in aquatic environments. With exposure to ultraviolet radiation (wavelengths from 400 to 100 nm), pure titanium dioxide is photocatalytic (increases the rate of chemical reaction without itself being consumed) (Wold, 1993). Hydrolysis of titanium tetrachloride ($TiCl_4$) and titanyl sulfate ($TiOSO_4$), upon contact with moisture in the air, transforms these compounds into titanium dioxide (ATSDR, 1997b; HSDB, 2013).

A.2.13.2 Human Health Toxicology of Titanium Dioxide

In using titanium dioxide training grenades, military personnel are likely to be exposed to airborne titanium dioxide particles in the fine size range (150–600 nm; NRC, 1999). The ACGIH has classified titanium dioxide as a "nuisance dust" with a threshold limit value of 10 mg/m^3 of total dust (<1% quartz). In a well-described study by Hilaski and colleagues (1992), a single 30 min exposure to titanium dioxide at average concentrations ranging from 1240 to 830 mg/m^3 produced no irreversible adverse effects in animals observed after 24 h or 14 days. Over the course of the 30 min exposure, rats experienced a 5 min average titanium dioxide concentration of 2260 mg/m^3 and a 15 min average of 1506 mg/m^3. In addition to this acute exposure study, the 4 h LC_{50} value for rats was found to be >6820 mg/m^3 of titanium dioxide (DuPont, 1979; Trochimowicz et al., 1988). Studies using ultrafine titanium dioxide particles (<100 nm) indicated that these particles would be expected to have a negative effect at a lower concentration than fine particles in the rat lung (NRC, 1999). Titanium dioxide was not mutagenic to *Salmonella typhimurium* TA1535, TA1537, TA1538, TA97, TA98, or TA100 or to *Escherichia coli* WP2, either in the presence or absence of an exogenous metabolic system from the livers of uninduced and Aroclor-induced rats, mice, and Syrian hamsters. Evidence for the carcinogenicity of titanium dioxide in humans was inadequate. There was limited evidence for the carcinogenicity of titanium dioxide in experimental animals. A significant increase in the incidence of lung adenomas and carcinomas was observed in rats exposed by inhalation to fine titanium dioxide particles for 6 h per day for 5 days per week for 24 months at a concentration of 250 mg/m^3 (Lee et al., 1985). No increase in the incidence of lung tumors was reported at titanium dioxide exposure concentrations of 10 and 50 mg/m^3 for the same periods of exposure. Hamsters and mice did not have an increased incidence of lung tumors, even when lung burdens of poorly soluble, low-toxicity particles were similar to those of rats. An overall evaluation concluded that titanium dioxide was not classifiable as to its carcinogenicity to humans (HSDB, 2013). Titanium dioxide was reported to be neither a dermal irritant nor a dermal sensitizer, and it evoked only mild irritation in the eye (NRC, 1999).

A.2.13.3 Ecotoxicology of Titanium Dioxide

In aquatic assays, titanium dioxide materials used in smoke grenades did not show any apparent toxic effects to *Daphnia* (*D. magna*) at concentrations up to 1000 mg/L. Microscopic examination (10× magnification) of the *Daphnia* after exposure to titanium dioxide showed that the organisms ingested the materials and passed them through the gut. Internal damage was not apparent after 48 h of exposure to titanium dioxide. After 24 h into the test, all of the titanium dioxide had settled to the bottom of the test chambers, except for one formulation (Tronox CRX). This material stayed in suspension, allowing ingestion to occur for the entire duration (48 h) of the test. In addition, no lethality was found in standard testing with up to and including >1000 mg/L of titanium dioxide exposure concentrations for midges (*Chironomus*

riparius), fathead minnows (*P. promelas*), and rainbow trout (*Oncorhynchus mykiss*). No significant effects were reported for titanium dioxide as applied orally to Norway rats (*Rattus norvegicus*) for measurement endpoints that included biomarkers, enzymes, and liver function (USEPA, 2013a).

A.3 CHARACTERIZATION, TOXICOLOGICAL SUMMARIES, AND FATE AND TRANSPORT SUMMARIES FOR REACTION PRODUCTS FROM CANDIDATE SMOKES

A.3.1 Aluminum Chloride

Aluminum is the most abundant metal in the earth's crust, comprising about 8% of its mass. Aluminum, in its elemental form, is very reactive and is never found as a free metal in nature. It is found combined with other elements, most commonly with oxygen, silicon, and fluorine. These chemical compounds are commonly found in soil, minerals (e.g., sapphires, rubies, turquoise), rocks (especially igneous rocks), and clays. Aluminum as a free metal is obtained by refining aluminum-containing minerals, primarily bauxite (ATSDR, 2008a). Aluminum chloride does not occur naturally in the environment. Municipal drinking water and wastewater treatment plants are the main users of aluminum chloride, as well as aluminum sulfate and other aluminum-based polymeric products. Aluminum salts are used as coagulants and flocculants to remove fine, suspended particles through sedimentation and filtration. Most of the sludge produced during this process is disposed of as waste, but a small amount may remain with the treated water as aluminum hydroxides. Aluminum chloride is used in either an anhydrous or hydrated form. In the anhydrous form, aluminum chloride is used as a catalyst in Friedel-Crafts reactions, in the manufacture of rubber, cracking of petroleum, manufacture of lubricants, and candidate smoke formulations. In its hydrated form, aluminum chloride is used by the pharmaceutical industry as an active ingredient in deodorants and antiperspirants. It is also used in wood preservation and in the manufacture of adhesives, paint pigments, resins, fertilizers, and astringents (EC, 2008).

A.3.1.1 Aluminum Chloride Environmental Fate and Transport

Aluminum chloride, hydrated in air, will react with moisture to produce hydrochloric acid and aluminum oxide. Aluminum chloride is highly soluble and will form various dissolved species in water; the solubility of these species in water is highly dependent on pH, and the chemistry is complex. In general, aluminum is a strongly hydrolyzing metal that is relatively insoluble in the neutral pH range (6.0–8.0). Aluminum solubility is enhanced in the presence of complexing ligands such as dissolved organic carbon and under acidic ($pH < 6$) and alkaline ($pH > 8$) conditions. At low-pH values, dissolved aluminum is present mainly as Al^{3+}. Sediment, where metals are generally considered to be less biologically available, is nonetheless an important medium for aluminum (EC, 2008). Experimental acidification of lakes and limnocorrals has shown that aqueous aluminum concentrations rapidly increase in response to acidification (Brezonick et al., 1990). Mass-balance studies have demonstrated that retention of aluminum by sediments decreases as pH decreases (Dillon et al., 1988). Under such conditions, sediments in acidified watersheds can provide a source of aluminum to the water column (Wong et al., 1989). Based on a calculation of fluxes in acidic lakes, Wong and colleagues (1989)

suggested that sediment is a source of aluminum in the overlying water column. Inputs of aluminum into soil solutions usually occur by mobilization of aluminum derived from the chemical weathering of soil minerals. The most-important reaction in the chemical weathering of the common silicate minerals is hydrolysis. However, aluminum is not very soluble over the normal soil pH range; therefore, it generally remains near its site of release to form clay minerals or precipitate as amorphous or crystalline oxides, hydroxides, or hydrous oxides (EC, 2008).

