EnSafe Environmental and Safety Designs, Inc.

Memphis, TN • Nashville, TN • Raleigh, NC • Pensacola, FL

5724 Summer Trees Dr. • P.O. Box 341315 • Memphis, TN 38184-1315

October 29, 1993

Commanding Officer,
Atlantic Division
Naval Facilities Engineering Command
Attn: Code 18231, Linda G. Berry
1510 Gilbert St.
Norfolk, Virginia 23511-2699

Subject: Contract N62470-93-D-4009, Indefinite Quantity Contract for Environmental Documentation and Investigation at Various Activities under the Cognizance of the Atlantic Division, Naval Facilities Engineering Command in Support or RCRA, SWDA, and TSCA Regulations— **Marine Corps Base, Camp Lejeune SWMU Assessment.**

Dear Ms. Berry:

Enclosed is a copy of EnSafe's Health and Safety Plan for the solid waste management unit assessment.

If you have any questions, please call me.

Sincerely,

Environmental and Safety Designs, Inc.

By: Robert Moser, P.E.

copy: Mr. Brynn Ashton, MCB Camp Lejeune (w/o)
Chris McKeeman

Telephone: 901-372-7962 • Facsimile: 901-372-2454

01.09-10/29/93-02361

HEALTH AND SAFETY PLAN

SOLID WASTE MANAGEMENT UNIT ASSESSMENT
MARINE CORPS BASE
CAMP LEJEUNE, NORTH CAROLINA

Prepared by:

Environmental and Safety Designs, Inc.
5724 Summer Trees Drive
Memphis, Tennessee 38134
(901) 372-7962

October 29, 1993

Table of Contents

List of Figures

List of Tables

Appendices

1.0 INTRODUCTION

The following is the Health and Safety Plan for conducting a Solid Waste Mangement Unit (SWMU) Assessment at Marine Corps Base Camp Lejeune, North Carolina. The SWMUs to be address include less-than 90-day hazardous waste storage areas, oil/water separators, waste oil tanks, and underground storage tank locations.

2.0 APPLICABILITY

The provisions of this plan are mandatory for Environmental and Safety Designs, Inc. (EnSafe) employees engaged in onsite operations who will be or have the potential to be exposed to hazardous substances.

Onsite personnel will be responsible for operating in accordance with the most current OSHA regulations including:

29 CFR 1910.95, Occupational Noise Exposure;

1910.120, Hazardous Waste Operations and Emergency Response;

1910.134, Respiratory Protection;

1910.135, Occupational Head Protection;

1910.136, Occupational Foot Protection;

1910.100, Air Contaminants;

1910.1030, Occupational Exposure to Bloodborne Pathogens;

1910.1200, Hazard Communications.

In addition to the above sections of 29 CFR 1910, construction operations will comply with the applicable section of 29 CFR 1926, Safety and Health Regulations for Construction.

Inadequate health and safety precautions by onsite personnel will cause EnSafe to suspend site work and evacuate the site until conditions have been corrected.

3.0 SITE CHARACTERIZATION

Marine Corps Base (MCB) Camp Lejeune is located within Onslow County in southeastern North Carolina, approximately 45 miles south of New Bern and 47 miles north of Wilmington. The County seat, as well as the primary commercial center, is the city of Jacksonville, which is the largest developed area in the County. MCB Camp Lejeune is situated on the southeast coast of North Carolina with a twelve-mile ocean front extending from Bear Inlet to New River Inlet and encompasses approximately 170 square miles of land and water. A vicinity and facility map have been submitted as Figures 3-1 and 3-2, respectively. These maps illustrate the location of MCB Camp Lejeune in relation to the state of North Carolina and the location of the centralized storage facility in relations to MCB Camp lejeune.

Established in 1941, MCB Camp Lejeune houses numerous Marine Corps and Navy Commands, including 2d Marine Division, 2d Force Service Support Group, Marine corps Air Station, New River, the Naval Hospital and Naval Dental Clinic.

The primary mission of MCB Camp Lejeune is to provide specialized schools and other training for Marines; provide housing, training facilities, logistic support and certain administrative support for Fleet Marine Force units and other units as assigned; received and process personnel as assigned and conduct individual combat training as directed; storage, maintenance, and the utilization of military equipment; and the storage and proper training of conventional munitions, pyrotechnics, and other associated ordnance items. MCB Camp Lejeune is the world's most complete Amphibious Training base

The site address of Marine Corps Base Camp Lejeune is:

Marine Corps Base Camp Lejeune
North Carolina, 28542-5001

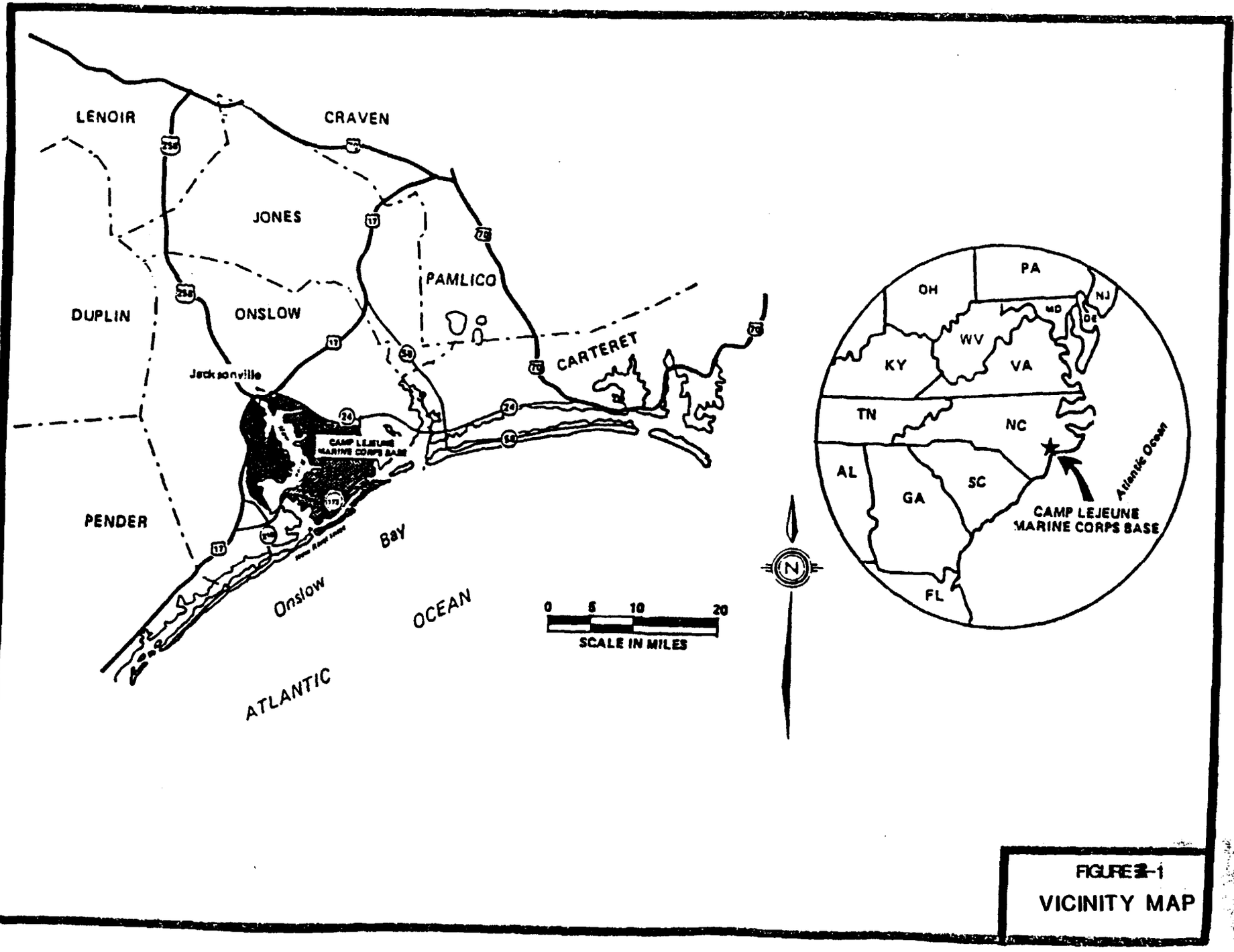
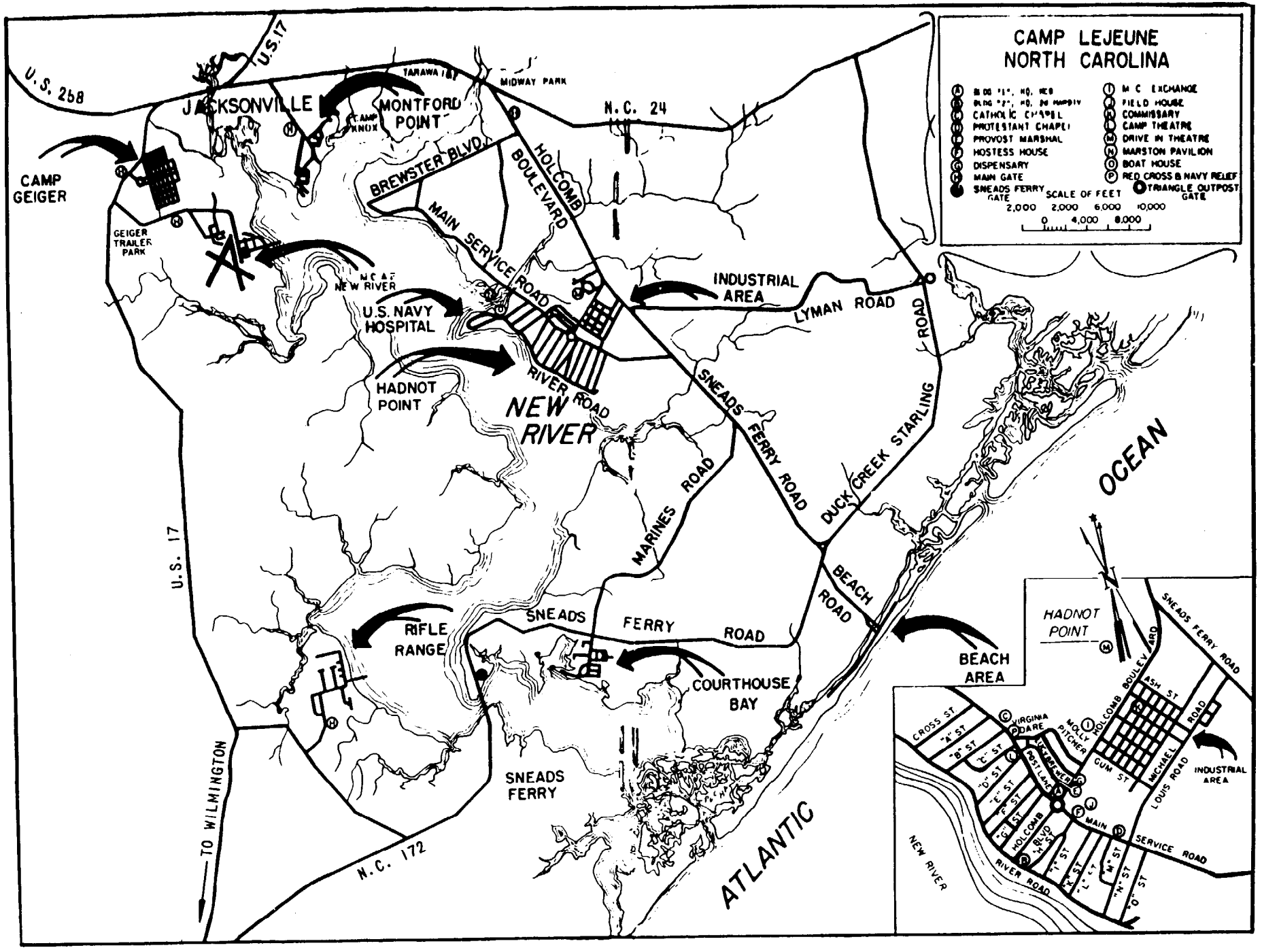


