DEPARTMENT OF THE ARMY
US ARMY CENTER FOR HEALTH PROMOTION AND PREVENTIVE MEDICINE
5158 BLACKHAWK ROAD
ABERDEEN PROVING GROUND MD 21010-5403

MCHB-TS-THE

29 October 2009

MEMORANDUM FOR Environmental Acquisition and Logistics Sustainment Program (AMSRD-MSF\Mr. Erik Hangeland), US Army Research, Development and Engineering Command, Aberdeen Proving Ground, MD 21010-5424

SUBJECT: Environmental Health Assessment for Work Unit PYRO 05-01, Toxicology Report No. 87-XE-074Z-09B, Environmentally Benign Black Smoke Formulations in Pyrotechnics

1. Five copies of the subject report with Executive Summary are enclosed.

2. Please contact us if this report or any of our services did not meet your expectations.

3. The US Army Center for Health Promotion and Preventive Medicine point of contact is Dr. Valerie H. Adams, Directorate of Toxicology, Health Effects Research Program. She may be contacted at DSN 584-5063 or commercial (410) 436-5063.

FOR THE COMMANDER:

Encl

GLENN J. LEACH
Acting Director, Toxicology

U.S. Army Center for Health Promotion and Preventive Medicine

ENVIRONMENTAL HEALTH ASSESSMENT
FOR WORK UNIT PYRO 05-01
TOXICOLOGY REPORT NO. 87-XE-074Z-09B
ENVIRONMENTALLY BENIGN BLACK SMOKE
FORMULATIONS IN PYROTECHNICS
AUGUST 2009

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Readiness Thru Health

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14. ABSTRACT

This Environmental Health Assessment is part of an ongoing effort to assess the human health and environmental impact of items of Army materiel during the development process. The objective of the program is to reduce the human health and environmental impact of newly developed Army end items with concomitant reductions in overall costs, without sacrificing performance. The naphthalene and perchlorate present in the current TITAN system have resulted in training restrictions, including the cessation of their use at ranges located in California. The benign Black Smoke formulation replaces naphthalene and perchlorate and has been evaluated in light of the current environmental laws and policies. The two dyes, Solvent Green 3 and Disperse Red 9, are of moderate concern due to possible inhalation toxicity. The remaining substances in the formulation are of negligible concern because they exhibit low toxicity, are combusted during use, or are used in small quantities. We recommend that the development of the benign Black Smoke formulation be continued.

15. SUBJECT TERMS
Environmental Health Assessment, TITAN, Black Smoke, Solvent Green 3, Disperse Red 9

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EXECUTIVE SUMMARY
ENVIRONMENTAL HEALTH ASSESSMENT FOR WORK UNIT PYRO 05-01
TOXICOLOGY REPORT NO. 87-XE-074Z-09B
ENVIRONMENTALLY BENIGN BLACK SMOKE FORMULATIONS IN PYROTECHNICS
AUGUST 2009

1. PURPOSE.

a. Research, development, testing, training, and use of substances potentially less hazardous to human health and the environment is vital to the readiness of the U.S. Army. Safeguarding the health of Soldiers, civilians, and the environment requires an assessment of alternatives before they are fielded. Continuous assessments begun early in the Research, Development, Testing and Evaluation (RDT&E) process can save significant time and effort during RDT&E, as well as over the life cycle of the items developed. Residues of pyrotechnics, propellants, explosives and incendiaries that cost the U.S. Army billions of dollars and were part of mission essential activities have been found in soil, air, surface and ground water samples, creating environmental problems and interfering with training activities.

b. The Army Environmental Quality Technology (EQT) Ordnance Environmental Program (OEP) is dedicated to finding replacements for substances causing environmental and/or occupational risks to health. As part of this program, each work unit is evaluated for environmental and occupational health impacts. The purpose of this work unit is to develop and prove-out an environmentally benign Black Smoke formulation that is also more conducive to human health. Previous black smoke formulations contained substances that presented environmental contamination concerns. One example is naphthalene, a compound that is under U.S. Environmental Protection Agency review and likely to be more strictly regulated. Continued inclusion of identified or emerging environmental contaminants in these formulations risks operational readiness and range sustainment. Black Smoke is used by the military in training and tactical situations resulting in potential exposures to the individual chemical components, as well as the combustion or waste products. Although the primary concern are environmental releases of compounds such as naphthalene and aromatic combustion products, exposures to both Soldiers and civilians could occur during manufacture, use, or demilitarization of these munitions.

2. CONCLUSIONS. An assessment was conducted of possible environmental quality and health effects impacts of a new Black Smoke formulation. Based upon this assessment, the proposed Black Smoke formulation reduces the impact of these munitions on both the environment and human health.

3. RECOMMENDATION. The current Black Smoke formulation should continue in development. Testing of the final formulation combustion products in *in vitro* systems for potential toxicity resulting from the combustion process is recommended.

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TOXICOLOGY REPORT NO. 87-XE-074Z-09B
ENVIRONMENTALLY BENIGN BLACK SMOKE
FORMULATIONS IN PYROTECHNICS

1. REFERENCES. See Appendix A for a listing of references used in this report.

2. PURPOSE. To provide environmental and occupational health information and program recommendations for new or replacement compounds in specific formulations in weapon systems or platforms used in explosives, propellants, and pyrotechnics. This information is necessary for work unit program assessment.

3. AUTHORITY. This Environmental Health Assessment addresses, in part, the environmental safety and occupational health (ESOH) requirements outlined in Army Regulations (AR) 200-1, (2007), 40-5 (2007), and 70-1 (2003); Department of Defense (DOD) Instruction 4715.4 (1996); and Army Environmental Research and Technology Assessment (AERTA) Requirement PP-3-02-04, *Compliant Ordnance Lifecycle for Readiness of the Transformation and Objective Forces*. It was performed as part of the on-going effort by the U.S. Army Environmental Quality Technology (EQT), Ordnance Environmental Program, Pollution Prevention Team to produce weapons systems that allow for greater range flexibility, ensure the health of Soldiers and do not damage the environment. This program is under the direction of the U.S. Army Research, Development and Engineering Command (USARDECOM) Environmental Acquisition Logistics & Sustainment Program (EALSP; Mr. Erik Hangeland) and EQT Pollution Prevention (Dr. John Beatty).

4. BACKGROUND.

a. Current regulations require assessment of human health and environmental effects arising from exposure to substances in soil, surface water, and ground water. When applied after an item has been fielded, these assessments can reveal the existence of adverse environmental and human health effects that must be addressed, often at substantial cost. It is more efficient to begin the evaluation of exposure, effects, and environmental transport of military-related compounds/substances early in the research, development, testing and evaluation (RDT&E) process in order to avoid unnecessary costs, conserve physical resources, and sustain the health of those potentially exposed. Moreover, such an evaluation may assist in preventing unwanted environmental consequences of use thereby assisting in range flexibility and lower restoration costs.

b. In an effort to support this preventive approach, the U.S. Army Center for Health Promotion and Preventive Medicine (USACHPPM) has been tasked with creation of a phased process to reduce adverse ESOH effects impacting readiness, training, and development costs. This is an on-going effort, and this report represents the status of information available as of the date of

publication. Summary interpretations of the preliminary information search results for this work unit were provided to the sponsor during the development effort.

c. Black smoke is used primarily in a training environment, and has two applications. The first application is as a marking device for direct-fire weapons; black smoke simulates fuel burning and indicates that a target has been successfully engaged. In this first application, there is little chance of human exposure to the smoke plume. A more recent application involves using Black Smoke to simulate an Improvised Explosive Device (IED). In this second application, troop exposure is more likely, as the smoke will be discharged in their immediate vicinity.

5. STATEMENT OF PROBLEM.

a. Current TITAN systems rely on the partial combustion of polyaromatic hydrocarbons (PAHs) such as naphthalene to produce black smoke. Naphthalene has come under scrutiny given recent toxicological data regarding the risk of cancer from exposure. Currently, regulatory agencies such as the U.S. Environmental Protection Agency (USEPA), California Environmental Protection Agency (CalEPA), and others, are reevaluating the environmental standards for naphthalene, and the lowering of the standard is under consideration. Likewise, many PAHs have been linked to increased risk of cancer to exposed individuals and may face similar review. By reducing environmental compliance constraints, a safer, more environmentally benign formulation can improve life-cycle cost effectiveness.

b. Previous Black Smoke composition(s) contained over 50 percent potassium perchlorate, which has numerous environmental and human health regulatory concerns, as well as dyes for which there are health concerns.

c. Previous applications of this system utilized black smoke as an indicator of target hit from training exercises with armed helicopters (force-on-target). In this application, the extent and probability of Soldier exposures were low. Subsequently, the TITAN system has been increasingly used in force-on-force training for improvised explosive device detection and training. This recent application has led to a much greater probability and extent for Soldier exposures that should be evaluated.

d. Given these relatively recent developments, a focus of the Ordnance Environmental Program has been the development and proving of a safe, environmentally benign Black Smoke and igniter formulation. The Armament Research, Development and Engineering Center (ARDEC) has been developing new formulations designed to eliminate known toxic components, such as naphthalene and perchlorate salts that were present in previous formulations, while maintaining the desired performance characteristics of the smoke. Functional, effective, and quality-engineered Black Smoke formulations using environmentally viable alternatives can make positive contributions to current and future readiness by being more conducive to human health and quality of life. In this report, potential toxicity and physical properties of individual

components of proposed formulations are assessed in relation to their environmental fate and toxicology.

6. METHODS.

a. In order to determine the human health and ecological impacts of compounds employed in these formulations, it is necessary to correctly and unambiguously identify each compound and to determine its physical, chemical, and toxicological properties. The primary means of identification employed for each compound in this program is its Chemical Abstracts Service Registry Number (CAS® RN; see Table 1). While all compounds do not necessarily have a single CAS RN, the CAS RN is an unambiguous way of accessing information for chemical substances. The CAS RN is readily used as a keyword for searching online databases, and is often cross-referenced with both systematic and trivial (i.e., "common") names for chemical substances. In some cases, synonyms and trade names are also used to identify structures. (CAS® is a registered trademark of the American Chemical Society.)

Table 1. Individual Chemicals, Representative Formulation Proportions, and Functions in Pyrotechnics Benign Black Smoke Formulations

Substance/ CAS number	**Percentage Formulation**	**Mixture Component**	**Component %**	**Function/ Use**
Igniter Formulation (~2 gm/charge)				
Charcoal (as Carbon) (CAS # 7440-44-0)	>14			Igniter
Gum Arabic CAS # 9000-01-5	>4			Igniter
Potassium Nitrate CAS # 7757-79-1	>35			Oxidizer
Black Powder CAS # none	15	Potassium Nitrate	~75	Fuel & Oxidizer
		Sulfur CAS # 7704-34-9	~10	
		Charcoal	~15	
Ethyl Acetate CAS # 141-78-6	0.05-0.1			Solvent (evaporated)
Black Smoke Formulation (~26 gm/charge)				
Magnesium Carbonate CAS # 546-93-0	13.2			Coolant
Potassium Chlorate CAS # 3811-04-9	28.1			Oxidizer
Disperse Red 9 CAS # 82-38-2	12.6			Dye
Solvent Green 3 CAS # 128-80-3	24.8			Dye
Stearic Acid CAS # 57-11-4	1			Lubricant

Table 1. Individual Chemicals, Representative Formulation Proportions, and Functions in Pyrotechnics Benign Black Smoke Formulations (continued)

Substance/ CAS number	Percentage Formulation	Mixture Component	Component %	Function/ Use
Sugar (sucrose) CAS # 57-50-1	19.2			Fuel
Vinyl Acetate Alcohol Resin (VAAR) CAS # 25213-24-5 27.6% solids 13% impurities	1	Methyl Acetate CAS # 79-20-9	72	Binder/base
		Vinyl Acetate CAS # 108-05-4	23	
		Polyvinyl Alcohol CAS # 9002-89-5	5	

b. This report addresses compounds investigated as part of this work unit through the end of fiscal year (FY) 2008. Basic physical and chemical properties are usually determined by consulting primary reference sources, such as the Merck Index, when such information is available. The properties necessary to assess fate and transport in the environment (FTE) include-

(1) Molecular weight (MW).