A.3.1.2 Human Health Toxicology of Aluminum Chloride

The oral LD_{50} values for aluminum chloride were determined to be 370 and 222 mg/kg for the rat and mouse, respectively (ATSDR, 2008a). Occupational exposure studies and animal studies suggested that the lungs and nervous system may be the most-sensitive targets of toxicity following inhalation exposure. The genotoxicity of various aluminum compounds was described in detail by Krewski and colleagues (2007) and ATSDR (2008a). Briefly, aluminum compounds produced negative results in most short-term, in vitro mutagenic assays including the Rec-assay using *Bacillus subtilis*; *S. typhimurium* TA92, TA 98, TA102, TA104, and TA1000 strains (with and without S9 metabolic activation); and *E. coli* (Krewski et al., 2007). The literature concerning oral exposure bioassays was very limited. An increase in gross tumors was reported in male rats and female mice in a one-dose study, but few study details were reported by Schroeder and Mitchener in 1975 (ATSDR 2008a). Two other studies reported no increased incidence of tumors in rats and mice that were exposed orally to aluminum compounds (ATSDR, 2008a). IARC did not classify specific aluminum compounds for carcinogenicity, but classified the exposure circumstances of aluminum production as carcinogenic to humans (Group 1) (IARC, 1999). No increased tumor incidence was observed in rats following inhalation of alumina fibers (aluminum oxide) at concentrations of up to 2.45 mg/m^3 (Krewski et al., 2007).

A.3.1.3 Ecotoxicology of Aluminum Chloride

Aluminum salts hydrolyze when added to water, and monomeric aluminum can be formed in the dissolved fraction (EC, 2008). It is the monomeric aluminum, and not the salts, that can adversely affect organisms (Baker et al., 1990; Driscoll et al., 1980; Parker et al., 1989). Aluminum exposure may also disrupt ionic balance and osmoregulation in aquatic invertebrates (Otto and Svensson, 1983). Aluminum reduced the sodium influx, and to a lesser extent, increased the sodium outflux in *D. magna*, which impaired osmoregulation (Havas and Likens, 1985). Respiratory effects can occur in aquatic organisms when acidic waters are rapidly neutralized, such as when an acidic tributary enters a larger, neutral receiving stream. This can lead to the formation of mononuclear and polynuclear aluminum species from the dissolved ion in the water (Gensemer and Playle, 1999). These aluminum species may bind to or precipitate onto the bodies of invertebrates and create a physical barrier to respiration. Aluminum has been reported to impair reproduction in *D. magna* (Beisinger and Christensen, 1972), although recent work with *Daphnia pulex* suggests that adaptive strategies, which heighten survivorship and fecundity, may occur after long-term exposure to sublethal levels of aluminum (Wold et al., 2005). The BCF for aluminum by *D. magna* varied from 10,000 at pH 6.5 down to 0 at pH 4.5 (Havas, 1985). Similar results (i.e., decreasing accumulation of aluminum with decreasing pH) were reported for crayfish, caddisflies, unionoid clams, and chironomids (EC, 2008). Other studies with clams and benthic insects showed no relationship between water pH and tissue

accumulation of aluminum (Sadler and Lynam, 1985; Servos et al., 1985). Studying the effects of aluminum on the mayfly (*Heptagenia sulphurea*), Frick and Herrmann (1990) found that the largest portion (70%) of aluminum was present in the exuvia, which indicated that the metal was largely adsorbed and was not incorporated into the organism. Van Gestel and Hoogerwerf (2001) found that the earthworm (*Eisenia andrei*) has an aluminum LC_{50} value of 359 mg/kg at pH 4.4. In acid soils (pH $\leq$ 4.9), aluminum has been shown to be toxic to plants. The most easily recognized symptom of aluminum toxicity is the inhibition of root growth, which has become a widely accepted measure of aluminum stress in plants. Many trivalent cations are toxic to plants, and because aluminum toxicity is largely restricted to acidic conditions, it is generally assumed that $A1^{3+}$ is the major phytotoxic species. However, this has been difficult to confirm and nearly all of the monomeric aluminum species listed in this report have been considered toxic in one study or another (EC, 2008).

A.3.2 Aluminum Oxide

Aluminum oxide is produced from bauxite; it is crushed, ground, and then leached with sodium hydroxide to form sodium aluminate, from which the aluminum hydrate is precipitated and then calcined to produce aluminum oxide (alumina) (ATSDR, 2008a; HSDB, 2013; Krewski et al., 2007). Aluminum oxide is used in the production of aluminum; more than 95% of alumina produced is used for this purpose. Aluminum oxide is also used in the manufacture of abrasives, refractories, ceramics, electrical insulators and resistors, catalysts, paper, spark plugs, laboratory materials, light bulbs, artificial gems, alloys, glass, heat resistant fibers, and food additives (ATSDR, 2008a; HSDB, 2013; Krewski et al., 2007).

A.3.2.1 Aluminum Oxide Fate and Transport

Aluminum levels in surface water are usually very low (<0.1 mg/L) due to low solubility (O'Neil, 2006). However, in acidic water or water high in humic or fulvic acid content, the concentration of soluble aluminum increases somewhat due to the increased solubilization of aluminum oxide and aluminum salts. The aluminum concentration in soils varies widely, ranging from about 7 to over 100 g/kg; however, the vast majority of naturally occurring aluminum in soil matrices is in forms that are unavailable to organisms (Hausenbuiller, 1972; Sparling and Lowe, 1996).

A.3.2.2 Human Health Toxicology of Aluminum Oxide

Because aluminum oxide is essentially insoluble in water (O'Neil, 2006), it is poorly absorbed through membranes. Reports have been made of the appearance of progressive, non-nodular interstitial fibrosis in the aluminum abrasives industry, where aluminum oxide and silicon are processed. This condition, known as Shaver's disease, is rapidly progressive and often fatal. However, because the exposure in the recorded cases was to aluminum oxide as a fume, and because the great mass of evidence indicates the comparative harmlessness of aluminum oxide, the role of aluminum in the etiology of Shaver's disease remains unclear (HSDB, 2013). Additionally, no significant increase in cancer morbidity or mortality was found in 521 Swedish workers who were exposed to aluminum oxide in an abrasive-manufacturing plant in a cohort that was followed up between 1958 and 1983 (HSDB, 2013).

A.3.2.3 Ecotoxicology of Aluminum Oxide

Data for micro- and macro-sized particles of aluminum oxide in aquatic systems are lacking. Exposure of the water flea (C. *dubia*) to nano-sized particles of aluminum oxide resulted in an LOEC of 100 mg/L for growth and mortality and an EC_{50} aluminum oxide concentration of 42 mg/L (USEPA, 2013a). Aluminum oxide is considerably less toxic than more-soluble forms of aluminum salts (e.g., aluminum salts of chloride, nitrate, and sulfate) (USEPA, 2003). Storer and Nelson (1968) observed no toxicity to poultry chicks that were fed up to 1.6% of their diet as aluminum oxide. Aluminum oxide is essentially insoluble in water (O'Neil, 2006).

A.3.3 Ammonium Chloride

The fate, transport, human health toxicology, and ecotoxicology of ammonium chloride has been reviewed in Section A.2 of this document.

A.3.4 Boron Trioxide

Boron trioxide is a noncombustible, odorless, colorless or white, brittle substance that occurs as hygroscopic granules, flakes, or powder. It is the anhydride form of boric acid. Because of its hygroscopic properties, boron trioxide reacts easily with water to form boric acid and hydrates. Commercially, boron trioxide is used in the production of different glass types, glass fibers, ceramics, and enamels.

Environmental fate, transport, human health effects, and ecotoxicological information for boron compounds, including the hydration product of boron trioxide (boric acid), are listed under Boron in Section A.2.3.

