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

30 September 2009

MEMORANDUM FOR Environmental Acquisition and Logistics Sustainment Program (AMSRD-MSF/Mr. Erik Hangeland), US Army Edgewood Chemical Biological Center, US Army Research, Development and Engineering Command, 5183 Blackhawk Road, Aberdeen Proving Ground, MD 21010-5424

SUBJECT: Environmental Health Assessment for Work Unit PYRO 06-08, Pyrotechnic Perchlorate Elimination/Mitigation Program for M118/M119 Simulators, Toxicology Report No. 87-XE-074Z-09C

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. Larry Williams, Directorate of Toxicology, Health Effects Research Program. He may be contacted at DSN (312) 584-7159 or commercial (410) 436-7159.

FOR THE COMMANDER:

Encl

GLENN J. LEACH
Acting Director, Toxicology

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14. ABSTRACT

The Army 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 primary purpose of this work unit (PYRO 06-08) is to eliminate potassium perchlorate from the flash composition of the formulations for the M118 and M119 simulators. These simulators are used in training to produce illumination or create a high-pitched whistle. The proposed formulations have successfully removed perchlorate from the mixtures, resulting in much more environmentally-friendly devices. The current formulations for the M118 and M119 simulators should continue in development.

15. SUBJECT TERMS

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Environmental Health Assessment for Work Unit PYRO 06-08
Pyrotechnic Perchlorate Elimination/Mitigation Program
For M118/M119 Simulators
Toxicology Report No. 87-XE-074Z-09C
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Final Report

Performing Laboratory

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DEPARTMENT OF THE ARMY
US ARMY CENTER FOR HEALTH PROMOTION AND PREVENTIVE MEDICINE
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EXECUTIVE SUMMARY
ENVIRONMENTAL HEALTH ASSESSMENT FOR WORK UNIT PYRO 06-08
PYROTECHNIC PERCHLORATE ELIMINATION MITIGATION PROGRAM
FOR M118/M119 SIMULATORS
TOXICOLOGY REPORT NO. 87-XE-074Z-09C
SEPTEMBER 2009

1. PURPOSE. To provide environmental and occupational health information on new or replacement energetic compounds for Army use in the research, development, testing, and evaluation (RDT&E) of alternatives under the Environmental Quality Technology (EQT) program. This information is necessary for work unit program evaluation.

a. Residues of pyrotechnics, propellants, explosives, and incendiaries that 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. As a consequence, research, development, testing, training, and use of substances potentially less hazardous to human health and the environment is vital to the readiness of the US Army. Safeguarding the health of Soldiers, civilians, and the environment requires an assessment of alternatives before they are fielded. Continuous assessment of the potential alternatives, begun early in the RDT&E process, can save significant time and effort during RDT&E, as well as over the life cycle of the items developed.

b. The Army 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 primary purpose of this work unit (PYRO 06-08) is to eliminate potassium perchlorate from the flash composition of the formulations for the M118 and M119 simulators. These simulators are used in training to produce illumination or create a high-pitched whistle.

2. CONCLUSION. The proposed formulations have successfully removed perchlorate from the mixtures, resulting in much more environmentally-friendly devices.

3. RECOMMENDATIONS. The current formulations for the M118 and M119 simulators should continue in development.

Toxicology Report No. 87-XE-074Z-09C

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ENVIRONMENTAL HEALTH ASSESSMENT FOR WORK UNIT PYRO 06-08 PYROTECHNIC PERCHLORATE ELIMINATION PROGRAM FOR M118/M119 SIMULATORS TOXICOLOGY REPORT NO. XE-87-074Z-09C SEPTEMBER 2009

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

2. PURPOSE. To provide environmental and occupational health information on new or replacement compounds and mixtures for Army use in explosives, propellants, and pyrotechnics. This information is necessary for work unit program evaluation.

3. AUTHORITY. This Environmental Health Assessment addresses, in part, the environmental safety and occupational health (ESOH) requirements outlined in Army Regulation (AR) 200-1 (2007); AR 40-5 (2007); and AR 70-1 (2003); Department of Defense Instruction (DoDI) 4715.4; and Army Environmental Research and Technology Assessment (AERTA, 2007) requirement PP-3-02-04, "Compliant Ordnance Lifecycle for Readiness of the Transformation and Objective Forces." This assessment was performed as part of an on-going effort by the US Army Environmental Quality Technology (EQT), Ordnance Environmental Program to reduce or eliminate the environmental impact from the life cycle use of new chemical formulations proposed for use in weapon systems or platforms. This program is under the direction of the Research Development Engineering Command (RDECOM) Environmental Acquisition Logistics & Sustainment Program (EALSP; Mr. Erik Hangeland) and EQT P2 Chair (Army Research Laboratory (ARL); Dr. John Beatty).

4. BACKGROUND.

a. Current regulations require assessment of human health and environmental effects arising from exposure to chemical substances in soil, surface water, and ground water. 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 RDT&E process in order to avoid unnecessary costs, conserve physical resources, and sustain the health of those potentially exposed.

b. In an effort to support this preventive approach, the US 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, development, and potential remediation costs and to sustain the environmental integrity of testing and training ranges. This is an ongoing effort, and this report represents the status of information available as of the date of publication. Summary interpretations of preliminary information search results for this work unit were provided to the sponsor during the course of the development effort.

c. Pyrotechnic simulators teach troops installation, detection, and caution by mimicking booby traps and other hazards a Soldier might encounter on the battlefield. The M118 simulates illumination from a parachute flare or artillery illumination round and the M119 produces a loud

whistle. The developer for these simulators is the US Army Armament Research, Development, and Engineering Center (ARDEC), Picatinny Arsenal, NJ. The principle investigator is Mr. Mark Motyka.

5. STATEMENT OF PROBLEM.

a. Research, develop, and prove-out perchlorate-free charges for the M118 and M119 simulators that are more efficient, are life-cycle cost effective, and more conducive to human health and environmental quality.

b. Potassium perchlorate is a major component of the current simulator formulations. The perchlorate anion is a strong and effective oxidizer and, as such, is an efficient component of propellant mixtures. Unfortunately, the perchlorate anion has numerous environmental and human health regulatory concerns. The greatest human health concern stems from the fact that perchlorate biochemically mimics iodine, competes with iodine for uptake by the body, specifically the sodium-iodine symporter (NIS), and inhibits the production of thyroid hormones. While individuals that consume recommended quantities of iodine in their diet appear not to be affected, persons who are iodine-deficient, nursing mothers, and infants are potentially at an increased risk of thyroid hormone deficiency. Thyroid hormone deficiency has been shown in the developing rat model to result in impaired neurological development (NRC, 2005). Ecological effects associated with perchlorate exposure are summarized in a previous publication (USACHPPM, 2007).

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) (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.

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 tertiary sources 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 US Department of Health and Human Services (DHHS), Agency for Toxic Substances and Disease Registry (ATSDR), and *The Merck Index*. The Chemical Propulsion Information Agency's (CPIA), *Hazards of Chemical Rockets and Propellants* (1985), and the US Environmental Protection Agency's (USEPA) *Drinking Water Health Advisory: Munitions* (1992), were also consulted. Commercial suppliers are sometimes contacted for results of in-house research that may not appear in the open literature.

e. Online sources include the US 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® 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 (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). 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. Some properties are not measured experimentally, but are estimated by Quantitative Structure-Activity Relationship (QSAR) models, or calculated from another value. Every effort was made to find original research reports describing the data and method of collection. Data from secondary and tertiary sources and databases were used when data were abundant and well-evaluated. When toxicity data were not available, criteria were modeled using QSAR approaches.

g. Potential products from combustion were estimated by Dr. Ross Sausa of the US Army Research Laboratory using the NASA/GLENN Chemical Equilibrium with Applications (CEA) code. Additional information on the methods and assumptions used in this code can be found at http://www.grc.nasa.gov/WWW/ CEAWeb/ceaWhat.htm. Results depend upon chemical-specific information contained within the model. When chemical-specific information is not available, data from the closest surrogate is used.

h. 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 1. Composition of Simulator Formulations

Substance/ Formulation	**CAS Number**	**WF-9 M118**	**WM-2 M119**	**WM-1**	**RNX-6**	**RNX-17**
Boron	7440-42-8					8%
Charcoal	1333-86-4	2%				
Epikure 3125™	NA				8.6%	7.3%
- >96% Polyamino Amide	68410-23-1					
- < 6% Triethylene Tetramine	112-24-3					
Epon 828™	25068-38-6				17.4%	14.7%
Iron Oxide Red (Ferric oxide)	1332-37-2, 1309-37-1				4%	
Potassium Benzoate	582-25-2		17%	17%		
Potassium Chlorate	3811-04-9		67%	80%		
Potassium Nitrate	7757-79-1	81%	13%		70%	70%
Red Gum	9000-20-8		3%	3%		
Silicon	7440-21-3	6%				
Sulfur	7704-34-9	8%				
Vinyl Alcohol Acetate Resin (VAAR)	25213-24-5	3%				

Table 2. Categorization Criteria used in the Development of Environmental Safety and Occupational Health Severity (Howe et al., 2006)

	LOW	MODERATE	HIGH
PERSISTENCE	Readily biodegrades (<28 days)	Degradation ½ life: water <40 days , soil <120 days	Degradation ½ life: water >40 days soil > 120 days
TRANSPORT	Water sol. < 10 mg/L log K_{OC} > 2.0	Water sol. 10-1000 mg/L log K_{OC} 2.0-1.0	Water sol. > 1000 mg/L log Koc <1.0
BIOACCUMULATION	log K_{OW} <3.0	log K_{OW} 3.0-4.5	log K_{OW} >4.5
TOXICITY	No evidence of carcinogenicity/ mutagenicity; Subchronic LOAEL > 200 mg/kg-d	Mixed evidence for carcinogenicity/mutagenicity (B2, 2); Subchronic LOAEL 5-200 mg/kg-d	Positive corroborative evidence for carcinogenicity /mutagenicity; LOAEL < 5 mg/kg-d
ECOTOXICITY	Acute $LC_{50}LD_{50}$ >1 mg/L or 1500 mg/kg; Subchronic EC_{50} >100 µg/L or LOAEL >100 mg/kg-d	Acute $LC_{50}LD_{50}$ 1-0.1 mg/L or 1500-150 mg/kg; Subchronic EC_{50} 100-10 µg/L or LOAEL – 10-100 mg/kg-d	Acute LC_{50},LD_{50}<100 µg/L or <150 mg/kg; Subchronic LOAEL <10 mg/kg-d

Notes:
mg/L - milligrams per liter
LOAEL - lowest-observed adverse effect level
LC_{50} – concentration expected to result in 50% lethality to a population of test animals.
LD_{50} - Lethal Dose 50 median concentration of a toxicant that will kill 50% of the test animals within a designated period
mg/kg-d - milligram per kilogram per day
µg/L - microgram per liter

7. RESULTS.

a. Physical and Chemical Properties. Physical and chemical properties are summarized in Table 3. Numerical toxicological data is tabulated in Table 4. Data for properties that could not be found are indicated by "nd" (no data). 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. From the available data, there were no indications of significant potential environmental problems (i.e. mobility in water, bioaccumulation, or persistence) for any component resulting from its physical or chemical properties. However, few data were found for some of the commercial plasticizers (e.g. Epon™, Epikure™). Although efforts were made to gather additional data from the manufacturers, little relevant information has been received. Given the relatively small amounts used in the formulation, it is unlikely these substances will significantly affect the conclusions of this report.

b. Summaries. 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) Boron. Boron is an element that exists as one of three allotropes at room temperature, including red crystals, black crystals with a metallic luster, and a black or dark brown amorphous powder (O'Neil, 2006). Very few toxicity data are available for elemental boron, since its occurrence in nature is rare. The most likely paths for boron exposure are via food – mainly fruits and vegetables, ground water, air, and consumer products (ATSDR, 1992). Individuals who reside in areas of the western United States that have natural boron-rich deposits may be exposed to higher-than-average levels of boron compounds (ATSDR, 1992). In soil, boron readily forms compounds with surrounding materials depending on the type of soil, its pH level, salinity, organic matter content, clay content, and other variables. Boron compounds are absorbed by plants through soil, and there is evidence that boron is an essential nutrient for some plants (WHO, 2005a).

(a) Acute oral. The acute oral LD_{50} for boron has been determined in several species. In mouse it is 2000 mg/kg for "presumably amorphous" boron (Stokinger, 1981) although a value of 560 mg/kg was reported for another "unspecified" form of boron, presumably elemental boron. LD50's in other species are: rat, 650 mg/kg; rabbit, 310 mg/kg; guinea pig, 310 mg/kg; dog, 310 mg/kg; and cat, 250 mg/kg (CIDPL, 2009a).

(b) Sub-chronic oral. No data found.

(c) Chronic oral. Based on developmental effects (decreased fetal weights) in rats from two studies (Heindell et al., 1992; Price et al., 1996), the EPA reference dose (RfD) for boron is 0.2 mg/kg/day (USEPA, 2006b). The No-Observed-Adverse-Effect Level (NOAEL) for boron is 9.6 mg/kg-day (Price et al., 1996), and the LOAEL is 13.3 mg/kg-day (Heindell et al., 1992).

(d) Acute inhalation. No data found.

(e) Sub-Chronic inhalation. Fifteen mice were exposed to concentrations averaging 72.8 mg/m^3 of elemental amorphous boron for 7 hours per day, 5 days per week over a 30-day period. Mean particle size was 0.67 μm with a standard deviation of 2.0 μm. At the end of the study, average boron concentrations in the mouse tissues were: 792 μg/g in the lung, 252 μg/g in the kidney, 106 μg/g in the liver, and 73 μg/g in the gastrointestinal tract. There was no evidence of toxicological effects. The mice had a mean weight loss of 7% during the first week, but they later recovered the lost weight in steady increases (Stokinger, 1981).

(f) Chronic inhalation. No data found.

(g) Dermal. No data found.

(h) Development and Reproduction. No data found.

(i) Mutagenesis. No data found.

(j) Carcinogenesis. Based on the lack of human studies and limited animal studies, the human carcinogenicity of elemental boron cannot be classified (WHO, 2005a).

(k) Ecotoxicology. Boron is an essential micronutrient for some plants. It is important in plant cell division, metabolism, and membrane structure and function (WHO, 2005a).

(2) Charcoal.

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

(b) 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 g/kg for 52 weeks (Pence et al., 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% carbon black in mice for 72 weeks were observed (Nau et al., 1976).

(c) 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 et al., 1985).

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) 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. 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, 2005). Few toxicity data exist for acute inhalational exposure to carbon black due to the greater concern over the potential for cancer from longer term exposures (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.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis. A recent review by Valberg and co-workers reassesses the International Agency for Research on Cancer'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--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).

(k) 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 et al., 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 (C. Driver, pers. comm.). ECOSAR-predicted endpoints from the EPA's assessment for carbon black (USEPA, 2005) are presented in Table 3.

Table 3. ECOSAR-predicted endpoints for carbon black.

Organism	Duration	Endpoint	Predicted mg/L
Fish	14 day	LC_{50}	250
Fish	96 hour	LC_{50}	170
Daphnid	48 hour	LC_{50}	170
Green algae	96 hour	EC_{50}	96
Fish	30 day	ChV	18
Daphnid	16 day	EC_{50}	5
Green algae	96 hour	ChV	5
Fish	96 hour	LC_{50}	22
Mysid shrimp	96 hour	LC_{50}	110
Earthworm	14 day	LC_{50}	240 (dry mass soil)

Key: ChV = chronic value estimate (survival/growth for fish; growth for algae

(3) Epikure™ 3125. Epikure™ 3125 is a curing agent manufactured by Hexion Specialty Chemicals, Columbus, OH, and is comprised of two components: polyaminoamide (>96%) and triethylenetetramine (<6%) (Hexion Specialty Chemicals, 2007). A request was made to Hexion for data that had been collected from any internal toxicological studies done with Epikure™ 3125, but no data has been forthcoming. SCORECARD, has Epikure™ 3125 listed as a high volume chemical with productions exceeding 1 million pounds annually in the US; it could not be designated with a hazard ranking because of lack of data (Scorecard). The majority of the information found was from Material Safety Data Sheets (MSDS) for specific products that

contained Epikure™ 3125. Information on the two ingredients of Epikure™ 3125 is provided below.

(a) Acute Oral. No data found.

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis. No data found.

(k) Ecotoxicology. No data found.

(4) Polyaminoamide. From the name, one can infer that this is a heterodisperse polyamide with basic (amino) side chains, structurally very similar to a protein with a high percentage of basic amino acids, such as histones or similar molecules. While the exact nature of the monomers involved is unknown, the combustion process will probably consume most of the material. Polyamino amides have been patented for use in protecting hair against atmospheric attack and light (US Patent 4842851, 1989).

(a) Acute Oral. The LD_{50} values for rats, mice, and rabbits have been determined to be 4340 mg/kg, 1600 mg/kg, and 5500 mg/kg respectively (Stavreva, 1979). The manufacturer's MSDS for Epikure™ 3125 (Hexion Specialty Chemicals, 2007) reports, that the mixture is expected to be of low toxicity, $LD_{50} > 2000$ mg/kg.

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. The manufacturer's MSDS for Epikure™ 3125 (Hexion Specialty Chemicals, 2007) reports, "Inhalation of vapors or mists may cause irritation."

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis. Lifelong dermal application to mice at 1.2 mg three times a week did not result in increased incidence of any tumors, nor did it have any effect on skin irritation (DePass et al., 1987).

(k) Ecotoxicity. No data found.

(5) Triethylenetetramine. Also known as triene, triethylenetetramine is used as an oral chelator in treatment of Wilson's Disease (toxic accumulation of copper) and biliary cirrhosis, especially when patients are unable to tolerate D-penicillamine. It is also used in detergents, synthesis of dyestuffs, pharmaceuticals, and drilling mud (Ormerod et al., 1989; HSDB, 2008).

(a) Acute oral. Use of triethylenetetramine in treatment of biliary cirrhosis has been associated with serious gastrointestinal and abdominal pain, anemia, and rhabdomyolysis (HSDB, 2008).

(b) Subchronic oral.

i. Greenman et al., administered triethylenetetramine to B6C3F1 mice and F344 rats in drinking water for up to 92 days at concentrations of 0, 120, 600, and 3000 ppm (Greenman et al., 1996). Animals were fed diets containing nutritionally-adequate amounts of copper. At 600 and 3000 ppm, rats fed a purified diet had somewhat lowered levels of plasma copper, but failed to demonstrate signs of copper deficiency. In female rats receiving 3000 ppm in water and a purified diet, an increased frequency of uterine dilatation was noted. In mice, toxicity was noted only at the highest concentration in animals receiving a purified diet. Rats receiving a cereal-based diet (NIH-31) showed reduced liver copper levels at 3000 ppm.

ii. In a 10-month study in rats, the "threshold dose for chronic toxicity" was reported to be 0.8 mg/kg (BUA, 1995).