(2) Henry's law constant (K_H).

(3) Octanol-water partition coefficient ($\log K_{OW}$).

(4) Water solubility.

(5) Boiling point (bp).

(6) Organic carbon partition coefficient ($\log K_{OC}$).

(7) Vapor pressure (vp).

c. Available information on combustion, explosion, and thermal decomposition products is also collected if available. Toxicological information needed to estimate potential human health risks includes reported toxicity effects of acute, subacute, subchronic, and chronic exposures; potential for mutagenesis and carcinogenesis; and mode(s) and mechanisms of toxicity. Toxicological information is derived directly from primary sources whenever possible.

d. Hardcopy sources used in this search included publications from the U.S .Department of Health and Human Services (DHHS), Agency for Toxic Substances and Disease Registry (ATSDR) and *The Merck Index* (O'Neil, 2006). The Chemical Propulsion Information Agency's (CPIA), *Hazards of Chemical Rockets and Propellants* (CPIA, 1984), the USEPA's, *Drinking Water Health Advisory: Munitions* (USEPA, 1992), American Conference of Governmental

Industrial Hygienists, Inc.'s (ACGIH®) *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices,* Code of Federal Regulations (CFR), the National Research Council's (NRC) *Drinking Water and Health,* were also consulted. Commercial suppliers are sometimes contacted for results of in-house research that may not appear in the open literature. (ACGIH® is a registered trademark of the American Conference of Governmental Industrial Hygienists.)

e. Online sources include the 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 (ICSC) developed by the National Institute for Occupational Safety and Health (NIOSH), and the U.S. National Library of Medicine's Toxicology Data Network (TOXNET®) that provides access to information from the National Institutes of Health and the USEPA. The TOXNET is a suite of individual databases including ChemIDplusLite® (CIDPL) and ChemIDplus® Advanced (i.e., chemical and registration numbers, and chemical identification and structure, searches respectively), Hazardous Substances Data Bank (HSDB®), Chemical Carcinogenesis Research Information System (CCRIS), Developmental and Reproductive Toxicology (DART/ETIC), Directory of Information Resources Online (DIRLINE®), Genetic Toxicology (GENE-TOX), Haz-Map (database linking chemicals, jobs and diseases), Household Products Databank (HPD) (potential health effects of chemicals in common household products), Integrated Risk Information System (IRIS), International Toxicity Estimates for Risk (ITER), Toxicology. Information Online (TOXLINE®), Toxic Release Inventory (TRI), and Lactation Database (LactMed) (database of drugs and other chemicals to which breastfeeding mothers may be exposed). The USEPA ECOTOXicology Database System (ECOTOX®) and the National Institute of Environmental Health Sciences (NIEHS) National Toxicology Program (NTP) databases were used. Primary sources are identified and retrieved using PubMed®, the Ovid® Technologies Journals, and the EBSCOhost® Research Database. (TOXNET®, ChemIDplusLite®, ChemIDplus®, DIRLINE®, TOXLINE®, PubMed®, are registered trademarks US National Library of Medicine; OVID®, is a registered trademark of Ovid Technologies, Inc.; and EBSCOhost® is a registered trademark of EBSCO Publishing).

f. Persistence, bioaccumulation, human health toxicity, and ecotoxicity were assigned to general categories of risk (e.g., low, moderate, and high) using criteria modified from Howe et al. (2006). Table 2 describes the criteria used in the categorization, though the relative proportions of each substance were also factored into the final assessment.

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

	LOW	MODERATE	HIGH
PERSISTANCE	Readily biodegrades (<28 d)	Degradation ½ life: water < 40 d soil < 120 d	Degradation ½ life: water > 40 d soil > 120 d
TRANSPORT	Water sol. < 10 mg/L Log Koc > 2.0	Water sol. 10-1000 mg/L Log Koc 2.0-1.0	Water sol. > 1000 mg/L Log Koc < 1.0

Table 2. Categorization Criteria Used in the Development of ESOH Severity (continued)

	LOW	MODERATE	
BIOACCUMULATION	log Kow < 3.0	log Kow 3.0-4.5	log Kow > 4.5
TOXICITY	No evidence of carcinogenicity/mutagenicity; Subchronic LOAEL > 200 mg/kg-d	Mixed evidence for carcinogenicity/mutagenicity (B2, 2); Subchronic LOAEL 5-200 mg/kg-d	Positive corroborative evidence for carcinogenicity/mutagenicity; LOAEL < 5 mg/kg-d
ECOTOXICITY	Acute LC(D)50 >1 mg/L or 1500 mg/kg; Subchronic EC50 > 100 µg/L or LOAEL > 100 mg/kg-d	Acute LC(D)50 1-0.1 mg/L or 1500-150 mg/kg; Subchronic EC50 100-10 µg/L or LOAEL = 10-100 mg/kg-d	Acute LC(D)50 <100 µg/L or < 150 mg/kg; Subchronic LOAEL <10 mg/kg-d

Legend:
mg/L – milligrams per liter
LOAEL – lowest-observed adverse effect level
mg/kg-d – milligrams per kilograms per day
LC(D) – lethal concentration dosage
µg/L – micrograms per liter
mg/kg – milligrams per kilograms

7. RESULTS.

a. Physical and Chemical Properties. Physical and chemical properties are summarized in Table 3. When data were not found, "nd" (no data) is inserted. In some cases the property named is not applicable ("n/a") to the substance being described. For example, if the compound is a nonvolatile solid or an inorganic salt, vapor pressure, K_{OW}, K_{OC}, and the Henry's Law constant (K_H) are typically negligible.

b. Summaries. Compiled toxicological data is collected in Table 4. Summaries of human health and environmental toxicology for each of the formula components are presented in Tables 5 and 6, respectively. Each characterization is generally based on the criteria set forth in Table 2. The final risk characterization also incorporates assessment of the uncertainty associated with available data, the amount of each compound present in the formulation, and the nature of potential exposure associated with use of the end item.

c. Compound Characterizations.

(1) Charcoal (as Carbon).

(a) Acute Oral. The LD_{50} (or lethal dose, 50%) for rats orally exposed to carbon black has been determined to be >15,400 mg/kg and >3000mg/kg in rabbits (RTECS, 2006).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. Female Sprague-Dawley rats and female CF1 mice treated with 1,2-dimethylhydrazine to induce adenocarcinomas of the colon were fed carbon black at 2.05 grams per kilogram (g/kg) for 52 weeks (Pence and Buddingh, 1985). No differences in tumor incidences were seen in rats or mice. Although exact amounts were not reported, no effects of a diet of 10 percent carbon black in mice for 72 weeks were observed (Nau et al., 1976).

(d) Chronic Oral. No differences in tumor incidences were observed in a two-year feeding study with 2.05 g/kg carbon black in female Sprague-Dawley rats and female CF1 mice (Pence and Buddingh, 1985).

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. Rhesus monkeys that were exposed at 1.5 milligrams per cubic feet (mg/ft^3) for 160 days did not have any impairment in pulmonary function (Nau et al., 1976). However, these monkeys did have accumulations of carbon black particles in the lymphatics surrounding the bronchiolar areas and were observed to have experienced destruction of the alveolar walls in the bronchioles and parenchyma surrounding the pulmonary veins.

(h) Chronic Inhalation.

i. Hamsters exposed to 3 mg/ft^3 black carbon for 172 days did not have any observable differences in pathological changes to the larynx, trachea, hypopharynx, or cervical esophagus compared to controls (Nau et al., 1976). Exposure to 1.5 mg/ft^3 did, however, result in edema in the subepithelial area of the thyroaryntenoid fold and retention of amorphous eosinophilic material in the subglotic glands. In the same study, Rhesus monkeys that were exposed to 1.5 mg/ft^3 for 160 days did not have any impairment in pulmonary function but did have accumulations of carbon black particles in the lymphatics surrounding the bronchiolar areas and were observed to have experienced destruction of the alveolar walls in the bronchioles and parenchyma surrounding the pulmonary veins (Nau et al., 1976).

ii. Carbon black is considered to be a non-specific irritant with toxic effects similar to other insoluble particulates (USEPA, 2005b). Due to the concern over the potential for cancer from longer term exposures, few toxicity data exist for acute inhalational exposure to carbon black (Heinrich et al., 1994; Driscoll et al., 1996). A description of the current views on the mechanism of inhalational carcinogenicity is discussed in the carcinogenesis section.

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. No data found.

(l) Carcinogenicity. A recent review by Valberg et al. (2006) reassesses the IARC's 1996 reclassification of carbon black from group 3 to group 2B (Valberg et al., 2006). The elucidated mechanism for carcinogenicity reveals that the particulate exposures result in macrophage activation of various signaling pathways that amplify inflammation (IARC, 1996). Mutations and fibrosis result from the chronic state of inflammation that help to cause metaplastic changes and lung tumors (Knappen et al., 2004). The carcinogenic properties are, therefore, similar to any poorly soluble particle (i.e., toxicity results from particle overload more than the molecules' chemistries). In light of the new mechanistic data for carbon black's potential carcinogenicity, it has been determined that there is inadequate evidence of cancer risk in humans and limited evidence in experimental animals (Valberg et al., 2006).

(m) Ecotoxicology. Ecotoxicity data were available for the common carp (*Cyprinus carpio*) using activated charcoal. Activated charcoal is used in many aquatic filtering systems; however, effects to the fry in a slurry had not yet been tested. No adverse effects were found (Kaviraj and Das, 1995). An inhalation study conducted with carbon fibers using northern bobwhite (*Colinus virginianus*) was found not to result in adverse effects except at high concentrations (Personal communication, C. Driver). The ECOSAR-predicted endpoints from the USEPA's assessment for carbon black are presented in Table 3 (USEPA, 2005b).

Table 3. ECOSAR-Predicted Endpoints for Carbon Black

Organism	Duration	End Pt.	Predicted mg/L
Fish	14-day	LC_{50}	249
Fish	96-hour	LC_{50}	167
Daphnid	48-hour	LC_{50}	164
Green Algae	96-hour	EC_{50}	96
Fish	30-day	ChV*	17.6
Daphnid	16-day	EC_{50}	4.9
Green Algae	96-hour	ChV	4.7
Fish	96-hour	LC_{50}	21.7
Mysid Shrimp	96-hour	LC_{50}	115
Earthworm	14-day	LC_{50}	235 (dry wt soil)

Legend:
LC_{50} – lethal concentration (50%)
EC – exposure concentration (50%)
ChV = chronic value

(2) Gum Arabic (Acacia). The U.S. Food and Drug Administration (FDA) has determined that gum arabic is generally recognized as safe and appropriate for use as a food additive (CFR, 2001). Gum Arabic is also used in pharmaceutical and cosmetic applications as an emulsifier and binding agent (HSDB, 2009c).