FIGURE 2-1
VICINITY MAP


CAMP LEJEUNE
NORTH CAROLINA
SCALE OF FEET
2,000 2,000 6,000 10,000
0 4,000 8,000
CAMP GEIGER
GEIGER TRAILER PARK
JACKSONVILLE
MONTFORD POINT
CAMP KNOX
TARAWA TER.
MIDWAY PARK
U.S. 17
U.S. 258
N.C. 24
BREWSTER BLVD.
HOLCOMB BOULEVARD
MAIN SERVICE ROAD
NCAF NEW RIVER
U.S. NAVY HOSPITAL
INDUSTRIAL AREA
LYMAN ROAD
HADNOT POINT
RIVER ROAD
NEW RIVER
SNEADS FERRY ROAD
DUCK CREEK STARLING ROAD
OCEAN
MARINES ROAD
BEACH ROAD
RIFLE RANGE
SNEADS FERRY ROAD
COURTHOUSE BAY
BEACH AREA
U.S. 17
TO WILMINGTON
SNEADS FERRY
N.C. 172
ATLANTIC
HADNOT POINT
ASH ST
GUM ST
MICHAEL ROAD
LOUIS ROAD
INDUSTRIAL AREA
MAIN SERVICE ROAD
NEW RIVER
RIVER ROAD
HOLCOMB BLVD
POST LANE
VIRGINIA DARE
MOLLY PITCHER
CROSS ST
"A" ST
"B" ST
"C" ST
"D" ST
"E" ST
"F" ST
"G" ST
"I" ST
"K" ST
"L" ST
"M" ST
"N" ST
"O" ST

The mailing address of Marine Corps Base Camp Lejeune is:

Commanding General
Marine Corps Base Camp Lejeune
North Carolina, 28542-5001

3.1 Work Areas

Where necessary site control will be established and maintained according to the recommendations set forth in the Environmental Protection Agency's *Interim Standard Operating Safety Guides*, Revised September 1982 and Section 2.5 of the EnSafe Corporate Health and Safety Manual. This will include where necessary, establishing an exclusion zone, a contamination reduction zone, and a support zone.

For the most part, there will be at least one EnSafe personnel and one MCB site personnel. No exclusion zones are anticipated.

3.2 Work Area Access/Egress

Personnel entering into each work zones must:

1. Check in with MCB's Hazardous Material Disposal Officer or Coordinator or site manager,
2. Provide the following information:
   a. Name
   b. Organization
   c. Destination
   d. Purpose for being onsite, and
3. Check out with the same person.

There will be approximately 265 work area locations at this site. A record will be maintained in the field log that indicates the date and location of work performed on that date.

3.3 Site Communications

Onsite Communications

Workers will communicate by voice and/or hand signals. During the daily site briefing, the use of hand signals will be discussed. Communications will be used to (1) alert site workers of emergency situations, (2) transmit safety information, (3) communicate changes in work schedules, and (4) maintain site control.

The hand signals outlined below will be used.

Hand Signal	Meaning
Hands clutching throat	Out of air/cannot breathe
Hand on top of head	Need assistance
Thumbs up	OK
Arms waving upright	No, negative
Grip partner's waist	Exit area immediately

Offsite Communications

Onsite and offsite personnel will communicate by telephone, fax machine, and a computer with a modem. This equipment is located in MCB's Environmental Management Division offices (Building 67) and will be used to coordinate emergency responses, file reports, and maintain contact with offsite personnel.

4.0 SITE ACTIVITIES

The activities to be performed at this site will include **only** visual observation. Details on field effort can be found in milestone work plan. Sampling will **not** be conducted.

5.0 HAZARD EVALUATION

This section includes a discussion of the chemical, mechanical equipment, electrical, physical, and temperature hazards present at the site.

5.1 Chemical Hazards

Several previous investigations have been conducted at this site. Table 5-1a, -1b, and -1c lists previously identified contaminants. Table 5-2, Exposure Guidelines for some of the contaminants, summarizes the exposure levels and critical information about these site chemical hazards.

5.2 Mechanical, Electrical, Physical, and Temperature Hazards

The mechanical, electrical, physical, and temperature hazards associated with field activities at this site are summarized in Table 5-3.

5.3 Methods for Dealing with Site Hazards

Chemical Hazards

MSDS contain information relating to the hazards of each contaminant and responses to spills, fires, and personnel exposures. These can be obtained at MCB's base safety office.

Mobile Equipment Operations

No self-propelled equipment will be operatored.