(c) Chronic oral. Most publications relating to doses in humans during treatment of Wilson's Disease indicate oral doses up to 2.3 g/day are well-tolerated (BUA, 1995).

(d) Acute inhalation. Exposure to hot vapor results in respiratory tract irritation (HSDB, 2008). Acute exposure to saturated vapor via inhalation was tolerated without impairment (OECD SIDS, 1998).

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal.

i. Exposure to hot vapor results in itching of the face with erythema and edema. Sensitization of workmen in an electrical equipment factory was also noted, with serious skin lesions, vesicular popular eczema (sometimes weeping), localized particularly on hands, forearms, genital, and inguinal regions (HSDB, 2008).

ii. In animals, triethylenetetramine has moderate toxicity on dermal application to rabbits; LD_{50} 550-820 mg/kg (BUA, 1995).

(h) Reproduction and Development.

i. Classified as FDA pregnancy category C (see box text). Numerous malformations and fetal abnormalities have been reported with use in animals (HSDB, 2008).

Pregnancy Category C: If animal reproduction studies have shown an adverse effect on the fetus, if there are no adequate and well-controlled studies in humans, and if the benefits from the use of the drug in pregnant women may be acceptable despite its potential risks, the labeling shall state: "Pregnancy Category C. (Name of drug) has been shown to be teratogenic (or to have an embryocidal effect or other adverse effect) in (name(s) of species) when given in doses (x) times the human dose. There are no adequate and well-controlled studies in pregnant women. (Name of drug) should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus." The labeling shall contain a description of the animal studies. If there are no animal reproduction studies and no adequate and well-controlled studies in humans, the labeling shall state: "Pregnancy Category C. Animal reproduction studies have not been conducted with (name of drug). It is also not known whether (name of drug) can cause fetal harm when administered to a pregnant woman or can affect reproduction capacity. (Name of drug) should be given to a pregnant woman only if clearly needed." The labeling shall contain a description of any available data on the effect of the drug on the later growth, development, and functional maturation of the child. (FDA: http://www.fda.gov/Cder/handbook/categc.htm)

ii. Keen and co-workers fed triethylenetetramine in the diet at levels of 0, 0.17, 0.83, or 1.66% to Sprague-Dawley rats (Keen et al., 1983). Frequency of resorptions and abnormal fetuses at term increased with increasing levels of the drug. Maternal and fetal tissue copper levels were significantly lower than controls, and decreased in a dose-dependent manner. Maternal liver iron was increased in the high-dose group, but fetal iron and both maternal and fetal manganese levels were not significantly affected by the drug. A follow-on study indicated that low copper levels were primarily responsible for the teratogenic response (Cohen et al., 1983).

iii. Administration of triethylentetramine to rats results in decreased fetal copper levels, and may result in increased fetal zinc (Keen et al., 1983).

iv. Tanaka and coworkers investigated the effects of triethylenetetramine in mice on gestational day 19 (Tanaka et al., 1992). Triethylenetetramine was given throughout pregnancy to the dams at levels of 0; 3000; 6000; and 12, 000 mg/L via drinking water. At the level of 12,000 mg/L, frequency of resorption of fetuses tended to be high, and fetal viability was low compared to controls. Fetal body and cerebrum weights decreased significantly at dosage rates of 6000 and 12,000 mg/L, however fetal liver weights remained unchanged. Fetal copper concentrations in liver and cerebrum were significantly lower than controls in the treated groups, changing is a dose-related manner. Changes in fetal zinc concentration varied by tissues, i.e. there was an increase in liver but no change in the cerebrum.

v. Tanaka and coworkers also investigated the effects of the dosage levels in the previous paragraph in mice on gestational day 19 (Tanaka et al., 1993). While mean litter size and live fetus per dam at birth were not affected, a dose-dependent increase in gross brain abnormalities such as hemorrhages, delayed ossification of the cranium, hydrocephaly, exencephaly, and microcephaly were noted. At the microscopic level, disorganization of neuronal cell layers, spongiform changes in white matter, and reduced myelin development were noted. Abnormalities increased in a dose-dependent manner.

(i) Mutagenicity.

i. Triethylenetetramine is mutagenic in the Ames *Salmonella* test in all 5 strains of testing bacteria, with or without S9 activation. The lowest positive dose tested was 0.033 mg/plate in TA100 without activation (Mortelmans et al., 1986; CCRIS, 2009c).

ii. Positive results have been obtained in the Sister Chromatid Exchange (SCE) and Unscheduled DNA Synthesis (UDS) tests, but triethylenetetramine was negative in the Sex-linked Recessive Lethal (SLRL) test in *Drosophila.* Triethylenetetramine is not clastogenic in the mouse micronucleus test *in vivo* following either oral or intraperitoneal administration (BUA, 1995).

(j) Carcinogenesis. No data found.

(k) Ecotoxicology. The 24-hour growth inhibition test on bacteria (*Pseudomonas fluorescens*) gave an Effective Concentration (EC_{50}) of 500 mg/L, and 500 mg/L has a marked cytostatic effect on the yeast *Saccharomyces cerevisiae*. Green algae (*Scenedesmus subspicatus*) showed a 72-hour EC_{10} of 2.5 mg/L based on biomass. The test of an inhibiting effect on mobility in *Daphnia magna* gave the following 24-hour values: EC_0: 22 mg/L, EC_{50}: 92.4 mg/L, and EC_{100}: 354 mg/L. In finfish, a test with golden orfes (*Leuciscus idus)* gave a 48-hour LC_0 of 200 mg/L and a 27-hour LC_{100} of 500 mg/L (BUA, 1995). In one study using guppies (*Poecilia reticulate*), the LC_{50} after a 96-hr exposure was determined to be 570 mg/L (OECD SIDS, 1998; Procolor MSDS, 1999). An LD_{50} for red-winged blackbird (*Agelaius phoenicus*) was estimated to be 101 mg/kg based on food consumption data over an 18-hour period (Schafer et al., 1983).

(l) Other. Yin and co-workers found that triethylenetetramine inhibits telomerase activity in HeLa cells in culture, with an Inhibitory Concentration (IC_{50}) of about 7.8 μM (Yin et al., 2004). The proportion of cells in the G1 phase of the cell cycle was found to increase with the dosage of triethylenetetramine.

(6) EPON 828 ™. Epon 828™ is a low-molecular weight epoxy resin. It is a yellow, flaky solid at room temperature, and is insoluble in water with no measurable vapor pressure (CCRIS, 2009a). Epon 828™ has a wide variety of uses in automotive products, home maintenance materials, and hobby/craft adhesives (HSDB, 2008). The reaction product of Epon 828 ™ is bisphenol-A-(epichlorhydrin) epoxy resin.

(a) Acute Oral. Based on a reported oral LD_{50} value of 11,400 mg/kg for rats (Hine et al., 1958), Epon 828™ is considered practically non-toxic to humans. Several other LD_{50} values have been reported for this chemical, including an oral LD_{50} of 15,600 mg/kg for mice, an intraperitoneal LD_{50} of 2400 mg/kg for rats, and an intraperitoneal LD_{50} of 4000 mg/kg for mice (Hine et al., 1958). Epon 828™ showed no chronic toxicity in the diet of male rats (Hine et al., 1958). The manufacturer (Hexion Specialty Chemicals) MSDS for Epon 828™ (Hexion Specialty Chemicals, 2006) reports, that the LD_{50} is expected to be of low toxicity, LD_{50} >2000 mg/kg.

(b) Subchronic Oral. No data found.

(c) Chronic Oral. Significant mortality was observed in male rats fed 5.0% Epon 828™ in their diet for 26 weeks. Female rats were not tested (Hine et al., 1958).

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. In rabbit, dermal exposure to 0.1 mL of a 20% suspension Epon 828™ polymer in propylene glycol resulted in an irritation grade of 2 for the eye. Epon 828™ was also tested for effect on the skin of rabbits. Compounds were introduced under patches of gauze on the clipped rabbit skin. The dose was not specified. After 24 hours, a first reading was made, and, after 72 hours, a second reading was made. Epon 828™ received an irritation grade of 0 for the skin. After 20 applications, ore irritation was observed (Hine et al., 1958).

(h) Reproduction and Development. No data found.

(i) Mutagenicity. Epon 828™ tested positive in the Ames assay (Hauptman et al., 1978). Chinese hamster ovary (CHO) cells tested positive for mutagenicity (structural changes) after in vitro exposure to 0.01-0.04 mg/mL Epon 828™ in dimethylsulfoxide solution for 24 hours continuous treatment (JCIETIC, 1996).

(j) Carcinogenesis.

i. A two-year bioassay with rats and mice exposed dermally to the diglycidyl ether of bisphenol A (BADGE) showed evidence of carcinogenicity to the skin (Hexion Specialty Chemicals, 2006). Epichlorohydrin has been found to be carcinogenic in rodents, though inadequate evidence has been determined for carcinogenesis in humans (IRIS, 2007).

ii. Female mice, CF-1 strain, tested positive for reticulum cell sarcoma and lymphoblastic lymphosarcoma tumors after the twice weekly dermal application of a 10% solution of Epon 828™ in acetone (0.2 mL total dose) for 2 weeks. There was no evidence that exposure of male mice to the epoxy resin affected their susceptibility to systemic tumor development. However, it is possible that the increased incidence of reticulum-cell sarcomas or lymphosarcomas may be due to the susceptibility of the CF-1 mice to a viral infection and is not a direct indication of any systemic carcinogenic potential of epoxy resins (Peristianis et al., 1988).

(k) Ecotoxicology. Aquatic data exists for epichlorohydrin tested in *Daphnia*, algae, and many fish species. Toxicity was observed is at relatively high concentrations (USEPA, 2006a). Aquatic data exists for bisphenol-A, though the results are variable. Mortality has been reported in rainbow trout at 96-hour exposures of 60 µg/L (Brooke, 1991).

(7) Iron Oxide Red. Known more commonly by the name ferric oxide, this is the common form of iron (III) oxide.

(a) Acute Oral. No data found.

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. In humans there was no effect of inhaled iron oxide particle aerosols. Inhalation did not cause an appreciable alteration of alveolar epithelial permeability, lung diffusing capacity, or pulmonary function in healthy subjects under the studied conditions (Lay et al., 2001).

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis. No data found.

(k) Ecotoxicology. No data found.

(8) Benzoic acid, sodium or potassium salt. Benzoic acid is a weak, organic acid with a pK_a of 4.204 (Dean, 1992). Accordingly, exposure of either benzoate salt to an aqueous environment will result in formation of benzoic acid ($K_a = 6.25 \times 10^{-5}$). The information below reflects both the protonated and salt forms of benzoic acid.

(a) Acute Oral. The rat LD_{50} for sodium benzoate is reported to be 1714 mg/kg (SCCP, 2005).

(b) Subchronic Oral. Sherman rats dosed with 16-1090 mg/kg-d sodium benzoate for 30 days had lesions of the adrenal glands, upper intestine, kidneys, liver, and spleen (Smyth et al., 1948; Smyth et al., 1969), and rats dosed with 1947-2195 mg/kg-d sodium benzoate for 3-6 weeks were observed to have severe reductions in growth rates compared to controls (White, 1941).

(c) Chronic Oral. No data found.

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. Pregnant CD-1 mice and Wistar rats received oral doses of sodium benzoate at 1.75, 8, 38, or 175 mg/kg on gestation days 6-15, pregnant hamsters received doses of 3, 14, 65, or 300 mg/kg on gestation days 6-10, and rabbits received doses of 2.5, 12, 54, or 250 mg/kg on gestation days 6-18 without any harmful effects observed on maternal or fetal survival or soft or skeletal fetal abnormalities (FDRL, 1972).

(i) Mutagenicity. Potassium benzoate showed no mutagenic activity in *in vitro* Ames tests (OECD SIDS, 2001).

(j) Carcinogenesis. No data found.

(k) Ecotoxicity. No change in mortality was observed in *Daphnia, Gammarus* (scud), *Pimphales promelas* (fathead minnow), *Helisoma* (ramshorn snail), flatworm, or *Lumbriculus* (oligochate worm) exposed to benzoic acid concentrations of up to 100 mg/L (Anderson, 1946; Geiger et al., 1985; Ewell et al., 1986).

(9) Potassium chlorate. Potassium chlorate is an inorganic salt that occurs as colorless crystals or as white powder or granules. It is soluble in water, alcohol, alkalies, glycerol, glycerin, and liquid ammonia and insoluble in acetone. Potassium chlorate is explosive and flammable, and as an oxidizing agent it is useful for various medical, and veterinary purposes (ATSDR, 2008). Potassium chlorate is approved by the European Union for use in toothpastes at concentrations of 5% or less, and for other uses at 3% or less. It has a Category II for over the counter (OTC) drugs. Heads of "strike anywhere" matches contain 24% potassium chlorate,

while "safety" or "strike-on-box" matches contain potassium chlorate as their chief ingredient. This chemical is incorporated in gargles, mouth washes, dentifrices, and throat lozenges (Kurokawa et al., 1985).

(a) Acute Oral. Several acute toxicity studies with rats indicate that potassium chlorate is slightly toxic. In humans, the lethal dose ranged from 5 g to 75 g potassium chlorate. Clinical descriptions of potassium chlorate poisonings in humans from accidental or suicidal ingestion include reports of rapid oxidative destruction of red blood cells, possibly followed or preceded by increased methemoglobin and eventual cyanosis and progressive kidney failure (NRC, 1980). The LD_{50} value for the rat was determined to be 1850 mg/kg (SciLab, 2009). From cases of accidental or suicidal ingestion, the probable oral lethal dose for adult humans is estimated to be between 50-500 mg/kg (Gosselin et al., 1977; NRC, 1980).

(b) Subchronic Oral.

i. Oral doses of potassium chlorate 0.05 to 0.25 g/kg were administered daily to cats for 25 to 32 day. 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 0.05 g/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 mM sodium chlorate. The mean drinking water consumption between exposure groups varied 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 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).

(c) Chronic Oral. In another study, groups of male and female rats were provided drinking water containing 125, 1000, or 2000 mg $NaClO_3$/L for two years, while male and female mice received 500, 1000, or 2000 mg $NaClO_3$/L. 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, 2005a).

(d) Acute Inhalation. Inhalation of potassium chlorate dust and contact with eyes or skin causes local irritation (NRC, 1980).

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis.

i. Few data exist on the carcinogenic potential of any of the chlorates in either humans or animals. Exposure to 1% potassium chlorate in the drinking water of rats for 25 weeks, which was assumed to be the maximum tolerated dose as determined in a 6-week toxicity test, did not promote rat renal carcinogenesis (Kurokawa et al., 1985).

ii. In another study, groups of male and female rats were provided drinking water containing 125, 1000, or 2000 mg $NaClO_3$/L for two years, while male and female mice received 500, 1000, or 2000 mg $NaClO_3$/L. 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 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, 2005b).

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

(k) Ecotoxicology.

i. Fourteen day-old soybean seedlings were exposed to up to 2.0 mM potassium chlorate for 24 hours at varying light levels (100, 67, 33 and 0% of full light which was 750 μEinstein/m^2-s) (Harper, 1981). Interveinal chlorosis was evident within 24 hours following addition of 0.5 mM, with the severity of the symptoms increasing with dose. Leaflet nitrate reductase activity was lower following exposures to concentrations of 0.5 mM and higher, indicating that ClO_3^-, or some reduction product of ClO_3^-, was detrimental to enzyme functionality. Sodium chlorate was not shown to greatly affect shoot growth in sorghum or cucumber (Sund et al., 1963).

ii. Changes in population growth rates were observed by Van Wijk and co-workers in brown algae (*Ectocarpus variabilis*) exposed 14 days to relatively small amounts of chlorate salt (0.005 mM) (Van Wijk et al., 1998). Similar changes were not seen in two species of green

algae (*Pseudokirchneriella subcapitata* and *Scenedesmus subspicatus*) at concentrations of up to 144 mg/L (USEPA, 2000) and 3137 mg/L (Brixham, 1995), respectively.

iii. Concentrations of sodium chlorate lethal to *Daphnia* (Dosdall et al., 1997) and many fish species (Matida et al., 1976; USEPA, 2000) were reported to be greater than 3000 mg/L, though variation exits in the data. Aquatic insects were also relatively resistant to chloric salt exposures where reports of lethality (LC_{50} values) were often greater than 30 mg/L (e.g. caddisfly, mayfly; (USEPA, 2000)).

iv. Median lethal dose (LD_{50}) was reported to be greater than 2500 mg/kg for mallard and northern bobwhite (USEPA, 2000). Median lethal concentration (LC_{50}) for the earthworm (*Lumbricus terrestis*) could not be determined for concentrations of $NaClO_3$ of up to 1000 mg/kg soil, though reductions in biomass may occur at lower levels (Hague et al., 1983).

(10) Potassium nitrate. 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 mmol nitrate/person/day (Shuval et al., 1972; Hotchkiss et al., 1992; Francis, 1995).

(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 et al., 1989).

(b) 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.

(c) Chronic Oral. Female guinea pigs were given 0, 300, 2500, 10,000 or 30,000 ppm potassium nitrate in drinking water for 204 days (Sleight et al., 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 estimated to have received a dose equivalent to 1130 mg/kg. One female in this dose group died with four mummified fetuses *in utero*. The fetal deaths were attributed to hypoxia caused by maternal methemoglobinemia.

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. Female guinea pigs were given 0, 300, 2500, 10,000 or 30,000 ppm potassium nitrate in drinking water for 204 days (Sleight et al., 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 nitrate at a dose equivalent to 1130 mg/kg.

(i) Mutagenicity. Potassium nitrate was positive in a Chinese Hamster Ovary (CHO) cell chromosome aberration test (Ishidate et al., 1977).

(j) Carcinogenesis. 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, 1991a). 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).

(k) Ecotoxicology. The LC_{50} for a 28-day exposure of annelid worms averages 2230 µg KNO_3/L (Reish, 1970). The 24-, 48-, and 96-hour LC_{50}'s for adult female mosquitofish (*Gambusia affinis*) have been determined to be 58,500; 31,100; and 22,500 µg/L, respectively (Wallen et al., 1957). Other data collected from *Daphnia*, algae, and many fish species suggest potassium nitrate toxicity is relatively low (USEPA, 2007b).