A.3.5 Carbon Dioxide

Carbon dioxide occurs naturally in the atmosphere from 0.03 to 0.06% (v/v; equivalent to 0.2 to 0.4 mmHg of atmospheric pressure) (HSDB, 2013). At normal temperature and pressure, carbon dioxide is an odorless, colorless, and heavier-than-air gas. Carbon dioxide is normally present as a dissolved gas in natural waters, and some spring waters are so charged with the gas under pressure that the water is effervescent. The average indoor concentration of carbon dioxide is 0.08 to 0.1%; the maximal acceptable concentration has been defined as between 0.5 and 3%, depending on the duration of exposure (HSDB, 2013). Carbon dioxide is produced during combustion, putrefaction, and fermentation balance (Guais et al., 2011). Active volcanoes emit large quantities of carbon dioxide (HSDB, 2013). Carbon dioxide is widely used in industries, especially in solid form (dry ice) for cooling and conserving food products and as a gas in medical applications when mixed with oxygen to avoid carbon dioxide tension in blood (Guais et al., 2011; HSDB, 2013). Pressurized carbon dioxide is used in fire extinguishers and as a propellant gas in some commercial aerosol products.

A.3.5.1 Carbon Dioxide Fate and Transport

In air, carbon dioxide is a very stable and nonflammable compound. Carbon dioxide is soluble in water, where it reacts to form dilute carbonic acid. Dissolved carbon dioxide in ambient water undergoes hydration according to the following reaction: $CO_2 + H_2O \rightarrow H_2CO_3 \rightarrow H^+ + HCO_3^-$. This reaction can affect the acid–base balance (Guais et al., 2011). The average indoor concentration of carbon dioxide is 0.08 to 0.1% (HSDB, 2013). Increasing atmospheric carbon dioxide concentrations in recent years has contributed to the greenhouse effect, and it has been hypothesized to be one of the primary gases responsible for acceleration of global warming (Cox et al., 2000; Lal, 2001). It has been calculated that terrestrial ecosystems effectively remove approximately 495 billion tons of carbon dioxide per year (123 ± 8 Pg of carbon per year) from the atmosphere (Beer et al., 2010). Soil is both a source and a substantial sink for atmospheric carbon dioxide (Lal, 2001).

A.3.5.2 Human Health Toxicology of Carbon Dioxide

Carbon dioxide is a product of respiration within the human body, and its partial pressure under normal conditions in pulmonary capillary blood (approximately 7%, or 46 mmHg) is greater than that in alveolar air (6%, or 40 mmHg). The gas is exchanged freely through the alveolar membrane and is thus released from the lungs by diffusion because of the concentration gradient existing between blood and air in the alveoli. Its free diffusion through lipid cell membranes allows it to be one of the main regulators of intracellular pH, whereby it acts as a stimulant or a retardant in numerous cellular processes. Because of its free diffusion through tissue membranes, the toxicological effects of carbon dioxide can appear very rapidly and are mainly observed in the blood pH, lungs, heart, and central nervous system, and the maximal acceptable concentration of carbon dioxide has been defined as between 0.5 and 3%, depending on the duration of exposure (Guais et al., 2011). Because carbon dioxide freely diffuses through the alveolar membrane to the blood, it results in an increase of the carbon dioxide tension in arterial blood ($PaCO_2$). Therefore, increases in $PaCO_2$ can cause acute or chronic respiratory acidosis. In acute respiratory acidosis, the $PaCO_2$ is elevated above the upper limit of the reference range (i.e., >6.75% or 45 mmHg), which results in acidosis (i.e., pH <7.35). In chronic respiratory acidosis, the value of the pH is subnormal secondary to renal compensation and an elevated concentration of serum bicarbonate (HSDB, 2013). Carter and colleagues (1959) measured plasma carbon dioxide contents of 45 and 52 mequiv/L in rats exposed to either 10 or 15% carbon dioxide for 11 days (chronic acidosis), respectively, and they estimated the $PaCO_2$ values to be 15% (102 mmHg CO_2) and 22% (148 mmHg CO_2) for the same rat exposures, respectively. Carbon dioxide is heavier than air and collects in low spots, displacing air in these locations. Hundreds of people have died of carbon dioxide asphyxiation near volcanoes in the past two decades, most of them in Cameroon, Africa and Indonesia (HSDB, 2013).

A.3.5.3 Ecotoxicology of Carbon Dioxide

Studies with Atlantic salmon (*Salmo salar*) resulted in an LOEC of 19,000 µg/L (4.3% mortality) after 63 days of exposure to carbon dioxide in freshwater, and a NOEC of 10,600 µg/L in saltwater. Carbon dioxide in ambient air is not expected to significantly harm

terrestrial organisms. Terrestrial plants take up carbon dioxide to convert it into organic constituents through photosynthesis, and it has been calculated that terrestrial ecosystems effectively remove approximately 495 billion tons of carbon dioxide per year (123 ± 8 Pg of carbon per year) from the atmosphere (Beer et al., 2010).

A.3.6 Carbon Monoxide

Carbon monoxide is a product of the incomplete combustion of carbon-containing fuels and is also produced by natural processes. Among the natural sources of atmospheric carbon monoxide are oxidation of methane, forest fires, terpene oxidation, and production by terrestrial and marine microorganisms. Biotransformation of halomethanes within the human body is also a source. Natural sources account for 90% of atmospheric carbon monoxide, whereas human activity produces about 10% (HSDB, 2013).

A.3.6.1 Carbon Monoxide Fate and Transport

Under ambient conditions, outdoors where sufficient oxygen is present, carbon monoxide reacts with oxygen to produce carbon dioxide.

A.3.6.2 Human Health Toxicology of Carbon Monoxide

As exposure to carbon monoxide increases, subtle effects can begin to occur, and exposure to high levels can result in death. The health effects of carbon monoxide are largely the result of the formation of carboxyhemoglobin, which impairs the oxygen-carrying capacity of the blood (HSDB, 2013). During typical daily activities, people encounter carbon monoxide in a variety of microenvironments—while traveling in motor vehicles; working at their jobs; visiting urban locations associated with combustion sources; cooking or heating with domestic gas, charcoal, or wood; as well exposure to carbon monoxide in tobacco smoke. Carbon monoxide is absorbed through the lungs, and the resulting concentration of carboxyhemoglobin in the blood will depend mainly on the concentrations of inspired carbon monoxide and oxygen. However, it will also depend on the duration of exposure, pulmonary ventilation, and the beginning concentration of carboxyhemoglobin within the blood (HSDB, 2013). The binding of carbon monoxide to hemoglobin, which produces the carboxyhemoglobin that decreases the oxygen-carrying capacity of blood, appears to be the principal mechanism of action underlying the induction of toxic effects of low-level carbon monoxide exposures. Carbon monoxide related cognitive effects include impairment of memory, attention, executive and motor functions, visual spatial acuity, and mental-processing speed.

A.3.6.3 Ecotoxicology of Carbon Monoxide

Ecotoxicological effects of carbon monoxide on tested species have been minimal. At concentrations of 28–350 mg/L in the air and a light intensity of 18,000 lux, carbon monoxide had no effect on growth in cultures of the aquatic algae *Chlorella*. However, at 4,500 lux, the lowest exposure concentration tested, carbon monoxide inhibited the algal growth rate. At levels of 100 ppm, carbon monoxide had negligible effects on the behavior of several *Enchytraeus* species including *Arion fasciatus*, *Tracheoniscus rathkei*, *Diploiulus* species,

Liobunum calcar, and several other forest-litter invertebrates; carbon monoxide had no effect on the health and biological functions of these organisms (ECHA, 2009). During times of illumination, plants and leaves of 35 species of temperate and tropical plants absorbed carbon monoxide from air containing 6 ppm carbon monoxide at an average rate of 190 nL/kg of plant fresh weight (ECHA, 2009). Carbon monoxide uptake from the air by nine plant species, with widely different rates of absorption, was proportional to the carbon monoxide concentration within the range tested (from 0 to 100 ppm). Absorbed carbon monoxide was metabolized by plants either by oxidation to carbon dioxide and fixation or by reduction and incorporation into serine. Carbon monoxide had various effects on the photosynthesis in the leaves of different species; it acted inhibitory at concentrations as low as 65 ppm, exerted no influence, or at 99% carbon monoxide in air, it increased the net carbon fixation within the plants (ECHA, 2009).