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
001	D002, D008	ELECTROLYTE BATTERY ACID	WASTE BATTERY FLUID ACID (CORROSIVE MATERIAL UN2796)
002	D002	ELECTROLYTE UNUSED	
003	NONE	INSECT REPELLENT	NONE
004	D002	SODIUM HYDROXIDE	WASTE BATTERY FLUID ALKALI (CORROSIVE MATERIAL UN2797)
005	D003	LITHIUM BATTERIES	WASTE LITHIUM BATTERIES FOR DISPOSAL (ORM-C)
006	D002, D006	NICKEL CADMIUM BATTERIES (WET)	WASTE BATTERY, ELECTRIC (CORROSIVE MATERIAL UN2795)
007	D006	NICAD BATTERIES (DRY)	WASTE BATTERY, ELECTIRC POTASSIUM HYDROXIDE SOILD (CORROSIVE MATERIAL NA1813)
008	D009	MERCURY BATTERIES	HAZARDOUS WASTE SOLID NOS (ORM-E NA9189)
009	D007	MAGNESIUM BATTERIES	HAZARDOUS WASTE SOLID NOS CHROMIUM, MAGNESIUM (ORM-E NA9189)
010	D002, D011	SILVER ZINC BATTERIES	WASTE BATTERY FLUID ALKALI (CORROSIVE MATERIAL UN2797)
011	NONE	PERMANONE INSECT REPELLENT	WASTE COMPRESSED GAS NOS (NONFLAMMALBE GAS UN1956)
012	D003	ZINC MIX PACKET-M272 KIT	WATER REACTIVE SOLID NOS (FLAMMABLE SOLID UN2813)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
013	NONE	SODIUM HYPOCHLORITE M72A2 KIT	HYPROXHLORITE SOLUTION (ORM-B NA1791)
014	D026	SOIL/SPILL RESIDUE WITH MUSTARD STIMULANT	HAZARDOUS WASTE SOLID NOS CRESOL (ORM-E NA9189)
015	D026	MUSTARD SIMULANT	HAZARDOUS WASTE LIQUID NOS CRESOL (ORM-E NA9189)
016	D001	M58 DECONTAMINATION KIT	WASTE FLAMMABLE LIQUID NOS ISOPROPANOL (FLAMMABLE LIQUID UN1993)
017	NONE	D5-2 SPILL	CORROSIVE SOLID NOS DIETHYLENE TRIAMINE AND HYDROXIDE (CORROSIVE MATERIAL UN1759)
018	D002	STB LIQUID SUPER TROPICAL BLEACH	HYPOCHLORITE SOLUTION (CORROSIVE MATTER UN1791)
019	NONE	STB GRANULAR	BLEACHING POWDER (ORM-C 2208)
020	D002	DS-2	WASTE ALKALINE LIQUID NOS DIETHYLENETRIAMINE ETHYLENE GLYCOLMONOETHYL SODIUM HYDROXIDE (CORROSIVE MATERIAL NA1791)
021	D001, D002, U103, U020	M72A2 CHEMICAL TEST KIT NBC TESTING	WASTE CORROSIVE LIQUID NOS ACEDTIC ACID, DIMETHYL SULFATE (CORROSIVE MATERIAL UN1760)
022	U103	DIMETHYL SULFATE-M72A2 KIT AMPULE H	WASTE DIMETHYL SULFATE (CORROSIVE MATERIAL UN1595)
023	D001	M-258 A1 DECON KIT	WASTE ETHYL ALCOHOL (FLAMMABLE LIQUID UN1170)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
024	D001, D009, P030	M256/M256A1 DETECTOR KITS	WASTE FLAMMABLE LIQUID POISONOUS NOS METHANOL, MERCURIC CYANIDE (FLAMMABLE LIQUID UN1992)
025	D001, D003, D009	M256 KIT METHANOL, MERCURIC CYANIDE AMPOULE NO. 3, POSITION 2	WASTE FLAMMALBE LIQUID NOS METHANOL, MERCURIC CYANIDE (FLAMMABLE LIQUID UN1992)
026	D001	M256 TRAINS	WASTE FLAMMABLE LIQUID NOS METHANOL (FLAMMABLE LIQUID UN1993)
027	D001, D002, D011, U088	M229 REFILL KIT-ENTIRE KIT	WASTE FLAMMABLE LIQUID CORROSIVE NOS ETHYLALCOHOL AND POTASSIUM HYDROXIDE (FLAMMABLE LIQUID UN2924)
028	D011	SILVER NITRATE M229 REFILL, FILTER PACKAGES	WASTE SILVER NITRATE (OXIDIZER UN1934)
029	D002	BUFFER SOLUTION M229 REFILL KIT, BUFFER SOLUTION CONTAINERS	WASTE ALKALINE LIQUID NOS POTASSIUM HYDROXIDE AND ETHYLENE GLYCOL (CORROSIVE MATERIAL NA1719)
030	D001	ETHYL ALCOHOL, M229 REFILL KIT FILTER PACKAGES	WASTE ETHYL ALCOHOL (FLAMMABLE LIQUID UN1170)
031	U088	DIETHYL PHTHALATE, M229 REFILL KIT SIMULANT SOLUTION CONTAINERS	HAZARDOUS WASTE LIQUID NOS DIETHYL PHTHALATE (ORM-E NA9189)
032	P030	POTASSIUM FERROCYANIDE M272 KITS TICKET PACKETS	WASTE CYANIDE MIXTURE DRY (POISON B UN1588)
033	U041	EPICHLOROHYDRIN M272 KIT YELLOW BAND	WASTE EPICHLOROHYDRIN (FLAMMABLE LIQUID UN2023)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
034	P098	POTASSIUM CYANIDE M272 KITS RED BAND TUBES	WASTE POTASSIUM CYANIDE SOLUTION (POISON B UN1680)
035	D009, P030	NBC M272 BLUE BAND TUBES MERCURIC CYANIDE	WASTE MERCURIC CYANIDE (POISON B UN 1636)
036	D004	SODIUM ARSENITE M272 KITS ORANGE BAND TUBES	WASTE SODIUM ARSENITE LIQUID (POISON B UN1686)
037	D004, D009, P030, P098, U041	M272 ENTIRE KITS	WASTE FLAMMABLE LIQUID POISONOUS NOS EPICHLOROHYDRIN AND POTASSIUM CYANIDE (FLAMMABLE LIQUID UN1992)
038	D004	M272 SODIUM ARSENITE ORANGE BAND TUBES-SOLID	WASTE, POISON B, SOLID NOS SODIUM ARSENITE (POISON B UN2811)
039	D001	KEROSENE	WASTE PAINT RELATED MATERIAL (COMBUSTIBLE LIQUID 1223)
040	D001	BREAK FREE-CLP	WASTE PETROLEUM DISTILLATE (COMBUSTIBLE LIQUID UN1268)
041	D001	DEVELOPER (NASHUA BRAND) PETROLEUM DISTILLATES	WASTE PETROLEUM DITILLATE (COMBUSTIBLE LIQUID UN1268)
042	D001	DRY CLEANING SOLVENT/UNUSED PRODUCT/PDG80	WASTE LIQUID PETROLEUM DISTILLATES (COMBUSTIBLE LIQUID UN1268)
043	D001	PD680 DRY CLEANING SOLVENT	WASTE PETROLEUM DISTILLATES (COMBUSTIBLE LIQUID UN 1268)
044	F001, D039	DRY CLEANING FILTERS	WASTE PERCHLOROETHYLENE (ORM-A UN1897)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
045	D010	COLEMAN FUEL	WASTE GASOLINE (FLAMMABLE LIQUID UN1203)
046	D001	BONDO DENT FILLER	WASTE FLAMMABLE LIQUID NOS STYRENE MONOMER AND POLYESTER RESIN (FLAMMABLE LIQUID UN1993)
047	D001	EPOXY	WASTE ADHESIVE (FLAMMABLE LIQUID UN1133)
048	D001	PLASTIC POLISH	WASTE FLAMMABLE LIQUID NOS MINERAL SPIRITS (FLAMMABLE LIQUID UN1993)
049	D001	RUBBER CEMENT	WASTE CEMENT RUBBER (FLAMMABLE LIQUID NA1133)
050	F003	ACETONE SPENT	WASTE ACETONE (FLAMMBLE LIQUID UN1090)
051	U002	ACETONE UNUSED	WASTE ACETONE (FLAMMALBE LIQUID UN1090)
052	NONE	SODIUM THIOCYANATE-M72A2 KIT AMPOULE B-2	NONE
053	U154, D001	METHANOL UNUSED	WASTE METHYL ALCOHOL (FLAMMABLE LIQUID UN1230)
054	F003	METHANOL - PAINTING	WASTE METHYL ALCOHOL (FLAMMABLE LIQUID UN1230)
055	U031	BUTANOL (BUTYL ALCOHOL)	WASTE BUTYL ALCOHOL (FLAMMABLE LIQUID NA1120)
056	D001	ISOPROPHYL ALCOHOL	WASTE ISOPROPANOL (FLAMMABLE LIQUID UN1219)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
057	D001	DENTURED ALCOHOL (ETHYL ALCOHOL)	WASTE DENATURED ALCOHOL (FLAMMABLE NA1986)
058	D001, F003	ALCOHOL	WASTE ALCOHOL NOS ISOPROPYL, METHYL AND ETHYL ALCOHOL (FLAMMABLE LIQUID UN1987)
059	NONE	SODIUM HYPOCHLORITE M72A2 KIT REMAINDER	WASTE ORM-B NOS SODIUM HYPOCHLORITE (ORM-B NA1760)
060	D002	ACETIC ACID	WASTE ACETIC ACID (CORROSIVE MATERIAL UN2790)
061	D001, D002	ACETIC ACID, GLACIAL	WASTE ACETIC ACID, GLACIAL (CORROSIVE UN2789)
062	U020	BENZENE SULFONYL CHLORIDE, AMPOUL G, CX	WASTE BENZENE SULPHONYL CHLORIDE (CORROSIVE MATERIAL UN2225)
063	D002, D700	BRIGHT DIP (CHROMIC ACID)	WASTE CHROMIC ACID SOLUTION (CORROSIVE MATERIAL UN1755)
064	D002	MOLYBDATE REAGENT	WASTE SULFURIC ACID (CORROSIVE MATERIAL UN1830)
065	D002	MURIATIC ACID (HYDROCHLORIC)	WASTE HYDROCHLORIC ACID (CORROSIVE MATERIAL UN1789)
066	D002	NAVAL JELLY	WASTE ACID LIQUID NOS PHOSPHORIC ACID (CORROSIVE MATERIAL NA1760)
067	D002, D007	PHOSCOTE	WASTE ACID LIQUID NOS PHOSPHORIC AND CHROMIC ACID (CORROSIVE MATERIAL NA1760)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
068	D002	NITRIC ACID	WASTE NITRIC ACID (OXIDIZER UN2031)
069	D002	NITRIC ACID	WASTE NITRIC ACID (CORROSIVE MATERIAL NA1760)
070	D002	SODIUM HYDROXIDE PARTS WASHER	WASTE SODIUM HYDROXIDE SOLUTION (CORROSIVE MATERIAL UN1824)
071	D002, D007	ALODINE	WASTE ACID LIQUID NOS CHROMIC ACID AND HYDROFLUORIC ACID (CORROSIVE MATERIAL NA1760)
072	D002	PHOSPHORIC ACID	WASTE PHOSPHORIC ACID (CORROSIVE MATERIAL UN1805)
073	D002	CLEANING COMPOUND	WASTE ALKALINE LIQUID NOS HYDROXIDE SOLUTION (CORROSIVE MATERIAL NA1719)
074	D001, D002, D007	PRIMER PRETREATMENT RESIN, ACID	FLAMMABLE LIQUID, CORROSIVE NOS PHOSPHORIC ACID (FLAMMABLE LIQUID UN2924)
075	D001	M280 DECON KIT	ETHYL ALCOHOL (FLAMMABLE LIQUID UN1170)
076	D002	AMMONIUM HYDROXIDE	WASTE AMMONIUM HYDROXIDE (CORROSIVE MATERIAL NA2672)
077	NONE	SODIUM HEXAMETAPHOSPHATE, TECHNICAL	NONE
078	NONE	CITRIC ACID, ANHYDRAOUS, TECHNICAL	NONE
079	D001	DE-ICING FLUID	WASTE COMBUSTIBLE LIQUID NOS METHYL CELLOSOLVEL (COMBUSTIBLE LIQLUID NA1993)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
080	U220	TOLUENE	WASTE TOLUENE (FLAMMABLE LIQUID UN1294)
081	NONE	OIL HERDER	COMBUSTIBLE LIQUID NOS ETHYLENE GLYCOL MONOBUTYL ETHER (COMBUSTIBLE LIQUID NA1993)