(a) Acute Oral. The NTP reports LD_{50}s to be 16 g/kg in rats and 80 g/kg in rabbits (Sax, 2001).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. Wistar rats were exposed to gum arabic in feed for 13 weeks (Anderson et al., 1982). A no-observed adverse effects level (NOAEL) was determined to be 5 g/kg-day, and the LOAEL was found to be 14 g/kg-day, indicated by significantly reduced growth rates and kidney weights in males.

(d) Chronic Oral. No data found.

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development.

i. Yegnanarayan and Joglekar performed a number of experiments that exposed male and female rats to 4 percent gum acacia though oral gavage (Yegnanarayan and Joglekar, 1978). No effects on insemination, duration of estrus cycle, litter size, and fetal resorption were observed.

ii. In a different rat study, the teratogenic potential of gum arabic was tested in male and female Osborne-Mendel rats during premating, mating, and throughout gestation. Rats exposed to as high as 15 percent gum arabic in feed (10,647 mg/kg body weight/day) had no dose-related teratogenic effects.

iii. In a study performed at the Food and Drug Research Laboratories (FDRL), female CD-1 mice received oral doses of 16, 75, 350, and 1600 mg/kg gum arabic on days 6 through 15 of gestation (FDRL, 1972). Pups were removed by caesarian section on day 17. No significant differences in nidation (implantation in the uterus), maternal or fetal survival, or abnormalities present in fetal soft or skeletal tissues were observed.

(k) Mutagenicity. Gum Arabic was not found to be mutagenic in the Ames assay either with or without S-9 activation (CCRIS, 2009b).

(l) Carcinogenicity. Gum arabic was not found to be carcinogenic for F344 rats or B6C3F1 mice of either sex (NTP, 1982).

(m) Ecotoxicology. No data found.

(3) Potassium Nitrate. For most alkaline nitrate salts, it is the nitrate ion and not the cation (e.g., sodium, potassium, calcium) that is considered to be the toxicant. Sodium and potassium nitrate are often considered together or viewed as interchangable in toxicity assessments. 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 are naturally exposed to the precursors as a part of a normal diet. The average Western diet contains 1-2 millimols (mmol) nitrate/person/day (Hotchkiss et al., 1992).

(a) Acute Oral. The LD_{50} values for rats and rabbits have been determined to be 3750 mg/kg and 1901 mg/kg respectively, and the estimated minimum lethal dose for cattle and sheep is 1000 mg/kg (Sax and Lewis, 1989).

(b) Subacute Oral. No systematic toxicology studies were found; however, there are considerable data on the toxicity of feed containing high nitrate levels (>1% by weight) to livestock (sheep, cows, horses, and so forth) (HSDB, 2009f).

(c) Subchronic Oral. In a study by Til et al., 6-week-old Wistar rats were given 100, 300, 1000, and 3000 mg potassium nitrite/L in drinking water for 90 days (Til et al., 1988). 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.

(d) Chronic Oral. Female guinea pigs were given 0, 300, 2500, 10,000, or 30,000 parts per million (ppm) 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 in animals in the 30,000 ppm dose group, which were approximated to have received an equivalent dose of 1130 mg/kg nitrate. One female in this dose group died with four mummified fetuses in utero. The fetal deaths were attributed to hypoxia caused by maternal methemoglobinemia.

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development. Female CD-1 mice were administered potassium nitrate for 10 days; at the highest dose of 400 mg/kg, no significant differences in survival, number of pregnancies, number of abortions, resorptions, dead fetuses or average fetal weight were observed (HSDB, 2009f).

(k) Mutagenicity. As reported in the HSDB, potassium nitrate was found to be negative in the Ames assay under aerobic conditions both with and without metabolic activation. It was also found to be negative in a Chinese hamster fibroblast assay (HSDB, 2009f).

(l) Carcinogenicity. 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, 1991b). The possible carcinogenicity of nitrate depends on the conversion of nitrate to nitrite and the reaction of nitrite with secondary amines, amides, and carbamates to form N-nitroso compounds that are carcinogenic (Bouchard et al., 1992). Potassium nitrate has been reported to cause mutagenic effects in various genetic toxicity tests (Ishidate and Odashima, 1977).

(m) Ecotoxicology. The LC_{50} for a 28-day exposure to annelid worms averages 2230 µg/L (Reish, 1970). The 24-, 48-, and 96-hour LC_{50}'s for adult female mosquitofish (*Gambusia affinis*) have been determined as 58,500; 31,100; and 22,500 µg/L (Wallen et al., 1957). Other data collected from *Daphnia*, algae, and many fish species suggest potassium nitrate toxicity is relatively low (USEPA, 2007b).

(4) Sulfur. In solid form, sulfur is insoluble in water and, thus, cannot be transported downward to the ground water table. If molten sulfur is spilled onto soil, it will solidify prior to any significant movement into the soil. The WHO lists sulfur as a technical grade active ingredient of pesticides unlikely to present acute hazard in normal use. Inhalation of high concentrations of sulfur may have harmful effects on the skin, eyes, and respiratory system. In humans, exposure to airborne sulfur may result in ulceration of the skin, conjunctivitis, inflammation of the nasal mucosa, shortness-of-breath, asthma, and tracheobronchitis (Ellenhorn and Barceloux, 1988). When burned in air, sulfur will produce the toxic oxides sulfur dioxide (CAS RN: 7746-09-5) and sulfur trioxide (CAS RN: 7746-11-9). When exposed to water, these gases will react to produce sulfurous and sulfuric acids, respectively. The primary routes of exposure to sulfur dioxide and sulfur trioxide are through inhalation of the gas or vapor particles of the acids.

(a) Acute Oral. Sulfur is reported to have an oral LD_{50} >5000 mg/kg in rats (Doull and Cross, 1977). No deaths were observed when rabbits were fed 98 percent sulfur at a single dose of 2000 mg/kg. The only adverse effect noted from acute oral exposure to sulfur is a mild laxative effect (USEPA, 1982). When given orally, such as the historical practice of a "spring cleansing"

of sulfur and molasses, sulfur is partially converted to sulfide by bacteria in the digestive tract, and is sufficiently stimulating to exert a cathartic effect (Harvey, 1975).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. A repeat-dose study in rabbits compared the effects of intravenous and oral dosing. Three rabbits received 3-5, 10 and 20 mg/kg of colloidal sulfur per day orally. Autopsies did not reveal pathological changes in any of the organs (Greengard and Woodley, 1940).

(d) Chronic Oral.

i. Epidemiological studies with mine workers who were exposed to sulfur dust and sulfur dioxide (SO_2) during their lifetimes revealed ocular disturbances as one of the principal toxicity signs. Chronic bronchitis was generally found in those individuals as well as chronic sinusal effects and respiratory disturbances (USEPA, 1991a).

ii. Groups of rats (4 per dose and 2 controls) were fed doses of 2, 5, 10 or 15 mg per day (15 mg-day/0.3kg $\approx$ 50mg/kg-day [NOAEL] at the higher dose level) colloidal sulfur in the form of a pill for 100 days. Treated rats grew normally and no abnormalities were found following gross necropsy. At maturity, the rats were mated to evaluate effects on reproductive capacity. The control and sulfur-fed rats gave birth to healthy litters which they nursed successfully (Greengard and Woodley, 1940).

(e) Acute Inhalation.

i. The acute inhalation LC_{50} for 98 percent sulfur in rats was found to be >2.56 mg/L and >5.74 mg/L for 80 percent sulfur (USEPA, 1982; USEPA, 1988). The NIOSH has established a short-term exposure limit (STEL) of 5 ppm (13 mg/m^3) for SO_2; the immediately dangerous to life or health (IDLH) level is 100 ppm. The NIOSH recommends use of a respirator at all levels of SO_2 exposure (NIOSH, 2009). Because of the extremely irritating nature of SO_2 and SO_3 gases, encounters with them tend to be self-limiting (Steuven et al., 1993).

ii. The approximate LC_{50} for a 30-minute exposure of Swiss albino mice to sulfur dioxide is estimated to be 3000 ppm (Hilado and Machado, 1977). Groups of rats exposed to levels of sulfur dioxide 925 ppm and higher experienced some degree of mortality. The lower limit of toxicity is somewhere between 590 ppm (NOAEL, 0 fatalities/8 animals) and 925 ppm (unbounded LOAEL, 3 fatalities/8 animals) (Cohen et al., 1973).

iii. Approximate LC_{50} values for male and female rats exposed to fuming sulfuric acid gas were and 420 and 347 ppm, respectively (Vernot et al., 1977). The lowest concentration of

sulfuric acid aerosols that resulted in the death of rats was 383 milligrams per cubic meter (mg/m^3) (Treon et al., 1950).

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. Nasopharyngitis and lipid peroxidation of lung tissue were observed in guinea pigs exposed to 10 ppm sulfur dioxide for 1 hour/day for 30 days (Haider, 1985). Mild bronchitic lesions were seen in 72 hamsters exposed to 650 ppm sulfur dioxide for 4 hours/day, 5 days/week, for 19-74 days (Goldring and Park, 1970). Subchronic inhalation exposure to sulfur in rats showed body weight depression, decreased blood sulfhydryl contents, decreased serum peroxidase levels, and increased serum albumin (USEPA, 1991a).

(h) Chronic Inhalation. The NIOSH time-weighted average (TWA) exposure limit is 2 ppm (5 mg/m^3) for SO_2. The corresponding Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL) is 5 ppm (13 mg/m^3). For sulfur trioxide (as sulfuric acid), the NIOSH TWA Recommended Exposure Limit (REL) and OSHA PEL are both 1 mg/m^3 (NIOSH, 2009).

(i) Dermal. LD_{50} for 98 percent sulfur from acute dermal rabbit study is >2000 mg/kg (Baker, 1976).

(j) Reproduction and Development. No data found.

(k) Mutagenicity. Assays of clastogenic effects in humans following occupational exposure to SO_2 and sulfur trioxide were largely negative (ATSDR, 1998a; ATSDR, 1998b).

(l) Carcinogenicity. There are no known risks of carcinogenic or oncogenic effects associated with the use of sulfur (USEPA, 1991a). There is no definitive evidence for an increased cancer potential from SO_2 or sulfur trioxide in humans (ATSDR, 1998a; ATSDR, 1998b).

(m) Ecotoxicology.

i. The 48-hour LC_{50} for *Daphnia magna* and the 96-hour LC_{50} for *Mysidopsis bahia* (shrimp) was reported to be > 5,000 and 736 ppm respectively in a study using 90 percent sulfur. A beneficial insect study on *Apis mellifera* (honey bee) demonstrated that a 98 percent dust and a 92 percent wettable powder is low in toxicity through contact and ingestion (Borthwich and Stanley, 1982).

ii. The 96-hour LC_{50} values for bluegill sunfish and rainbow trout are >180 ppm using a 99.5 percent sulfur dust formulation (USEPA, 1982). Exposing goldfish to 16,000 ppm sulfur for 5 hours in turbid water conditions resulted in 100 percent mortality; exposure to 1600 ppm in tap water for 3.5-5.25 hours also proved fatal (HSDB, 2008a).

iii. An 8-day oral LD_{50} for bobwhite quail was reported to be >5620 ppm in a study using 95 percent sulfur wettable powder formulation (USEPA, 1991a). Grützmacher and co-workers (Grützmacher et al., 2004) found the sulfur fungicide Kumulus® DF to eliminate parasitism (i.e. eliminated reproduction) by the hymenoptera egg parasitoid *Trichogramma cacoeciae* used in biological pest control, at both 600 g/l00 L and 2.40 micrograms per square centimeter ($\mu g/cm^2$). (Kumulus® is a registered trademark of BASF AG.)