A.3.7 Lithium Chloride

Lithium chloride is a hygroscopic, white cubic crystalline material. It is soluble in water, ethanol, acetone, pyridine, and nitrobenzene. Lithium chloride is used in the manufacture of mineral waters, in pyrotechnics, as flux-soldering aluminum, in refrigeration machines, as an additive in the chemical synthesis of some organic compounds, as a relative humidity standard in the calibration of hygrometers, and as a desiccant (HSDB, 2013). Refer to Section A.2 in this document and Aral and colleagues (2008) for more-detailed information regarding the fate and toxicology of all lithium compounds.

A.3.7.1 Lithium Chloride Environmental Fate and Transport

Lithium chloride in nature is found in small concentrations in seawater and mineral wells, salt brines, and salars (Garrett, 2004).

A.3.7.2 Human Health Toxicology of Lithium Chloride

Ingestion of lithium chloride has been reported to produce drowsiness, weakness, tremors, anorexia, nausea, blurred vision, and muscle spasms in humans. Exposure to lithium chloride has been shown to impair reproductive function and to produce developmental toxicity in laboratory animals (Moore, 1995). The lithium chloride oral LD_{50} value for the rat is 526 mg/kg (Peterson, 1980). There was no lethality observed for rats exposed by inhalation to up to 5.57 mg/L lithium chloride for 4 h (RTECS, 2013). The absorption of lithium through the skin is considered to be very poor. The dermal LD_{50} value for the rat was experimentally determined to be >2000 mg/kg (RTECS, 2013). Lithium chloride is not listed as a mutagen or carcinogen.

A.3.7.3 Ecotoxicology of Lithium Chloride

The 96 h LC_{50} value for lithium chloride is 158 mg/L for rainbow trout (*O. mykiss*) (ECHA, 2014a). The 26 day LC_{50} value for lithium chloride is 6.4 mg/L for fathead minnow (*P. promelas*) embryos (RTECS, 2013). Earthworms (*E. fetida*) exhibited 90% mortality when exposed to 5 mmol/kg of lithium chloride in soil (Fischer and Molnar, 1997). Lithium chloride (and lithium sulfate) produced foliar injury symptoms in sweet orange plants exposed to ≥2 mg/kg of lithium chloride in soil (Aldrich et al., 1951). Other citrus species have also shown

foliar injury symptoms (Aldrich et al., 1951; Bingham et al., 1964). Injury symptoms attributable to excess lithium in soils have not been found for any plants other than citrus species.

A.3.8 Lithium Oxide

When elemental lithium metal is burned in the air and combines with oxygen, lithium oxide is formed (Greenwood and Earnshaw, 1984).

A.3.8.1 Lithium Oxide Environmental Fate and Transport

When elemental lithium metal is burned in air and combines with oxygen, lithium oxide forms, along with small amounts of lithium peroxide (Greenwood and Earnshaw, 1984). Lithium oxide is hygroscopic and reacts with water in air, water bodies, and soil to form lithium hydroxide, which is a corrosive chemical (HSDB, 2013).

A.3.8.2 Human Health Toxicology of Lithium Oxide

Lithium hydroxide is listed as a corrosive chemical (HSDB, 2013), and for this reason, lithium oxide is also listed as corrosive chemical on most lithium oxide material safety data sheets. No other data was found specifically for lithium oxide. Refer to sections in this document that describe the human health toxicology of other lithium compounds.

A.3.8.3 Ecotoxicology of Lithium Oxide

No ecotoxicity data was found specifically for lithium oxide. Refer to sections in this document that describe ecotoxicology of other lithium compounds.

A.3.9 Magnesium Chloride

Magnesium accounts for approximately 2% of the earth's crust, it is eighth in elemental abundance, and it is widely distributed in the environment as a variety of compounds (HSDB, 2013). Its concentration averages 1.8 and 1.6% in igneous and sedimentary rocks, respectively. In igneous rocks, magnesium is typically a constituent of the dark-colored ferro-magnesium minerals (e.g., olivine, pyroxenes, amphiboles, and dark-colored micas), along with other less-common minerals. In metamorphic rocks, magnesium minerals such as chlorite, montmorillonite, and serpentine occur (HSDB, 2013). Sedimentary rocks of magnesium include carbonates (e.g., magnesite and hydromagnesite), hydroxides (e.g., brucite), and mixtures of magnesium and calcium carbonate (e.g., dolomite). Magnesium is also found in silicate minerals (e.g., olivine, serpentine, and asbestos). Rocks and minerals generally contain a higher percentage of magnesium than do soils as a result of the loss of magnesium due to leaching during weathering. Magnesium chloride comprises 17% of sea salt (HSDB, 2013).

Magnesium is an essential element in humans, animals, and plants, and is naturally present in many foods, added to other food products, available as a dietary supplement, and present in some medicines such as antacids and laxatives. Magnesium is also a cofactor in more than 300 enzyme systems that regulate diverse biochemical reactions in the body, including

protein synthesis, muscle and nerve function, blood glucose control, and blood pressure regulation (NIH, 2013). Magnesium is required for energy production, oxidative phosphorylation, and glycolysis. It contributes to the structural development of bone and is required for the synthesis of DNA, RNA, and the antioxidant glutathione. Magnesium also plays a role in the active transport of calcium and potassium ions across cell membranes, which is a process that is important for nerve impulse conduction, muscle contraction, and normal heart rhythm (NIH, 2013). Chloride is an essential micronutrient for humans, animals, and plants (NIH, 2013; Hausenbuiller, 1972).

A.3.9.1 Magnesium Chloride Fate and Transport

Given its high solubility in water, magnesium chloride in aqueous solution dissociates into magnesium cation (Mg^{2+}) and chloride anions (Cl^{-}). The dissociated magnesium cation can then form complexes with dissolved ligands present in natural waters or become bound to sediment or soil particles by ion exchange or precipitation reactions. Chloride anions in the soil tend to increase the concentration of available cations in solution. Because chloride is an anion that carries a negative charge, it generally does not adsorb to most soil or sediment particles that also carry negative charges; therefore, chloride tends to move with water within soil (Hausenbuiller, 1972). Magnesium is widespread in living cells and generally does not bioconcentrate in aquatic organisms, and chloride is also present in living cells as an essential trace element. Environmental fate analysis, based on log K_{OW} and log K_{OC} values and typical fugacity modeling, is not applicable for magnesium chloride as it is an inorganic compound. Photodegradation and biodegradation also do not apply to inorganic metal salts such as magnesium chloride (OECD, 2011). Magnesium chloride is released into the atmosphere from sea spray.

A.3.9.2 Human Health Toxicology of Magnesium Chloride

Magnesium is absorbed mainly in the small intestine after oral exposure; however, the colon also absorbs some. Magnesium absorption following oral ingestion is reported to range from 40 to 60%, with a lower percentage of absorption at higher daily intakes (OECD, 2011). Magnesium decreases the absorption of fluoride, and excess calcium may partially inhibit the absorption of magnesium. In the plasma, the level of magnesium is about 65% in ionic form, with the remainder bound to plasma proteins. Of the approximately 20 g body burden of magnesium, most is stored primarily in bone and muscle after absorption. Magnesium is excreted into the digestive tract by bile, pancreatic, and intestinal juices (OECD, 2011). A small amount of radiomagnesium, given intravenously, appears in the gastrointestinal tract, and the resulting serum levels are remarkably constant. There is an apparent obligatory urinary loss of magnesium, which amounts to about 12 mg of magnesium per day, and urine is the major route of excretion under normal conditions. Magnesium excretion can also occur via sweat and breast milk (OECD, 2011). Unabsorbed ingested magnesium is excreted in the feces.