082	D001, P044	DIMETHOATE 2E SYTEMIC INSECTICIDE	WASTE ORGANOPHOSPHORUS NOS DIMETHOATE (FLAMMABLE LIQUID 2784)
083	D001	ETHYLENE GLYCOL MONOBUTYL ETHER (BUTYL CELLOSOLVE)	WASTE PAINT RELATE MATERIAL WASTE ETHYLENE GLYCOL MONOBUTYL ETHER (COMBUSTABLE LIQUID NA1263)
084	D001	PURGE 111 INSECTICIDE	WASTE COMPRESSED GAS NOS PROPANE(FLAMMABLE GAS 1954)
085	U117	ETHER MEDICAL	WASTE ETHYL ETHER (FLAMMABLE LIQUID UN1155)
086	D001, D039	PARTS CLEANER/GASOLINE	WASTE FLAMMABLE LIQUID NOS TETRACHLOROETHYLENE (FLAMMABLE LIQUID UN1993)
087	D001, F003	XYLENE MEDICAL LAB	WASTE XYLENE (FLAMMABLE LIQUID UN1307)
088	F003	XYLENE	WASTE XYLENE (FLAMMABLE LIQUID UN1307)
089	U239	XYLENE UNUSED	WASTE XYLENE (FLAMMABLE LIQUID UN1307)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
090	F003	METHYL ISOBUTYL KETONE-SPENT PAINTING OR DEGREASING	WASTE METHYL ISOBUTYL KETONE (FLAMMABLE LIQUID UN1245)
091	U161	METHYL ISOBUTYL KETONE UNUSED	WASTE METHYL ISOBUTYL KETONE (FLAMMABLE LIQUID UN1245)
092	D001, D005, D035	CARC PAINT-POLYESTER RESIN, TITANIUM DIOXIDE BUTYL ACETATE, ETHYLENE GLYCOL MONOETHYL ETHER ACETONE, PROPYLENEGLYCOMOMOMETHYL ETHER ACETONE, METHYL ETHYL KETONE, HOMOPOLYER OF HEXAMETHYLENE	WASTE PAINT (FLAMMABLE LIQUID UN1263)
093	D001, D007, D035	CARC PAINT-HOMOGLYMER OF HEXAMETHYLENE DIISOVYANATE, METHYL ISOAMYL KETONE BUTYL ACETATE, TITANIUM DIOXIDE RESIN, CHROMIUM OXIDE	WASTE PAINT (FLAMMABLE LIQUID UN1263)
094	D001, D007, D008, D035	CARC PAINT-POLYESTER RESIN TITANIUM DIOXIDE BUTYL ACETATE, ETHYLENE GLYCOL MONOMETHYL ETHER ACETATE, PROPYLENE GLYCOLMONOMETHYL ETHER ACETATE, HOMOPLYMER OF HEXAMETHYLENE, CLAY SUSPENSION (XYLENE), METHYLISOBUTYL KETONE	WASTE PAINT (FLAMMABLE LIQUID UN1263)
095	D001, D035	CARC PAINT, WHITE-METHYLETHY KETONE, ALIPHATIC ISOCYANATES, ETHYL 3-ETHOXYPROPIONATE, ETHYL ACETATE, N-BUTYL ACETATE	WASTE PAINT (FLAMMABLE LIQUID UN1993)
096	D001, D007, D035	EPOXY PAINT	WASTE PAINT (FLAMMABLE LIQUID UN1263)
097	NONE	ISOPAC, ISOLATING PASTE	NONE
098	NONE	SEALING COMPOUND	NONE
099	D001	POLYESTER RESIN VARNISH	RESIN SOLUTION (FLAMMABLE LIQLUID UN1866)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
100	D001, D035	AIRCRAFT COATING THINNER-XYLENE, TOLUENE, OXOHEXYL ACETATE, N-BUTYL ACETATE, METHYL ETHYL KETONE, 4-METHYL 2-PENTENONE, PROPYLENE GLYCOL MONOMETHYL EHTER, ALIPHATIC HYDROCARBONS, AROMATIC HYDROCARBONS	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN1263)
101	D001, D007, D008, D035	DIESEL FUEL/PAINT	WASTE PAINT RELATED MATERIAL (COMBUSTIBLE LIQUID NA1263)
102	D001, D035	EPOXY THINNER-METHYL ETHYL KETONE, METHYL ISOBUTYL KETONE, METHYL ETHER, ETHYLENE GLYCOL, ETYLENE GLYCOL MONOMETHYL ETHER	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN1263)
103	D001, D035, F003, F005	KEROSENE AND PAINT AND PAINT SOLVENT-	WASTE PAINT RELATED MATERIAL (COMBUSTIBLE LIQUID NA 1263)
104	D001, D004-D011, D035, F003, F005	PAINT AND SOLVENT-VM&P NAPTHA, ALCOHOLS, ACETONE, XYLENE, ALIPHATIC, AROMATIC HYDROCARBONS, GLYCOLS, LEAD CHROMATE, PIGMENTS	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN1263)
105	D001, D007, D008, D035	PAINT ENAMEL ALKYD-ETHYL ALCOHOL, BUTYL ALCOHOL, ACETONE, METHYL ETHYL KETONE, XYLENE	WASTE PAINT (FLAMMABLE LIQUID UN1263)
106	D001, D005, D006, D007, D008, D035	PAINT-POLYESTER RESIN, TITANIUM DIOXIDE, BUTYL ACETATE, ETHYLENEGLYCOLMONOMETHYLETHER ACETATE, PROPYLENE GLYCOMONOMETHLY ETHER ACETATE, METHYLTHYL KETONE, HOMOPOLYMER OF HEXAMETHYLEN	WASTE PAINT(FLAMMABLE LIQLUID UN1263)
107	D007, D008	PAINT SPILL CLEAN UP-CLAY ABORBENT, FIBER ABSORBENT, METAL CANS, PAINT PIGMENT, RESINS	HAZARDOUS WASTE SOLID NOS CHROMIUM LEAD (ORM-E NA9189)
108	D001, D007, D008, D035	AEROSOL PAINT CANS-METAL CAN, PAINT PIGMENTS, PROPANE, ISOBUTANE, XYLENE, TOLUENE, METHYL ISOBUTYL KETONE, RESINS, ZINC CHROMATE, LEAD CHROMATE	WASTE COMPRESSED GAS NOS (FLAMMABLE GAS UN1954)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
109	D001, D007, D008, D035	PAINT AND PAINT CANS NON AEROSOL-METAL CANS, POLYESTER RESINS, TITANIUM DIOXIDE, BUTYL ACETATE, ETHYLENEGLYCOL MONOMETHYL ETHER ACETATE	WASTE PAINT (FLAMMABLE LIQUID UN1263)
110	D001	MINERAL SPIRITS	WASTE NAPTHA (FLAMMABLE LIQUID UN2553)
111	D007, D008, D035, F002, F003	PAINT STRIPPER AND SOLVENTS-TOLUENE, XYLENE, METHYL ETHYL KETONE, PAINT PIGMENTS, METHYLENECHLORIDE, LEAD CHROMATE, MINERAL SPIRITS	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN1263)
112	D001	WALKWAY-BUTYL ACETATE, VM&P NAPTHA, MINERAL SPIRITS, BUTYL CELLOSOLVE, BUTYLRALDEHYDE OXIME	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN 1263)
113	D004, D011, F001, F005	DRY CLEANING SOLVENT/FINGERPRINT REMOVER-METHYLENE CHLORIDE, PARAFFIN HYDROCARONS, NAPTHENIC HYDROCARBONS, AROMATIC HYDROCARBONS, METHYLAMYL ALCOHOL	WASTE ORM-A NOS METHYLENE CHLORIDE (ORM-A NA1693)
114	D035, D004-D011, F001-F005	PAINT RELATED MATERIAL AND WATER-METHYL ETHYL KETONE, PAINT, WATER	HAZARDOUS WASTE LIQUID NOS METHYL ETHYL KETONE (ORM-E 9189)
115	D001	MODEL ENGINE FUEL-METHYL ALCOHOL, NITROMETHANE, OTHER NITROPARAFFINS	WASTE FLAMMABLE LIQUID NOS METHYL ALCOHOL AND NITROMETHANE (FLAMMABLE LIQLUID UN1993)
116	D007, D008	PAINT IN PAINT CANS (SOLID OR SEMI-SOLID)-METAL CANS, SYNTHETIC RESINS, PIGMENTS, CLAY SUSPENSION, LEAD CHROMATE MINERAL SPIRITS	HAZARDOUS WASTE SOILD NOS CHROMIUM, LEAD (ORM-E NA9189)
117	NONE	CLEANING AGENT	COMBUSTIBLE LIQUID NOS ISOPROPANOL (COMBUSTIBLE LIQUID NA1993)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
118	D001	CITRA-SOLV-SATURATED ALIPHATIC HYDROCARBONS, AROMATIC HYDROCARBONS, CITRIC ACID, OTHER PETROLEUM DISTILLATES	WASTE FLAMMABLE LIQUID NOS ALIPHATIC AND AROMATIC HYDROCARBONS (FLAMMABLE LIQUID UN1993)
119	D001	WASTE RAGS CONTAMINATED WITH NAPTHA 40%	WASTE RAGS CONTAMINATED WITH NAPTHA 40% (FLAMMABLE SOLID UN2553)
120	D001	NAPTHA (BLANKET WASH)	WASTE NAPTHA (COMBUSTIBLE LIQUID UN2553)
121	D001	NAPTHA	WASTE NAPTHA (FLAMMABLE LIQUID UN 2553)
122	NONE	USE OIL ON SPECIFICATION	USE OIL (NONE)
123	D001	VARSOL	WASTE PETROLEUM DISTILLATES (COMBUSTIBLE LIQUID UN1268)
124	D001	LUBRICANT, SOLID FILM (AEROSOL)	WASTE COMPRESSED GAS NOS XYLENE, METHYL EHTYL KETONE (FLAMMABLE GAS 1954)
125	D001	RIFLE BORE CLEANER-PETROLEUM HYDROCARBON, LIGHTWEIGHT OIL, EMULSIFIER, ALCOHOL, SOAP	WASTE COMPOUND CLEANING LIQUID (COMBUSTIBLE LIQUID NA 1993)
126	D001	TONER	WASTE PETROLEUM DISTILLATES (COMBUSTIBLE LIQUID UN1268)
127	D001	CORROSIVE PREVENTIVE COMPOUND	WASTE PETROLEUM DISTILATES NOS (FLAMMABLE LIQUID UN1268)
128	D001, D008	CORROSION PREVENTION COMPOUND-OXIDIZED ASPHALT, STODDARD SOLVENT, LEAD TALLATE, NAPTHA	HAZARDOUS WASTE LIQUID NOS LEAD (ORM-E 9189)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
129	NONE	PD680	COMBUSTIBLE LIQUID NOS
130	D001	SOLDER FLUX	WASTE FLAMMABLE LIQUID NOS ALCOHOLS AND RESINS (FLAMMABLE LIQUID UN1993)
131	D001	DIISORYANATES	WASTE COMBUSTIBLE LIQUID NOS DIISOCYANATES (COMBUSTIBLE LIQUID NA1993)
132	D003	GAS PAK-DISPOSABLE HYDROGEN AND CARBON DIOXIDE GENERATOR MEDICAL	WASTE REACTIVE SOLID NOS SODIUM BOROHYDRIDE (FLAMMABLE SOLID UN2813)
133	D001	PRESERVATIVE COATING COMPOUND	COMBUSTIBLE LIQUID NOS (COMBUSTIBLE LIQUID NA 1993)
134	D001, D035	ADHESIVE-ISOMETRIC HYDROCARBONS, TOLUENE, METHYL ETHYL KETONE, ACETONE, ALCOHOLS, RESINS	WASTE ADHESIVE (FLAMMABLE LIQUID UN1133)
135	D001	TENT PATCH KIT-NATURAL RUBBER, ACETONE, TOLUENE	WASTE ADHESIVE (FLAMMABLE LIQUID UN1133)
136	D001, D035	ASPHALT ADHESIVE-RESINS, METHYL ETHYL KETONE, ASPHALT, TARS, RUBBERS	WASTE ADHESIVE (FLAMMABLE LIQUID UN1133)
137	NONE	CORROSION PREVENTIVE-FINGERPRINT REMOVER-METHYLAMYL ALCOHOL, ALIPHATIC, AROMATIC	COMBUSTIBLE LIQUID NOS METHYAMYOL ALCOHOL (COMBUSTIBLE NA1993)
138	NONE	INSTPACK PART A-POLYMERIC ISOCYANATE DIPHENYLMETHANT-4,4-DIISOCYONATE (MDI)	NONE
139	F002	FUEL OIL	HAZARDOUS WASTE LIQUID NOS HALOGENATED SOLVENT (COMBUSTIBLE LIQUID 1270)