(11) Red Gum (Eucalyptus Oil). Red Gum may refer to any of a number of species of Eucalyptus or the closely related *Corymbia spp.*, including River Red Gum (*Eucalyptus camaldulensis*) and Mari (*Corymbia calophylla*). It is obtained by steam distillation of fresh leaves of the *E. globulus* tree, which is native to Australia and cultivated in subtropical regions of Europe, Asia, Africa, and the United States. Red gum is a mixture of natural organic compounds, including eucalyptol (70-80%), α-pinene, *d*-limonene, *p*-cymene, α-phellandrene, and 1-α-terpineol. It is practically insoluble in water but can be dissolved in 5 volumes of 70% alcohol (O'Neil, 2006). Eucalyptus oil has a long history of safe medicinal uses and has been classified by Food and Drug Administration (FDA) as a Generally Recognized as Safe (GRAS) substance and permitted as a direct additive to foods for human consumption. The primary constituent of eucalyptus oil is cineole or 1,3,3-trimethyl-2-oxabicyclo[2.2.2]octane (CAS RN: 470-82-6). It is used as a component of decongestant products, as an expectorant component of cough and cold products. Red gum is used as an astringent and fragrance in soaps, lotions, and cosmetics, such as Noxema® skin cream (HPD, 2007). Traditionally, red gum has been used as a topical antiseptic and analgesic as well as an expectorant. The amount of eucalyptus oil allowed in cough drops is 2000 ppm (USEPA, 2006c).

(a) Acute Oral.

i. Accidental ingestion is the most common means of human exposure to red gum, through its traditional use as an antiseptic and analgesic and its industrial use as an astringent and fragrance. Ingestion of red gum depresses the central nervous system and respiratory system and causes abdominal pain, vomiting, and diarrhea (Tibballs, 1995).

ii. The LD_{50} values for eucalyptus oil is reported as 4400 mg/kg for rats and 3320 mg/kg for mice (USEPA, 2006c). Red gum is considered slightly toxic based on the oral LD_{50} value of 2480 mg/kg for rats (Ford et al., 1988).

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No genotoxicity was observed following exposure of eucalyptus oil to *Salmonella* strains TA100, TA1535, TA1537, and TA98 in tests with or without activation by rat and hamster liver S9 fractions (NTP, 2009). An additional study reported that eucalyptol, the main component of eucalyptus oil, was negative for mutation in *S. typhimurium* strains TA100, TA97A, TA98, and TA102 (Gomes-Carneiro, 1998).

(j) Carcinogenesis. There are no chronic or sub-chronic effects of red gum reported in the literature, but studies show that red gum produces weak tumor-promoting activity on mouse skin (Roe et al., 1965).

(k) Ecotoxicity. Population growth rate was affected in blue-green algae exposed to cineole for 5-days at 100 μM (Schrader et al., 1998). Mortality occurred in fathead minnow (*Pimphales promelas*) at exposures exceeding 95,400 μg/L for four days (Geiger et al., 1988). Lettuce exposed to cineole for 72 hours was reported to have an effect on germination and growth. An EC_{50} was reported at 13.2 mM concentration (Reynolds, 1987).

(12) Elemental Silicon. Silicon, also known as polycrystalline silicon powder, does not occur free in nature. Silicon is found as silica (quartz, sand, sandstone) or as silicate (feldspar or orthoclase, koalinite, anorthorite, etc.). It constitutes about 27% of the earth's crust, and is the second most abundant element on earth (O'Neil, 2006). Silicon is prepared industrially by carbon reduction of silica in an electric arc furnace. Very pure silicon is obtained by decomposition of silicon tetraiodide. For the past century, silicon has been produced almost exclusively by carbothermal reduction of silicon dioxide. Silicon is practically insoluble in water but is soluble in molten alkali oxides. It reacts to fluorine and chlorine and is decomposed by hydrofluoric and nitric acids. Silicon is also used in the manufacturing of transistors, silicon diodes and semiconductors, and for making such alloys as ferrosilicon, silicon bronze, and silicon copper. It is also used as a reducing agent like aluminum in high temperature reactions (O'Neil, 2006). The chemical dangers associated with silicon are that it reacts violently with oxidants and halogens causing a fire hazard, and reacts on heating with water forming flammable/explosive gas. The occupational exposure limits for silicon expressed as Threshold Limit Value (TLV) is 10 mg/m^3, for Occupational Safety and Health Administration (OSHA) is a Time Weighted Average (TWA) of 5-15 mg/m^3, and for National Institute for Occupational

Safety and Health (NIOSH) the TWA is 5-10 mg/m^3 (NIOSH, 2009). Silicon occurs naturally as oxides. In humans, silicon exposure has been found to cause increased renal silicon levels and can deposit in the eyes, ears, and nasal passages. In rats, dogs, and guinea pigs, silicon is only slightly toxic. Although the International Agency for Research on Cancer does not have a cancer classification for silicon, crystalline silica inhaled in the form of quartz or cristobalite from occupational sources is carcinogenic to humans (Group 1), and amorphous silica is not classifiable as to its carcinogenicity to humans (Group 3) (WHO, 1997). No ecotoxicity information was found pertaining to silicon.

(a) Acute Oral. The LD_{50} in rats is 3,160mg/kg (SciLab, 2009).

(b) Subchronic Oral. No data found.

(c) Chronic Oral. In humans, silicon exposure has been found to cause increased renal silicon levels and can deposit in the eyes, ears, and nasal passages (WHO, 1997).

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenesis. No data found.

(k) Ecotoxicology. No data found.

(13) 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 World Health Organization (WHO) lists sulfur as a technical grade active ingredient of pesticides unlikely to present an 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 et al., 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 et al., 1977). No deaths were observed when rabbits were fed 98% 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) 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 et al., 1940).

(c) 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, 1991b).

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 ≈ 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 et al., 1940).

(d) Acute Inhalation.

i. The acute inhalation LC_{50} for 98% sulfur in rats was found to be >2.56 mg/L and >5.74 mg/L for 80% sulfur (USEPA, 1982; USEPA, 1988). NIOSH has established a short-term exposure limit of 5 ppm (13 mg/m^3) for sulfur dioxide; the Immediately Dangerous to Life or Health (IDLH) level is 100 ppm. 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 et al., 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 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 mg/m^3 (Treon et al., 1950).

(e) 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 sufur dioxide for 4 hours/day, 5 days/week, for 19-74 days (Goldring et al., 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, 1991b).

(f) Chronic Inhalation. The NIOSH TWA exposure limit is 2 ppm (5 mg/m^3) for sulfur dioxide. The corresponding 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).

(g) Dermal. LD_{50} for 98% sulfur from acute dermal rabbit study is >2,000 mg/kg (Baker, 1976).

(h) Reproduction and Development. No data found.

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

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

(k) 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% sulfur. A beneficial insect study on *Apis mellifera* (honey bee) demonstrated that a 98% dust and a 92% wettable powder is low in toxicity through contact and ingestion (Borthwich et al., 1982).

ii. The 96-hour LC_{50} values for bluegill sunfish and rainbow trout are >180 ppm using a 99.5% sulfur dust formulation. In the same report, the 48-hour LC_{50} for *Daphnia* and the 96-hour LC_{50} for mysid shrimp were quoted as >5000 ppm and 736 ppm, respectively, in a study using 90% sulfur (USEPA, 1982). Exposing goldfish to 16,000 ppm sulfur for 5 hours in turbid water conditions resulted in 100% mortality; exposure to 1600 ppm in tap water for 3.5-5.25 hours also proved fatal (HSDB, 2008).

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

(14) 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. The molecular weight of VAAR is variable. Using the VAAR CAS RN, a search of the TOXNET database returns one CIDPL record which contains no toxicity data (CIDPL, 2009d). The VAAR product is used in the formulation for black smoke (Pyro 05-01), as well as several other systems. The formulation for the current Black Smoke configuration (as of 7/22/2009) contains 1% VAAR. The percent composition for VAAR is 72% methyl acetate, 23% vinyl acetate, 5% polyvinyl alcohol, and 13% 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.

(15) 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.

(a) Acute Oral. From the CIDPL database, the oral LD_{50} for rat is >5 g/kg and for rabbit is 3705 mg/kg (CIDPL, 2009b).

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. Inhalation data include rat Lethal Concentration Low (LCLo) 32000 ppm/4 hrs, mouse LCLo 34 gm/m^3/4 hrs, and human Toxic Concentration Low (TCLo) 15000 mg/m^3 (CIDPL, 2009b).

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. Methyl acetate is negative in Ames mutagenicity assay both with and without S9 activation (CCRIS, 2009b).

(j) Carcinogenicity. No data found.

(k) Ecotoxicity. Little aquatic toxicity data is available; fathead minnow LC_{50} was reported to be between 320,000-408,000 ug/L (USEPA, 2007a).

(16) Vinyl acetate (CAS RN 108-05-4).

(a) Acute Oral. The acute toxicity of vinyl acetate is low: oral LD50's for rat and mouse are 2900mg/kg and 1600 mg/kg respectively (CIDPL, 2009e).

(b) Subchronic Oral. No data found.

(c) Chronic Oral. No data found.

(d) Acute Inhalation. LC50 inhalation values are 11,400 mg/m3/4 hr (rat), 1550 ppm/4 hr (mouse), and 2500 ppm/4hr (rabbit) (CIDPL, 2009e).

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. A group of 96 Sprague-Dawley rats (sex not specified) were exposed to vinyl acetate for 4 hours/day, 5 days/week for 52 weeks to a maximum tolerated concentration of 8.8 g/m^3 (2500 ppm) vinyl acetate in air. No tumors were reported to have occurred during 135 weeks. Early mortality was high; 49 animals survived for 26 or more weeks (IARC, 1995).

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. 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).

(j) Carcinogenicity.

i. Vinyl acetate is rapidly transformed into acetaldehyde in human blood and animal tissues. There is sufficient evidence in experimental animals for the carcinogenicity of acetaldehyde. Both vinyl acetate and acetaldehyde induce nasal cancer in rats after administration by inhalation (IARC, 1995).

ii. Chronic and reproductive toxicology studies have been performed for vinyl acetate. In inhalation studies using rats and mice, animals were exposed to 50, 200, or 600 ppm vinyl acetate in air. A small percentage of early deaths in mice, attributable to respiratory lesions, were possibly associated with inhalation of 600 ppm vinyl acetate. Body weight gain was lower in both rats and mice at higher concentrations. In the nasal cavity, atrophy of the olfactory epithelium with replacement by different cell types was noted in both species at 200 and 600 ppm. Epithelial hyperplasia of the trachea was noted at 200 and 600 ppm in mice only. In the lungs of both species, foamy histiocytes, epithelial exfoliation and fibroepithelial tags were noted at 600 ppm. Mice demonstrated epithelial hyperplasia at 600 ppm and fibroepithelial tags were noted at 200 ppm. Changes were noted at 53 weeks into the study and showed little progression for the remainder of the time. Eleven tumors were found in the nasal cavities of rats at the 600 ppm exposures; eight were benign papillomas of various cell types and three were squamous carcinomas. There was no evidence that vinyl acetate exposure caused adverse systemic effects and 50 ppm was a clear no observable effect (NOEL) level (Clary, 1988).

iii. In a chronic 2-year toxicity/carcinogenicity study following *in utero* exposure of rats to vinyl acetate via drinking water, dose-related reductions in water and food intake, and body weight gain were seen. An increase in relative kidney weight in high dose males was the only

treatment-related organ effect. A two-generation reproduction study was conducted in rats exposed via drinking water. A decrease in fertility was seen in the 5000 ppm group, attributable to males (Mebus et al., 1995).

v. There is little evidence that vinyl acetate causes developmental or reproductive effects. Teratogenicity was evaluated in mated female Sprague Dawley rats (23/group) orally exposed to vinyl acetate in their drinking water at concentrations of 0, 200, 1000 or 5000 ppm on gestation days (GD) 6-15 (Hazelton, 1980). There was a significant difference observed between treated and control animals in increased mean pregnancy rates (high-dose group). There were no significant differences observed between treated and control animals in the following: maternal mortality, body weight, body weight gain and food intake, mean pregnancy rates, numbers of corpora lutea/dam and fetal sex ratio, pre- or post-implantation losses, macroscopic fetal changes, mean litter weight and fetal weight, external/visceral and skeletal defects and crown rump length.

iv. 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 et al., 1992).

(k) Ecotoxicity. 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 370000 ug/L; behavioral effects in *Daphnia magna* were observed at an EC_{50} of 52000 ug/L and *Daphnia* mortality EC_{50} was 330000 ug/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 ug/L; guppy (*Poecilia retculata*) 31,080 ug/L, bluegill (Lepomis macrochirus) 18,000 ug/L, and goldfish (*Carassius auratus*) 42,330 ug/L (USEPA, 2007a). Significant quantities were also necessary for toxicity measured by changes in growth/biomass in corn and bean (*Phaseolus vulgaris*; (Miller et al., 1980)).

(17) Polyvinyl alcohol (CAS RN 9002-89-5). Polyvinyl alcohol is a polymer prepared from polyvinyl acetates. It is used as a pharmaceutic aid and ophthalmic lubricant and in the manufacture of surface coatings (CIDPL, 2009c).

(a) Acute Oral. The rat oral LD_{50} is > 20000 mg/kg and the mouse oral LD_{50} is 14,270 mg/kg (FAO/WHO, 2004).

(b) Subchronic Oral. Based on rat 90-day and two-generation toxicity studies, the FAO/WHO identified a NOEL of 5000 mg/kg and calculated an acceptable daily intake (ADI) of 50 mg/kg-day (FAO/WHO, 2004).

(c) Chronic Oral. No data found.

(d) Acute Inhalation. No data found.

(e) Subchronic Inhalation. No data found.

(f) Chronic Inhalation. No data found.

(g) Dermal. No data found.

(h) Reproduction and Development. No data found.

(i) Mutagenicity. No data found.

(j) Carcinogenicity. No data found.

(k) Ecotoxicity. No data found.

(18) Combustion Products. The NASA/GLENN model was used to predict production of combustion for five compounds (or their surrogates) that had properties within the database (potassium nitrate, silicon oxide, aluminum, sulfur, and boric acid). Products of complete combustion consist primarily of aluminum oxides, borates, aluminum and silicon oxides, potassium and nitrogen oxides. The predominant products were oxides of aluminum and potassium; both are considered relatively benign from an environmental perspective. A detailed listing of the results is provided at Appendix B.

Table 4. List of Physical and Chemical Properties.

Compound	MW	bp (°C)	Aq. sol. (mg/L)	log Kow[26]	log Koc	Henry's Law Constant	vp (mmHg)
Boron	10.8[1]	2550 [2]	Insoluble in water[1]	nd	nd	n/a	1.1856 x10^{-2} at 2140 °C (exp)[3]
Charcoal	12.01[4]	4200[5]	Insoluble[23]	n/a	n/a	n/a	n/a
Epikure 3125™	nd	nd	nd	nd	nd	nd	< 0.013 at 20 °C [4]
Polyamino Amide	varies	n/a	n/a	n/a	n/a	n/a	n/a
Triethylenetetramine	146.1[4,8]	266.5[4]	4.77 x 10^{6} [4,8]	2.65 (Est) [4]	nd	1.66 x 10^{-11} [4]	< 1 mmHg@20°C [4,8]
Epon 828™	320.8[6]	>260 [15]	Negligible[7]	nd	nd	nd	0.03 @ 77°C [4]
Iron Oxide - Red	159.76[8]	n/a	Insoluble[8]	n/a	n/a	n/a	n/a
Potassium Benzoate	112.13[9]	249.2@760 mmHg[9]	3.5x10^{3}, [10]	1.87 [11]	nd	0.3x10^{-8} atm-cu m/mole at 25°C (estimated) [12]	7.0x10^{-4}@25°C[13]
Potassium Chlorate	122.55[1]	400[14]	71 70,000 at 25°C (exp) [15, 16]	-4.22 (est)[17]	n/a	n/a	6.2 x10^{-17} [18]
Potassium Nitrate	101.10[19]	400[20]	1g/2.8ml[1]	n/a	n/a	n/a	n/a
Red Gum	variable	variable	variable	nd	nd	nd	variable
Silicon	28.09[1]	2355 [9]	insoluble[1]	n/a	n/a	n/a	n/a
Sulfur	32.06 [9]	444.6[21]	insoluble[1]	n/a	n/a	n/a	3.95x10^{-6}@30°C[22]

[1] O'Neil, 2006
[2] USEPA, 2006b
[3] Searcy, et al., 1957
[4] SciLab, 2009
[5] Sax, et al., 1989
[6] NCBI,
[7] CCOHS, 2007
[8] HSDB, 2008
[9] Lide, 2006-2007
[10] Yalkowsky, et al., 1992
[11] Hansch, et al., 1995
[12] USEPA, 2005
[13] McEachern, et al., 1973
[14] Clayton, et al., 1993
[15] WHO, 2005b
[16] Shiu, et al., 1990
[17] Meylan, et al., 1991
[18] Ohe, 1976
[19] Dean, 1992
[20] Weast, 1979
[21] EnvCan, 1981
[22] Ashford, 1994

Compound	MW	bp (°C)	Aq. sol. (mg/L)	log Kow[26]	log Koc	Henry's Law Constant	vp (mmHg)
Table 4. List of Physical and Chemical Properties - Continued							
Vinyl/polyvinyl Acetate (VAAR) (components listed below)	Monomer 86.09[8]	72.7[8]	insoluble[23]	n/a	60[8]	5.1×10^{-4} [8]	90.2@20 °C [8]
Methyl acetate[24]	74.08[1]	56.8[26]	Soluble[1]	0.18[26]		1.15×10^{-4} [26]	216 [26]
Vinyl acetate[24]	86.09[25]	72.3[1]	2.0×10^{4} [1]	0.73 [26]		5.11×10^{-4} [26]	90.2 [26]
Polyvinyl alcohol[24]	varies [1]	200 decom.[1]	varies [1]	unk [1]		unk [1]	unk [1]

n/a = not applicable nd = no data found

[23] Hawley, 1981
[24] Surrogate component of vinyl/polyvinyl acetate
[25] Thurman, et al., 1992

Table 5. Toxicological data.