(5) Ethyl Acetate. Ethyl acetate is used in foods as an artificial fruit essence, as a solvent, as a carminative antispasmodic. In the proposed formulation,it is used in very small quantities and allowed to evaporate from the preparation.

(a) Acute Oral. The LD_{50} values for rats, mice, rabbits, and guinea pigs were reported to be 5620, 4100, 4935, and 5500 mg/kg, respectively (NTP, 2009).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. Rats were gavaged daily with 0, 300, 900, or 3600 mg/kg ethyl acetate for 90 days (USEPA, 1987). The LOAEL was determined to be 3600 mg/kg based upon significant loss of weight, appetite, and mortality in males; treated females were not different from controls at this dose. The NOAEL was reported as 900 mg/kg.

(d) Chronic Oral. No data found.

(e) Acute Inhalation. The LC_{50} values for rats, mice, cats, and guinea pigs were reported as 1,600 ppm/8 hours, 45.0 gm/m^3/2 hours, 61.0 gm/m^3/hour, and 77.0 mg/m^3/hour, respectively (NTP, 2009). Bowen et al. (Bowen and Balster, 1997) exposed male CFW mice to up to 2000 ppm ethyl acetate for 20 minutes and determined the LOAEL to be 2000 ppm based on decreases in locomotor activity. Changes in posture, decreased arousal and rearing, increased tonic and clonic movements, and decreased mobility and righting reflexes were also observed.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. Christoph et al. (Christoph et al., 2003) exposed rats to 0, 350, 750 or 1500 ppm of ethyl acetate for 6 hours/day, 5 days/week for 13 weeks. Diminished behavioral responses to unexpected auditory stimuli, and reduced body weight, body weight gain, feed consumption, and feed efficiency were experienced at 750 and 1500 ppm in both sexes. Reductions in body weight gain and feed efficiency were observed in male rats exposed to 350 ppm. Reduced motor activity was observed in the females exposed to 1500 ppm ethyl acetate. The authors determined a LOEL of 350 ppm for systemic toxicity based on decreased body weight gain in male rats, and a LOEL of 1500 ppm for neurotoxicity based on transient reduction in motor activity in female rats.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. Ethyl acetate was negative for mutagenicity in Ames *Salmonella typhimurium* assay with and without metabolic activation (Zeiger et al., 1992).

(l) Carcinogenicity. Ethyl acetate was negative in the CHO-LB cell line and bone marrow polychromatic erythrocyte test systems (Hayashi et al., 1988; Loveday et al., 1990).

(m) Ecotoxicology.

i. The 48-hour LC_{50} for the Mexican axolotl (*Ambystoma laevis*) and the clawed toad (*Xenopus laevis*) were reported to be 150,000 and 180,000 µg/L, respectively (Slooff and Baerselman, 1980).

ii. The 48-hour LC_{50} for the leech (*Erpobdella octoculata*), hydra (*Hydra oligactis*), and aquatic sowbug (*Ascellus aquaticus*) were determined to be 1,200,000; 1,350,000; and 1,600,000 µg/L, respectively (Slooff, 1983).

iii. The 48-, 72-, and 96-hour LC_{50} for the Indian catfish (*Heteropneustes fossilis*) were determined to be 275,000; 225,000; and 212,500 µg/L, respectively (Gupta and Srivastava, 1982), and the 48-hour LC_{50} for the fathead minnow (*Pimephales promelas*) is 270,000 µg/L (Slooff and Canton, 1983).

iv. The 48-hour LC_{50} for the first and third instar mosquito (*Aedes aegypti*) was found to be 270,000 µg/L (Slooff, 1983).

v. Opdyke (Opdyke, 1979) injected yolks of chicken eggs with 9, 22.5, 45, or 90 mg ethyl acetate/egg. Hatchability was observed to be 85, 50, 35, and 15%, respectively.

(6) Magnesium Carbonate. Magnesium carbonate is listed as a direct food substance affirmed by the FDA as Generally Recognized As Safe (GRAS) when used in food and is deemed to be used "with no limitation other than current good manufacturing practice" (CFR, 2003). As a food additive, magnesium carbonate is GRAS and is included in the list of antacid products for over-the-counter human use (HSDB, 2009d). Magnesium carbonate is not particularly toxic when inhaled, but is considered to be a nuisance particulate. The OSHA PEL for General Industry for magnesium carbonate is 15 mg/m^3 TWA, and the NIOSH REL has been determined to be 10 mg/m^3 TWA (ICSC, 1995).

(a) Acute Oral. No data found.

(b) Subacute Oral. No data found.

(c) Subchronic Oral. No data found.

(d) Chronic Oral. No data found.

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. No data found.

(l) Carcinogenicity. No data found.

(m) Ecotoxicology. No data found.

(7) Potassium Chlorate. As the chlorate ion and not the sodium or potassium ion is considered to be the toxicant, chlorate salts (either sodium or potassium) are often considered together in toxicity assessment. Additionally, sodium chlorate toxicity evaluations are sometimes substituted for potassium chlorate or "chlorate salts" are grouped together as in the reference book Clinical Toxicology of Commercial Products (Gosselin et al., 1984). Potassium chlorate is a component of matchheads and accidental poisoning by ingestion has been reported (Olson, 2007). Inhalation of potassium chlorate dust and contact with eyes or skin causes local irritation. When dosed orally in aqueous solution, chlorate is rapidly eliminated in urine.

(a) Acute Oral. The LD_{50} value for the rat was determined to be 1870 mg/kg (CIDPL, 2009d). From cases of accidental or suicidal ingestion, the probable lethal oral dose for adult humans is estimated to be between 50-500 mg/kg (or 5 to 30 g) (Gosselin et al., 1976; NRC, 1980).

(b) Subacute Oral. No data found.

(c) Subchronic Oral.

i. Oral doses from 50 to 250 mg/kg were administered daily to cats for 25 to 32 days. Although one of the common effects of oral toxicity is an increase in methemoglobin, none of the cats had any demonstrable changes in plasma levels. Upon necropsy, all cats receiving greater than 50 mg/kg showed fibrosis and atrophy of distal renal tubules (NRC, 1980).

ii. Male and female Sprague-Dawley rats were exposed to drinking water containing 3.0, 12.0, or 48.0 mmol sodium chlorate. The mean drinking water consumption varied between exposure groups from 100-200 ml/kg-day. There were no compound-related deaths; however, both males and females in the high exposure groups had significant weight loss during the 90-day exposure period. Also, in these same groups females had mild but significant decreases in the relative organ weights of adrenals, thymus, and spleen, while the relative brain weight was increased. In males, the weights of heart, kidneys, and liver were mildly decreased while the brain and testes were mildly increased. Red blood cell counts and percent hematocrit were decreased in both sexes in the high dose group. Pituitary gland (pars distalis) vacuolization and thyroid gland colloid depletion were prominent in both sexes in mid- and/or high-dose animals. A NOAEL of 0.36 mM chlorate/kg-day in males and 0.50 mM chlorate/kg-day in females were established (McCauley et al., 1995).

(d) Chronic Oral. Groups of male and female rats were provided drinking water containing 125, 1000, or 2000 mg sodium chlorate/L for two years, while male and female mice received 500, 1000, or 2000 mg sodium chlorate $NaClO_3$/L (NTP, 2005). Other groups of animals received plain tap water and served as controls. At the end of the study, tissues from more than 40 sites were examined for every animal. Male and female rats receiving sodium chlorate had higher rates of follicular cellular hypertrophy of the thyroid gland, and the groups receiving 2000 mg/L had higher rates of thyroid gland cancer, compared with the control group. Female mice exposed to sodium chlorate had a few pancreatic islet cell tumors. Based on these results, it was concluded that sodium chlorate caused some thyroid gland neoplasia in male and female rats (NTP, 2005).

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. The *in-vitro* genotoxic effects of potassium chlorate were evaluated using the alkaline comet assay and micronucleus assay on CHO cells (HSDB, 2009e). No evidence of genotoxicity was reported.

(l) Carcinogenicity.

i. Using a rat renal carcinogenesis promoter model, Kurokawa and coworkers exposed male F344 rats to either sodium chlorate or potassium chlorate 2 weeks after pretreatment with the tumor initiator EHEN (N-ethyl-N-hydroxyethylnitrosamine (Kurokawa et al., 1985). As controls, additional groups of rats were not treated with EHEN prior to chlorate exposure. Animals were exposed to 1 percent chlorate (approximately 670 mg/kg/day) in the drinking water for 25 weeks; the 1 percent chlorate dose levels were considered to be the maximum tolerated doses based on subacute (6 weeks) toxicity testing performed by the same laboratory. All rats survived the experimental period and there were no significant differences in dysplastic foci or renal cell tumors between any of the treatments and control groups. Rats treated with chlorates (with or without EHEN) were found to weigh slightly, but significantly less, than untreated controls: 7 percent less for sodium chlorate and 4.4 percent less for potassium chlorate (Kurokawa et al., 1985).

ii. In another study, groups of male and female rats were provided drinking water containing 125, 1000, or 2000 mg sodium chlorate/L for two years, while male and female mice received 500, 1000, or 2000 mg sodium chlorate/L. Other groups of animals received plain tap water and served as controls. At the end of the study, tissues from more than 40 anatomical sites were examined for every animal. Male and female rats receiving 2000 mg/L had higher rates of thyroid gland cancer, compared with the control group. Female mice exposed to sodium chlorate had a few pancreatic islet cell tumors. Based on these results, it was concluded that sodium chlorate caused some thyroid gland neoplasia in male and female rats (NTP, 2005).

iii. Khan and coworkers found that administration of sodium chlorate in conjunction with ammonium perchlorate potentiated thyroid effects (Khan et al., 2005). Effects included thyroid hypertrophy and hyperplasia, colloid depletion, and reduction in circulating thyroxine (T4) levels.