Table 5-1a
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
140	D001	DIESEL FUEL WITH DRY CLEANING SOLVENT PD680-DIESEL FUEL,	COMBUSTIBLE LIQUID NOS DIESEL (COMBUSTIBLE LIQUID NA 1993)
141	NONE	FUEL OIL #6	PETROLEUM OIL NOS FUEL OIL #6(COMBUSTIBLE LIQUID NA 1270)
142	NONE	FUEL OIL WITH DIRT AND SPILL RESIDUE	#6 OIL SPILL RESIDUE (NONE)
143	D001	OIL/FUEL	WASTE PETROLEUM OIL (COMBUSTIBLE LIQUID NA1270)
144	D001, D004-D011	USE OIL/FUEL OFF SPECIFICATION	WASTE PETROLEUM OILS (FLAMMABLE LIQUID 1270)
145	NONE	OIL, SEPARATOR SKIMMINGS	USED OIL (NONE)
146	F002	COMPRESSOR OIL AND REFRIGERANT	HAZARDOUS WASTE LIQUID NOS OIL/FREON (COMBUSTIBLE LIQUID NA 1270)
147	D008, F001	OIL/CONTAMINATED WITH HALOGENS AND LEAD	WASTE OIL (COMBUSTIBLE LIQUID NA 1270)
148	F002	OIL/CONTAMINATED WITH HALOGENS	WASTE OIL (COMBUSTIBLE LIQUID NA1270)
149	F001	HYDRAULIC FLUID AND FREON	HAZARDOUS WASTE LIQUID NOS HYDRAULIC FLUID AND FREON (ORM-F NA9189)
150	D008	OIL/CONTAMINATED WITH LEAD	WASTE OIL (COMBUSTIBLE LIQUID NA 1270)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
151	F002	METHYLENE CHLORIDE - PAINT REMOVER	WASTE METHYLENE CHLORIDE (ORM-A UN1593)
152	F001	1,1,1-THRICHLOROETHANE - SPENT PRODUCT USED IN DEGREASING	WASTE 1,1,1-TRICHLOROETHANE (ORM-A UN2831)
153	F002	1,1,1-TRICHLOROETHANE - SPENT PRODUCT	WASTE 1,1,1-TRICHLOROETHANE (ORM-A UN2831)
154	F002	FREON 113, TRICHLOROTRIFLUOROETHANE - INSTRUMENT CALIBRATION, DRYING	WASTE ORM-S NOS TRICHLOROTRIFLUOROETHANE (ORM-A NA1693)
155	D039, F001	PERCHLOROETHYLENE - DEGREASING OR DRY CLEANING	WASTE PERCHLOROETHYLENE (ORM-A UN1897)
156	U080	METHYLENE CHLORIDE - UNUSED PRODUCT	WASTE METHYLENE CHLORIDE (ORM-A UN1593)
157	U226	1,1,1-TRICHLOROETHANE (METHYL CHLOROFORM) UNUSED	WASTE 1,1,1-TRICHLOROETHANE (ORM-A UN2831)
158	F002	SPILL RESIDUE-METHYLENE CHLORIDE	HAZARDOUS WASTE SOLID NOS METHYLENE CHLORIDE SPILL RESIDUE (ORM-E NA9189)
159	D001, D009	PVA FIXTATIVE DISCARDED MEDICAL ITEM	WASTE FLAMMABLE LIQUID NOS ALCOHOLS AND MERCURIC CHLORIDE (FLAMMABLE LIQUID UN1993)
160	D001, D022, D039, F002	STRIPPER - HALOGENATED ALIPHATIC/AROMATIC, NONHALOGENATED ALIPHATIC/AROMATIC, METHYLENE CHLORIDE, DICHLOROPRANE	WASTE FLAMMABLE LIQUID NOS METHYLENE CHLORIDE, DICHLOROPROPANE (FLAMMABLE LIQUID UN1993)
161	D001	GRAM STAIN SET - BACTO GRAM DECOLORIZER DISCARDED MEDICAL ITEM ACETONE, ISOPROPYL ALCOHOL	WASTE FLAMMABLE LIQUID NOS ACETONE AND ISOPROPANOL (FLAMMABLE LIQUID UN1993)
162	D001	PAINT - BUILDING MAINTENANCE PIGMENTS AND SOLUBLE ORG.	WASTE PAINT (FLAMMABLE LIQUID UN1263)
163	D008	VENTILATION SYSTEM PAINT WASTE - VEHICLE PAINTING OIL AND PAINT PIGMENTS	WASTE PAINT RELATED MATERIAL (COMBUSTIBLE LIQUID NA1263)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
164	D001	BUTANE	WASTE LIQUIFIED PETROLEUM (FLAMMABLE GAS UN1075)
165	D001	PAINT ENAMEL/ALKYD	WASTE PAINT (COMBUSTIBLE LIQUID UN1263)
166	D001	GIESMA STAIN DISCARDED MEDICAL METHANOL	WASTE FLAMMABLE LIQUID NOS GLYCERIN AND METHANOL (FLAMMABLE LIQUID UN1992)
167	D001, D007, D008, D035, F003, F005	PAINT AND SOLVENT SOLVENT DISTILLING PROCESS TOLUENE, VM&P NAPTHA, ALCOHOL, ACETONE, XYLENE, ALIPHATIC AROMATIC HYDROCARBONS, GLYCOLS, LEAD CHROMATE	WASTE PAINT RELATED MATERIAL (FLAMMABLE LIQUID UN1263)
168	NONE	BREAK FREE-CLP SPENT SOLVENT	COMBUSTIBLE LIQUID NOS (COMBUSTIBLE LIQUID NA1993)
169	U112	ETHYL ACETATE MEDICAL ITEM	WASTE ETHYL ACETATE (FLAMMABLE LIQUID UN1173)
170	D001	PROPANE DISCARDED	WASTE LIQUIFIED PETROLEUM (FLAMMABLE UN1075)
171	D006, D008, D018	OIL MIXED WITH SOLVENT PARTS CLEANING PETROLEUM OILS AND STODDAR SOLVENTS	HAZARDOUS WASTE LIQUID NOS LEAD/CADMIUM (ORM-E 9189)
175		HALOGENATED AEORSOL, CORROSION INHIBITOR AND LUBRICANTS DICHLORODIFLUOROMETHANE, TRICHLOROTRIFLOUROMETHANE, TRICHLOROETHYLENE, METHYLENE CHLORIDE, TRICHLOROETHANE, TRICHLOROEFLUOREMETHANE, TETRACHLOROETHYLENE	WASTE COMPRESSED GAS NOS FREON (NON-FLAMMABLE GAS UN1956)
178	NONE	INSECTICIDE, D-PHENOTHRINE 2% AEROSOL	INSECTICIDE GAS, NOS DICHLORODIFLUOROMETHANE, TRICHLOROFLUOROMETHANE (1968)
179	U248	RODENTICIDE (BAIT, ANTI-COAGULANT) UNUSED	HAZARDOUS WASTE SOLID NOS WARFARIN (ORM-E NA9189)
180	U129	LINDANE DISCARDED	WASTE LINDANE (ORM-A NA2761)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
181		INSECTICIDE DISCARDED PYRETHINE AND PETROLEUM DISTILLATES	INSECTICIDE LIQUID NOS PYRETHRINE (POISON B NA2902)
182	U061	DDT	WASTE DDT, MIXTURE RQ (ORM-A NA2761)
190	D009	MERCURY DISCARDED MERCURY, METALIC	WASTE MERCURY METALLIC (ORM-E NA2809)
192	D009	SPILL RESIDUE MERCURY THERMOMETERS	HAZARDOUS WASTE SOLID NOS SPILL RESIDUE MERCURY (ORM-E NA9189)
195	D001	AMMONIUM SULFIDE SOLUTION	WASTE AMMONIUM SULFIDE SOLUTION (FLAMMABLE LIQUID UN2683)
196	D003, D006, D008	CLEANING COMPOUND PARTS CLEANING SODIUM SULFIDES, CYANIDES, CADMIUM, LEAD, SODIUM HYDROXIDE	HAZARDOUS WASTE LIQUID NOS CADMIUM AND LEAD (ORM-E NA9189)
200	D003	CALCIUM HYDRIDE	WASTE WATER REACTIVE SOLID NOS CALCIUM HYDRIDE (FLAMMABLE SOLID UN2813)
202	D001, U162	METHYL METHACRYLATE DISCHARDED	WASTE METHYL METHACRYLATE MONOMER INHIBITED (FLAMMABLE LIQUID UN1247)
205	D002	LAB PACK CHEMICALS	WASTE CORROSIVE LIQUID NOS (CORROSIVE MATERIAL UN1760)
206	D001, D005, D008	LAB PACK CHEMICALS	WASTE OXIDIZER NOS (OXIDIZER UN1479)
208A	D003,	LAB PACKS CHEMICALS	WASTE FLAMMABLE SOLID NOS (FLAMMABLE SOLID UN1325)
208B	NONE	LAB PACKS CHEMICALS	NONE
208C	NONE	LAB PACKS CHEMICALS	CORROSIVE SOLID NOS (CORROSIVE MATERIAL UN1759)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
208D	D003	LAB PACKS CHEMICALS	WASTE WATER REACTIVE SOLID NOS CALCIUM CARBIDE (FLAMMABLE LIQUID UN2813)
208G	D001	LAB PACKS CHEMICALS	WASTE OXIDIZER NOS (OXIDIZER UN1479)
210	U117	ENGINE STARTING FLUID ETHYL ETHER, ISOPROPANE, BUTANE	WASTE COMPRESSED GAS NOS ETHER, ISOPROPANE, BUTANE (FLAMMABLE GAS UN1954)
212	D001, F005	SOLVENT MIXTURE ISOPROPYL ALCOHOL, TOLUENE, ACETONE, PETROLEUM ETHER, ISO-OCTANE, PETROLEUM FUEL	WASTE FLAMMABLE LIQUID NOS (FLAMMABLE LIQUID UN1993)
222	D001, D002	M258/M258A1 ETHYL ALCOHOL, PHENOL, SODIUM HYDROXIDE, AMMONIA	WASTE FLAMMABLE LIQUID CORROSIVE NOS ETHER, ALCOHOL PHENOL, SODIUM HYDROXIDE, AMMONIA (FLAMMABLE LIQUID UN2924)
223	D001	M258/M258A1 ETHYL ALCOHOL, ZINC CHLORIDE, AMPOULE CHLORAMINE B	WASTE FLAMMABLE LIQUID NOS ETHYLE ALCOHOL, ZINC CHLORIDE, CHLORAMINE B (FLAMMABLE LIQUID UN 1993)
225	P029	COPPER CYANIDE DISCARDED PLATING MATERIAL	WASTE COPPER CYANIDE (POISON B UN1587)
227	P104, D011	SILVER CYANIDE DISCARDED PLATING MATERIAL	WASTE SILVER CYANIDE (POISON B UN1684)
229	P106	SODIUM CYANIDE DISCARDED PLATING MATERIAL	WASTE SODIUM CYANIDE (POISON B UN1689)
230	NONE	CUPRIC SULFATE	CUPRIC SULFATE (NONE)
231	NONE	POTASSIUM HYDROXIDE GRANULAR	POTASSIUM HYDROXIDE GRANULAR (CORROSIVE MATERIAL UN1813)
232	NONE	SODIUM CHLORIDE	SODIUM CHLORIDE (NONE)
233	NONE	KODAK RPX-OMAT DEVELOPER AND REPLENISHER, PART A	ALKALINE LIQUID NOS POTASSIUM HYDROXIDE AND POSTASSIUM SULFITE (CORROSIVE MATERIAL NA1719)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
234	NONE	KODAK RPX-OMAT DEVELOPER AND REPLENISHER, PART C	ACID LIQUID NOS ACETIC ACID AND GLUTARALDEHYDE (CORROSIVE MATERIAL NA160)
235	NONE	KODAK FIXER AND REPLENSIHER PART A	AMMONIUM THIOSULFATE SOLUTION (NONE)
236	NONE	KODAK GBX DEVELOPER AND REPLENISHER	SODIUM SULFITE SOLUTION (NONE)
237	NONE	KODAK DEVELOPER HC-110	KODAK DEVELOPER (NONE)
238	NONE	KODAK X-RAY DEVELOPER ACID BASE	X-RAY DEVELOPER (NONE)
239	NONE	KODAK 55 DEVELOPER	DEVELOPING SOLUTION (NONE)
240	NONE	KODAK RAPID FIXER, PART A	KODAK RAPID FIXER (NONE)
248	NONE	NON-HAZARDOUS SPILLS/NON-RCRA BENZOIN TINCTURE AND BETADINE	NON-REGULATED SOLID (NONE)
250	NONE	NON-HAZARDOUS SPILLS/NON-RCRA OILS AND FLUIDS, SYN AND NATURAL	NON-REGULATED SOLID (NONE)
251	NONE	GREASE	NON-REGULATED SOLID (NONE)
252	NONE	EPOXY RESIN (ADHESIVE)	NON-REGULATED LIQUID (EPOXY) (NONE)
260	U122, D001	FORMALDEHYDE SOLUTION DISCARDED METHANOL	WASTE FORMALDEHYDE SOLUTION (ORM-A UN1198)
275	NONE	USED DIESEL FUEL BLEND	COMBUSTIBLE LIQUID NOS DIESEL (COMBUSTIBLE LIQUID NA1993)
280	D008	USED OIL FILTERS TERNE PLATED AND ALLOY OF TIN AND LEAD	HAZARDOUS WASTE SOLID NOS LEAD (ORM-E NA9189)
285	D001	CORROSION PREVENTIVE COMPOUND P-L	WASTE SOLID NOS NAPTHA AND ASPHALT (COMBUSTIBLE LIQUID NA1993)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
286	D001, D008	CORROSION PREVENTIVE COMPOUND P-2	HAZARDOUS WASTE SOLID NOS STODDARD SOLVENT AND ASPHALT (ORM-E NA9189)
287	D001, D008	CORROSION PREVENTIVE COMPOUND P-2 SPILL CLEAN UP	HAZARDOUS WASTE SOLID NOS STODDARD SOLVENT AND ASPHALT (ORM-E NA9189)
288	D001, D005	CORROSION PREVENTIVE-FINGERPRINT REMOVER	WASTE COMBUSTIBLE LIQUID NOS METHYLAMYI ALCOHOL (COMBUSTIBLE LIQUID NA1993)
289	D001	FINGERPRINT REMOVER WEAPONS CLEANER	WASTE PETROLEUM DISTILLATE (COMBUSTIBLE LIQUID UN1268)
300	D006, D007, D008	CITRIKLEEN SLUDGE ETHANOLMINE	HAZARDOUS WASTE LIQUID NOS CITRIKLEEN (OR-E NA9189)
305	D008	GREASE	HAZARDOUS WASTE SOLID NOS GREASE AND LEAD (ORM-E NA9189)
	D004, D005, D006, D007, D008, D009, D010, D011	METAL CARBON CARTDIDGE/FILTERS	HAZARDOUS WASTE SOLID NOS(NA3077)
310	D007	CHARCOAL FILTERS DISCARDED GAS MASK FILTERS	HAZARDOUS WASTE SOLID NOS CHARCOAL FILTERS (ORM-E NA9189)
313	NONE	GRINDING/SANDING RESIDUES	NON-REGULATED SOLID (NONE)
314	NONE	FINE GRADE POWDER-GRAY DENTAL WASTE	NON-RCRA REGULATED DENTAL WASTE (NONE)
315	D006, D008	SAND BLAST GRIT	HAZARDOUS WASTE SOLID NOS CADMIUM AND LEAD (ORM-E NA9189)
316	NONE	SOIL CONTAMINATED WITH PETROLEUM HYDROCARBONS	SOIL CONTAMINATED WITH PETROLEUM HYDROCARBONS (NONE)