Compound	Acute LD_{50} (mg/kg)	Sub-acute (mg/kg-d)	Sub-chronic (mg/kg-d)	Chronic (mg/kg-d)	Carcinogenicity	Mutagenicity
Boron	560[26]	nd	>72.8 mg/m^3 [27]	LOAEL 13.[28]	nd	nd
Charcoal	>2,000[29]	nd	>2,000[30]	>2,000 [30]	nd	nd
Epikure 3125™	nd[31]	nd	nd	nd	nd	
Polyamino Amide	>2,000 (rat)[32]	nd	n/a	n/a	Positive [33]	
Triethylenetetramine	nd	nd	nd	>2,000[34]	nd	Positive[34,,35]
Epon 828™	>2,000[36]	nd	n/a	n/a	Positive [37]	Positive[38]
Iron Oxides Red	nd	nd	nd	nd	nd	nd
Potassium Benzoate	LD_{50} - 1714[39]	nd	16-1090[40]	nd	nd	Negative[41]
Potassium Chlorate	LD_{50} - 1,500[42]	nd	50 [42]	nd	2000 mg/L[43]	nd
Potassium Nitrate	LD_{50} - 3750[44]	nd	methemoglobin @ 300[45]	methemoglobinemia at 1130[46]	equivocal[47]	Positive[48]

[26] CIDPL, 2009a
[27] Stokinger, 1981.
[28] Heindell, et al., 1992.
[29] RTECS, 2006
[30] Nau, et al., 1976
[31] No data found
[32] Hexion Specialty Chemicals, 2005
[33] DePass, et al., 1987
[34] BUA, 1995
[35] Mortelmans, et al., 1986
[36] Hine, et al., 1958
[37] IRIS, 2008
[38] Hauptman, et al., 1978
[39] SCCP, 2005
[40] Smyth, et al., 1969
[41] OECD SIDS, 2001
[42] Gosselin, et al., 1977, NRC, 2005, SciLab, 2009
[43] NTP, 2005b
[44] Sax, et al., 1989
[45] Til, et al., 1988
[46] Sleight, et al., 1968
[47] Bouchard, et al., 1992
[48] Ishidate, et al., 1977

Compound	Acute LD_{50} (mg/kg)	Sub-acute (mg/kg-d)	Sub-chronic (mg/kg-d)	Chronic (mg/kg-d)	Carcinogenicity	Mutagenicity
Red Gum	LD_{50} .2480[49]	nd	na	na	Negative[50]	Negative[51]
Table 5. Toxicological data – Continued.						
Silicon	LD_{50} .3160[52]	nd	na	na	nd	nd
Sulfur	LD_{50} .>5000[53]		No effect @ 20 [54]	No effect @ 50 for 100d[54]	Negative[55]	Negative[55]
Vinyl/polyvinyl Acetate (VAAR) (components listed below)	nd	nd	nd	nd	Positive[56]	Positive[57]
Methyl acetate[58]	> 5000 (rat) 3705 (rabbit)[59] LCLo 32,000 ppm/ 4hr [59]	n/a	n/a	n/a	Negative [60]	nd
Vinyl acetate[24]	2900 (rat) 1600 (mouse) 11,400 mg/m3/4hr (rat), 1550 ppm/4hr (mouse), and 2500 ppm/4hr (rabbit) [59]	TWA 10 ppm STEL 15 ppm [59]	n/a	n/a	Negative [61]	Pos. micronucleus and SCE [61] Carcinogenicity data is confounded; vinyl acetate is rapidly metabolized in vivo to acetaldehyde [62]
Polyvinyl alcohol[24]	> 20,000 (rat) 14,270 (mouse) [63]	n/a	n/a	ADI 50 mg/kg/bw/d [63]	nd	nd

n/a= not applicable; nd = no data

[49] Ford, et al., 1988
[50] Roe, et al., 1965
[51] NTP, 1982
[52] ARS, 1966
[53] Doull, et al., 1977
[54] Greengard, et al., 1940
[55] ATSDR, 1998a, ATSDR, 1998b
[56] Clary, 1988
[57] IARC, 1995
[58] Surrogate component of vinyl/polyvinyl acetate
[59] CIDPL, 2009b
[60] CCRIS, 2009b
[61] IARC, 1995
[62] Thurman, et al., 1992
[63] FAO/WHO, 2004

Table 6. Human Health Impact Assessment

Compound	Acute toxicity	Sub-chronic toxicity	Acute inhalation	Sub-chronic inhalation	Cancer probability	Comments
Boron	Low	Low	Low	nd	unknown	
Charcoal	Low	Low	Low	Low	Low	Effects similar to other non-soluble irritants.
Epikure 3125™	Low	Low	Low-moderate	n/a	Low	Body weight reductions, hemapoetic cell proliferations, non-specific, mild effects, in general.
Polyamino Amide	nd	nd	nd	nd	nd	
Tetraethylenetetramine	Low	Low	nd	nd	Low	Positive in AMES mutagenicity test, dermal sensitizer; teratogenic in animals.
Epon 828™	Low	unknown	unknown	unknown	Moderate	Reticulum cell sarcoma and lymphoblastic lymphosarcoma tumors observed after dermal application of a 10% solution (female mice) Shell Corp. contacted for additional data.
Iron Oxide Red	Low	Low	Low	Low	Low	
Potassium Benzoate	Low	Low	unknown	unknown	Low	Few adverse effects; reproductive and developmental studies conducted w/o adverse effects.
Potassium Chlorate	Low-Mod	Low-Mod	Low-Mod	Low	Low	May cause erythrolysis at high doses (anemia, kidney effects). Some states regulate as "chlorates" (e.g. Maine = 7 ug/L water guideline for chlorate ion; CA = 800 ug/L). No cancerous effects noted (Kurokawa et al., 1985). Toxic dose to a human varies between 5 grams and 35 grams
Potassium Nitrate	Low	Low	nd	nd	Low	Causes erythrolysis (anemia) at high concentrations, some nitrates may from N-nitroso compounds in gut leading to carcinogenesis.
Red Gum	Low	Low	nd	nd	Low	Classified by FDA as GRAS substance. Weak tumor promoting activity on mouse skin (Roe et al., 1960).
Elemental silicon	Low	Low	Low	Low	Low	
Sulfur	Low	n/a	Low-Mod	n/a	Low	Mild laxative effect from acute exposures. SO_2: STEL = 2 ppm; REL = 5 ppm; PEL = 13 ppm. Respirator recommended.
Polyvinyl Acetate (VAAR)	Low	Low	Low-Mod	Moderate	Low	Vinyl acetate is a possible concern if VAAR is a major component of formulation.

Table 7. Ecotoxicology Impact Assessment.

Compound	Aquatic	Invertebrate	Plants	Mammalian	Avian	Comments
Boron	Low	Low	Low	Low	Low	
Charcoal	Low (carp)	Low (modeled)	Low (expected)	Low (rats, mice)	Low (bobwhite, inhal.)	Non reactive in many biological systems.
Epikure 3125™	Low-moderate (fish; triethyleneamine)	nd	nd	Low (expected based on LD_{50})	Low (based on LD_{50} for triethylenea mine)	Not expected to be toxic given the amounts used in combustion formulations.
Polyamino Amide	nd	nd	nd	Low	nd	
Triethylenetetramine	Mod	nd	Low	Low	nd	
Epon 828™	nd	nd	nd	Low	nd	
Iron Oxide - Red	Low (mayfly)	Low (mayfly)	Low-moderate (chlorophyll; soybean, oat)	Low (small mammals, mink)	nd	Data from both oxides integrated in assessment.
Potassium Benzoate (Benzoic acid, sodium salt)	Low (fish, *Daphnia*, snail, worms)	Low (snail, copepods, worms)	nd	Low (mice, rats)	nd	Both potassium and sodium benzoate produce benzoic acid in water; data from both integrated in assessment.
Potassium Chlorate (chloric acid, potassium salt)	Low (*Daphnia*, mayfly, caddisfly, fish)	Low (*Daphnia*, mayfly, caddisfly, earthworm)	Low-moderate (algae, soybean, sorghum, cucumber)	Low (mice, rats)	Low (mallard, bobwhite)	Few subchronic (sublethal) data.
Potassium/Sodium Nitrate	Low (*Daphnia*, algae, fish spp.)	Low (earthworms, *Daphnia*)	Low (algae)	Low (mice, rats)	nd	Any toxicity would be expected from the nitrate anion (expected low for all species).
Red Gum	Low (*Pimphales*, algae)	nd	Low (algae, lettuce)	Low (mice, rats)	nd	Toxicity expected to be for other organisms.
Elemental Silicon	Low	Low	Low	Low	Low	Determination based on the inert properties of environmental silica and not from experimental data per se.
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.
Vinyl/Polyvinyl Acetate (VAAR)	Low	Low	Unk	Low	unk	Data represent those available for vinyl acetate, a primary component of VAAR.

8. DISCUSSION.

a. Environmental problems associated with perchlorate use are widespread and in the forefront of public awareness (Mayer et al., 2006). Perchlorate has been found in the environment at concentrations shown to elicit adverse effects in some species (USACHPPM, 2007). Awareness of concentrations of perchlorate found in ground waters and surface waters in the southwestern and northeastern United States (Callahan et al., 1998; Sterner et al., 1998) have halted training exercises and may have a direct impact on the readiness of our forces. Because of the impact on military training and operations, replacements for these compounds and rigorous assessment of alternatives are essential.

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 the simulator formulations resulted in the information below. Industrial standards set by the NIOSH and the OSHA are also included. No additional requirements were discovered.

(1) Boron. EPA has established a RfD for humans for boron of 0.2 mg/kg-day (USEPA, 2006b), based on developmental effects in rats from two studies. Based on an oral LD_{50} value of 650 mg/kg for rats, boron is classified as slightly toxic to humans (CIDPL, 2009a).

(2) Triethylenetetramine. Workplace Environmental Exposure Level (WEEL): 8-hour TWA 1 ppm skin (HSDB, 2008). Primary concerns for tetraethylenetetramine are in the area of dermal exposure and possible teratology as indicated in animal studies.

(3) Iron Oxide, Red. NIOSH has established a PEL of TWA 15 mg/m^3 for total particulates, and TWA 5 mg/m^3 for respirable particles (NIOSH, 2009).

(4) Potassium chlorate. Potassium chlorate is subject to state drinking water guidelines in California (800 µg chlorate/L) and Maine (7 µg chlorate/L) (HSDB, 2008).

(5) Potassium nitrate. 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, 2008).

(6) Silicon. Elemental silicon does not occur naturally. It is considered a nuisance particulate. NIOSH has established a TWA REL at 10 mg/m^3 (5 mg/m^3 respirable particles). OSHA has established a PEL of 15 mg/m^3 (5 mg/m^3 resipirable particles) (NIOSH, 2009).

(7) Sulfur. Sulfur is generally considered safe in applications involving pesticides, and is approved as an indirect food additive as a component of adhesives (HSDB, 2008). Elemental sulfur is not regulated by NIOSH or OSHA. Toxicity of elemental sulfur is very low; its low

solubility in water makes its primary hazard one of exposure to solid sulfur applied as a pesticide. The combustion products SO_2 and SO_3 represent a greater risk, but if quantities of sulfur in the formulation are not large, and the devices are used in an open-air environment, there is little risk to humans. Extended use in the same area could eventually result in acidification of soil and water, so monitoring during periods of heavy or extended use may be desirable.

(8) VAAR. 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, 2009e). The FDA has approved the use of vinyl acetate as a food starch modifier (21CFR172.892) and as the copolymer vinyl acetate/crotonic acid for food packaging (21CFR175.350).

(9) Combustion Products. While the NASA/GLENN modeling gives some indication of combustion products, it is not completely accurate as the modeling is based on equilibrium concentrations at very high temperatures, as in the combustion temperatures of rocket and missile propellants. However, combustion of any compound containing carbon, nitrogen or sulfur will certainly produce oxides of those elements. When present in sufficient concentration in an enclosed place, such oxides should be considered toxic. However, if the ignition of the device occurs in open air, normal wind dispersion of the combustion products is expected to make toxic impact of the combustion products negligible.

c. Environmental degradation products depend upon many factors, including but not limited to ignition efficiency, ambient temperature and humidity, site-specific media characteristics (e.g. soil pH, organic carbon content, cation exchange capacity, etc.) and other media and site specific parameters.

d. Regardless, from an environmental perspective the proposed formulation is a great improvement over previous formulations that integrate perchlorate as an oxidizer. Replacing antimony compounds also has added benefit in that antimony is persistent and has environmental toxicological properties. Many of the proposed alternative compounds are relatively benign from an environmental fate and effects viewpoint.

e. Areas of uncertainty.

(1) The mode of action of potassium chlorate is unknown, but appears to be different from that of perchlorate in that it has been reported to affect red blood cells and cause anemia at relatively high exposure levels. The chlorate anion is mobile in the environment, and may reach ground water if sufficient quantities are used. Some states regulate as "chlorates" (e.g. Maine = 7 µg/L water guideline for chlorate ion; CA = 800 µg/L), though the standards vary between jurisdictions.

(2) There is some concern over triethylenetetramine, especially with respect to dermal sensitization. However, this is primarily a manufacturing concern, and should be addressable through appropriate engineering controls or personal protective equipment (PPE). The total amount present in the formulation is likely to lead to negligible risk upon functioning of the simulator.

(3) Toxicity information was not available for Epon and Epikure.

(4) Despite apparently being little studied, VAAR appears not to be considered hazardous as such. Furthermore, in this 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 M118-M119 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.

9. RECOMMENDATIONS. At this time Work Unit PYRO 06-08 appears to integrate a relatively benign environmental and human health, perchlorate-free alternative group of formulations. There do not appear to be significant health issues resulting from the use of these replacement compounds. The toxicology of chlorates and the synthetic plasticizers and binders merit closer scrutiny. However, the available evidence and relatively small amounts used in these formulations suggest health risks are relatively low. It is recommended that this formulation proceed to program implementation.

APPENDIX A

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APPENDIX B

PRODUCTS OF COMBUSTION: NASA/GLENN PREDICTIONS

Combustion Products of WF-9

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*************************************************************************
*

     NASA-GLENN CHEMICAL EQUILIBRIUM PROGRAM CEA, NOVEMBER 5, 1999
          BY  BONNIE MCBRIDE And SANFORD GORDON
     REFS: NASA RP-1311, PART I, 1994 And NASA RP-1311, PART II, 1996

*************************************************************************
*



reac

  name   S   S 1          wt%=8        t,k=298.15    h,kcal/mol=0
  name   Si  Si 1           wt%=6        t,k=298.15    h,kJ/mol=0
  name CC 1              wt%=2        t,k=298.15    h,kcal/mol=0
  name   KNO3 K 1 N 1 O 3       wt%=81       t,k=298.15    h,kcal/mol=-118.08
  name VAAR   C 34 O 17 H 54          wt%=3      t,k=298.15    h,kJ/mol=-2664.9

prob  case=wf9      hp eqil p(psia)=14.7
outp cal massf
end

 OPTIONS: TP=F  HP=T  SP=F  TV=F  UV=F  SV=F  DETN=F  SHOCK=F  REFL=F
INCD=F
 RKT=F  FROZ=F  EQL=T  IONS=F  SIUNIT=F  DEBUGF=F  SHKDBG=F  DETDBG=F
TRNSPT=F

 TRACE= 0.00E+00  S/R= 0.000000E+00  H/R= 0.000000E+00  U/R= 0.000000E+00

 P,BAR =    1.013525

   REACTANT        WT.FRAC   (ENERGY/R),K   TEMP,K  DENSITY
     EXPLODED FORMULA
N: S             0.080000   0.000000E+00   298.15  0.0000
       S  1.00000
N: Si            0.060000   0.000000E+00   298.15  0.0000
       SI 1.00000
```

```
N: C          0.020000  0.000000E+00  298.15  0.0000
     C  1.00000
N: KNO3       0.810000  -0.594198E+05  298.15  0.0000
     K  1.00000  N  1.00000  O  3.00000
N: VAAR       0.030000  -0.320512E+06  298.15  0.0000
     C 34.00000  O 17.00000  H 54.00000
```

SPECIES BEING CONSIDERED IN THIS SYSTEM
(COndENSED PHASE MAY HAVE NAME LISTED SEVERAL TIMES)
LAST thermo.txt UPDATE: 11/08/99