The magnesium chloride acute oral LD_{50} value for female rats is between 300 and 2000 mg/kg of bw/day and was estimated to be 1085 mg/kg of bw/day (OECD, 2011). Dead animals had gastric filling with the test substance. At 300 mg/kg of bw/day, no treatment-related clinical signs and no mortality were observed. There were normal body weight gains in all

animals. At the end of the study, necropsy was conducted on all animals, and no abnormal gross findings were observed. The 24 h dermal LD_{50} value for magnesium chloride was in excess of 2000 mg/kg of bw/day in male and female rats. Signs of toxicity, such as diarrhea and watery diarrhea, were observed at 2000 mg/kg of bw/day. Skin sensitization to magnesium chloride was investigated in the female guinea pig (5 weeks) following the study, and no evidence was found (OECD, 2011).

A.3.9.3 Environmental Toxicology of Magnesium Chloride

Magnesium is an essential nutrient required in relatively large quantities in plants (Barker and Pilbeam, 2006). Magnesium plays two very essential roles in the processes of photosynthesis and carbohydrate metabolism. Magnesium is the metal atom in the chlorophyll molecule, without which photosynthesis would not occur. Magnesium also serves as an activator in carbohydrate metabolism. Magnesium serves as an activator for those enzymes involved in the synthesis of the nucleic acids (RNA and DNA) from nucleotide polyphosphates. Magnesium may also be involved in protein synthesis, serving as a binding agent in the microsomal particles (Davis, 1983).

Chloride has also been established as an essential plant micronutrient and is thought to play a role in photosynthesis (Barker and Pilbeam, 2006). When excessive amounts of magnesium chloride are used as a de-icing salt in winter and as a dust suppressant in spring and summer, magnesium chloride has been shown to be toxic to roadside trees (Goodrich, 2008). High concentrations of magnesium chloride in the soil may be toxic or change water relationships such that plants cannot easily accumulate water and other essential nutrients. Although chloride is a micronutrient in plants, excess chloride is considered to be the primary toxicant (Hausenbuiller, 1972). Once inside the plant, excess chloride moves through the water-conducting system and accumulates at the margins of leaves or needles, where dieback occurs first. Leaves are weakened or killed, which can lead to the death of the tree in extreme cases. Severe foliar damage has been reported in coniferous species containing 4,000 to 20,000 mg/kg of chloride in leaf tissues within 91 m (300 ft) from roads (Goodrich, 2008). Magnesium chloride is relatively nontoxic to aquatic organisms (HSDB, 2013). The 48 h LD_{50} values for magnesium chloride were 1330 mg/L and 2840 mg/L for *Daphnia* and the fathead minnow (*P. promelas*), respectively (Mount et al., 1997).

A.3.10 Magnesium Oxide

Magnesium oxide, also known as magnesia, is the second-most abundant compound in the earth's crust, next to silicon dioxide (Greenwood and Earnshaw, 1984). Magnesium oxide is used in making crucibles and insulating materials; refining some metals from their ores; and in the manufacture of some types of cements, high-temperature refractories, electrical insulation (Shand, 2006), food packaging (Ash, 2004; Othman, 2014), cosmetics, and pharmaceuticals (Ash, 2004). When mixed into water, magnesium oxide forms magnesium hydroxide, which is only mildly alkaline due to its low solubility in water (McGraw-Hill, 2003). A suspension of magnesium hydroxide in water is commercially known as milk of magnesia, and is commonly used as a nonprescription antacid or laxative (Ash, 2004). See Section 3.3.5 for a description of magnesium other magnesium-containing compounds.

A.3.10.1 Magnesium Oxide Fate and Transport

Magnesium oxide is only slightly soluble in water, at 6.2 mg/L at 20 °C (McGraw-Hill, 2003); therefore, it is not expected to be very mobile or highly bioavailable in surface waters, sediments, or soils.

A.3.10.2 Human Health Toxicology of Magnesium Oxide

The primary human health hazard associated with magnesium oxide is development of "metal fume fever" in production plants, which causes influenza-like symptoms including cough, chest pain, and fever, if it is inhaled in sufficient quantity (HSDB, 2013). The symptoms of metal fume fever may be delayed for 4–12 h following exposure. Proper personal protective equipment and engineering controls help avoid excess inhalation of magnesium oxide.

A.3.10.3 Environmental Toxicology of Magnesium Oxide

Magnesium is an essential nutrient that is required in relatively large quantities in plants (Barker and Pilbeam, 2006). No ecotoxicity studies were found specifically for magnesium oxide toxicity to aquatic or terrestrial organisms.

A.3.11 Potassium Carbonate

Potassium carbonate is used in manufacturing soap, liquid shampoos, glass, and pottery; in process engraving and lithography; in tanning and finishing leather; for removal of water from organic liquids; in analytical chemistry; and as a general-purpose food additive (HSDB, 2013). Potassium is an essential nutrient required in relatively large quantities in plants (Hausenbuiller, 1972) and is the third major nutrient for plants after nitrogen and phosphorus (Jasinski, 2010). Since antiquity, soluble salts of potassium have been added to soil as potash fertilizer to increase fertility (Jasinski, 2010); the original primary ingredient of potash fertilizer was potassium carbonate (Davy, 1840). Today, potash fertilizer may contain potassium carbonate as well as any of the soluble salts of potassium (Jasinski, 2010).

A.3.11.1 Potassium Carbonate Fate and Transport

Potassium carbonate is a naturally occurring compound, with deposits found in southeastern New Mexico (HSDB, 2013). It is one of the major inorganic particle components of cigarette smoke (Churg and Stevens, 1992). Potassium carbonate is soluble in water (Haynes, 2010). In water, potassium carbonate dissociates into potassium cations (K^+) and carbonate anions (CO_3^{2-}), and neither of these ions is expected to adsorb significantly to sediment. Carbonate ions subsequently react with water, resulting in the formation of bicarbonate and hydroxide, until an equilibrium is established. The ions do not volatilize into air from water. When added to water or soil, potassium carbonate will increase alkalinity with a corresponding tendency to raise the pH value (HERA, 2005).

A.3.11.2 Human Health Toxicology of Potassium Carbonate

As an alkaline corrosive, potassium carbonate may cause liquefaction necrosis; it can saponify the fats in the cell membrane, which destroys the cell and allows deep penetration of the chemical into mucosal tissue. In gastrointestinal tissue, an initial inflammatory phase may be followed by tissue necrosis (sometimes resulting in perforation), then granulation, and finally stricture formation (HSDB, 2013). The Ames test for mutagenicity was negative. Carcinogenicity tests with potassium carbonate were not found; the compound is not listed as a carcinogen.

A.3.11.3 Ecotoxicology of Potassium Carbonate

Studies with potassium carbonate and the aquatic invertebrates, *C. dubia* and *D. magna*, yielded 48 h LC_{50} values of 630 and 650 mg/L, respectively (Mount et al., 1997). A 96 h LC_{50} value of 510 mg/L was determined for fathead minnows (*P. promelas*) exposed to potassium carbonate (Mount et al., 1997). The minimum lethal concentration for *Daphnia* was also shown to be 300 mg/L at 17 °C, and at 800 mg/L, all of these exposed organisms were killed (HERA, 2005).

A.3.12 Potassium Chloride

The fate and transport, human health toxicology, and ecotoxicology of potassium chloride are summarized in Section A.2 of this document (Characterization, Toxicological Summaries, and Fate and Transport Summaries for Component Compounds of Candidate Smokes). Toxicological endpoints are listed in Tables A.1 and A.3.

A.3.13 Potassium Metaborate

Environmental fate, transport, human health effects, and ecotoxicological information for boron compounds are listed under Boron in Section A.2.3.