Table 5-1b
Marine Corps Base Camp Lejeune
Identified Wastes

WID #	EPA HW #	WASTE NAME	WASTE DOT SHIPPING NAME
317	NONE	SOIL CONTAMINATED WITH PETROLEUM HYDROCARBONS	SOIL CONTAMINATED WITH PETROLEUM HYDROCARBONS (NONE)
318	D007; D008	SPILL RESIDUE CONTAMINATED WITH PAINT ENAMEL/ALKYD	HAZARDOUS WASTE SOLID NOS (ORM-E NA9189)
319	NONE	RESIN	COMBUSTIBLE LIQUID NOS FUEURYL ALCOHOL, FORMALDEHYDE, RESIN (COMBUSTIBLE LIQUID NA1993)
320	D007	TICONIUM METAL AND WASTE DENTAL WORK	WASTE POISON B SOLID NOS CHROMIUM AND BERYLLIUM (POISON B UN2811)
321	NONE	5% AMMONIA PASTE GEL	NON-REGULATED LIQUID (NONE)
600	NONE	AMMONIUM CHLORIDE SOLID	AMMONIUM CHLORIDE (NONE)
601	NONE	HYPOSULPHITE OF SODA	HYPOSULPHITE OF SODA (NONE)
610	NONE	NICKEL SALTS	NICKEL SALTS (NONE)
611	NONE	BORIC ACID SOLID	BORIC ACID (NONE)
616	NONE	DEVELOPER X-RAY	X-RAY DEVELOPER (NONE)
650	NONE	MIRACHEM 100 CLEANER/DEGREASER AROMATIC HYDROCARBONS	MIRACHEM 100 CLEANER/DEGREASER (NONE)
655	NONE	ACRYLIC POLYMER	ACRYLIC POLYMER AQUEOUS SOLUTION (NONE)
656	NONE	DISENFECTANT ISOPROPYL ALCOHOL, 4&6 CHLORO2-PHENYLPHENAL	DISENFECTANT (NONE)

Table 5-1c
Marine Corps Base Camp Lejeune
Identified Medical Wastes

WID #	EPA HW #	MEDICAL WASTE NAME	MEDICAL WASTE DOT SHIPPING NAME
039	D001	GRAM SAIN SET - SAFRANIN STAIN	FLAMMABLE LIQUID NOS (FLAMMABLE LIQUID)
001	D001, D002	ACETIC ACID, GLACIAL	WASTE ACETIC ACIT, GLACIAL (CRROSIVE)
003	NONE	ANTIVENIN POYVALENT - SNAKE BITE KITS	NONE
005	NONE	BBL GAS PACK-DISPOSABLE ANAEROBIC INDICATOR-AQUEOUS SOLUTION	NONE
007	D001	BENZOIN TINCTURE - BENZOIN, ISOPROPYL ALCOHOL	WASTE ISOPROPYL ALCOHOL (FLAMMABLE LIQUID)
009	NONE	BETADINE OINTMENT	BETADINE OINTMENT (NONE)
011	NONE	BETADINE SOLUTION	BETADINE SOLUTION (NONE)
013	DOO1	BUTANE	WASTE LIQUIFIED PETROLEUM (FLAMMABLE GAS)
015	D001	CAMCO QUICK STAIN	WASTE FLAMMABLE LIQUID NOS (FLAMMABLE LIQUID)
017	NONE	CIDEX	GLUTARALDEHYDE SOLUTION (NONE)
019	NONE	CRYSTAL VIOLET	CRYSTAL VIOLET (NONE)
021	D009	TPI-TITRATOR REAGENT MERCURIC NITRATE SOLUTION	HAZARDOUS WASTE LIQUID NOS MERCURY (ORM-E)
023	D001	DIATEX SYNTHETIC MOUNTING AGENT	WASTE FLAMMABLE LIQUID NOS TOLUENE, ACRYLIC RESIN (FLAMMABLE LIQUID)
025	U112	ETHYL ACETATE	WASTE ETHYL ACETATE (FLAMMABLE LIQUID)
027	D001	FAST RAY SELF CURING PLASTIC-SOLVENT	WASTE FLAMMABLE LIQUID NOS XYLENE AND TETRAHYDROFURAN (FLAMMABLE LIQUID)
029	D001	FORMALIN, FORMALDEHYDE SOLUTION	WASTE FORMALDEHYDE (ORM-A)