```
g 7/97  *C          tpis79  *CH          g 8/99  CH2
g 8/99  CH3         g12/92  CH2OH        g10/92  CH3O
g 8/99  CH4         g 8/88  CH3OH        g 8/99  *CN
g12/89  CNN         tpis79  *CO          g 6/95  COS
g 9/99  *CO2        tpis91  COOH         g 7/99  CS
g 6/95  CS2         tpis91  *C2          g 1/91  C2H
g 6/89  CHCO,ketyl      g12/89  C2H2,vinylidene  g 1/91  C2H2,acetylene
g 5/90  CH2CO,ketene    g 2/92  C2H3,vinyl      g12/92  CH3CN
g 6/96  CH3CO,acetyl    g 1/91  C2H4           g 8/88  C2H4O,ethylen-o
g 8/88  CH3CHO,ethanal  g 8/88  CH3COOH        g12/92  C2H5
g 8/88  C2H6            g 8/88  CH3N2CH3       g 8/88  C2H5OH
g12/92  CH3OCH3         g12/92  CCN           tpis91  CNC
tpis79  C2N2          g12/89  C2O           tpis79  *C3
x 4/98  C3H3,1-propynl  x 4/98  C3H3,2-propynl  g12/92  C3H4,allene
g12/92  C3H4,propyne    g 5/90  C3H4,cyclo-     bur 92  C3H5,allyl
g 2/95  C3H6,propylene  g 1/93  C3H6,cyclo-     g 6/90  C3H6O
g 6/90  C3H7,n-propyl   g 9/85  C3H7,i-propyl   g 6/90  C3H8
g 9/88  C3H8O,1propanol g 9/88  C3H8O,2propanol g 7/88  C3O2
g 7/88  C4            g 2/93  C4H2          g 5/90  C4H4,1,3-cyclo-
x10/92  C4H6,butadiene  x10/93  C4H6,1-butyne   x10/93  C4H6,2-butyne
g 5/90  C4H6,cyclo-     x 4/88  C4H8,1-butene   x 4/88  C4H8,cis2-buten
x 4/88  C4H8,tr2-butene x 4/88  C4H8,isobutene  g 5/90  C4H8,cyclo-
g 6/90  (CH3COOH)2      x10/84  C4H9,n-butyl    x10/84  C4H9,i-butyl
g 1/93  C4H9,s-butyl    g 1/93  C4H9,t-butyl    g 6/90  C4H10,isobutane
g 6/90  C4H10,n-butane  j 3/61  C4N2          g 7/88  C5
g 5/90  C5H6,1,3cyclo-  g 1/93  C5H8,cyclo-     x 4/87  C5H10,1-pentene
g 6/90  C5H10,cyclo-    x10/84  C5H11,pentyl    g 1/93  C5H11,t-pentyl
x10/85  C5H12,n-pentane x10/85  C5H12,i-pentane x10/85  CH3C(CH3)2CH3
g 2/93  C6H2            g 1/91  C6H5,phenyl     g 6/90  C6H5O,phenoxy
g 1/91  C6H6            g 6/90  C6H5OH,phenol   g 1/93  C6H10,cyclo-
x 4/87  C6H12,1-hexene  g 6/90  C6H12,cyclo-    x10/83  C6H13,n-hexyl
g 6/96  C6H14,n-hexane  g 1/93  C7H7,benzyl     g 1/93  C7H8
g 1/93  C7H8O,cresol-mx x 4/87  C7H14,1-heptene x10/83  C7H15,n-heptyl
x10/85  C7H16,2-methylh x10/85  C7H16,n-heptane x 4/89  C8H8,styrene
x10/86  C8H10,ethylbenz x 4/87  C8H16,1-octene  x10/83  C8H17,n-octyl
```

x 4/85 C8H18,n-octane x 4/85 C8H18,isooctane x10/83 C9H19,n-nonyl
g 8/93 C10H8,naphthale x10/83 C10H21,n-decyl g12/84 C12H9,o-bipheny
g12/84 C12H10,biphenyl g 6/97 *H g 7/88 HCN
g 9/96 HCO tpis89 HCCN g11/92 HNC
g 2/96 HNCO g 5/99 HNO tpis89 HNO2
g 5/99 HNO3 g 5/99 HO2 tpis78 *H2
g 8/88 HCHO,formaldehy g 8/88 HCOOH g 8/89 H2O
g 6/99 H2O2 tpis89 H2S tpis89 H2SO4
g 8/88 (HCOOH)2 g 7/97 *K J 3/66 KCN
J 3/63 KH tpis82 KNO2 tpis82 KNO3
J12/67 KO g 9/97 KOH j12/83 K2
tpis82 K2CO3 J 3/66 K2C2N2 tpis82 K2O
tpis82 K2O2 g 9/97 K2O2H2 g10/99 K2SO4
g 5/97 *N g 2/96 NCO g 4/99 *NH
g 5/99 NH2 tpis89 NH3 tpis89 *NO
g 4/99 NO2 j12/64 NO3 tpis78 *N2
g12/89 NCN g 5/99 N2H2 tpis89 NH2NO2
g 4/99 N2H4 g 4/99 N2O g 4/99 N2O3
tpis89 N2O4 g 4/99 N2O5 tpis89 N3
g 4/99 N3H g 5/97 *O tpis78 *OH
tpis89 *O2 tpis89 O3 g 5/97 *S
tpis89 SH tpis89 SN tpis89 SO
tpis89 SO2 tpis89 SO3 tpis89 S2
tpis89 S2O tpis89 S3 tpis89 S4
tpis89 S5 tpis89 S6 tpis89 S7
tpis89 S8 g 8/97 *Si tpis91 SiC
tpis91 SiC2 J12/60 SiC4H12 g 3/99 SiH
g 3/99 SiH2 g 3/99 SiH3 tpis91 SiH4
g 5/99 SiN tpis91 SiO tpis91 SiO2
tpis91 SiS tpis91 SiS2 tpis91 Si2
tpis91 Si2C J 3/67 Si2N g 7/95 Si3
x 4/83 C(gr) x 4/83 C(gr) x 4/83 C(gr)
g 8/89 H2O(s) g 8/89 H2O(L) J 9/77 H2SO4(L)
coda89 K(cr) coda89 K(L) J 3/66 KCN(s)
J 3/66 KCN(L) J 3/66 KCN(L) tpis82 KH(cr)
tpis82 KH(L) tpis82 KNO2(II) tpis82 KNO2(I)
tpis82 KNO2(L) tpis82 KNO3(a) tpis82 KNO3(b)
tpis82 KNO3(L) g 8/97 KOH(a) g 8/97 KOH(b)
g 8/97 KOH(c) g 8/97 KOH(L) tpis82 KO2(b)
tpis82 KO2(a) tpis82 KO2(L) tpis82 K2CO3(a)
tpis82 K2CO3(b) tpis82 K2CO3(L) tpis82 K2O(c)
tpis82 K2O(b) tpis82 K2O(a) tpis82 K2O(L)
tpis82 K2O2(cr) tpis82 K2O2(L) J 3/78 K2S(1)
J 3/78 K2S(1) J 3/78 K2S(2) J 3/78 K2S(3)
J 3/78 K2S(L) tpis82 K2SO4(II) tpis82 K2SO4(II)
tpis82 K2SO4(I) tpis82 K2SO4(L) tpis82 K2SiO3(cr)

tpis82 K2SiO3(cr) tpis82 K2SiO3(L) tpis82 K2Si2O5(a)
tpis82 K2Si2O5(b) tpis82 K2Si2O5(c) tpis82 K2Si2O5(L)
tpis89 S(cr1) tpis89 S(cr2) tpis89 S(L)
tpis89 S(L) tpis89 S(L) tpis89 S(L)
tpis89 S(L) tpis91 Si(cr) tpis91 Si(cr)
tpis91 Si(L) tpis91 SiC(b) tpis91 SiC(b)
tpis91 SiC(L) tpis91 SiO2(a-qz) tpis91 SiO2(b-qz)
tpis91 SiO2(b-crt) tpis91 SiO2(L) tpis91 SiS(cr)
tpis91 SiS(L) tpis91 SiS2(cr) tpis91 SiS2(cr)
tpis91 SiS2(L) g 7/95 Si2N2O(s) g 7/95 Si2N2O(s)
tpis91 Si3N4(cr) tpis91 Si3N4(cr)

O/F = 0.000000

	EFFECTIVE FUEL	EFFECTIVE OXIDANT	MIXTURE
ENTHALPY	h(2)/R	h(1)/R	h0/R
(KG-MOL)(K)/KG	-0.48913461E+03	0.00000000E+00	-0.48913461E+03

KG-FORM.WT./KG	bi(2)	bi(1)	b0i
*S	0.24948544E-02	0.00000000E+00	0.24948544E-02
*Si	0.21363337E-02	0.00000000E+00	0.21363337E-02
*C	0.30533480E-02	0.00000000E+00	0.30533480E-02
*K	0.80116127E-02	0.00000000E+00	0.80116127E-02
*N	0.80116127E-02	0.00000000E+00	0.80116127E-02
*O	0.24728921E-01	0.00000000E+00	0.24728921E-01
*H	0.22047345E-02	0.00000000E+00	0.22047345E-02

POINT	ITN	T	S	SI	C	K
		N	O	H		
1	41	1598.385	-39.911	-28.300	-31.019	-20.197
		-13.753	-15.402	-17.924		
ADD SiO2(b-crt)						
1	11	2109.888	-25.426	-34.025	-24.762	-19.134
		-14.220	-15.629	-16.049		
PHASE CHANGE, REPLACE SiO2(b-crt) WITH SiO2(L)						
1	2	2106.153	-25.483	-34.134	-24.786	-19.139
		-14.215	-15.630	-16.053		
ADD K2Si2O5(L)						
1	7	2482.171	-21.902	-27.477	-22.172	-22.302
		-14.454	-15.684	-13.612		
REMOVE SiO2(L)						
1	8	2150.656	-24.324	-37.851	-24.541	-19.582
		-14.245	-15.572	-15.542		
ADD K2SO4(L)						
1	3	2196.799	-24.003	-36.640	-24.162	-19.593
		-14.270	-15.580	-15.360		