A.3.14 Titanium Tetrachloride

Titanium tetrachloride is a colorless to pale-yellow liquid that has fumes with a strong odor. If it comes in contact with water, it transforms into hydrochloric acid and titanium compounds. In 1990, approximately 1.5 million tons of titanium tetrachloride were produced in the United States. Titanium tetrachloride is not found naturally in the environment and is made from minerals that contain titanium. Titanium tetrachloride is used to make titanium metal and other titanium-containing compounds, such as titanium dioxide, which is used as a white pigment in paints and other products and as an intermediary to produce other chemicals (ATSDR, 1997b). Titanium tetrachloride is a visible obscurant that is meant to be disseminated from aircraft or ships but is seldom used because of its corrosivity and its propensity to clog lines because of its hygroscopic nature (U.S. Army, 2000).

A.3.14.1 Titanium Tetrachloride Fate and Transport

Titanium tetrachloride enters the environment primarily as air emissions from facilities that make or use it in various chemical processes, from white smoke generation in military operations, or as a result of spills. If moisture is present in the air, titanium tetrachloride reacts with the moisture to form hydrochloric acid and other titanium compounds, such as titanium hydroxide, titanium oxychlorides, and titanium dioxide. The end products produced when titanium tetrachloride reacts with water are titanium dioxide and hydrochloric acid. The hydrochloric acid may react to break down or be carried in the air. When titanium tetrachloride is released into water, it rapidly hydrolyzes to hydrochloric acid, titanium oxychloride, and titanium dioxide; titanium oxychloride usually further hydrolyzes to hydrochloric acid and titanium dioxide (ATSDR, 1997b). In water, hydrochloric acid dissociates to the hydrogen cation (H^+) and the chloride anion (Cl^-). Titanium dioxide is insoluble in water and may settle out into sediment or soil. No information was located on the degradation of titanium tetrachloride released into soils or sediments; however, based on the rapid hydrolysis of this compound in moist air or in water, it may be expected that titanium tetrachloride will also hydrolyze upon contact with moisture in soil and sediment, as described herein. Therefore, it is not expected to bioaccumulate (ECHA, 2014b).

A.3.14.2 Human Health Toxicology of Titanium Tetrachloride

Titanium tetrachloride can be very irritating to the skin, eyes, mucous membranes, and lungs. Titanium tetrachloride is corrosive because it reacts strongly with water to produce hydrochloric acid. The reaction products, especially hydrochloric acid, can cause harmful health effects including burns that can occur after exposure to titanium tetrachloride. Breathing in large amounts of titanium tetrachloride can injure the lungs seriously enough to cause death (ATSDR, 1997b). After short-term exposure to titanium tetrachloride, less serious respiratory system effects can include coughing and tightness in the chest. More severe effects can include chemical bronchitis or pneumonia and congestion of the mucous membranes of the upper respiratory tract. These effects can cause long-term effects such as the narrowing of the vocal cords, windpipe, and upper airways. Accidental exposure to liquid titanium tetrachloride can result in skin burns and can cause permanent damage to the eyes if they are not protected. (ATSDR, 1997b). Limited information was located regarding the lethal effects in animals after inhalation exposure to titanium tetrachloride. The 4 h and 2 min inhalation LC_{50} values in rats exposed head-only were 460 and 108,000 mg/m^3, respectively (DuPont, 1980); other exposure durations have LC_{50} values that fall in between these. Death was attributed to pulmonary edema and occurred during exposures and up to 1 week post-exposure. After or during a single 2 h inhalation exposure to various levels of titanium tetrachloride and its hydrolysis products (titanium oxychloride, titanium dioxide, and hydrochloric acid), 9 of 15 mice died. Some laboratory animals that breathed titanium tetrachloride fumes for 2 years developed lung tumors of a special type. However, there is no evidence that chronic exposure to titanium tetrachloride causes cancer in humans. There is not enough information to determine if titanium tetrachloride causes birth defects or affects reproduction. Titanium tetrachloride has not been classified for its carcinogenic properties.

A.3.14.3 Ecotoxicology of Titanium Tetrachloride

The substance is not expected to be acutely toxic to aquatic invertebrates. Titanium tetrachloride rapidly hydrolyzes in water, which results in the formation of titanium dioxide and hydrochloric acid that causes an increase in acidity. The resulting titanium dioxide is very poorly soluble in water at neutral pH (<0.1 µg/L); excess titanium dioxide will be present as an insoluble precipitate. Aquatic toxicity data for titanium dioxide show an absence of acute and chronic effects in fish and invertebrates at nominal concentrations that were several orders of magnitude higher than the soluble concentration. In moderately buffered water (250 mg/L as calcium carbonate), a shift from pH 7.0 to 5.0, which might be biologically intolerable, would only occur when the added concentration of titanium tetrachloride exceeded 100 mg/L (ECHA, 2014b). Standardized tests using concentrations of titanium tetrachloride equal to or exceeding levels found in the environment were nontoxic to terrestrial micro- and macro-invertebrates, microorganisms, and plants (ECHA, 2014b).

Table A.1. Human Health Toxicology Data for Component Compounds

Compound	Acute Oral (mg/kg)	Chronic Oral (mg/kg/day)	Inhalation (mg/L)	Dermal	Ocular	Mutagenicity	Carcinogenicity
Potassium chloride	LD_{50} 2,600 (guinea pig, rat)[a]; 383 (mouse)[a]	n/a	nd; toxicity expected to be low due to low VP[b]	nd; toxicity expected to be low due to low log K_{OW} [b]; irritant at 60 % solution[a]	Mild irritation (500 mg, 24h)[a]	Negative Ames[a]	Negative 2 year rat study[a]
Potassium nitrate	LD_{50} 3,750 (rat)[c]; 1,901 (rabbit)[c]; >22.8 mg/L (infant methemoglobinemia)[b]	365 NOAEL (rat lifetime)[c]	nd; toxicity expected to be low due to low VP[b]	Probable irritant[c]	Probable irritant[c]	Negative Ames[c]	Equivocal; conversion to nitrite increases reactivity of molecule[d]
Boron (elemental)	nd; toxicity expected to be low[b]	nd; toxicity expected to be low[b]	low; toxicity expected to be low due to low VP[b]	nd; toxicity expected to be low due to low log K_{OW} [b]	Mechanical irritant[e]	Not listed[f]	Not listed[g]
Boron carbide	nd; toxicity expected to be low[b]	nd; toxicity expected to be low[b]	nd; toxicity expected to be low due to low VP[b]	nd; toxicity expected to be low due to low log K_{OW} [b]	Mechanical irritant[e]	Not listed[f]	Not listed[g]
Calcium stearate	nd; toxicity expected to be low, stearic acid LD_{10} 4,600 (rat)[h]	nd; toxicity expected to be low, stearic acid LD_{10} 4,600 (rat)[h]	nd; toxicity expected to be low due to low VP[b]	nd; toxicity expected to be low due to low log K_{OW} [b]	nd; toxicity expected to be low[b]	Negative Ames[g]	Negative[g]

table continued

Table A.1. Human Health Toxicology Data for Component Compounds (continued)