(m) Ecotoxicology. Chlorates are herbicides; thus, it is expected that plant species will be sensitive to chlorate exposure. In plants, chlorate ions are believed to interfere with nitrate metabolism. A review of the ECOTOX database reveals that brown algae are more sensitive than green and blue-green algae species (Van Wijk et al., 1998; USEPA, 2007a).

i. Fourteen day-old soybean seedlings were exposed to up to 2.0 mmol potassium chlorate with varying light levels (100, 67, 33 and 0 percent of full light which was 750 μ Einstein*/m^2/s for 24 hours (Harper, 1981). Interveinal chlorosis was evident within 24 hours following addition of 0.5 mmol, chlorate, with the severity of the symptom increasing with dose. Leaflet nitrate

* An Einstein is a measurement unit for irradiance, regardless of frequency. One Einstein is equal to one mol of photons; m^2/s = square meters per second.

reductase activity was lower following exposures to concentrations of 0.5 mmol and higher, indicating that chlorate, or some reduction product of chlorate, was detrimental to enzyme functionality. Sodium chlorate was not shown to greatly affect shoot growth in sorghum or cucumber (Sund and Nomura, 1963).

ii. Changes in population growth rates were observed by Van Wijk et al. (1998) in brown algae (*Ectocarpus variabilis*) exposed 14 days to relatively small amounts (5 microMolar) of chlorate salt. Similar changes were not seen in two species of green algae (*Pseudokirchner-iella subcapitata* and *Scenedesmus subspicatus*) to concentrations up to 144 mg/L and 3137 mg/L, respectively (USEPA, 2007a) .

iii. The concentration of sodium chlorate lethal to *Daphnia* and many fish species were reported to be greater than 3000 mg/L; however, brown trout are approximately 1000-fold more sensitive (USEPA, 2007a). Aquatic insects were also relatively resistant to chlorate salt exposures where reports of lethality (LC_{50} values) were often greater than 30 mg/L (e.g., caddisfly, mayfly; (USEPA, 2007a)).

iv. The LD_{50} was reported to be greater than 2500 mg/kg for mallard and northern bobwhite (USEPA, 2007a). The LC_{50} for the earthworm (*Lumbricus terrestis*) could not be determined for concentrations of sodium chlorate of up to 1000 mg/kg soil, though reductions in biomass may occur at lower levels (Hague and Ebing, 1983).

(8) Disperse Red 9. Disperse Red 9 (DR9, 1-methylaminoanthraquinone) is an anthracenedione dye. It is used as a coloring agent for synthetic fabrics and plastics; however, it has not been certified for use in cosmetics, food, or drugs (Davidson and Hovatter, 1987b).

(a) Acute Oral. The DR9 was tested for acute oral toxicity in dogs (Martin et al., 1983). Dogs (number of animals is indicated in parentheses) were dosed at 0 (2), 4000 (3), and 8000 (2) mg/kg body weight over an 8-24 hour period. The extended dosing period was necessary to account for the low density of the dye. Blood was collected for clinical chemistry and hematology, and the dogs were euthanized and necropsied at 14 days post treatment. Feces and urine were strongly pigmented for the first 48 hours after exposure; however, there were no changes in animal behavior, clinical values, or findings at necropsy. Martin et. al. (Martin et al., 1983) also performed metabolism studies using sheep treated with 50 mg/kg body weight DR9. Due to limitations of the study, only colored metabolites of DR9 were measured and Martin and colleagues concluded that about 40% was excreted as either unmetabolized DR9 or its glucuronide conjugate.

(b) Subacute Oral. No data found.

(c) Subchronic Oral. No data found.

(d) Chronic Oral. As part of a 35 compound carcinogenicity experiment, DR9 (500 mg/rat/dose 10 doses at 3 day intervals for 30 days) was fed to female rats followed by 9 months of observation (Griswold et al., 1968). No organ toxicity was observed upon necropsy.

(e) Acute Inhalation. The DR9 (as a constituent of a M18 smoke grenade) was tested for acute inhalation toxicity in seven mammalian species (monkey, dog, goat, swine, rabbit, rat, and guinea pig) (Owens and Ward, 1974). Each species was exposed between 10 and 240 minutes to concentrations of smoke between 2758-17,946 mg/m^3. The combined LC_{50} for all species was calculated to be 647,470 mg/min/m^3 (slope=2.96). Observations of exposed animals included signs of upper respiratory tract irritation, salivation, and gagging. Urine was reported to be dark red for 24 hours post-exposure in non-rodent species. In an earlier study, Weeks and Yevich tested several military pyrotechnic devices, one of which contained DR9 (Weeks and Yevich, 1960). Groups of 10 male rats, 10 female mice, 5 male guinea pigs, and 5 female guinea pigs were exposed to 1 or 3 pyrotechnic units in a 700 L chamber (1000 L = 1 m^3) or 4 pyrotechnic units in a 20,000-L chamber. Exposure was whole body and oral ingestion of the combusted products was not accounted for in the study. All animals exposed to the 3 units/700-L treatment died within 7 days of exposure, while only 3 of the 10 mice died in the same time span under the 1 unit/700-L treatment. No animals exposed to the 4 units/20,000-L treatment died. Symptoms of exposure are similar to the observations made by Owens (Owens and Ward, 1974). Microscopic evaluation of lung tissue revealed only alveolar edema.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. The DR9 was tested as a component of a brown smoke mixture (Marrs et al., 1984). Mice, rats, and guinea pigs were exposed for 20 weeks (1 hr/day 5 days/week; total exposures = 100) and observed for an additional 20-51 weeks. The brown smoke consisted of Solvent Yellow 33, DR9, and Solvent Green 3; animals were exposed to 0, 6.3, 18.6, or 60.7 grams per minute per cubic meter (g/min-m^3) daily dose. High dose effects included an increase in mortality for guinea pigs and an increase in the lung weight for mice and rats. Microscopic evaluation of lung sections from mice found that Solvent Green 3 was retained in the lungs, but DR9 and Solvent Yellow 33 were not. Pathological lung changes for the middle and high dose groups (mice and rats) included macrophage infiltration and alveolitis. The significant non-lung findings were fatty changes to the liver (mice-high dose), biliary hyperplasia (rats-high dose), and adrenal haemangioma (rats-high dose). The authors concluded that the general lung response represented a "foreign body" reaction and that below 6.3 g/min-m^3 daily dose little organ-directed toxicity was observed. Additionally, because the smoke tested was a mixture, any toxic effects can not be attributed solely to DR9.

(i) Dermal. In a study by Martin and coworkers, rabbits were exposed to 2 g/kg DR9 following a standard Draize protocol, no signs of toxicity or irritation were noted (Martin et al.,

1983). In a different study, Weeks and Yevich (Weeks and Yevich, 1960) applied DR9 to either the abraded or unabraded skin of guinea pigs. When the dye was tested by itself, no reaction was observed; however, when the complete mixture, including the binder (polyvinyl acetate in methylene chloride) or binder alone was tested sensitivity was observed.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. The DR9 was one of 90 anthraquinone derivatives screened for mutagenicity using the Ames test. The DR9 tested negative for mutagenicity both with and without microsomal activation (Brown and Brown, 1976). Corroborative results were obtained by Brusick and Matheson (1978).

(l) Carcinogenicity. The DR9 was tested in a battery of *in vitro* and *in vivo* assays to determine its carcinogenic activity (Brusick and Matheson, 1978). While DR9 was negative in the Ames test, it was positive in the mouse lymphoma assay in the range of 200-500 μg/mL. The DR9 was found to have greater activity under +S9 conditions. The DR9 was also positive in the unscheduled DNA synthesis assay at 50-100 μg/ml. Additionally, it was also noted that DR9 was very cytotoxic to the assay systems. *In vivo* dominant lethal assays were performed using mouse and rat. The mouse test results were negative at 200, 670, and 2000 mg/kg. Rat results were considered negative, based on the evaluation of unusual negative control values. Griswold et al. (1968) tested DR9 along with 34 other compounds for mammary carcinogenicity. The DR9 (500 mg/rat/dose, 10 doses at 3 day intervals for 30 days) was fed to 20 female rats followed by 9 months of observation (Griswold et al., 1968). For DR9, mammary tumor incidence was not higher than in control animals. One tubular adenocarcinoma of the kidney, a rare form of cancer for this strain of rat, was observed in DR9-treated rats but the significance of the finding was not addressed. Two minor (12 percent) combustion products of DR9 have been identified: 1-aminoanthraquinone (1-AA) and 2-aminoanthraquinone (2-AA) (Lundy and Eaton, 1994). The 1-AA is described as a likely carcinogen; 2-AA was studied in detail and found to be a carcinogen in male rats (2-AA dose to induce tumors in half the test animals TD_{50}=101 mg/kg-day) and in both male and female mice (TD_{50}=1190 mg/kg-day). The 2-AA was also tested in the Griswold et al study (Griswold et al., 1968). No mammary tumors were observed, however at the dose tested (100 mg/rat/dose, 10 doses at 3 day intervals for 30 days) a high proportion of the treated animals had cystic changes to the kidneys.

(m) Ecotoxicology. The transformation of DR9 in anoxic sediment was examined by Baughman and Weber (1994). They conclude that DR9 will not persist in the environmental conditions tested and determined the half-life to be <2 days. The sediment reaction products identified using the gas chromatograph/mass spectrometer (GC-MS) from the transformation of DR9 were anthraceneone/anthrone isomers with predicted half-lives of 87 days. Low water solubility and hydrophobicity (K_{oc} = 4.1; (Garrison, 1992)) suggest DR9 will not stay in the water column but be adsorbed to the soil. The potential for bioaccumulation is moderately high with a

BCF estimated to be 1000. However, photo degradation and rapid transformation in soils suggest DR9 persistence is low.

(9) Solvent Green 3. Solvent Green 3 (SG3) is an anthracenedione dye. A very extensive review of the compound, its manufacture, and environmental fate was prepared by Davidson and Hovatter (1987a), some of whose details are presented below. According to this report, 90-95 percent of the dye in a smoke grenade formulation sublimes during operation of the grenade. The remaining 5-10 percent decomposes to polynuclear aromatic materials, including polynuclear aromatic hydrocarbons, but no ring-opening reactions occur. Some of the decomposition products are reported to be 1-*p*-toluidinoanthracenedione, 1-*p*-toluidino-4-aminoanthracenedione, *p*-toluidine, 1-aminoanthracenedione, and 1-*p*-toluidineanthrone (Chin and Borer, 1983; Chin et al., 1984).

(a) Acute Oral. The LD_{50} for SG3 is reported to be 3660 mg/kg in rats (Marhold, 1972), >10,000 mg/kg in rabbits (Dacre et al., 1979; Davidson and Hovatter, 1987a), and >1000 mg/kg in dogs (Davidson and Hovatter, 1987a).

(b) Subacute Oral. No data found.

(c) Subchronic Oral.

i. In a 6-week range-finding study in rats, SG3 was administered in the diet at concentrations between 0.1 and 3.0 percent. The daily doses normalized against body weight were 119-3540 mg/kg at week 1 and 63.9-2270 mg/kg at week 6. There were no deaths or gross signs of toxicity. Some animals were found to have small thyroid glands, but no histopathological abnormalities were noted on necropsy. Gross degenerative changes in the liver were observed and confirmed by histopathological examination, which showed an increase in vacuolated cells around the hepatic central vein. No other effects were observed (Davidson and Hovatter, 1987a).

ii. In a 90-day subchronic study, two dogs were fed a diet containing 1 percent SG3 during weeks 1 and 2 (290 mg/kg-d). The concentration was increased to 2 percent during week 3 (570 and 500 mg/kg-d), and 3 percent during week 5. The weight-normalized doses fluctuated between 610 and 1400 mg/kg-d during the remainder of the experiment. There were no gross signs of toxicity, and no significant changes in body weight. Necropsy revealed only an accumulation of dye in the pelvis of the kidney, in the mucosa of the small and large intestines, in adipose tissue, and in the gall bladder. No histopathological alterations were observed (Davidson and Hovatter, 1987a).

(d) Chronic Oral. Cichowicz and Wentsel (1983) described a study in which rats and dogs were administered SG3 in their diets at concentrations of 0.25, 1.0, or 3.0 percent for 2 years (Cichowicz and Wentsel, 1983). No effects were found, even at the highest concentrations.