```
ADD  K2SiO3(L)
  1   4   2152.914    -23.888    -37.181    -24.683    -20.429
            -14.233    -15.478    -14.946
REMOVE  K2Si2O5(L)
  1   3   2170.762    -23.860    -37.109    -24.479    -20.135
            -14.247    -15.514    -15.063
```

THERMODYNAMIC EQUILIBRIUM COMBUSTION PROPERTIES AT ASSIGNED PRESSURES

CASE = wf9

REACTANT		WT FRACTION (SEE NOTE)	ENERGY CAL/MOL	TEMP K
NAME	S	0.0800000	0.000	298.150
NAME	Si	0.0600000	0.000	298.150
NAME	C	0.0200000	0.000	298.150
NAME	KNO3	0.8100000	-118080.000	298.150
NAME	VAAR	0.0300000	-636926.386	298.150

O/F= 0.00000 %FUEL= 0.000000 R,EQ.RATIO= 0.828069 PHI,EQ.RATIO= 0.000000

THERMODYNAMIC PROPERTIES

P, ATM	1.0003
T, K	2170.76
RHO, G/CC	3.9125-4
H, CAL/G	-972.02
U, CAL/G	-1033.93
G, CAL/G	-3907.63
S, CAL/(G)(K)	1.3523
M, (1/n)	69.673
MW, MOL WT	60.537
(dLV/dLP)t	-1.32829
(dLV/dLT)p	6.1599
Cp, CAL/(G)(K)	2.6240
GAMMAs	1.0919
SON VEL,M/SEC	531.8

MASS FRACTIONS

*CO	0.00111
*CO2	0.13260
H2O	0.00311
*K	0.02830
KNO2	0.00021
KO	0.00214
KOH	0.10298
K2	0.00001
K2CO3	0.00012
K2O	0.00009
K2O2H2	0.00015
K2SO4	0.09341
*NO	0.00244
*N2	0.11104
*O	0.00019
*OH	0.00034
*O2	0.06339
SO	0.00016
SO2	0.12320
SO3	0.00021
K2SO4(L)	0.00522
K2SiO3(L)	0.32959

* THERMODYNAMIC PROPERTIES FITTED TO 20000.K

PRODUCTS WHICH WERE CONSIDERED BUT WHOSE MASS FRACTIONS WERE LESS THAN 5.000000E-06 FOR ALL ASSIGNED COndITIONS

*C *CH CH2 CH3 CH2OH
CH3O CH4 CH3OH *CN CNN
COS COOH CS CS2 *C2
C2H CHCO,ketyl C2H2,vinylidene C2H2,acetylene CH2CO,ketene
C2H3,vinyl CH3CN CH3CO,acetyl C2H4 C2H4O,ethylen-o
CH3CHO,ethanal CH3COOH C2H5 C2H6 CH3N2CH3
C2H5OH CH3OCH3 CCN CNC C2N2
C2O *C3 C3H3,1-propynl C3H3,2-propynl C3H4,allene
C3H4,propyne C3H4,cyclo- C3H5,allyl C3H6,propylene C3H6,cyclo-
C3H6O C3H7,n-propyl C3H7,i-propyl C3H8 C3H8O,1propanol
C3H8O,2propanol C3O2 C4 C4H2 C4H4,1,3-cyclo-
C4H6,butadiene C4H6,1-butyne C4H6,2-butyne C4H6,cyclo- C4H8,1-butene
C4H8,cis2-buten C4H8,tr2-butene C4H8,isobutene C4H8,cyclo- (CH3COOH)2
C4H9,n-butyl C4H9,i-butyl C4H9,s-butyl C4H9,t-butyl C4H10,isobutane
C4H10,n-butane C4N2 C5 C5H6,1,3cyclo- C5H8,cyclo-
C5H10,1-pentene C5H10,cyclo- C5H11,pentyl C5H11,t-pentyl C5H12,n-pentane
C5H12,i-pentane CH3C(CH3)2CH3 C6H2 C6H5,phenyl C6H5O,phenoxy
C6H6 C6H5OH,phenol C6H10,cyclo- C6H12,1-hexene C6H12,cyclo-

C6H13,n-hexyl C6H14,n-hexane C7H7,benzyl C7H8 C7H8O,cresol-mx
C7H14,1-heptene C7H15,n-heptyl C7H16,2-methylh C7H16,n-heptane C8H8,styrene
C8H10,ethylbenz C8H16,1-octene C8H17,n-octyl C8H18,n-octane C8H18,isooctane
C9H19,n-nonyl C10H8,naphthale C10H21,n-decyl C12H9,o-bipheny C12H10,biphenyl
*H HCN HCO HCCN HNC
HNCO HNO HNO2 HNO3 HO2
*H2 HCHO,formaldehy HCOOH H2O2 H2S
H2SO4 (HCOOH)2 KCN KH KNO3
K2C2N2 K2O2 *N NCO *NH
NH2 NH3 NO2 NO3 NCN
N2H2 NH2NO2 N2H4 N2O N2O3
N2O4 N2O5 N3 N3H O3
*S SH SN S2 S2O
S3 S4 S5 S6 S7
S8 *Si SiC SiC2 SiC4H12
SiH SiH2 SiH3 SiH4 SiN
SiO SiO2 SiS SiS2 Si2
Si2C Si2N Si3 C(gr) H2O(s)
H2O(L) H2SO4(L) K(cr) K(L) KCN(s)
KCN(L) KH(cr) KH(L) KNO2(II) KNO2(I)
KNO2(L) KNO3(a) KNO3(b) KNO3(L) KOH(a)
KOH(b) KOH(c) KOH(L) KO2(b) KO2(a)
KO2(L) K2CO3(a) K2CO3(b) K2CO3(L) K2O(c)
K2O(b) K2O(a) K2O(L) K2O2(cr) K2O2(L)
K2S(1) K2S(2) K2S(3) K2S(L) K2SO4(II)
K2SO4(I) K2SiO3(cr) K2Si2O5(a) K2Si2O5(b) K2Si2O5(c)
K2Si2O5(L) S(cr1) S(cr2) S(L) Si(cr)
Si(L) SiC(b) SiC(L) SiO2(a-qz) SiO2(b-qz)
SiO2(b-crt) SiO2(L) SiS(cr) SiS(L) SiS2(cr)
SiS2(L) Si2N2O(s) Si3N4(cr)

NOTE. WEIGHT FRACTION OF FUEL IN TOTAL FUELS And OF OXIDANT IN TOTAL OXIDANTS

Combustion Products of WM-2

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      NASA-GLENN CHEMICAL EQUILIBRIUM PROGRAM CEA, NOVEMBER 5, 1999
            BY  BONNIE MCBRIDE And SANFORD GORDON
       REFS: NASA RP-1311, PART I, 1994 And NASA RP-1311, PART II, 1996

*************************************************************************
*



reac
  name KBenz    K 1 C 7 H 5 O 2  wt%=17   h,kJ/mol=-610.94  t,k=298.15
  name KNO3     K 1 N 1 O 3    wt%=13  h,cal/mol=-117760.0  t,k=298.15
  name KClO3    K 1 Cl 1 O 3   wt%=67  h,kJ/mol=-389       t,k=298.15
  name RedGum   C 10 H 18 O 1   wt%=3    h,kJ/mol=-276      t,k=298.15
prob case=WM2  hp  equil  p(psia)=14.7
outp cal massf
end

 OPTIONS: TP=F  HP=T  SP=F  TV=F  UV=F  SV=F  DETN=F  SHOCK=F  REFL=F
INCD=F
 RKT=F  FROZ=F  EQL=T  IONS=F  SIUNIT=F  DEBUGF=F  SHKDBG=F  DETDBG=F
TRNSPT=F

 TRACE= 0.00E+00  S/R= 0.000000E+00  H/R= 0.000000E+00  U/R= 0.000000E+00

 P,BAR =    1.013525

   REACTANT        WT.FRAC  (ENERGY/R),K   TEMP,K  DENSITY
     EXPLODED FORMULA
 N: KBenz          0.170000  -0.734788E+05   298.15  0.0000
       K  1.00000  C  7.00000  H  5.00000  O  2.00000
 N: KNO3           0.130000  -0.592588E+05   298.15  0.0000
       K  1.00000  N  1.00000  O  3.00000
 N: KClO3          0.670000  -0.467857E+05   298.15  0.0000
       K  1.00000  CL 1.00000  O  3.00000
 N: RedGum         0.030000  -0.331950E+05   298.15  0.0000
       C 10.00000  H 18.00000  O  1.00000

 SPECIES BEING CONSIDERED IN THIS SYSTEM
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(COndENSED PHASE MAY HAVE NAME LISTED SEVERAL TIMES)
LAST thermo.txt UPDATE: 11/08/99

g 7/97 *C g 8/99 CCL g 8/99 CCL2
x12/93 CCL3 tpis91 CCL4 tpis79 *CH
g 9/99 CHCL x12/93 CHCL2 g 7/99 CHCL3
g 8/99 CH2 g 2/96 CH2CL tpis91 CH2CL2
g 8/99 CH3 tpis91 CH3CL g12/92 CH2OH
g10/92 CH3O g 8/99 CH4 g 8/88 CH3OH
g 8/99 *CN g12/89 CNN tpis79 *CO
tpis91 COCL tpis91 COCL2 tpis91 COHCL
g 9/99 *CO2 tpis91 COOH tpis91 *C2
tpis91 C2CL tpis91 C2CL2 tpis91 C2CL3
tpis91 C2CL4 tpis91 C2CL6 g 1/91 C2H
tpis91 C2HCL tpis91 C2HCL3 g 6/89 CHCO,ketyl
g12/89 C2H2,vinylidene g 1/91 C2H2,acetylene tpis91 C2H2CL2
g 5/90 CH2CO,ketene g 2/92 C2H3,vinyl tpis91 C2H3CL
g12/92 CH3CN g 6/96 CH3CO,acetyl g 1/91 C2H4
g 8/88 C2H4O,ethylen-o g 8/88 CH3CHO,ethanal g 8/88 CH3COOH
g12/92 C2H5 g 8/88 C2H6 g 8/88 CH3N2CH3
g 8/88 C2H5OH g12/92 CH3OCH3 g12/92 CCN
tpis91 CNC tpis79 C2N2 g12/89 C2O
tpis79 *C3 x 4/98 C3H3,1-propynl x 4/98 C3H3,2-propynl
g12/92 C3H4,allene g12/92 C3H4,propyne g 5/90 C3H4,cyclo-
bur 92 C3H5,allyl g 2/95 C3H6,propylene g 1/93 C3H6,cyclo-
g 6/90 C3H6O g 6/90 C3H7,n-propyl g 9/85 C3H7,i-propyl
g 6/90 C3H8 g 9/88 C3H8O,1propanol g 9/88 C3H8O,2propanol
g 7/88 C3O2 g 7/88 C4 g 2/93 C4H2
g 5/90 C4H4,1,3-cyclo- x10/92 C4H6,butadiene x10/93 C4H6,1-butyne
x10/93 C4H6,2-butyne g 5/90 C4H6,cyclo- x 4/88 C4H8,1-butene
x 4/88 C4H8,cis2-buten x 4/88 C4H8,tr2-butene x 4/88 C4H8,isobutene
g 5/90 C4H8,cyclo- g 6/90 (CH3COOH)2 x10/84 C4H9,n-butyl
x10/84 C4H9,i-butyl g 1/93 C4H9,s-butyl g 1/93 C4H9,t-butyl
g 6/90 C4H10,isobutane g 6/90 C4H10,n-butane j 3/61 C4N2
g 7/88 C5 g 5/90 C5H6,1,3cyclo- g 1/93 C5H8,cyclo-
x 4/87 C5H10,1-pentene g 6/90 C5H10,cyclo- x10/84 C5H11,pentyl
g 1/93 C5H11,t-pentyl x10/85 C5H12,n-pentane x10/85 C5H12,i-pentane
x10/85 CH3C(CH3)2CH3 g 2/93 C6H2 g 1/91 C6H5,phenyl
g 6/90 C6H5O,phenoxy g 1/91 C6H6 g 6/90 C6H5OH,phenol
g 1/93 C6H10,cyclo- x 4/87 C6H12,1-hexene g 6/90 C6H12,cyclo-
x10/83 C6H13,n-hexyl g 6/96 C6H14,n-hexane g 1/93 C7H7,benzyl
g 1/93 C7H8 g 1/93 C7H8O,cresol-mx x 4/87 C7H14,1-heptene
x10/83 C7H15,n-heptyl x10/85 C7H16,2-methylh x10/85 C7H16,n-heptane
x 4/89 C8H8,styrene x10/86 C8H10,ethylbenz x 4/87 C8H16,1-octene
x10/83 C8H17,n-octyl x 4/85 C8H18,n-octane x 4/85 C8H18,isooctane
x10/83 C9H19,n-nonyl g 8/93 C10H8,naphthale x10/83 C10H21,n-decyl

g12/84 C12H9,o-bipheny g12/84 C12H10,biphenyl g 7/97 *CL
g 6/95 CLCN tpis89 CLO g 7/93 CLO2
tpis89 CL2 tpis89 CL2O g 6/97 *H
g 7/88 HCN g 9/96 HCO tpis89 HCCN
tpis89 HCL g11/92 HNC g 2/96 HNCO
g 5/99 HNO tpis89 HNO2 g 5/99 HNO3
tpis89 HOCL g 5/99 HO2 tpis78 *H2
g 8/88 HCHO,formaldehy g 8/88 HCOOH g 8/89 H2O
g 6/99 H2O2 g 8/88 (HCOOH)2 g 7/97 *K
J 3/66 KCN J 3/66 KCL J 3/63 KH
tpis82 KNO2 tpis82 KNO3 J12/67 KO
g 9/97 KOH j12/83 K2 tpis82 K2CO3
J 3/66 K2C2N2 tpis82 K2CL2 tpis82 K2O
tpis82 K2O2 g 9/97 K2O2H2 g 5/97 *N
g 2/96 NCO g 4/99 *NH g 5/99 NH2
tpis89 NH3 tpis89 *NO g 4/99 NOCL
g 4/99 NO2 g 4/99 NO2CL j12/64 NO3
tpis78 *N2 g12/89 NCN g 5/99 N2H2
tpis89 NH2NO2 g 4/99 N2H4 g 4/99 N2O
g 4/99 N2O3 tpis89 N2O4 g 4/99 N2O5
tpis89 N3 g 4/99 N3H g 5/97 *O
tpis78 *OH tpis89 *O2 tpis89 O3
x 4/83 C(gr) x 4/83 C(gr) x 4/83 C(gr)
g 8/89 H2O(s) g 8/89 H2O(L) coda89 K(cr)
coda89 K(L) J 3/66 KCN(s) J 3/66 KCN(L)
J 3/66 KCN(L) tpis82 KCL(cr) tpis82 KCL(cr)
tpis82 KCL(L) tpis82 KH(cr) tpis82 KH(L)
tpis82 KNO2(II) tpis82 KNO2(I) tpis82 KNO2(L)
tpis82 KNO3(a) tpis82 KNO3(b) tpis82 KNO3(L)
g 8/97 KOH(a) g 8/97 KOH(b) g 8/97 KOH(c)
g 8/97 KOH(L) tpis82 KO2(b) tpis82 KO2(a)
tpis82 KO2(L) tpis82 K2CO3(a) tpis82 K2CO3(b)
tpis82 K2CO3(L) tpis82 K2O(c) tpis82 K2O(b)
tpis82 K2O(a) tpis82 K2O(L) tpis82 K2O2(cr)
tpis82 K2O2(L) BAR 73 NH4CL(a) BAR 73 NH4CL(b)

O/F = 0.000000

	EFFECTIVE FUEL	EFFECTIVE OXIDANT	MIXTURE
ENTHALPY	h(2)/R	h(1)/R	h0/R
(KG-MOL)(K)/KG	-0.41640624E+03	0.00000000E+00	-0.41640624E+03

KG-FORM.WT./KG	bi(2)	bi(1)	b0i
*K	0.78141024E-02	0.00000000E+00	0.78141024E-02
*C	0.93725755E-02	0.00000000E+00	0.93725755E-02
*H	0.88063060E-02	0.00000000E+00	0.88063060E-02

*O	0.22575701E-01	0.00000000E+00	0.22575701E-01
*N	0.12858144E-02	0.00000000E+00	0.12858144E-02
*CL	0.54671920E-02	0.00000000E+00	0.54671920E-02

POINT	ITN	T	K	C	H	O	N	CL
1	28	2370.406	-20.999	-19.360	-12.602	-17.140	-15.523	-25.450

THERMODYNAMIC EQUILIBRIUM COMBUSTION PROPERTIES AT ASSIGNED PRESSURES

CASE = WM2

REACTANT		WT FRACTION (SEE NOTE)	ENERGY CAL/MOL	TEMP K
NAME	KBenz	0.1700000	-146018.164	298.150
NAME	KNO3	0.1300000	-117760.000	298.150
NAME	KClO3	0.6700000	-92973.231	298.150
NAME	RedGum	0.0300000	-65965.583	298.150

O/F= 0.00000 %FUEL= 0.000000 R,EQ.RATIO= 1.068989 PHI,EQ.RATIO= 0.000000

THERMODYNAMIC PROPERTIES

P, ATM	1.0003
T, K	2370.41
RHO, G/CC	2.3668-4
H, CAL/G	-827.49
U, CAL/G	-929.84
G, CAL/G	-4722.57
S, CAL/(G)(K)	1.6432
M, (1/n)	46.025
(dLV/dLP)t	-1.01572
(dLV/dLT)p	1.2796
Cp, CAL/(G)(K)	0.5372
GAMMAs	1.1311
SON VEL,M/SEC	695.9

MASS FRACTIONS

*CO	0.04484
*CO2	0.34204
*CL	0.00015
*H	0.00002
HCL	0.00120
*H2	0.00025
H2O	0.06188
*K	0.03312
KCL	0.40330
KH	0.00001
KO	0.00053
KOH	0.08562
K2CL2	0.00151
K2O	0.00001
K2O2H2	0.00003
*NO	0.00043
*N2	0.01781
*O	0.00022
*OH	0.00189
*O2	0.00513

* THERMODYNAMIC PROPERTIES FITTED TO 20000.K

PRODUCTS WHICH WERE CONSIDERED BUT WHOSE MASS FRACTIONS WERE LESS THAN 5.000000E-06 FOR ALL ASSIGNED COndITIONS

*C CCL CCL2 CCL3 CCL4
*CH CHCL CHCL2 CHCL3 CH2
CH2CL CH2CL2 CH3 CH3CL CH2OH
CH3O CH4 CH3OH *CN CNN
COCL COCL2 COHCL COOH *C2
C2CL C2CL2 C2CL3 C2CL4 C2CL6
C2H C2HCL C2HCL3 CHCO,ketyl C2H2,vinylidene
C2H2,acetylene C2H2CL2 CH2CO,ketene C2H3,vinyl C2H3CL
CH3CN CH3CO,acetyl C2H4 C2H4O,ethylen-o CH3CHO,ethanal
CH3COOH C2H5 C2H6 CH3N2CH3 C2H5OH
CH3OCH3 CCN CNC C2N2 C2O
*C3 C3H3,1-propynl C3H3,2-propynl C3H4,allene C3H4,propyne
C3H4,cyclo- C3H5,allyl C3H6,propylene C3H6,cyclo- C3H6O
C3H7,n-propyl C3H7,i-propyl C3H8 C3H8O,1propanol C3H8O,2propanol
C3O2 C4 C4H2 C4H4,1,3-cyclo- C4H6,butadiene
C4H6,1-butyne C4H6,2-butyne C4H6,cyclo- C4H8,1-butene C4H8,cis2-buten
C4H8,tr2-butene C4H8,isobutene C4H8,cyclo- (CH3COOH)2 C4H9,n-butyl
C4H9,i-butyl C4H9,s-butyl C4H9,t-butyl C4H10,isobutane C4H10,n-butane
C4N2 C5 C5H6,1,3cyclo- C5H8,cyclo- C5H10,1-pentene
C5H10,cyclo- C5H11,pentyl C5H11,t-pentyl C5H12,n-pentane C5H12,i-pentane

CH3C(CH3)2CH3 C6H2 C6H5,phenyl C6H5O,phenoxy C6H6
C6H5OH,phenol C6H10,cyclo- C6H12,1-hexene C6H12,cyclo- C6H13,n-hexyl
C6H14,n-hexane C7H7,benzyl C7H8 C7H8O,cresol-mx C7H14,1-heptene
C7H15,n-heptyl C7H16,2-methylh C7H16,n-heptane C8H8,styrene C8H10,ethylbenz
C8H16,1-octene C8H17,n-octyl C8H18,n-octane C8H18,isooctane C9H19,n-nonyl
C10H8,naphthale C10H21,n-decyl C12H9,o-bipheny C12H10,biphenyl CLCN
CLO CLO2 CL2 CL2O HCN
HCO HCCN HNC HNCO HNO
HNO2 HNO3 HOCL HO2 HCHO,formaldehy
HCOOH H2O2 (HCOOH)2 KCN KNO2
KNO3 K2 K2CO3 K2C2N2 K2O2
*N NCO *NH NH2 NH3
NOCL NO2 NO2CL NO3 NCN
N2H2 NH2NO2 N2H4 N2O N2O3
N2O4 N2O5 N3 N3H O3
C(gr) H2O(s) H2O(L) K(cr) K(L)
KCN(s) KCN(L) KCL(cr) KCL(L) KH(cr)
KH(L) KNO2(II) KNO2(I) KNO2(L) KNO3(a)
KNO3(b) KNO3(L) KOH(a) KOH(b) KOH(c)
KOH(L) KO2(b) KO2(a) KO2(L) K2CO3(a)
K2CO3(b) K2CO3(L) K2O(c) K2O(b) K2O(a)
K2O(L) K2O2(cr) K2O2(L) NH4CL(a) NH4CL(b)

NOTE. WEIGHT FRACTION OF FUEL IN TOTAL FUELS And OF OXIDANT IN TOTAL OXIDANTS

Combustion Products of WM-1

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      NASA-GLENN CHEMICAL EQUILIBRIUM PROGRAM CEA, NOVEMBER 5, 1999
            BY  BONNIE MCBRIDE And SANFORD GORDON
     REFS: NASA RP-1311, PART I, 1994 And NASA RP-1311, PART II, 1996

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 reac
   name KBenz    K 1 C 7 H 5 O 2  wt%=17   h,kJ/mol=-610.94  t,k=298.15
   name KClO3    K 1 Cl 1 O 3   wt%=80  h,kJ/mol=-389       t,k=298.15
   name RedGum    C 10 H 18 O 1   wt%=3    h,kJ/mol=-276     t,k=298.15
 prob  case=WM1  hp  equil  p(psia)=14.7
 outp cal massf
 end

 OPTIONS: TP=F  HP=T  SP=F  TV=F  UV=F  SV=F  DETN=F  SHOCK=F  REFL=F
INCD=F
 RKT=F  FROZ=F  EQL=T  IONS=F  SIUNIT=F  DEBUGF=F  SHKDBG=F  DETDBG=F
TRNSPT=F

 TRACE= 0.00E+00  S/R= 0.000000E+00  H/R= 0.000000E+00  U/R= 0.000000E+00

 P,BAR =    1.013525

   REACTANT        WT.FRAC  (ENERGY/R),K   TEMP,K  DENSITY
     EXPLODED FORMULA
 N: KBenz          0.170000  -0.734788E+05   298.15  0.0000
       K  1.00000  C  7.00000  H  5.00000  O  2.00000
 N: KClO3          0.800000  -0.467857E+05   298.15  0.0000
       K  1.00000  CL 1.00000  O  3.00000
 N: RedGum         0.030000  -0.331950E+05   298.15  0.0000
       C 10.00000  H 18.00000  O  1.00000

 SPECIES BEING CONSIDERED IN THIS SYSTEM
(COndENSED PHASE MAY HAVE NAME LISTED SEVERAL TIMES)
 LAST thermo.txt UPDATE:   11/08/99
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g 7/97 *C g 8/99 CCL g 8/99 CCL2
x12/93 CCL3 tpis91 CCL4 tpis79 *CH
g 9/99 CHCL x12/93 CHCL2 g 7/99 CHCL3
g 8/99 CH2 g 2/96 CH2CL tpis91 CH2CL2
g 8/99 CH3 tpis91 CH3CL g12/92 CH2OH
g10/92 CH3O g 8/99 CH4 g 8/88 CH3OH
tpis79 *CO tpis91 COCL tpis91 COCL2
tpis91 COHCL g 9/99 *CO2 tpis91 COOH
tpis91 *C2 tpis91 C2CL tpis91 C2CL2
tpis91 C2CL3 tpis91 C2CL4 tpis91 C2CL6
g 1/91 C2H tpis91 C2HCL tpis91 C2HCL3
g 6/89 CHCO,ketyl g12/89 C2H2,vinylidene g 1/91 C2H2,acetylene
tpis91 C2H2CL2 g 5/90 CH2CO,ketene g 2/92 C2H3,vinyl
tpis91 C2H3CL g 6/96 CH3CO,acetyl g 1/91 C2H4
g 8/88 C2H4O,ethylen-o g 8/88 CH3CHO,ethanal g 8/88 CH3COOH
g12/92 C2H5 g 8/88 C2H6 g 8/88 C2H5OH
g12/92 CH3OCH3 g12/89 C2O tpis79 *C3
x 4/98 C3H3,1-propynl x 4/98 C3H3,2-propynl g12/92 C3H4,allene
g12/92 C3H4,propyne g 5/90 C3H4,cyclo- bur 92 C3H5,allyl
g 2/95 C3H6,propylene g 1/93 C3H6,cyclo- g 6/90 C3H6O
g 6/90 C3H7,n-propyl g 9/85 C3H7,i-propyl g 6/90 C3H8
g 9/88 C3H8O,1propanol g 9/88 C3H8O,2propanol g 7/88 C3O2
g 7/88 C4 g 2/93 C4H2 g 5/90 C4H4,1,3-cyclo-
x10/92 C4H6,butadiene x10/93 C4H6,1-butyne x10/93 C4H6,2-butyne
g 5/90 C4H6,cyclo- x 4/88 C4H8,1-butene x 4/88 C4H8,cis2-buten
x 4/88 C4H8,tr2-butene x 4/88 C4H8,isobutene g 5/90 C4H8,cyclo-
g 6/90 (CH3COOH)2 x10/84 C4H9,n-butyl x10/84 C4H9,i-butyl
g 1/93 C4H9,s-butyl g 1/93 C4H9,t-butyl g 6/90 C4H10,isobutane
g 6/90 C4H10,n-butane g 7/88 C5 g 5/90 C5H6,1,3cyclo-
g 1/93 C5H8,cyclo- x 4/87 C5H10,1-pentene g 6/90 C5H10,cyclo-
x10/84 C5H11,pentyl g 1/93 C5H11,t-pentyl x10/85 C5H12,n-pentane
x10/85 C5H12,i-pentane x10/85 CH3C(CH3)2CH3 g 2/93 C6H2
g 1/91 C6H5,phenyl g 6/90 C6H5O,phenoxy g 1/91 C6H6
g 6/90 C6H5OH,phenol g 1/93 C6H10,cyclo- x 4/87 C6H12,1-hexene
g 6/90 C6H12,cyclo- x10/83 C6H13,n-hexyl g 6/96 C6H14,n-hexane
g 1/93 C7H7,benzyl g 1/93 C7H8 g 1/93 C7H8O,cresol-mx
x 4/87 C7H14,1-heptene x10/83 C7H15,n-heptyl x10/85 C7H16,2-methylh
x10/85 C7H16,n-heptane x 4/89 C8H8,styrene x10/86 C8H10,ethylbenz
x 4/87 C8H16,1-octene x10/83 C8H17,n-octyl x 4/85 C8H18,n-octane
x 4/85 C8H18,isooctane x10/83 C9H19,n-nonyl g 8/93 C10H8,naphthale
x10/83 C10H21,n-decyl g12/84 C12H9,o-bipheny g12/84 C12H10,biphenyl
g 7/97 *CL tpis89 CLO g 7/93 CLO2
tpis89 CL2 tpis89 CL2O g 6/97 *H
g 9/96 HCO tpis89 HCL tpis89 HOCL
g 5/99 HO2 tpis78 *H2 g 8/88 HCHO,formaldehy
g 8/88 HCOOH g 8/89 H2O g 6/99 H2O2

g 8/88 (HCOOH)2	g 7/97 *K	J 3/66 KCL
J 3/63 KH	J12/67 KO	g 9/97 KOH
j12/83 K2	tpis82 K2CO3	tpis82 K2CL2
tpis82 K2O	tpis82 K2O2	g 9/97 K2O2H2
g 5/97 *O	tpis78 *OH	tpis89 *O2
tpis89 O3	x 4/83 C(gr)	x 4/83 C(gr)
x 4/83 C(gr)	g 8/89 H2O(s)	g 8/89 H2O(L)
coda89 K(cr)	coda89 K(L)	tpis82 KCL(cr)
tpis82 KCL(cr)	tpis82 KCL(L)	tpis82 KH(cr)
tpis82 KH(L)	g 8/97 KOH(a)	g 8/97 KOH(b)
g 8/97 KOH(c)	g 8/97 KOH(L)	tpis82 KO2(b)
tpis82 KO2(a)	tpis82 KO2(L)	tpis82 K2CO3(a)
tpis82 K2CO3(b)	tpis82 K2CO3(L)	tpis82 K2O(c)
tpis82 K2O(b)	tpis82 K2O(a)	tpis82 K2O(L)
tpis82 K2O2(cr)	tpis82 K2O2(L)	

O/F = 0.000000

	EFFECTIVE FUEL	EFFECTIVE OXIDANT	MIXTURE
ENTHALPY	h(2)/R	h(1)/R	h0/R
(KG-MOL)(K)/KG	-0.38984060E+03	0.00000000E+00	-0.38984060E+03

KG-FORM.WT./KG	bi(2)	bi(1)	b0i
*K	0.75890864E-02	0.00000000E+00	0.75890864E-02
*C	0.93725755E-02	0.00000000E+00	0.93725755E-02
*H	0.88063060E-02	0.00000000E+00	0.88063060E-02
*O	0.21900654E-01	0.00000000E+00	0.21900654E-01
*CL	0.65279904E-02	0.00000000E+00	0.65279904E-02

POINT	ITN	T	K	C	H	O	CL
1	27	2524.309	-21.864	-19.220	-12.501	-16.827	-24.015

THERMODYNAMIC EQUILIBRIUM COMBUSTION PROPERTIES AT ASSIGNED PRESSURES

CASE = WM1

REACTANT		WT FRACTION (SEE NOTE)	ENERGY CAL/MOL	TEMP K
NAME	KBenz	0.1700000	-146018.164	298.150

NAME	KClO3	0.8000000	-92973.231	298.150
NAME	RedGum	0.0300000	-65965.583	298.150

O/F= 0.00000 %FUEL= 0.000000 R,EQ.RATIO= 1.070663 PHI,EQ.RATIO= 0.000000

THERMODYNAMIC PROPERTIES

P, ATM	1.0003
T, K	2524.31
RHO, G/CC	2.2268-4
H, CAL/G	-774.70
U, CAL/G	-883.48
G, CAL/G	-4923.24
S, CAL/(G)(K)	1.6434

M, (1/n)	46.113
(dLV/dLP)t	-1.02052
(dLV/dLT)p	1.4407
Cp, CAL/(G)(K)	0.7146
GAMMAs	1.1169
SON VEL,M/SEC	713.0

MASS FRACTIONS

*CO	0.06272
*CO2	0.31393
*CL	0.00107
*H	0.00006
HCL	0.00521
*H2	0.00037
H2O	0.06593
*K	0.02106
KCL	0.47253
KH	0.00001
KO	0.00049
KOH	0.03851
K2CL2	0.00122
*O	0.00074
*OH	0.00400
*O2	0.01213

* THERMODYNAMIC PROPERTIES FITTED TO 20000.K

PRODUCTS WHICH WERE CONSIDERED BUT WHOSE MASS FRACTIONS WERE LESS THAN 5.000000E-06 FOR ALL ASSIGNED COndITIONS

*C CCL CCL2 CCL3 CCL4
*CH CHCL CHCL2 CHCL3 CH2
CH2CL CH2CL2 CH3 CH3CL CH2OH
CH3O CH4 CH3OH COCL COCL2
COHCL COOH *C2 C2CL C2CL2
C2CL3 C2CL4 C2CL6 C2H C2HCL
C2HCL3 CHCO,ketyl C2H2,vinylidene C2H2,acetylene C2H2CL2
CH2CO,ketene C2H3,vinyl C2H3CL CH3CO,acetyl C2H4
C2H4O,ethylen-o CH3CHO,ethanal CH3COOH C2H5 C2H6
C2H5OH CH3OCH3 C2O *C3 C3H3,1-propynl
C3H3,2-propynl C3H4,allene C3H4,propyne C3H4,cyclo- C3H5,allyl
C3H6,propylene C3H6,cyclo- C3H6O C3H7,n-propyl C3H7,i-propyl
C3H8 C3H8O,1propanol C3H8O,2propanol C3O2 C4
C4H2 C4H4,1,3-cyclo- C4H6,butadiene C4H6,1-butyne C4H6,2-butyne
C4H6,cyclo- C4H8,1-butene C4H8,cis2-buten C4H8,tr2-butene C4H8,isobutene
C4H8,cyclo- (CH3COOH)2 C4H9,n-butyl C4H9,i-butyl C4H9,s-butyl
C4H9,t-butyl C4H10,isobutane C4H10,n-butane C5 C5H6,1,3cyclo-
C5H8,cyclo- C5H10,1-pentene C5H10,cyclo- C5H11,pentyl C5H11,t-pentyl
C5H12,n-pentane C5H12,i-pentane CH3C(CH3)2CH3 C6H2 C6H5,phenyl
C6H5O,phenoxy C6H6 C6H5OH,phenol C6H10,cyclo- C6H12,1-hexene
C6H12,cyclo- C6H13,n-hexyl C6H14,n-hexane C7H7,benzyl C7H8
C7H8O,cresol-mx C7H14,1-heptene C7H15,n-heptyl C7H16,2-methylh C7H16,n-heptane
C8H8,styrene C8H10,ethylbenz C8H16,1-octene C8H17,n-octyl C8H18,n-octane
C8H18,isooctane C9H19,n-nonyl C10H8,naphthale C10H21,n-decyl C12H9,o-bipheny
C12H10,biphenyl CLO CLO2 CL2 CL2O
HCO HOCL HO2 HCHO,formaldehy HCOOH
H2O2 (HCOOH)2 K2 K2CO3 K2O
K2O2 K2O2H2 O3 C(gr) H2O(s)
H2O(L) K(cr) K(L) KCL(cr) KCL(L)
KH(cr) KH(L) KOH(a) KOH(b) KOH(c)
KOH(L) KO2(b) KO2(a) KO2(L) K2CO3(a)
K2CO3(b) K2CO3(L) K2O(c) K2O(b) K2O(a)
K2O(L) K2O2(cr) K2O2(L)

NOTE. WEIGHT FRACTION OF FUEL IN TOTAL FUELS And OF OXIDANT IN TOTAL OXIDANTS

Combustion Products of RNX-6

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      NASA-GLENN CHEMICAL EQUILIBRIUM PROGRAM CEA, NOVEMBER 5, 1999
            BY  BONNIE MCBRIDE And SANFORD GORDON
       REFS: NASA RP-1311, PART I, 1994 And NASA RP-1311, PART II, 1996

*************************************************************************
*



reac
   name KNO3  K 1 N 1 O 3    wt%=70.0  t,k=298.15  rho,g/cc=2.109  h,cal/mol=-11
7760.0
   name Fe2O3  Fe 2 O 3      wt%=4.0   t,k=298.15  rho,g/cc=5.24   h,kJ/mol=-8
25.50
   name epoxy  C 21 H 24 O 1  wt%=26.0  t,k=298.15  rho,g/cc=1.1    h,kJ/mol=-1
662.39
prob case=rnx-6  hp  equil  p(psia)=14.7
outp  cal  massf
end

 OPTIONS: TP=F  HP=T  SP=F  TV=F  UV=F  SV=F  DETN=F  SHOCK=F  REFL=F
INCD=F
 RKT=F  FROZ=F  EQL=T  IONS=F  SIUNIT=F  DEBUGF=F  SHKDBG=F  DETDBG=F
TRNSPT=F

 TRACE= 0.00E+00  S/R= 0.000000E+00  H/R= 0.000000E+00  U/R= 0.000000E+00

 P,BAR =    1.013525

   REACTANT        WT.FRAC  (ENERGY/R),K   TEMP,K  DENSITY
     EXPLODED FORMULA
 N: KNO3          0.700000  -0.592588E+05   298.15  2.1090
       K  1.00000  N  1.00000  O  3.00000
 N: Fe2O3         0.040000  -0.992843E+05   298.15  5.2400
       FE 2.00000  O  3.00000
 N: epoxy         0.260000  -0.199938E+06   298.15  1.1000
       C 21.00000  H 24.00000  O  1.00000

 SPECIES BEING CONSIDERED IN THIS SYSTEM
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(COndENSED PHASE MAY HAVE NAME LISTED SEVERAL TIMES)
LAST thermo.txt UPDATE: 11/08/99

g 7/97 *C tpis79 *CH g 8/99 CH2
g 8/99 CH3 g12/92 CH2OH g10/92 CH3O
g 8/99 CH4 g 8/88 CH3OH g 8/99 *CN
g12/89 CNN tpis79 *CO g 9/99 *CO2
tpis91 COOH tpis91 *C2 g 1/91 C2H
g 6/89 CHCO,ketyl g12/89 C2H2,vinylidene g 1/91 C2H2,acetylene
g 5/90 CH2CO,ketene g 2/92 C2H3,vinyl g12/92 CH3CN
g 6/96 CH3CO,acetyl g 1/91 C2H4 g 8/88 C2H4O,ethylen-o
g 8/88 CH3CHO,ethanal g 8/88 CH3COOH g12/92 C2H5
g 8/88 C2H6 g 8/88 CH3N2CH3 g 8/88 C2H5OH
g12/92 CH3OCH3 g12/92 CCN tpis91 CNC
tpis79 C2N2 g12/89 C2O tpis79 *C3
x 4/98 C3H3,1-propynl x 4/98 C3H3,2-propynl g12/92 C3H4,allene
g12/92 C3H4,propyne g 5/90 C3H4,cyclo- bur 92 C3H5,allyl
g 2/95 C3H6,propylene g 1/93 C3H6,cyclo- g 6/90 C3H6O
g 6/90 C3H7,n-propyl g 9/85 C3H7,i-propyl g 6/90 C3H8
g 9/88 C3H8O,1propanol g 9/88 C3H8O,2propanol g 7/88 C3O2
g 7/88 C4 g 2/93 C4H2 g 5/90 C4H4,1,3-cyclo-
x10/92 C4H6,butadiene x10/93 C4H6,1-butyne x10/93 C4H6,2-butyne
g 5/90 C4H6,cyclo- x 4/88 C4H8,1-butene x 4/88 C4H8,cis2-buten
x 4/88 C4H8,tr2-butene x 4/88 C4H8,isobutene g 5/90 C4H8,cyclo-
g 6/90 (CH3COOH)2 x10/84 C4H9,n-butyl x10/84 C4H9,i-butyl
g 1/93 C4H9,s-butyl g 1/93 C4H9,t-butyl g 6/90 C4H10,isobutane
g 6/90 C4H10,n-butane j 3/61 C4N2 g 7/88 C5
g 5/90 C5H6,1,3cyclo- g 1/93 C5H8,cyclo- x 4/87 C5H10,1-pentene
g 6/90 C5H10,cyclo- x10/84 C5H11,pentyl g 1/93 C5H11,t-pentyl
x10/85 C5H12,n-pentane x10/85 C5H12,i-pentane x10/85 CH3C(CH3)2CH3
g 2/93 C6H2 g 1/91 C6H5,phenyl g 6/90 C6H5O,phenoxy
g 1/91 C6H6 g 6/90 C6H5OH,phenol g 1/93 C6H10,cyclo-
x 4/87 C6H12,1-hexene g 6/90 C6H12,cyclo- x10/83 C6H13,n-hexyl
g 6/96 C6H14,n-hexane g 1/93 C7H7,benzyl g 1/93 C7H8
g 1/93 C7H8O,cresol-mx x 4/87 C7H14,1-heptene x10/83 C7H15,n-heptyl
x10/85 C7H16,2-methylh x10/85 C7H16,n-heptane x 4/89 C8H8,styrene
x10/86 C8H10,ethylbenz x 4/87 C8H16,1-octene x10/83 C8H17,n-octyl
x 4/85 C8H18,n-octane x 4/85 C8H18,isooctane x10/83 C9H19,n-nonyl
g 8/93 C10H8,naphthale x10/83 C10H21,n-decyl g12/84 C12H9,o-bipheny
g12/84 C12H10,biphenyl g 5/97 *Fe J 3/78 FeC5O5
J 9/66 FeO J12/66 Fe(OH)2 g 6/97 *H
g 7/88 HCN g 9/96 HCO tpis89 HCCN
g11/92 HNC g 2/96 HNCO g 5/99 HNO
tpis89 HNO2 g 5/99 HNO3 g 5/99 HO2
tpis78 *H2 g 8/88 HCHO,formaldehy g 8/88 HCOOH
g 8/89 H2O g 6/99 H2O2 g 8/88 (HCOOH)2

g 7/97 *K	J 3/66 KCN	J 3/63 KH
tpis82 KNO2	tpis82 KNO3	J12/67 KO
g 9/97 KOH	j12/83 K2	tpis82 K2CO3
J 3/66 K2C2N2	tpis82 K2O	tpis82 K2O2
g 9/97 K2O2H2	g 5/97 *N	g 2/96 NCO
g 4/99 *NH	g 5/99 NH2	tpis89 NH3
tpis89 *NO	g 4/99 NO2	j12/64 NO3
tpis78 *N2	g12/89 NCN	g 5/99 N2H2
tpis89 NH2NO2	g 4/99 N2H4	g 4/99 N2O
g 4/99 N2O3	tpis89 N2O4	g 4/99 N2O5
tpis89 N3	g 4/99 N3H	g 5/97 *O
tpis78 *OH	tpis89 *O2	tpis89 O3
x 4/83 C(gr)	x 4/83 C(gr)	x 4/83 C(gr)
j 3/78 Fe(a)	j 3/78 Fe(a)	j 3/78 Fe(a)
j 3/78 Fe(a)	j 3/78 Fe(c)	j 3/78 Fe(d)
j 3/78 Fe(L)	J 3/78 FeC5O5(L)	J 3/78 FeC5O5(L)
J 6/65 FeO(s)	J 6/65 FeO(s)	J 6/65 FeO(L)
J 6/66 Fe(OH)2(s)	J 6/66 Fe(OH)2(s)	J 6/66 Fe(OH)3(s)
J 6/66 Fe(OH)3(s)	J 6/65 Fe2O3(s)	J 6/65 Fe2O3(s)
J 6/65 Fe3O4(s)	J 6/65 Fe3O4(s)	g 8/89 H2O(s)
g 8/89 H2O(L)	coda89 K(cr)	coda89 K(L)
J 3/66 KCN(s)	J 3/66 KCN(L)	J 3/66 KCN(L)
tpis82 KH(cr)	tpis82 KH(L)	tpis82 KNO2(II)
tpis82 KNO2(I)	tpis82 KNO2(L)	tpis82 KNO3(a)
tpis82 KNO3(b)	tpis82 KNO3(L)	g 8/97 KOH(a)
g 8/97 KOH(b)	g 8/97 KOH(c)	g 8/97 KOH(L)
tpis82 KO2(b)	tpis82 KO2(a)	tpis82 KO2(L)
tpis82 K2CO3(a)	tpis82 K2CO3(b)	tpis82 K2CO3(L)
tpis82 K2O(c)	tpis82 K2O(b)	tpis82 K2O(a)
tpis82 K2O(L)	tpis82 K2O2(cr)	tpis82 K2O2(L)

O/F = 0.000000

	EFFECTIVE FUEL	EFFECTIVE OXIDANT	MIXTURE
ENTHALPY	h(2)/R	h(1)/R	h0/R
(KG-MOL)(K)/KG	-0.61292957E+03	0.00000000E+00	-0.61292957E+03

KG-FORM.WT./KG	bi(2)	bi(1)	b0i
*K	0.69236159E-02	0.00000000E+00	0.69236159E-02
*N	0.69236159E-02	0.00000000E+00	0.69236159E-02
*O	0.22411460E-01	0.00000000E+00	0.22411460E-01
*Fe	0.50097628E-03	0.00000000E+00	0.50097628E-03
*C	0.18672114E-01	0.00000000E+00	0.18672114E-01
*H	0.21339559E-01	0.00000000E+00	0.21339559E-01

POINT ITN	T	K	N	O	FE

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                C        H
 1  42    538.285   -24.301   -12.584   -62.106    36.385
           8.715   -12.490
ADD  C(gr)
 1  10    661.435   -22.452   -12.792   -49.511    18.359
          -1.040    -9.736
 1   0    661.439   -22.452   -12.792   -49.511    18.359
          -1.040    -9.736
ADD  Fe(a)
 1   5    687.029   -21.859   -12.842   -48.171    -4.179
          -1.075    -9.635
ADD  K2CO3(a)
 1  10    948.468   -26.553   -13.267   -39.399    -4.943
          -1.449    -9.186
PHASE CHANGE, REPLACE  K2CO3(a)     WITH  K2CO3(b)
 1   2    951.100   -26.429   -13.273   -39.345    -4.950
          -1.453    -9.188
 1   0    951.102   -26.429   -13.273   -39.345    -4.951
          -1.453    -9.188
ADD  FeO(s)
 1   3    976.309   -25.333   -13.301   -38.924    -5.024
          -1.489    -9.179
REMOVE  Fe(a)
 1   3    959.045   -26.083   -13.282   -39.206    -5.263
          -1.464    -9.184
```