Compound	Acute Oral (mg/kg)	Chronic Oral (mg/kg/day)	Inhalation (mg/L)	Dermal	Ocular	Mutagenicity	Carcinogenicity
Dechlorane Plus	LD_{50} >25,000 (rat)[i]	NOAEL 5,000 (rat)[i]	4 h LC_{50} >2.25 (rat)[i]; 1 h LC_{50} >300 (rat)[i]	LD_{50} >8,000 (rabbit)[i]	Minor irritant[i]	Negative Ames[i]	Negative[e]
Ammonium chloride	LD_{50} 1,630 (rat)[j]	LD_{50} >684[j]	nd; minor irritant[j]	nd; minor irritant[j]	Minor irritant[j]	Negative[j]	Negative (rat)[j]
Lithium carbonate	LD_{50} 531 (mouse)[k]; 525 (rat)[k]	30 day LTD 1980 (rat)[k]	LD_{50} 1,700 (rat)[l]	LD_{50} >2,000 (rabbit)[k]	nd; toxicity expected to be low[m]	Negative Ames, positive Chinese hamster mutation assay[e]	Not listed by IARC, NTP, OSHA or ACGIH[e]
Lithium perchlorate	LD_{50} 1,900 (rabbit)[n]	No effect[n]	No effect[n]	nd	nd	Negative[n]	Negative[n]
Azodicarbonamide	LD_{50} >5,000 (rat)[o]	14 day LTD 1,250 (mice, rats)[o]	LC_{50} >6,100[o]	LD_{50} >2,000 (rabbit)[o]	Negative[o]	Negative Ames[o]	Negative[g]

Notes: n/a, not applicable; nd, no data; VP, vapor pressure; ACGIH, American Conference of Governmental Industrial Hygienists
[a]OECD, 2001; [b]Haynes, 2010; [c]Sax, 1989; [d]Bouchard et al., 1992; [e]HSDB, 2013; [f]WHO, 1998; [g]CCRIS, 2013; [h]CIDPL, 2009; [i]USEPA, 2011a; [j]OECD, 2004; [k]NIOSH, 2014; [l]Greenspan et al., 1986; [m]Opresko, 1995; [n]ATSDR, 2008b; [o]WHO, 1999.

Table A.2. Human Health Toxicology Data for Reaction Products

Compound	Acute Oral (mg/kg)	Chronic Oral (mg/kg/day)	Inhalation (mg/L)	Dermal	Ocular	Mutagenicity	Carcinogenicity
Potassium metaborate	nd	nd	nd	nd	nd	Negative[a]	Negative[b]
Potassium chloride	LD_{50} 2,600 (guinea pig, rat), 383 (mouse)[a]	n/a	nd; toxicity expected to be low due to low VP[c]	nd; toxicity expected to be low due to low log K_{OW} Irritant at 60% solution[d]	Mild irritation (500 mg, 24 h)[d]	Negative Ames[b]	Negative (2 year rat study)[e]
Potassium carbonate	LD_{50} 2,570 (mouse), 1,870 (rat)[b]	nd	Irritant and caustic[b]	Irritant and caustic[b]	Irritant and caustic[b]	Negative Ames[b]	Negative[e]
Titanium tetrachloride	LD_{50} >2,900 (rat)[f]	nd	2 h LD_{50} 0.10 (mouse); 4 h LD_{50} 0.46 (rat); corrosive[f]	Corrosive[f]	Corrosive[f]	Negative[f]	Negative[e]
Carbon dioxide	n/a	n/a	"Respiratory acidosis" at >66%[b]	n/a	n/a	Negative[b]	Negative[e]
Magnesium chloride	LD_{50} 2,800 (rat)[b]	nd	Minor irritant[b]	Minor irritant[b]	Minor irritant[b]	Negative[b]	Negative[e]
Aluminum chloride	LD_{50} 222 (mouse)[b]; 370 (rat)[b]	No adverse effect[b]	Minor irritant[b]	Minor irritant[b]	Minor Irritant[b]	Negative[b]	Negative[e]
Magnesium oxide	LD_{50} 3,870 (rat)[b]; 810 (mouse)[b]	No adverse effect[b]	Minor irritant[b]	Minor irritant[b]	Minor Irritant[b]	Negative Ames[b]	Negative[e]
Aluminum oxide	LD_{50} >5,000 (rat)[b]	nd	No effect (rat)[g]	Irritant[b]	Irritant[b]	Negative[b]	Negative[e]

table continued

Table A.2. Human Health Toxicology Data for Reaction Products (continued)

Compound	Acute Oral (mg/kg)	Chronic Oral (mg/kg/day)	Inhalation (mg/L)	Dermal	Ocular	Mutagenicity	Carcinogenicity
Carbon	LD_{50} >10,000 (rat)[b]	nd	nd	LD_{50} 970 (mouse)[b]	nd	Negative[b]	Negative[e]
Carbon monoxide	n/a	n/a	LD_{50} 1,807/4 h (rat)[b]; 2,444/4 h (mouse)[b]	n/a	n/a	May be teratogenic[b]	Negative[e]
Lithium chloride	LD_{50} 526 (rat)[k]; 850 (rabbit)[b]; 1,165 (mouse)[b]	LTD 96 mg/kg/4 days (rat)[i]	No effect; 0.6–1.8 mg/m^3; 4–8 weeks, 5 days/week, 6 h/day (rabbit)[h]	Corrosive; LD_{50} >2000 mg/kg (rat)[i]	Irritant[b]	nd	Negative[e]
Lithium oxide	nd	nd	nd	nd	nd	Negative[b]	Negative[e]
Ammonium chloride	LD_{50} 1,410 (rat)[c]; LTD 500 (rabbit)[j]	Low[j]	Minor irritant[j]	Minor irritant[j]	Mild to severe[j]	nd	Negative[e]
Boron	nd	nd	nd	nd	nd	nd	nd
Boron trioxide	nd	nd	nd	nd	nd	nd	nd
Water	n/a	n/a	n/a	n/a	n/a	n/a	Negative[e]
Nitrogen gas	n/a	n/a	No effect[b]	n/a	n/a	Negative[b]	Negative[e]

[a]WHO, 1999; [b]HSDB, 2013; [c]Haynes, 2010; [d]Lewis, 2004; [e]CCRIS, 2013; [f]ATSDR, 1997b; [g]ATSDR, 2008a; [h]Johansson et al., 1988; [i]RTECS, 2013; [j]OECD, 2004; [k]Peterson, 1980.

Table A.3. Ecotoxicology Data for Component Compounds

Compound	Aquatic (µg/L)	Soil Invertebrate (mg/kg)	Terrestrial Plants	Mammals	Birds
Potassium chloride	24 h LC_{50} 630,000 (*Ceriodaphnia*)[a]; 96 h LC_{50} 134,000 (*Hyalella*)[a]; LD_{50} (fathead minnow) 950,000[a]	LC_{50} 5,595 (earthworm)[a]	Low[a]	Low[a]	Low[a]
Potassium nitrate	96 h LC_{50} 39,000 (*Daphnia*)[a]; 162,000 (mosquitofish)[a]	LC_{50} 144 µg/cm^2 (earthworm filter paper)[a]	LOEL 30 mM root length (durum wheat)[a]	LOEL 120 mg/L (Norway rat)[a]	nd
Boron (elemental)	nd	nd	nd	nd	nd
Boron carbide	nd	nd	nd	nd	nd
Calcium stearate	nd	nd	nd	nd	nd
Hexachloroethane	48 h LC_{50} 1,360 (*Daphnia*)[a]; LC_{50} 2,440 (bullfrog)[a]	48 h LC_{50} 19 µg/cm^2 (earthworm, filter paper)[a]	nd	nd	nd
Titanium dioxide (>100 nm diameter)	48 h NOEL 1,000,000 (*Daphnia*, midge, fathead minnow, rainbow trout)[a]	nd	nd	48 h NOEL 1,000,000 (Norway rat)[a]	nd
Magnesium metal	nd	nd	nd	nd	nd
Aluminum metal	24 h LC_{50} 2,600 (*Daphnia*)[a]; 96 h LC_{50} 160 (rainbow trout)[a]	NS ≤200 (earthworm survival, soil)[a]	nd	nd	8 day NOEL 10,000[a]
Dechlorane Plus	TL_{50} >100 (trout and sunfish)[a]	nd	nd	nd	nd

table continued

Table A.3. Ecotoxicology Data for Component Compounds (continued)

Compound	Aquatic (µg/L)	Soil Invertebrate (mg/kg)	Terrestrial Plants	Mammals	Birds
Ammonium chloride	48 h LC_{50} 101 (*Daphnia*)[a]; 96 h LC_{50} 96.2 (fathead minnow)[a]	14 day LC_{50} 160 (earthworm, soil)[a]	nd	nd	nd
Lithium carbonate	48 h LD_{50} 8,100 (mumichog)[a]	nd	nd	nd	nd
Azodicarbonamide	96 h NOEC 50,000 (fathead minnow)[a]; 48 h NOEC 4,800[a]; 48 h EC_{50} 11,000 (immobilization, *Daphnia*)[a]	nd	nd	nd	nd
Lithium perchlorate[†]	48 h LC_{50} 396,000 (*Daphnia*)[a]; 5 day LC_{50} 869,500[a]	nd	nd	nd	nd

Notes: NS, not statistically significant ($p > 0.05$) compared with control; LOEL, lowest-observed-effect level.
[†]No data for lithium perchlorate, values are for ammonium perchlorate.
[a]USEPA, 2013a.