(e) Acute Inhalation. Short-term inhalation studies on SG3 were reported by Henderson, et al. (1985b) and Medinsky, et al. (1986). Fisher 344 rats were exposed for 60 minutes to aerosols of Solvent Yellow 33/Solvent Green 3 mixture by inhalation. The average final concentration of dye in the exposure chamber was 246 mg/m^3, and the mean particle size was 2.6 μm. The concentration of SG3 in the mixture was calculated to be 154 mg/m^3. Animals were sacrificed at times up to 72 hours to determine the amount of SG3 deposited in the lungs. A total of 360 μg was found to have been deposited in the entire respiratory tract, with 310 μg in the upper respiratory tract and 50 μg in the lungs and bronchi. Solvent Green 3 was not cleared from the lungs during the sampling period.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation.

i. Marrs, et al. (1984) exposed female mice, rats, and guinea pigs to a smoke formulation containing 19 percent Solvent Green 3, 13 percent Solvent Yellow 33, and 16 percent Disperse Red 9 for 1 hour/day, 5 days/week, for 20 weeks, or a total of 100 exposures. Three levels of exposure were employed for each group, with the low group being approximately 106 μg/L, the median group 319 μg/L, and the high group 1012-1162 μg/L. They noted that SG3 was retained in the lungs, and marked alveolar macrophages were found, especially in the rats. Several other adverse findings were noted including adenocarcinomas of the breast in the high-dose group and a dose-related incidence of biliary hyperplasia in females of all three species. Fatty change was noted in the livers of the mice. A large number of high-dose guinea pigs died during the exposure period, resulting in cessation of the testing for that group after only 16 exposures; guinea pigs at lower doses were unaffected. All test species showed reduction in growth during the exposure period.

ii. Several publications from a single group reported on subchronic inhalation studies conducted with rats (Henderson et al., 1985a; Henderson et al., 1985b; Sun et al., 1987). Solvent Green 3 was retained in the lungs of rats exposed repeatedly to aerosols of Solvent Yellow 33/Solvent Green 3 (30:70) mixture at concentrations of 11, 49, or 210 mg/m^3 for 6 hours/day, 5 days/week for 4 weeks, and at 1.1, 10.2, or 101 mg/m^3 6 hours/day, 5 days/week for 13 weeks (90 days). Three rats from each group were sacrificed 16 hours after the last exposure, the lungs excised and homogenized, and measurements made of the amount of SG3 present. A large fraction of the dye was retained: 14-33 percent after the 4-week exposure and 16-21 percent after the 13-week exposure. In the 4-week study, animals exposed at the highest concentration showed a 7% reduction in body weight with respect to controls. They also exhibited reduced pulmonary gas exchange efficiency, airflow obstruction, mild pulmonary inflammation, slight Type II pulmonary epithelial cell hyperplasia, and proliferation of vacuolated alveolar macrophages.

iii. In the 13-week study, animals exposed at the highest concentrations demonstrated a 9 percent loss of body weight relative to controls and had accumulations of vacuolated alveolar

macrophages in the lungs. Rats exposed to the highest dye concentrations also had indications of mild pulmonary inflammation and slight Type II pulmonary epithelial cell hyperplasia. Significant amounts of the SG3 component were detected in the lungs after any exposures, while the Solvent Yellow 33 was rapidly cleared. In order to determine the rate of clearance of SG3 from the lungs, animals exposed to the highest dose were sacrificed at various times after the last day of exposure. Clearance half-times were determined to be 277 ± 67 days for males and 289 ± 40 days for females. Because the aerosols of Solvent Yellow 33/Solvent Green 3 mixtures caused microscopic lesions in rats at the medium concentration (10 mg/m^3) but no exposure-related lesions at the low concentration it was determined that the NOAEL was 1 mg/m^3 (Henderson et al., 1985a).

(h) Chronic Inhalation. Although chronic inhalation studies were identified as a data gap by Davidson and Hovatter (1987a), no appropriate studies appear to have been done in the intervening 21 years.

(i) Dermal.

i. Subchronic and chronic dermal toxicity tests revealed that 500 mg/kg of SG3 in petroleum or hydrophilic base applied to rabbit skin for 13 weeks, or single weekly applications of 1 mg of SG3 in 0.1 mL of benzene to the skin of mice for 95 weeks did not cause significant local or systemic effects. The thyroid glands in the male mice were enlarged, but histopathological examination did not show an effect (Davidson and Hovatter, 1987a).

ii. Technical grade formulations of SG3 may induce hypersensitivity. Fujii (2003) sensitized guinea pigs with 1 percent commercial grade dye, and found a dose-dependent hypersensitivity reaction on subsequent challenge. Dye purified by recrystallization failed to induce a similar response. Further investigation found that an intermediate in the synthesis of the dye, quinizarin, was the source of the induced hypersensitivity.

(j) Reproduction and Development. Solvent Green 3 is not teratogenic in rats or rabbits (Davidson and Hovatter, 1987a; USEPA, 2005a).

(k) Mutagenicity. Solvent Green 3 was negative in the Ames *Salmonella* test (Brown and Brown, 1976).

(l) Carcinogenicity. Solvent Green 3 is not carcinogenic in the Mouse Lung Tumor bioassay (Davidson and Hovatter, 1987a).

(m) Ecotoxicology.

i. Aquatic Toxicity. The USEPA conducted structure-activity analysis for concentrations from 1-200 parts per billion (ppb) in aquatic environments and found no concerns related to aquatic toxicity at these concentrations (USEPA, 2005a). At a concentration of 10 mg/L, SG3 is

reported to cause a transient reduction in the growth of *Selenastrum capricornutum* (green algae) (Davidson and Hovatter, 1987a).

ii. The hydrophobic nature of SG3 indicates that when released into aquatic systems, the dye will occur in a particulate form that may be deposited in bottom sediments (Davidson and Hovatter, 1987a).

iii. Static acute bioassays to determine the mean tolerance level (TL_{50}) of *Pimephales promelas* (fathead minnow) to SG3 have been conducted. Because of its insolubility, the dye was first dispersed as a 15 percent (by weight) solution in Reax 83-A solvent. Ten fish per group were exposed for 96 hours to 10, 18, 32, 56, or 100 mg/L. Temperature and pH were monitored during the exposure and found to be relatively constant during testing. Dissolved oxygen decreased during testing, so tanks were aerated for 5 minutes at 48 hours. Alkalinity of the water at the start of testing was determined to be 19 mg/L as calcium carbonate. The 96-hour TL_{50} was found to be >100 mg/L (Davidson and Hovatter, 1987a).

iv. Fisher, et al. (1987) studied the acute toxicity of a Solvent Yellow 33/Solvent Green 3 (30:70 ratio) mixture in eight aquatic species of fish and invertebrates (Fisher et al., 1987). Fish species tested were *Pimephales promelas* (fathead minnow), *Ictalurus punctatus* (channel catfish), *Lapomis macrochirus* (bluegill), and *Salmo gairdneri* (rainbow trout). Invertebrate species tested were *Daphnia magna* (water flea), *Gammarus pseudolimnaeus* (amphipod), *Hexagenia bilineata* (mayfly larvae), and *Paratanytarsus parthenogeneticus* (midge larvae). All species were tested at the aqueous solubility limit of the dyes at various temperatures. The SG3 component was very insoluble and was not detected at the detection limit of 0.002 mg/L of the high-performance liquid chromatography (HPLC) system employed to monitor dye concentrations in this experiment. No mortality was observed in any of the test species at the solubility limits of the dyes.

(10) Stearic Acid. Stearic acid is a naturally-occurring fatty acid of animal origin. It is used in the manufacture of soaps and cosmetics, and as a hardener in candles. Stearic acid exists as an amorphous solid or leaflet, and it has a slight odor and tastes of tallow. Because of its production and use in stearates and stearate driers, lubricants, soaps, pharmaceuticals and cosmetics, shoe and metal polishes, coatings, food packaging, suppositories, ointments, as an accelerator activator, and as a dispersing agent and softener in rubber compounds, it is expected to be released into the environment from several sources. In the ambient environment, stearic acid exists in both vapor and particulate phases, and it has an extrapolated vapor pressure of 7.2×10^{-7} millimeter of mercury (mmHg) at 25° Celsius (Daubert and Danner, 1989). In addition, a K_{oc} value of 7.2×10^{5} indicates that stearic acid is immobile in soil, and a Henry's Law constant of 4.8×10^{-7} atm-m^3/mol suggests that volatization from dry soil or water is not expected. It is poisonous by intravenous route, and is a human skin irritant. In rats, it is highly toxic when presented intravenously, and it can cause convulsions and affect the rats' seizure threshold. However, it is only slightly toxic in rats when administered orally. In humans, it is practically nontoxic (Drill and Lazer, 1977).

(a) Acute Oral. The LD_{10} for rats has been determined to be 4600 mg/kg (CIDPL).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. No data found.

(d) Chronic Oral. No data found.

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. No data found.

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. Results of Ames tests performed with a wide variety of *Salmonella* strains have been negative for stearic acid (CCIRS, 2009).

(l) Carcinogenicity. 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 and Krieger, 1992). Neither the NTP nor the IARC has a human cancer classification for stearic acid. Preliminary data indicates that stearic acid may inhibit tumor development in rats (Habib et al., 1987).

(m) Ecotoxicology. The 96-hour LC_{50} for Coho salmon (*Oncorhynchus kisutch*) was found to be 12 mg/L (Leach and Thakore, 1977).

(11) Sucrose. Sucrose, common table sugar, is listed as a direct food substance affirmed by the FDA as GRAS when used in food with good manufacturing practice.

(a) Acute Oral. The rat oral LD_{50} value is reported as 29,700 mg/kg (Sax, 1984).

(b) Subacute Oral. No data found.

(c) Subchronic Oral. No data found.

(d) Chronic Oral. No data found.

(e) Acute Inhalation. No data found.

(f) Subacute Inhalation. No data found.

(g) Subchronic Inhalation. No data found.

(h) Chronic Inhalation. Sucrose is not particularly toxic when inhaled but is considered to be a nuisance particulate. The OSHA PEL for sucrose is 5 mg/m^3 TWA, and the NIOSH REL has been determined to be 5 mg/m^3 TWA (ICSC, 2003).

(i) Dermal. No data found.

(j) Reproduction and Development. No data found.

(k) Mutagenicity. No data found.

(l) Carcinogenicity. Sucrose is not classifiable as a human carcinogen (ACGIH, 2005).

(m) Ecotoxicology. No data found

(12) Vinyl Acetate Alcohol Resin (VAAR). Vinyl Alcohol Acetate Resin is prepared by partial hydrolysis of polyvinyl acetate polymer and consists of methyl acetate, vinyl acetate monomer, polyvinyl alcohol, and impurities. Due to the polymeric nature of VAAR, the molecular weight is variable. The VAAR product is used in the formulation for Black Smoke (PYRO 05-01), as well as several other systems. The current Black Smoke formulation contains 1 percent VAAR. The percent composition for VAAR is 72 percent methyl acetate (technical grade; ~82 percent purity), 23 percent vinyl acetate, 5 percent polyvinyl alcohol, and 13 percent impurities. Toxicity data for VAAR could not be found; therefore as a substitute, toxicity data for the components of VAAR were reviewed and are summarized in the following section.