THERMODYNAMIC EQUILIBRIUM COMBUSTION PROPERTIES AT ASSIGNED PRESSURES

CASE = rnx-6

REACTANT		WT FRACTION (SEE NOTE)	ENERGY CAL/MOL	TEMP K
NAME	KNO3	0.7000000	-117760.000	298.150
NAME	Fe2O3	0.0400000	-197299.235	298.150
NAME	epoxy	0.2600000	-397320.746	298.150

REACTANT DENSITY= 1.736388 G/CC

O/F= 0.00000 %FUEL= 0.000000 R,EQ.RATIO= 2.330383 PHI,EQ.RATIO= 0.000000

THERMODYNAMIC PROPERTIES

P, ATM	1.0003
T, K	959.04
RHO, G/CC	5.9375-4
H, CAL/G	-1218.02
U, CAL/G	-1258.82
G, CAL/G	-2624.65
S, CAL/(G)(K)	1.4667

M, (1/n)	46.714
MW, MOL WT	30.885
(dLV/dLP)t	-1.08676
(dLV/dLT)p	2.5220
Cp, CAL/(G)(K)	1.5634
GAMMAs	1.0945
SON VEL,M/SEC	432.2

MASS FRACTIONS

CH4	0.00742
*CO	0.15871
*CO2	0.09125
*H2	0.01619
H2O	0.03085
NH3	0.00003
*N2	0.09696
C(gr)	0.08418
FeO(s)	0.03599
K2CO3(b)	0.47844

* THERMODYNAMIC PROPERTIES FITTED TO 20000.K

PRODUCTS WHICH WERE CONSIDERED BUT WHOSE MASS FRACTIONS WERE LESS THAN 5.000000E-06 FOR ALL ASSIGNED COndITIONS

*C *CH CH2 CH3 CH2OH
CH3O CH3OH *CN CNN COOH
*C2 C2H CHCO,ketyl C2H2,vinylidene C2H2,acetylene
CH2CO,ketene C2H3,vinyl CH3CN CH3CO,acetyl C2H4
C2H4O,ethylen-o CH3CHO,ethanal CH3COOH C2H5 C2H6
CH3N2CH3 C2H5OH CH3OCH3 CCN CNC
C2N2 C2O *C3 C3H3,1-propynl C3H3,2-propynl
C3H4,allene C3H4,propyne C3H4,cyclo- C3H5,allyl C3H6,propylene
C3H6,cyclo- C3H6O C3H7,n-propyl C3H7,i-propyl C3H8
C3H8O,1propanol C3H8O,2propanol C3O2 C4 C4H2
C4H4,1,3-cyclo- C4H6,butadiene C4H6,1-butyne C4H6,2-butyne C4H6,cyclo-