Table 5-1c
Marine Corps Base Camp Lejeune
Identified Medical Wastes

WID #	EPA HW #	MEDICAL WASTE NAME	MEDICAL WASTE DOT SHIPPING NAME
031	D003	GAS PAK - DISPOSABLE HYDROGEN AND CARBON DIOXIDE GENERATOR	WASTE WATER REACTIVE SOLID NOS SODIUM BOROHYDRIDE (FLAMMABLE SOLID)
033	NONE	GENTAMICIN SULFATE	GENTAMICIN SULFATE (NONE)
035	D001	GIESMA STAIN	WASTE FLAMMABLE LIQUID NOS GLYCERIN AND METHANOL (FLAMMABLE LIQUID)
037	NONE	GLYCERIN	GLYCERIN (NONE)
039	NONE	GRAM STAIN SET-SAFRAIN STAIN	COMBUSTIBLE LIQUID NOS (COMBUSTIBLE LIQUID)
041	NONE	GRAM STAIN SET-CRYSTAL VIOLET STAIN	CRYSTAL VIOLET STAIN (NONE)
043	D001	GRAM STAIN SET-BACTO GRAM DECOLORIZER	WASTE FLAMMABLE LIQUID NOS ACTONE AND ISOPROPANOL (FLAMMABLE LIQUID)
045	NONE	GRAM STAIN SET-GRAM IODINE	IODINE SOLUTION (NONE)
047	D001	HAND PIECE CLEANER	WASTE FLAMMABLE LIQUID NOS
049	D001	HEMA TEST-TEST FOR BLOOD IN FECES	WASTE OXIDIZER NOS STRONTIUM (OXIDIZER)
051	D001	HEMOCULT	WASTE FLAMMABLE LIQUID NOS ETHYL ALCOHOL (FLAMMABLE LIQUID)
053	D002	HYDROCHLORIC ACID	WASTE HYDROCHLORIC ACID (CORROSIVE MATERIAL)
055	D001	ISOPROPHYL ALCOHOL	WASTE ISOPROPHL ALCOHOL (FLAMMABLE LIQUID)
057	NONE	IODINE SCRUBS	IODINE SCRUB (NONE)
059	D001	JET KIT ACRYLIC TOOTH SHADES-RESIN LIQUID	WASTE RESIN SOLUTION (FLAMMABLE LIQUID)
061	NONE	JET KIT ACRYLIC TOOTH SHADES-POWDER ACRYLICS	ACRYLIC POWDER (NONE)

Table 5-1c
Marine Corps Base Camp Lejeune
Identified Medical Wastes

WID #	EPA HW #	MEDICAL WASTE NAME	MEDICAL WASTE DOT SHIPPING NAME
069	NONE	MANNITOL	MANNITOL SOLUTION (NONE)
071	NONE	MANNITOL SALT AGAR	MANNITOL SALT AGAR (NONE)
073	U154	METHYL ALCOHOL	WASTE METHYL ALCOHOL (FLAMMABLE LIQUID)
075	NONE	METHYLENEBLUE INDICATOR	?
077	NONE	MICROFIBRILLAR COLLAEGEN (AVITENE)	COLLAGEN (NONE)
079	NONE	MIF PROCEDURE KIT (FOR SPECIMENS)	IODINE SOLUTION (NONE)
081	NONE	N-MULTISTIX, AMES REAGENT STRIPS	NONE
083	NONE	ISONIAZIDE TABLETS, ISONICOTONIC ACID HYDROZIDE	NIACIN (NONE)
085	NONE	PARA CHLORO PHENOL	POISON B SOLID NOS (POISON UN2811)
086	U188	PHENOL	WASTE PHENOL(POISON B UN1671)
087	NONE	PIERCE UREA NITROGEN RAPID STAT KIT-UREA NITROGEN STANDARD	UREA NITROGEN STANDARD (NONE)
089	D002	PIERCE UREA NITROGEN RAPID STAT KIT-ACID REAGENT	WASTE ACID LIQUID NOS SULFURIC ACID AND FERRIC CHLORIDE (CORROSIVE MATERIAL NA1760)
091	NONE	PIERCE UREA NITROGEN RAPID STAT KIT-COLOR REAGENT	COLOR REAGENT (NONE)
095	NONE	POTASSIUM IODINE	POTASSIUM IODATE (NONE)
097	NONE	POTASSIUM PHOSPHATE TRIBASIC	POTASSIUM PHOSPHATE TRIBASIC (NONE)
099	NONE	POTASSIUM PHOSPHATE DIBASIC	POTASSIUM PHOSPHATE DIBASIC (NONE)
101	NONE	POTASSIUM PHOSPHATE MONOBASIC	POTASSIUM PHOSPHATE MONOBASIC (NONE)

Table 5-1c
Marine Corps Base Camp Lejeune
Identified Medical Wastes

WID #	EPA HW #	MEDICAL WASTE NAME	MEDICAL WASTE DOT SHIPPING NAME
102	D001, D009	PVA FIXTATIVE	WASTE FLAMMABLE LIQUID NOS ALCOHOLS AND MERCURIC CHLORIDE (FLAMMABLE LIQUID UN1993)
103	NONE	REOGRAFIN 60	RENOGRAFIN 60 (NONE)
107	NONE	SODIUM CITRATE	SODIUM CITRATE (NONE)
109	NONE	SODIUM PHOSPHATE, TRIBASIC	SODIUM PHOSPHATE, TRIBASE (NONE)
111	NONE	SODIUM PHOSPHATE, DIBASIC	SODIUM PHOSPHATE, DIBASIC (NONE)
113	NONE	SODIUM PHOSPHATE MONBASIC	SODIUM PHOSPHATE, MONOBASIC (NONE)
115	D001, D011	SILVER NITRATE APPLICATOR	WASTE OXIDIZER NOS SILVER NITRATE (OXIDIZER UN1479)
117	NONE	SULFOSALICYLIC ACIDE DIHYDRATE	SULFOSALICYLIC ACID DIHYDRATE (NONE)
119	NONE	TEST KIT SERUM CARBON DIOXIDE DETERMINATION	TEST KIT ...(NONE)
120	D001	TURPENTINE IN XYLENE	WASTE FLAMMABLE LIQUID NOS XYLENE AND TURPENTINE (FLAMMABLE LIQLUID UN1993)
121	NONE	UNDECYLENIC ACID	UNDECYLENIC ACID (NONE)
123	NONE	WESCODYNE	DISINFECTANT CLEANING COMPOUND (NONE)
125	U239	XYLENE	WASTE XYLENE (FLAMMABLE LIQLUID UN1307)
127	NONE	ZINC UNDECYLENATE	ZINC UNDECYLENATE (NONE)
129	D001, D009	TPI-TITRATOR REAGENT MERCURINC NITRATE SOLUTION	WASTE MERCURIC NITRATE (OXICIZER UN1625)
130	D002	TPI-TITRATOR REAGENT NITRIC ACID	WASTE NITRIC ACID (CORROSIVE MATERIAL NA1760)

Table 5-1c
Marine Corps Base Camp Lejeune
Identified Medical Wastes

WID #	EPA HW #	MEDICAL WASTE NAME	MEDICAL WASTE DOT SHIPPING NAME
131	NONE	TPI-TITRATOR REAGENT - CHLORIDE INDICATOR	CHLORIDE INDICATOR (NONE)
153	D001, D002	KIDAK RPX-OMAT DEVELOPER REPLENISHER PART B	WASTE ACID LIQUID NOS ACETIC ACID AND 1-PHENYL-3-PYROZALIDINONE (CORROSIVE MATERIAL NA1607)
159	D002	KODAK-FIXER AND REPLENISHER-PART B	WASTE ACID LIQUID NOS ACETIC AND GLUCONIC ACID (CORROSIVE MATERIAL NA1760)
167	D002	KODAK-X-RAY-DEVELOPER	WASTE ALKALINE LIQUID NOS POTASSIUM HYDROXIDE AND POTASSIUM SULFITE (CORROSIVE MATERIAL NA1719)
173	D001	KODAK-RAPID FIXER PART B	WASTE ACID LIQUID NOS SULFURIC ACID AND ALUMINUM SULFATE (CORROSIVE MATERIAL NA1760)

Table 5-2
Exposure Guidelines for Suspected Site Contaminants

Chemical	Odor Threshold (PPM)	OSHA PEL (PPM)	ACGIH TLV (PPM)	NIOSH REL (PPM)	Autoignition Temperature	Flammable Range (% by Volume)
BENZENE	4.68 ppm	1 ppm 5 ppm (STEL)	10 ppm	0.1 ppm 1.0 ppm (Ceiling)	1,588.6 °F	1.3% to 7.1%
CHLOROBENZENE	0.21 ppm	75 ppm	10 ppm	not listed	1,675.4°F	1.3% to 7.1%
CHLOROFORM	200 ppm	2 ppm	10 ppm	2 ppm (Ceiling)	N/A	N/A
CIS 1,2 DICHLOROETHENE	0.085 ppm	200 ppm	200 ppm 250 ppm (STEL)	200 ppm	1,352°F	9.7% to 12.8%
ETHYL BENZENE	140 ppm	100 ppm 125 ppm (STEL)	100 ppm 125 ppm (STEL)	100 ppm 125 ppm (STEL)	1,351.4°F	1.0% to 6.7%
METHYL CHLORIDE	100 ppm	50 ppm 100 ppm (STEL)	50 ppm 100 ppm (STEL)	N/A	1,661.6°F	7.6% to 19%
TETRACHLOROETHENE	5 ppm	100 ppm 200 ppm (Ceiling)	50 ppm 200 ppm (STEL)	N/A	N/A	N/A
TOLUENE	40 ppm	100 ppm 150 ppm (STEL)	100 ppm 150 ppm (STEL)	100 ppm 200 ppm (Ceiling)	996.5°F	1.3% to 7.1%