Table A.4. Ecotoxicology Data for Reaction Products

Compound	Aquatic (mg/L)	Soil Invertebrate (mg/kg)	Terrestrial Plants	Mammals	Birds
Potassium metaborate	nd	nd	nd	nd	nd
Potassium chloride	24 h LC_{50} 630 (*Ceriodaphnia*)[a]; 96 h LC_{50} 134 (*Hyalella*)[a]; LD_{50} (fathead minnow) 950[a]	14 day LC_{50} 5595 (earthworm)[a]	Low[a]	Low[a]	Low[a]
Potassium carbonate	24 h LC_{50} 200 (*Ceriodaphnia*)[a]; 96 h LC_{50} 300 (Bluegill sunfish)[a]	nd	nd	nd	nd
Titanium tetrachloride	nd	nd	nd	nd	nd
Carbon dioxide	19,000 LOEC (salmon, fresh water; 4.3% mortality)[a]	nd	nd	nd	nd
Carbon monoxide	nd	nd	nd	nd	nd
Magnesium chloride	48 h LC_{50} 1,330 (*Daphnia*)[a]; 2,840 (fathead minnow)[a]	nd	4000 mg/kg in leaf tissue (lodgepole pine)[a]	nd	nd
Aluminum chloride	48 h LC_{50} 3.99 (*Ceriodaphnia*)[a]; 105 (rainbow trout)[a]	14 day EC_{50} 359, soil pH 4.2–4.9 (earthworm)[a]	6 day LOEC 1.0 mM (mung bean)[a]	nd	nd
Magnesium oxide	nd	nd	nd	nd	nd
Aluminum oxide	48 h LC_{50} 420 (*Ceriodaphnia*)[a]	nd	18 day NOEL 4000 mg/L, plant (*Arabidopsis*)[a]	nd	nd
Carbon	nd	nd	nd	nd	nd
Lithium chloride	96 h LC_{50} 158 (rainbow trout); 26 day LC_{50} 6.4 (fathead minnow embryo)[a]	7 week 90% mortality 5 mmol/kg (earthworm)[a]	LOAEL 2 mg/kg in soil (sweet orange)[a]	nd	nd

table continued

Table A.4. Ecotoxicology Data for Reaction Products (continued)

Compound	Aquatic (mg/L)	Soil Invertebrate (mg/kg)	Terrestrial Plants	Mammals	Birds
Lithium oxide	nd	nd	nd	nd	nd
Ammonium chloride	48 h LC_{50} 101 (*Daphnia*)[a]; 96 h LC_{50} 96.2 (fathead minnow)[a]	14 day LC_{50} 160 (earthworm, soil)[a]	nd	nd	nd
Water	n/a	n/a	n/a	n/a	n/a
Nitrogen gas	n/a	n/a	n/a	n/a	n/a

[a]USEPA, 2013a.

DISTRIBUTION LIST

The following individuals and organizations were provided with one Adobe portable document format (pdf) electronic version of this report:

U.S. Army Edgewood Chemical Biological Center (ECBC)
Molecular Toxicology Branch
RDCB-DRT-M
ATTN: Simini, M.
Kristovich, R.
Checkai, R.
Haley, M.
Kuperman, R.

ECBC Analytical Toxicology Branch
RDCB-DRT-T
ATTN: Chester, N.

ECBC Operational Toxicology Branch
RDCB-DRT-O
ATTN: Whalley, C.

ECBC Smoke and Target Defeat Branch
RDCB-DRT-S
ATTN: Bickford, L.
Wise, D.

ECBC Obscuration & Nonlethal Engineering Branch
RDCB-DRT-N
ATTN: Lyons, T.
Pan, C.

ECBC Pyrotechnics Branch
RDCB-DRT-P
ATTN: Bonavito, N.

ECBC Senior Project Leader for RI&A
RDCB-DEJ-P
ATTN: Domanico, J.

U.S. Army RDECOM-RDEC
Pyrotechnics Technology and Prototyping Division
RDAR-MEE-T
ATTN: Shaw, A.

G-3 History Office
U.S. Army RDECOM
ATTN: Smart, J.

ECBC Technical Library
RDCB-DRB-BL
ATTN: Foppiano, S.
Stein, J.

Office of the Chief Counsel
AMSRD-CC
ATTN: Upchurch, V.

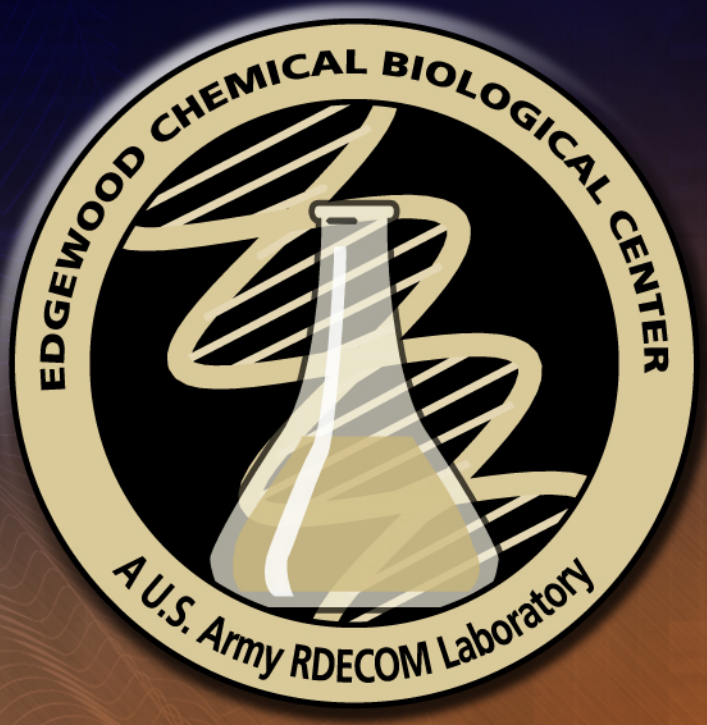
Defense Technical Information Center
ATTN: DTIC OA

ECBC Rock Island
RDCB-DES
ATTN: Lee, K.
RDCB-DEM
ATTN: Grodecki, J.

Defense Threat Reduction Agency
J9-CBS
ATTN: Graziano, A.

Department of Homeland Security
RDCB-PI-CSAC
ATTN: Negron, A.
DHS-S&T-RDP-CSAC
ATTN: Strang, P.

U.S. Army Public Health Center
MCHB-PH-TOX
ATTN: Johnson, M.
Adams, V.
Eck, W.


EDGEWOOD CHEMICAL BIOLOGICAL CENTER
A U.S. Army RDECOM Laboratory