(a) Methyl Acetate (CAS RN 79-20-9). Methyl acetate is a solvent for many resins. There are data on the low toxicity of methyl acetate. From the CIDPL database, the oral LD_{50} for rat is >5 g/kg and for rabbit is 3705 mg/kg (CIDPL, 2009b). Inhalation data include rat lethal concentration low (LC_{Lo}) 32,000 ppm/4hr, mouse LC_{Lo} 34 gm/m^3/4 hour, and human toxic concentration low (TC_{Lo}) 15,000 mg/m^3 (CIDPL, 2009b). Methyl acetate is negative in the Ames mutagenicity assay both with and without S9 activation (CCRIS, 2009c). Little aquatic toxicity data is available; fathead minnow LC_{50} was reported to be between 320,000-408,000 µg/L (USEPA, 2007a).

(b) Vinyl Acetate (CAS RN 108-05-4). There is considerable toxicity data for vinyl acetate. The acute toxicity of vinyl acetate is low: oral LD_{50}'s for rat and mouse are 2900mg/kg and 1600 mg/kg, respectively (CIDPL, 2009f). The LC_{50} inhalation values are 11,400 mg/m^3/4

hour (rat), 1550 ppm/4 hour (mouse), and 2500 ppm/4 hour (rabbit) (CIDPL, 2009f). Vinyl acetate was negative in the *Salmonella typhimurium* Ames mutagenicity assay both with and without S9 activation; however, it was positive in lymphoma assays (micronucleus and sister chromatid exchange) (IARC, 1995). A dose-dependant increase in nasal lesions and tumors was observed in chronic inhalation exposure studies of rats and mice (IARC, 1995). Carcinogenicity data is confounded by evidence that vinyl acetate is rapidly metabolized in vivo to acetaldehyde (Thurman and Kauffman, 1992). Because vinyl acetate is so widely used in a variety of manufacturing processes there are established occupational exposure limits; the TWA is 10 ppm and STEL is 15 ppm (CIDPL, 2009f). The FDA has approved the use of vinyl acetate as a food starch modifier (21 CFR 172.892) and as the copolymer vinyl acetate/crotonic acid for food packaging (21 CFR 175.350).

(c) Vinyl acetate toxicity has been tested on several aquatic species. Population effects in algae species was observed at an lowest-observed effect concentration (LOEC) of 370,000 µg/L; behavioral effects in *Daphnia magna* were observed at an EC_{50} of 52,000 µg/L and Daphnia mortality EC_{50} was 330,000 µg/L (USEPA, 2007a). The toxicity to several fish species has been evaluated as well. The LC_{50} for fathead minnow (*Pimephales promelas*) is approximately 24,000 µg/L; guppy (*Poecilia retculata*) 31,080 µg/L, bluegill (*Lepomis macrochirus*) 18,000 µg/L, and goldfish (*Carassius auratus*) 42,330 µg/L (USEPA, 2007a).

(d) Polyvinyl Alcohol (CAS RN 9002-89-5). Polyvinyl alcohol is a polymer prepared from polyvinyl acetates. It is used as a pharmaceutics aid and ophthalmic lubricant and in the manufacture of surface coatings (CIDPL, 2009c). The rat oral LD_{50} is > 20,000 mg/kg, and the mouse oral LD_{50} is 14,270 mg/kg. Based on rat 90-day and 2 generation toxicity studies, the FAO/WHO identified a no-observed effect level (NOEL) of 5000 mg/kg and calculated an acceptable daily dose (ADI) of 50 milligrams per kilograms per body weight per day (mg/kg bw/day) (FAO/WHO, 2004). Estimated U.S. dietary intake is approximately 8.3 mg/kg bw/day (FAO/WHO, 2004). A search of the ECOTOX database returned zero results (USEPA, 2007a).

Table 4. List of Physical and Chemical Properties

Compound	MW	bp (°C)	Aq. sol. (mg/L) @ 25°C	log Kow	log Koc	Henry's Law Constant (atm-m³/m @ 25°C)	vp (mmHg)
Igniter							
Charcoal/carbon CAS # 7440-44-0	12.01[a]	>4300[b]	Insoluble	n/a	n/a	n/a	n/a
Gum Arabic CAS # 9000-01-5	240,000[b]	m.p. ~70°[a] b.p. 383°[a]	0.597[c]	n/a	n/a	n/a	n/a
Potassium Nitrate CAS # 7757-79-1	101.1[d]	m.p. 333°[d] Decomp 400°[d]	357[d]	n/a	n/a	n/a	n/a
Sulfur CAS # 7704-34-9	32.06[a]	444.6[e]	Insoluble[a]	n/a	n/a	n/a	3.95E-06@30°C[f]
Ethyl Acetate CAS # 141-78-6	88.12	77°[c]	8.0E+04[c]	0.73[c]	1.26[g]	1.34E-04[c]	93.4[c]
Black Smoke							
Magnesium Carbonate CAS # 546-93-0)	84.3[d]	Decomp 350°[d]	103 (cold water)[d]	n/a	n/a	n/a	n/a
Potassium Chlorate CAS # 3811-04-9	122.54[d]	Decomp 400°[d]	73[d]	n/a	n/a	n/a	Negligible @20C[d]
Disperse Red 9 CAS # 82-38-2	237.26[g]	m.p. 171C[c]	0.119[c]	4.10[h] Exp.	4.03[g] Est.	2.97E-08[c] Est.	7.0E-09[g]
Solvent Green 3 CAS # 128-80-3	418.5[g]	m.p. 218[c]	Insoluble[g] Est.	8.690[c] Exp.	6.63[g] Est.	1.47E-16[g] Est.	7.05E-13[g] Est.
Stearic Acid CAS # 57-11-4	284.48[a]	383°[a]	0.568[c]	8.23[i] Exp.	4.71[g] Est	4.76E-7[g] Exp.	7.22E-07[g] Ext.
Sugar (Sucrose) CAS # 57-50-1	342.3[g]	185.5[c]	2.1E+06[c]	-3.82[i] Est.	-2.21[g] Est.	4.47E-22[g] Est.	5.15E-17[c] Est.
Vinyl alcohol acetate resin (VAAR) CAS # 25213-24-5	Varies; large	nd	nd	nd	nd	n/a	n/a

n/a = not applicable; nd = no data; Est.= estimated; Exp= experimental; Ext.= extrapolated

[a] O'Neil, 2006
[b] HSDB, 2009a
[c] CIDPL, 2009a
[d] IPCS-ICSC, 2009
[e] EnvCan, 1981
[f] Ashford, 1994
[g] USEPA, 2008
[h] Hou, 1991
[i] Tetko, et al., 2005

Table 5. Toxicological Properties

Compound	Acute LD_{50} (mg/kg)	Sub-acute LD50 (mg/kg/d)	Subchronic NOAEL/LOAEL (mg/kg/d)	Chronic NOAEL/LOAEL (mg/kg/d)	Mutagenicity	Carcinogenicity
Igniter						
Charcoal CAS # 7440-44-0	>15,400[a] (rats) >3000[a] (rabbits)	n/a	n/a	n/a	n/a	Inhalation of particulates can activate immune response.
Gum Arabic CAS # 9000-01-5	16, 000 (rat) 80,000 (rabbit)[b]	n/a	n/a	NOAEL 5 g/kg-d [c] LOAEL 14 g/kg-d [c]	nd	Negative in rats and mice
Potassium Nitrate CAS # 7757-79-1	3750 (rats) [d] 1901 (rabbit) [d]	nd	nd	365 mg/kg/day NOAEL (rat-lifetime)[e]	Negative Ames [e]	Equivocal- conversion to nitrite increases reactivity of molecule.
Sulfur CAS # 7704-34-9	> 5 mg/kg [f] >2.56 mg/L [f]	n/a	n/a	n/a	Negative Ames [f]	Negative [f]
Ethyl Acetate CAS # 141-78-6	5620 (rat) [g] 4935 (rabbit)[g] 4100 (mouse) [g] 5500 (guinea pig) [g]	n/a	LOAEL 3600 NOAEL 900 (rat) [g]	nd	Negative Ames [g]	Negative CHO [g] Negative bone marrow [g]
Black Smoke						
Magnesium Carbonate CAS # 546-93-0	n/a	n/a	n/a	n/a	nd	nd
Potassium Chlorate CAS # 3811-04-9	1870 (rat; LD50)[h] 1200 (dog; LDLo) [h] 2000 (rabbit; LDLo) [h]	nd	32 (rat-male; NOAEL)[i,j] 45 (rat-female; NOAEL)[h,j] 670 (rat; MTD) [h,j]	nd	nd	2000mg/L (rat; increased thyroid neoplasia)[k]
Disperse Red 9 CAS # 82-38-2	>8000 (dog/oral); >2000 (rabbit/skin) >2000 mg/m^3 (4 h mult. species)[l]	nd	nd	Estimated >55 mg/kg/day NOAEL[m]	Negative Ames [n]	Pos. In vitro Neg. dom. Lethal Neg. mammary carcinogenesis [n]

[a] Registry of Toxic Effects of Chemical Substances (RTECS), 2006
[b] Sax, 2001
[c] Anderson, et al., 1982
[d] Sax, et al., 1989
[e] HSDB, 2008a
[f] USEPA, 1991a
[g] Thurman, et al., 1992
[h] CIDPL, 2009a
[i] McCauley, et al., 1995
[j] Reported as sodium chlorate
[k] NTP, 2005
[l] Owens, et al., 1974
[m] Griswold, et al., 1968
[n] Brusick, et al., 1978

Table 5. Toxicological Properties (continued)

Compound	Acute LD_{50} (mg/kg)	Sub-acute LD50 (mg/kg/d)	Subchronic NOAEL/LOAEL (mg/kg/d)	Chronic NOAEL/LOAEL (mg/kg/d)	Mutagenicity	Carcinogenicity
Solvent Green 3 CAS # 128-80-3	3660 (rat)[o] >10,000 (rabbit) [p] >1000 (dog) [p]	n/a	n/a	n/a	Negative [q]	Negative [q]
Stearic Acid CAS # 57-11-4	LD_{10} 4640 (rat) [h]	n/a	nd	nd	Negative [r]	Negative [r]
Sucrose CAS # 57-50-1	29,700 mg/kg (rat) [h]	n/a	n/a	n/a	Negative mouse lymphoma [h]	n/a
Vinyl alcohol acetate resin (VAAR) Components						
Methyl acetate	> 5000 (rat) 3705 (rabbit)[s] LCLo 32,000 ppm/ 4hr [s]	n/a	n/a	n/a	Negative [t]	nd
Vinyl acetate	2900 (rat) 1600 (mouse) 11,400 mg/m3/4hr (rat), 1550 ppm/4hr (mouse), and 2500 ppm/4hr (rabbit) [s]	TWA 10 ppm STEL 15 ppm [s]	n/a	n/a	Negative [u]	Pos. micronucleus and SCE [u] Carcinogenicity data is confounded; vinyl acetate is rapidly metabolized in vivo to acetaldehyde [v]
Polyvinyl alcohol	> 20,000 (rat) 14,270 (mouse) [w]	n/a	n/a	ADI 50 mg/kg/bw/d [w]	nd	nd

n/a= not applicable; nd = no data

[o] Marhold, 1972
[p] Davidson, et al., 1987a
[q] HSDB, 2009a
[r] CCRIS, 2009a
[s] CIDPL, 2009b
[t] CCRIS, 2009c
[u] IARC, 1995
[v] Thurman, et al., 1992
[w] FAO/WHO, 2004