C4H8,1-butene C4H8,cis2-buten C4H8,tr2-butene C4H8,isobutene C4H8,cyclo-
(CH3COOH)2 C4H9,n-butyl C4H9,i-butyl C4H9,s-butyl C4H9,t-butyl
C4H10,isobutane C4H10,n-butane C4N2 C5 C5H6,1,3cyclo-
C5H8,cyclo- C5H10,1-pentene C5H10,cyclo- C5H11,pentyl C5H11,t-pentyl
C5H12,n-pentane C5H12,i-pentane CH3C(CH3)2CH3 C6H2 C6H5,phenyl
C6H5O,phenoxy C6H6 C6H5OH,phenol C6H10,cyclo- C6H12,1-hexene
C6H12,cyclo- C6H13,n-hexyl C6H14,n-hexane C7H7,benzyl C7H8
C7H8O,cresol-mx C7H14,1-heptene C7H15,n-heptyl C7H16,2-methylh C7H16,n-heptane
C8H8,styrene C8H10,ethylbenz C8H16,1-octene C8H17,n-octyl C8H18,n-octane
C8H18,isooctane C9H19,n-nonyl C10H8,naphthale C10H21,n-decyl C12H9,o-bipheny
C12H10,biphenyl *Fe FeC5O5 FeO Fe(OH)2
*H HCN HCO HCCN HNC
HNCO HNO HNO2 HNO3 HO2
HCHO,formaldehy HCOOH H2O2 (HCOOH)2 *K
KCN KH KNO2 KNO3 KO
KOH K2 K2CO3 K2C2N2 K2O
K2O2 K2O2H2 *N NCO *NH
NH2 *NO NO2 NO3 NCN
N2H2 NH2NO2 N2H4 N2O N2O3
N2O4 N2O5 N3 N3H *O
*OH *O2 O3 Fe(a) Fe(c)
Fe(d) Fe(L) FeC5O5(L) FeO(L) Fe(OH)2(s)
Fe(OH)3(s) Fe2O3(s) Fe3O4(s) H2O(s) H2O(L)
K(cr) K(L) KCN(s) KCN(L) KH(cr)
KH(L) KNO2(II) KNO2(I) KNO2(L) KNO3(a)
KNO3(b) KNO3(L) KOH(a) KOH(b) KOH(c)
KOH(L) KO2(b) KO2(a) KO2(L) K2CO3(a)
K2CO3(L) K2O(c) K2O(b) K2O(a) K2O(L)
K2O2(cr) K2O2(L)

NOTE. WEIGHT FRACTION OF FUEL IN TOTAL FUELS And OF OXIDANT IN TOTAL OXIDANTS

Combustion Products RNX-17

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      NASA-GLENN CHEMICAL EQUILIBRIUM PROGRAM CEA, NOVEMBER 5, 1999
            BY  BONNIE MCBRIDE And SANFORD GORDON
     REFS: NASA RP-1311, PART I, 1994 And NASA RP-1311, PART II, 1996

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reac
   name KNO3  K 1 N 1 O 3    wt%=70.0  t,k=298.15  rho,g/cc=2.109  h,cal/mol=-11
7760.0
   name B     B 1          wt%=8.0 t,k=298.15  rho,g/cc=2.355  h,cal/mol=0
   name epoxy  C 21 H 24 O 1  wt%=22.0  t,k=298.15  rho,g/cc=1.1    h,kJ/mol=-1
662.39
insert B2O3(L)
prob  case=rnx-4  hp  equil  p(psia)=14.7
outp  cal  massf
end

 OPTIONS: TP=F  HP=T  SP=F  TV=F  UV=F  SV=F  DETN=F  SHOCK=F  REFL=F
INCD=F
 RKT=F  FROZ=F  EQL=T  IONS=F  SIUNIT=F  DEBUGF=F  SHKDBG=F  DETDBG=F
TRNSPT=F

 TRACE= 0.00E+00  S/R= 0.000000E+00  H/R= 0.000000E+00  U/R= 0.000000E+00

 P,BAR =     1.013525

   REACTANT         WT.FRAC   (ENERGY/R),K   TEMP,K  DENSITY
     EXPLODED FORMULA
 N: KNO3           0.700000  -0.592588E+05   298.15  2.1090
       K  1.00000  N  1.00000  O  3.00000
 N: B              0.080000   0.000000E+00   298.15  2.3550
       B  1.00000
 N: epoxy          0.220000  -0.199938E+06   298.15  1.1000
       C 21.00000  H 24.00000  O  1.00000

 SPECIES BEING CONSIDERED IN THIS SYSTEM
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(COndENSED PHASE MAY HAVE NAME LISTED SEVERAL TIMES)
LAST thermo.txt UPDATE: 11/08/99

g 9/98 *B g 9/98 BC g 9/98 BC2
g 9/98 *BH tpis96 BH2 tpis96 BH3
tpis96 BH3NH3 g 9/98 BN g 9/98 *BO
tpis96 BOH g10/97 BO2 g 9/98 B(OH)2
g 9/98 B2 g 9/98 B2C tpis96 B2H6
g 9/98 B2O tpis96 B2O2 tpis96 B2O3
g 9/98 B2(OH)4 tpis96 B3N3H6 g 7/97 *C
tpis79 *CH g 8/99 CH2 g 8/99 CH3
g12/92 CH2OH g10/92 CH3O g 8/99 CH4
g 8/88 CH3OH g 8/99 *CN g12/89 CNN
tpis79 *CO g 9/99 *CO2 tpis91 COOH
tpis91 *C2 g 1/91 C2H g 6/89 CHCO,ketyl
g12/89 C2H2,vinylidene g 1/91 C2H2,acetylene g 5/90 CH2CO,ketene
g 2/92 C2H3,vinyl g12/92 CH3CN g 6/96 CH3CO,acetyl
g 1/91 C2H4 g 8/88 C2H4O,ethylen-o g 8/88 CH3CHO,ethanal
g 8/88 CH3COOH g12/92 C2H5 g 8/88 C2H6
g 8/88 CH3N2CH3 g 8/88 C2H5OH g12/92 CH3OCH3
g12/92 CCN tpis91 CNC tpis79 C2N2
g12/89 C2O tpis79 *C3 x 4/98 C3H3,1-propynl
x 4/98 C3H3,2-propynl g12/92 C3H4,allene g12/92 C3H4,propyne
g 5/90 C3H4,cyclo- bur 92 C3H5,allyl g 2/95 C3H6,propylene
g 1/93 C3H6,cyclo- g 6/90 C3H6O g 6/90 C3H7,n-propyl
g 9/85 C3H7,i-propyl g 6/90 C3H8 g 9/88 C3H8O,1propanol
g 9/88 C3H8O,2propanol g 7/88 C3O2 g 7/88 C4
g 2/93 C4H2 g 5/90 C4H4,1,3-cyclo- x10/92 C4H6,butadiene
x10/93 C4H6,1-butyne x10/93 C4H6,2-butyne g 5/90 C4H6,cyclo-
x 4/88 C4H8,1-butene x 4/88 C4H8,cis2-buten x 4/88 C4H8,tr2-butene
x 4/88 C4H8,isobutene g 5/90 C4H8,cyclo- g 6/90 (CH3COOH)2
x10/84 C4H9,n-butyl x10/84 C4H9,i-butyl g 1/93 C4H9,s-butyl
g 1/93 C4H9,t-butyl g 6/90 C4H10,isobutane g 6/90 C4H10,n-butane
j 3/61 C4N2 g 7/88 C5 g 5/90 C5H6,1,3cyclo-
g 1/93 C5H8,cyclo- x 4/87 C5H10,1-pentene g 6/90 C5H10,cyclo-
x10/84 C5H11,pentyl g 1/93 C5H11,t-pentyl x10/85 C5H12,n-pentane
x10/85 C5H12,i-pentane x10/85 CH3C(CH3)2CH3 g 2/93 C6H2
g 1/91 C6H5,phenyl g 6/90 C6H5O,phenoxy g 1/91 C6H6
g 6/90 C6H5OH,phenol g 1/93 C6H10,cyclo- x 4/87 C6H12,1-hexene
g 6/90 C6H12,cyclo- x10/83 C6H13,n-hexyl g 6/96 C6H14,n-hexane
g 1/93 C7H7,benzyl g 1/93 C7H8 g 1/93 C7H8O,cresol-mx
x 4/87 C7H14,1-heptene x10/83 C7H15,n-heptyl x10/85 C7H16,2-methylh
x10/85 C7H16,n-heptane x 4/89 C8H8,styrene x10/86 C8H10,ethylbenz
x 4/87 C8H16,1-octene x10/83 C8H17,n-octyl x 4/85 C8H18,n-octane
x 4/85 C8H18,isooctane x10/83 C9H19,n-nonyl g 8/93 C10H8,naphthale
x10/83 C10H21,n-decyl g12/84 C12H9,o-bipheny g12/84 C12H10,biphenyl

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 g 6/97  *H          tpis96  HBO          tpis96  HBO2
 g 7/88  HCN          g 9/96  HCO          tpis89  HCCN
g11/92  HNC          g 2/96  HNCO          g 5/99  HNO
tpis89  HNO2         g 5/99  HNO3          g 5/99  HO2
tpis78  *H2          g 9/98  HBOH          g 8/88  HCHO,formaldehy
 g 8/88  HCOOH         g 8/89  H2O          g 6/99  H2O2
tpis96  H2BOH        tpis96  HB(OH)2        tpis96  H3BO3
tpis96  H3B3O3       tpis96  H3B3O6         g 8/88  (HCOOH)2
 g 7/97  *K          tpis82  KBO2          J 3/66  KCN
 J 3/63  KH          tpis82  KNO2          tpis82  KNO3
 J12/67  KO           g 9/97  KOH          j12/83  K2
tpis82  K2CO3         J 3/66  K2C2N2         tpis82  K2O
tpis82  K2O2          g 9/97  K2O2H2         g 5/97  *N
 g 2/96  NCO          g 4/99  *NH           g 5/99  NH2
tpis89  NH3          tpis89  *NO           g 4/99  NO2
 j12/64  NO3         tpis78  *N2           g12/89  NCN
 g 5/99  N2H2         tpis89  NH2NO2          g 4/99  N2H4
 g 4/99  N2O          g 4/99  N2O3          tpis89  N2O4
 g 4/99  N2O5         tpis89  N3           g 4/99  N3H
 g 5/97  *O          tpis78  *OH          tpis89  *O2
tpis89  O3           j 6/83  B(b)          j 6/83  B(b)
 j 6/83  B(L)        tpis96  BN(cr)         tpis96  BN(cr)
tpis96  BN(L)         tpis96  B2O3(cr)        tpis96  B2O3(L)
 J 3/65  B3O3H3(cr)     J 3/65  B3O3H3(cr)      tpis96  B4C(cr)
tpis96  B4C(cr)       tpis96  B4C(cr)        x 4/83  C(gr)
 x 4/83  C(gr)        x 4/83  C(gr)         tpis96  HBO2(cr)
tpis96  HBO2(L)        g 8/89  H2O(s)         g 8/89  H2O(L)
tpis96  H3BO3(cr)      tpis96  H3BO3(L)        coda89  K(cr)
coda89  K(L)         tpis82  KBO2(cr)       tpis82  KBO2(cr)
tpis82  KBO2(L)        J 3/66  KCN(s)         J 3/66  KCN(L)
 J 3/66  KCN(L)       tpis82  KH(cr)         tpis82  KH(L)
tpis82  KNO2(II)       tpis82  KNO2(I)         tpis82  KNO2(L)
tpis82  KNO3(a)        tpis82  KNO3(b)         tpis82  KNO3(L)
 g 8/97  KOH(a)        g 8/97  KOH(b)         g 8/97  KOH(c)
 g 8/97  KOH(L)        tpis82  KO2(b)         tpis82  KO2(a)
tpis82  KO2(L)         tpis82  K2CO3(a)        tpis82  K2CO3(b)
tpis82  K2CO3(L)        tpis82  K2O(c)         tpis82  K2O(b)
tpis82  K2O(a)         tpis82  K2O(L)         tpis82  K2O2(cr)
tpis82  K2O2(L)
B2O3(L)      INSERTED

O/F =  0.000000

                EFFECTIVE FUEL    EFFECTIVE OXIDANT      MIXTURE
ENTHALPY              h(2)/R          h(1)/R            h0/R
(KG-MOL)(K)/KG    -0.56071005E+03   0.00000000E+00   -0.56071005E+03
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KG-FORM.WT./KG	bi(2)	bi(1)	b0i
*K	0.69236159E-02	0.00000000E+00	0.69236159E-02
*N	0.69236159E-02	0.00000000E+00	0.69236159E-02
*O	0.21523204E-01	0.00000000E+00	0.21523204E-01
*B	0.73998705E-02	0.00000000E+00	0.73998705E-02
*C	0.15799481E-01	0.00000000E+00	0.15799481E-01
*H	0.18056550E-01	0.00000000E+00	0.18056550E-01

POINT	ITN	T	K	N	O	B	C	H
1	21	1153.597	-17.788	-13.982	-41.567	-11.374	3.133	-9.780

ADD C(gr)

1	6	1138.172	-19.260	-13.607	-37.263	-18.646	-1.716	-9.457

ADD KBO2(cr)

1	7	1463.950	-20.064	-13.945	-34.870	-8.859	-2.145	-9.762

PHASE CHANGE, REPLACE KBO2(cr) WITH KBO2(L)

1	3	1502.684	-20.223	-13.973	-34.644	-8.020	-2.194	-9.788

ADD BN(cr)

1	5	1394.090	-21.968	-14.711	-34.539	-11.659	-2.056	-9.721

REMOVE B2O3(L)

1	6	1478.046	-18.176	-14.044	-34.568	-11.318	-2.163	-9.772

THERMODYNAMIC EQUILIBRIUM COMBUSTION PROPERTIES AT ASSIGNED PRESSURES

CASE = rnx-4

	REACTANT	WT FRACTION (SEE NOTE)	ENERGY CAL/MOL	TEMP K
NAME	KNO3	0.7000000	-117760.000	298.150
NAME	B	0.0800000	0.000	298.150
NAME	epoxy	0.2200000	-397320.746	298.150

REACTANT DENSITY= 1.767156 G/CC

O/F= 0.00000 %FUEL= 0.000000 R,EQ.RATIO= 2.564156 PHI,EQ.RATIO= 0.000000

THERMODYNAMIC PROPERTIES

P, ATM	1.0003
T, K	1478.05
RHO, G/CC	3.3731-4
H, CAL/G	-1114.25
U, CAL/G	-1186.07
G, CAL/G	-3705.18
S, CAL/(G)(K)	1.7529
M, (1/n)	40.900
MW, MOL WT	28.180
(dLV/dLP)t	-1.29681
(dLV/dLT)p	6.6343
Cp, CAL/(G)(K)	5.6197
GAMMAs	1.0914
SON VEL,M/SEC	572.7

MASS FRACTIONS

CH4	0.00016
*CO	0.24405
*CO2	0.00011
HBO	0.00003
HBO2	0.00004
HCN	0.00012
*H2	0.01814
H2O	0.00012
*K	0.01522
KBO2	0.26997
KCN	0.00878
KH	0.00002
KOH	0.00007
K2	0.00001
K2C2N2	0.00002
*N2	0.08100
BN(cr)	0.02483
C(gr)	0.08329
KBO2(L)	0.25402

* THERMODYNAMIC PROPERTIES FITTED TO 20000.K

PRODUCTS WHICH WERE CONSIDERED BUT WHOSE MASS FRACTIONS WERE LESS THAN 5.000000E-06 FOR ALL ASSIGNED COndITIONS

*B BC BC2 *BH BH2
BH3 BH3NH3 BN *BO BOH
BO2 B(OH)2 B2 B2C B2H6
B2O B2O2 B2O3 B2(OH)4 B3N3H6
*C *CH CH2 CH3 CH2OH
CH3O CH3OH *CN CNN COOH
*C2 C2H CHCO,ketyl C2H2,vinylidene C2H2,acetylene
CH2CO,ketene C2H3,vinyl CH3CN CH3CO,acetyl C2H4
C2H4O,ethylen-o CH3CHO,ethanal CH3COOH C2H5 C2H6
CH3N2CH3 C2H5OH CH3OCH3 CCN CNC
C2N2 C2O *C3 C3H3,1-propynl C3H3,2-propynl
C3H4,allene C3H4,propyne C3H4,cyclo- C3H5,allyl C3H6,propylene
C3H6,cyclo- C3H6O C3H7,n-propyl C3H7,i-propyl C3H8
C3H8O,1propanol C3H8O,2propanol C3O2 C4 C4H2
C4H4,1,3-cyclo- C4H6,butadiene C4H6,1-butyne C4H6,2-butyne C4H6,cyclo-
C4H8,1-butene C4H8,cis2-buten C4H8,tr2-butene C4H8,isobutene C4H8,cyclo-
(CH3COOH)2 C4H9,n-butyl C4H9,i-butyl C4H9,s-butyl C4H9,t-butyl
C4H10,isobutane C4H10,n-butane C4N2 C5 C5H6,1,3cyclo-
C5H8,cyclo- C5H10,1-pentene C5H10,cyclo- C5H11,pentyl C5H11,t-pentyl
C5H12,n-pentane C5H12,i-pentane CH3C(CH3)2CH3 C6H2 C6H5,phenyl
C6H5O,phenoxy C6H6 C6H5OH,phenol C6H10,cyclo- C6H12,1-hexene
C6H12,cyclo- C6H13,n-hexyl C6H14,n-hexane C7H7,benzyl C7H8
C7H8O,cresol-mx C7H14,1-heptene C7H15,n-heptyl C7H16,2-methylh C7H16,n-heptane
C8H8,styrene C8H10,ethylbenz C8H16,1-octene C8H17,n-octyl C8H18,n-octane
C8H18,isooctane C9H19,n-nonyl C10H8,naphthale C10H21,n-decyl C12H9,o-bipheny
C12H10,biphenyl *H HCO HCCN HNC
HNCO HNO HNO2 HNO3 HO2
HBOH HCHO,formaldehy HCOOH H2O2 H2BOH
HB(OH)2 H3BO3 H3B3O3 H3B3O6 (HCOOH)2
KNO2 KNO3 KO K2CO3 K2O
K2O2 K2O2H2 *N NCO *NH
NH2 NH3 *NO NO2 NO3
NCN N2H2 NH2NO2 N2H4 N2O
N2O3 N2O4 N2O5 N3 N3H
*O *OH *O2 O3 B(b)
B(L) BN(L) B2O3(cr) B2O3(L) B3O3H3(cr)
B4C(cr) HBO2(cr) HBO2(L) H2O(s) H2O(L)
H3BO3(cr) H3BO3(L) K(cr) K(L) KBO2(cr)
KCN(s) KCN(L) KH(cr) KH(L) KNO2(II)
KNO2(I) KNO2(L) KNO3(a) KNO3(b) KNO3(L)
KOH(a) KOH(b) KOH(c) KOH(L) KO2(b)
KO2(a) KO2(L) K2CO3(a) K2CO3(b) K2CO3(L)
K2O(c) K2O(b) K2O(a) K2O(L) K2O2(cr)
K2O2(L)

NOTE. WEIGHT FRACTION OF FUEL IN TOTAL FUELS AND OF OXIDANT IN TOTAL OXIDANTS