Table 6. Human Health Impact Assessment

Compound	Acute Toxicity	Sub-chronic Toxicity	Acute Inhalation	Sub-chronic Inhalation	Cancer Probability	Comments
Igniter						
Charcoal	Low	Low	Low	Low	Low	Lung irritant in high concentrations.
Gum Arabic	Low	Low	Low (expected)	Low (expected)	Low	Acacia is a common natural ingredient found in food and cosmetics. GRAS for oral consumption may be lung irritant if aerosolized or as powder.
Potassium Nitrate	Low	Low	Unknown	Unknown	Low	Causes erythrolysis (anemia) at high concentrations, some nitrates may from N-nitroso compounds in gut leading to carcinogenesis.
Sulfur	Low	Low	Low	Low	Low	Ubiquitous element
Ethyl Acetate	Low	Unk	Low	Mod	Low	Unhydrolyzed esters cause mild local irritation and Central Nervous System depression
Black Powder	Low	Low	Low	Low-Mod.	Low	No toxicity data on black powder was located, however none of its components are believed to be particularly toxic. Quantity used is small and consumed during ignition.
Black Smoke						
Magnesium Carbonate	Low	Low	Low	Low	Low	Substance added directly to human food affirmed as generally recognized as safe (GRAS).
Potassium Chlorate	Low – Mod.	Low – Mod.	Unknown	Unknown	Low – Mod.	May cause erythrolysis at high doses (anemia, kidney effects). Some states regulate as "chlorates" (e.g. Maine = 7 µg/L water guideline for chlorate ion; CA = 800 µg/L).
Disperse Red 9	Low	Unknown	Unknown	Low	Low – Mod.	Reported to be an irritant; NAS deems experimental data insufficient to assess disperse red 9's toxic effects.
Solvent Green 3	Low	Unknown	Low	Moderate	Low – Mod.	Main toxicity concern is of inhalational exposures, where the ½-life in the lung = 280 d.
Stearic Acid	Low	Low	Low (expected)	Low (expected)	Low	Reversible lipogranulomas in adipose tissue occurred in rats following 24 w exposure to 50 g/kg-d.
Sucrose	Low	Low	Low	Low	Low	Generally accepted as safe (GRAS) ingestion item.
Vinyl alcohol acetate resin (VAAR)	Low	Low (expected)	Low-Mod.	Moderate	Low	Few data exist for polyvinyl acetate; data for vinyl acetate suggest low toxicity, but possible carcinogenicity.

Table 7. Assessment of Ecotoxicological Data

Compound	Aquatic	Invertebrate	Plants	Mammalian	Avian	Comments
Igniter						
Charcoal data used: Carbon	Low (carp)	Low (modeled)	Low (expected)	Low (rats, mice)	Low (bobwhite, inhal.)	Non reactive in many biological systems.
Gum Arabic	Low (expected)	Low (expected)	Low (expected)	Low	Low (expected)	Generally recognized as safe (GRAS) ingestion item
Sulfur	Low (fish, *Daphnia*, shrimp)	Low (shrimp)	Low (suspected)	Low (rats, rabbits)	Low (bobwhite)	Few toxicity data exist for elemental sulfur; however, toxicity is suspected to be low.
Potassium Nitrate	Low (*Daphnia*, algae, fish spp.)	Low (earthworms, *Daphnia*)	Low (algae)	Low (rats, mice)	nd	Any toxicity would be expected from the nitrate anion (expected low for all species).
Ethyl Acetate	Low-catfish, algae; Mod-*Daphnia*	Mod-*Daphnia*	Low-algae	Low-rats, mice, rabbits	Low-chicken eggs	Environmental half-life expected to be short.
Black Powder	Low (expected)	Low (expected)	Low (expected)	Low (expected)	Low (expected)	No toxicity data on black powder was located; however none of its components are believed to be particularly toxic.
Black Smoke						
Magnesium Carbonate	Low	Low	Low	Low	Low	Generally recognized as safe (GRAS) ingestion item.
Potassium Chlorate	Low (*Daphnia*, mayfly, caddisfly, fish)	Low (*Daphnia*, mayfly, caddisfly, earthworm)	Low-moderate (algae, soybean, sorghum, cucumber)	Low (rats, mice)	Low (mallard, bobwhite)	Few subchronic (sublethal) data.
Disperse Red 9	nd	nd	nd	Low (rats, dogs)	nd	Insufficient data; low solubility and short half-life suggest little environmental exposure.
Solvent Green 3	nd	nd	nd	Low (rats)	nd	Inhalational exposure of concern due to poor solubility in the lung.
Stearic Acid	Low (salmon)	nd	nd	Low (rats)	nd	Relatively safe saturated fatty acid.
Sugar (Sucrose)	Low	Low	Low	Low (rats)	Low	Generally accepted as safe (GRAS) ingestion item.
Vinyl alcohol acetate resin (VAAR)	Low (fish, algae, Daphnia, shrimp)	Low (Daphnia, shrimp)	Low (algae, corn, bean)	Low (rats)	nd	Data represent those available for vinyl acetate, a primary component of VAAR.

nd= no data

8. DISCUSSION.

a. Black Smoke System. The current black smoke system produces black smoke through the incomplete combustion of PAHs. This formulation along with the products of combustion creates many compounds which are of regulatory concern. Some of these compounds are known toxicants that fall under different regulations and agreements. The proposed formulation operates differently by the assimilation of dye to produce black colored smoke. Based upon known, or estimated, properties of components or similar substances, projected range amounts, and conditions of use, none of the PYRO 05-01 work unit's proposed alternative Black Smoke components are considered to be of immediate environmental or human health concern and the proposed formulation, as a whole, is an improvement over the current formulation. Although not in these specific combinations or use(s), the majority of the PYRO 05-01 work unit compounds have established manufacturing, occupational safety, regulatory history and human health histories.

b. Regulatory Considerations. A search for regulatory requirements under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), Toxic Substances Control Act (TSCA), Resource Conservation and Recovery Act (RCRA), and Comprehensive Environmental Response, Compensation, and Liability Act/Superfund Amendments and Reauthorization Act (CERCLA/SARA) related to components of this Black Smoke formulation resulted in the information below. Industrial standards set by NIOSH and OSHA are also included. No additional requirements were discovered.

(1) Gum Arabic is exempted from a tolerance under FIFRA when used as a surfactant, suspending agent, and dispersing agent when used according to good agricultural practices as an inert ingredient applied as part of a pesticide formulation or to raw agricultural commodities after harvest. Gum Arabic is GRAS as a component of human food by the FDA (HSDB, 2009c).

(2) Potassium nitrate is approved as a component of rodenticides under FIFRA. Potassium nitrate is used as a curing agent for cod roe, and may not exceed 200 ppm in the finished roe. Drinking water standards exist at the Federal level, and for the states of Maine and Minnesota, with all three standards being identical at 10,000 μg nitrate ion/L (HSDB, 2008a).

(3) Ethyl acetate is exempt from requirements for a tolerance under FIFRA when used as a solvent or co-solvent in accordance with good agricultural practices. Ethyl acetate is GRAS for human consumption when used in small quantities in inks used for marking fruits and vegetables, or in synthetic flavoring substances. Releases of over 5000 lb (2270 kg) are reportable under CERCLA; spent ethyl acetate must be handled as a hazardous waste under RCRA (HSDB, 2009b). The NIOSH REL and OSHA PEL are both 400 ppm (1400 mg/m^3) TWA (Lewis, 2004).

(4) Magnesium carbonate is exempt from the requirement of a tolerance with used as an anticaking or conditioning agent in accordance with good agricultural practices as inert (or

occasionally active) ingredients in pesticide formulations under FIFRA. As a food additive, magnesium carbonate is GRAS and is included in the list of antacid products for over-the-counter human use (HSDB, 2009d). Magnesium carbonate has a NIOSH REL of 10 mg/m^3 TWA for total particulates, and 5 mg/m^3 TWA for respirable particulates. The OSHA PEL is 15 mg/m^3 TWA for total particulates and 5 mg/m^3 TWA for the respirable fraction (ICSC, 1995).

(5) Potassium chlorate is subject to state drinking water guidelines in California (800 µg chlorate/L) and Maine (7 µg chlorate/L) (HSDB, 2009e).

(6) Stearic acid added to human food is GRAS (HSDB, 2008b).

(7) Sucrose is exempt for requirement for a tolerance under FIFRA when used as a solid diluent, carrier, or safener according to good agricultural practices as inert (or occasionally active) ingredients in pesticide formulations applied to growing crops, raw agricultural commodities, or animals. Added to human food, it is GRAS (HSDB, 2008c). The OSHA has set a PEL of 10 mg/m^3 TWA for total particulates and 5 mg/m^3 TWA for the respirable fraction., and the NIOSH REL has been determined to be 15 mg/m^3 TWA for total particulates and 5 mg/m^3 TWA for the respirable fraction (HSDB, 2008c).

c. Areas of Uncertainty.

(1) Historically, the dyes used in signaling functions of military smoke formulations have been scrutinized as potential toxicants. Most of the dyes used are based on the anthraquinone molecule and the potential for toxicity and carcinogenicity is owed, in part, to the functional groups' identity and position on the triple-ringed anthraquinone structure. For the proposed Black Smoke formulation, the two dyes, DR9 and SG3, have molecular structures that are predicted to be less toxic than many other anthraquinone derivatives. The toxicological data currently available on the dyes employed in this formulation suggest they appear to be of low toxicity individually; however, there are no data on exposure to the dye mixture. It is possible that under the deployment conditions of the pyrotechnic device, recombined products may be generated or novel interactions may occur that modify the toxicity profile. For example, the persistence of SG3 in lungs may act synergistically with the irritant properties of DR9 yielding an increased toxicity. Inhalation testing of the "as used" formulation is recommended to investigate the potential for increased toxicity of the combusted formulation.

(2) Potassium nitrate is an example of a substance whose by-products might be of concern; however, due to the small amounts of potassium nitrate employed and the projected routes of exposure, the risks appear to be small. It would take decades of heavy use of these formulations to generate enough nitrites to be of concern comparable to that of regulated application of fertilizer on a golf-course of comparable area.

(3) Despite apparently being little studied, VAAR appears not to be considered hazardous as such. Furthermore, in this Black Smoke formulation it is used in small quantities. It is widely used in products to which people and the environment are far more likely to be exposed than production or demilitarization of the Black Smoke pyrotechnics. However, from a routine production standpoint, the extensive use of VAAR in manufacturing operations may be associated with the release of volatiles. Thus, the use of VAAR may have industrial hygiene concerns or regulatory compliance issues associated with atmospheric releases.

(4) Black powder, a pyrotechnic and ordnance staple for centuries, is a mixture of compounds addressed herein and is not expected to cause adverse health effects if used as intended.

(5) Respiratory irritation and sensitization is a common feature for several of the components of the formulation. While transient exposure to Black Smoke is likely to be relatively harmless, repeated or long-term exposure to either high concentrations of the smoke or occupational exposure to the complete formulation should be evaluated and appropriate personal protection equipment should be identified, if necessary.

(6) Magnitude of exposure of black smoke constituents and smoke products is largely unknown for force-on-force applications. It is expected that those individuals involved with using this system in day-to-day training activities would have the greatest propensity for exposure.

9. RECOMMENDATIONS. Development of the proposed Black Smoke formulation should continue. Additional data should be collected relating to potential products of combustion and their inhalation toxicity in mammalian systems of both the dyes and the complete formulation. In addition, it is recommended that *in vitro* toxicity testing and aqueous ecotoxicity testing be conducted for the dye mixture.

APPENDIX A

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