Table 5-2
Exposure Guidelines for Suspected Site Contaminants

Chemical	Odor Threshold (PPM)	OSHA PEL (PPM)	ACGIH TLV (PPM)	NIOSH REL (PPM)	Autoignition Temperature	Flammable Range (% by Volume)
TRICHLOROETHENE	50 ppm	50 ppm 200 ppm (STEL)	50 ppm 200 ppm (STEL)	25 ppm	N/A	11% to 41%
VINYL CHLORIDE	260 ppm	1 ppm 5 ppm (STEL)	1 ppm 5 ppm (STEL)	lowest feasible conc.	1,373°F	3.6% to 33%
XYLENE	0.4 to 20 ppm	100 ppm 150 ppm (STEL)	100 ppm 150 ppm (STEL)	100 ppm 200 ppm (Ceiling)	1,357°F	1% to 7%
ARSENIC	N/A	0.01 mg/m^3	0.2 mg/m^3	0.002 mg/m^3	N/A	N/A
BARIUM	N/A	not in table Z-1-A	not listed	not listed	N/A	N/A
CADMIUM	N/A	0.5 mg/m^3	0.5 mg/m^3	0.01 mg/m^3	N/A	N/A
CHROMIUM	N/A	1 mg/m^3	5 mg/m^3	not listed	N/A	N/A
COPPER	N/A	1 mg/m^3	1 mg/m^3	not listed	N/A	N/A
LEAD	N/A	50 $\mu g/m^3$	0.15 mg/m^3	0.1 mg/m^3 (in air)	N/A	N/A
MERCURY	N/A	0.05 mg/m^3	1 mg/m^3	0.05 mg/m^3	N/A	N/A
NICKEL	N/A	1 mg/m^3	1 mg/m^3	0.015 mg/m^3	N/A	N/A
SELENIUM	N/A	0.2 mg/m^3	0.2 mg/m^3	not listed	N/A	N/A

Table 5-2
Exposure Guidelines for Suspected Site Contaminants

Chemical	Odor Threshold (PPM)	OSHA PEL (PPM)	ACGIH TLV (PPM)	NIOSH REL (PPM)	Autoignition Temperature	Flammable Range (% by Volume)
ZINC	N/A	not in table Z-1-A	not listed	not listed	N/A	N/A

Table 5-3
Mechanical, Electrical, Physical and Temperature Hazards

Field Activity	Mechanical Equipment Used	Electrical Hazard	Physical Hazard	Temperature Hazard
Visual Observation	None	None	Slip, trip, fall Insect bites	Heat/Cold (H/C) Stress

Physical Hazards

While conducting field operations on foot, personnel must walk even during emergency situations. Running greatly increases the probability of slipping, tripping, and falling. When approaching the edges of ditches, ponds, or ravines/gullies, personnel should carefully examine the edges while approaching. Certain activities present a hazard level that must be dealt with on a case by case basis. These activities are not covered by this plan. Examples of such activities are: confined space entry; moving of unknown drums or containers; and entering excavations or trenches that are more than 3 feet deep. Should the Project Manager deem it necessary to perform an activity such as those listed above, it is that person's responsibility to contact the Project Health and Safety Coordinator and request an addendum to this plan specifying the health and safety procedures, training, and conditions necessary for undertaking task. These activities are prohibited until this plan addendum is provided.

Temperature Hazards

Temperature exposure guidelines are located in Appendix D of the EnSafe Corporate Health and Safety Manual.

Noise Hazards

Noise level exposures may be evaluated for the various site activities. Where noise levels exceed an 8-hour time weighted average of 85 dB as measured on the A scale (slow response), hearing conservation measures will be required. Because of the types of operations at this site, it is not anticipated that administrative or engineering controls will be feasible. Therefore, if hearing conservation measures are required, ear muff type hearing protectors complying with ANSI S12.6-1984 will be required.

Eye Hazards

This program applies to EnSafe employees. It provides for compliance with federal and EnSafe eye protection standards. Suitable eye protection equipment will be required where machines or operations present the hazard of chips, other flying objects, glare, compressed gases, liquids, injurious radiation, or a combination of these hazards to the eye. Workers will wear safety glasses or goggles while working in these area. Personnel will wear sealed chemical protective goggles when exposed to chemical reactions and spills or fluids under pressure.

6.0 EMPLOYEE PROTECTION

Employee protection for this project includes Standard Safe Work Practices and Personal Protective Equipment (PPE) which will be addressed in this section. Extreme Weather Condition Procedures, Work Limitations, and Exposure Evaluation will be covered in Sections 7 through 9 of this report.

6.1 Standard Safe Work Practices

Standard safe work and personal hygiene practices that will be followed include:

- Eating, drinking, chewing gum or tobacco, smoking or any activity that increases the probability of hand-to-mouth transfer and ingestion of material is prohibited in any area designated as contaminated, unless authorized by the site health and safety manager.
- Hands and face must be thoroughly washed upon leaving the work area.
- Contact lenses shall not be worn onsite.
- Contact with surfaces suspected of being contaminated should be avoided. Whenever possible, employees should not walk through puddles, leachate or discolored surfaces, or lean, sit or place equipment on drums, containers or soil suspected of being contaminated.
- Medicine and alcohol can exacerbate the effects of exposure to toxic chemicals. Prescribed drugs should not be taken by personnel on cleanup or response operations where the potential for absorption, inhalation, or ingestion of toxic substances exists unless specifically approved by a qualified physician. Consumption of alcoholic beverages or use of illegal drugs will not be allowed before or during operations.

6.2 Personal Protective Equipment (PPE)

The selection of personal protective equipment is based on information collected from the site screening.

Section 2.10 of the EnSafe Corporate Health and Safety Manual contains a general description of:

Selection of Protective Clothing and Accessories (Section 2.10.1)

Selection of Chemical-Protective Clothing (CPC) (Section 2.10.2)

- Permeation and Degradation
- Heat Transfer Characteristics
- Other Considerations
  - — Durability
  - — Flexibility
  - — Temperature effects
  - — Ease of decontamination
  - — Compatibility with other equipment
  - — Duration of use

Special Conditions

- Protective Clothing and Accessories (Tables 2.2, 2.3, 2.4, 2.5, 2.6, 2.7, and 2.8)
- Selection of Ensembles (Level of protection)
- Reasons to upgrade
- Reasons to downgrade
- Sample Protective Ensembles (Tables 2.9, 2.10, and 2.11)
- Training (Section 2.11.1)
- Work Mission Duration (section 2.11.2)
  - — Air Supply Consumption
  - — Suit/Ensemble Permeation and Penetration
  - — Ambient Temperature
  - — Coolant Supply
- Personal Use Factors (Section 2.12)
- Donning an Ensemble (Section 2.12.1)

- In-Use Monitoring (Section 2.12.2)
- Doffing and Ensemble (Section 2.12.3)
- Clothing Reuse (Section 2.12.4)
- Inspection (Section 2.12.5)
- Storage (Section 2.12.6)
- Maintenance (Section 2.12.6)

Based on the concentration level of the suspected site contaminants and the types of operations that will be conducted, a modified Level D Protective Clothing Ensemble will be used for work on this site. Section 2.9 of the EnSafe Corporate Health and Safety Manual provides general guidance and action levels for upgrading levels of PPE. Additional specific information (MSDS) about some of the site contaminants has been provided in Appendix A of this health and safety plan.

Level D Protective Clothing Ensemble

- Eye protection, meeting the ANSI Z87.1-1968 standard
- Chemical-resistant, steel toe, steel shank boots (or equivalent), meeting the ANSI Z41.1-1967 standard
- Optional Equipment as needed:
  - Coveralls

The following criteria are also provided to assist in determining when levels of protection must be upgraded:

Site Conditions That Require Upgrading to Level C PPE

- Concentration of unknown organic vapors exceeds 5 ppm in the breathing zone.
- Concentration of any constituent exceeds 50 percent of the OSHA Permissible Exposure Limit (PEL) in the breathing zone.

- Airborne particulates (dusts) exceed 5 mg/m^3 respirable particulates in the breathing zone.

Criteria for Use of Level C PPE

- Work area must contain at least 19.5 percent but less than 23.0 percent oxygen content by volume.
- Contaminants have been identified, concentrations determined, and have a distinctive odor or taste.
- Cartridges or canisters exist that will provide protection from the contaminants at the concentrations present.
- Site conditions do not indicate the use of Level A or B PPE.
- General site entry conditions for combustible gases and radiation are within the guidelines contained in the EnSafe Corporate Health and Safety Manual, Table 2.1.

Personal Hygiene

- Eating, drinking, chewing gum or tobacco, taking medication, and smoking are prohibited in contaminated or potentially contaminated areas or where the possibility for transfer of contamination exists.
- When leaving contaminated or potentially contaminated areas, hands and face must be thoroughly washed immediately after decontamination. A thorough shower and washing of the body may also be required.
- Avoid contact with potentially contaminated substances. Do not walk through puddles, pools, muds, or other wet areas of the site. Whenever possible, avoid kneeling on the ground and leaning or sitting on the ground, drums, or equipment (other than the operators seat).

Personal Protection

Persons entering this site will be familiar with:

- Standard safe operating procedures.
- Adhere to instructions in this site health and safety plan.
- The location of site telephones.
- The procedures for obtaining emergency medical assistance.
- The symptoms of heat stress and cold exposure and know what actions to take when an individual shows these symptoms.
- Protective clothing required on this site and how to wear and operate protective equipment.

Site Operations

- During routine operations and emergency incidents verbal and hand signals as established in this plan will be used.
- Prior to personnel entering this site, a safety briefing will be conducted. At a minimum, the safety briefing will cover safe work practices, communications, and emergency procedures.
- Site entrance and exit routes and emergency routes will be discussed with each site manager prior to each site entry.

6.3 Safety Equipment

Site personnel must be adequately protected from potential health and safety hazards. Therefore, the EnSafe site manager will maintain an inventory of safety equipment in order to meet anticipated hazards. Site personnel must be instructed in the proper use of this equipment before entering the work area.

6.4 Decontamination

As part of the system to prevent or reduce physical transfer of contaminants by personnel onsite and offsite, provision will be made for decontaminating personnel. The general guidelines for the decontamination procedures can be found in Section 2.18 of the EnSafe Corporate Health

and Safety Manual and in the protocols contained in "Hazardous Activities," and USEPA guidance document.

Typical solutions for personnel decontamination will be soap and water.

7.0 TEMPERATURE EXPOSURE GUIDELINES

Temperature exposure guidelines are located in Appendix D of the EnSafe Corporate Health and Safety Manual.

8.0 WORK LIMITATIONS

Site activities will be conducted only during daylight hours. Persons scheduled to perform site work will have completed initial health and safety training and supervised field training as specified in 29 CFR 1910.120(e). The site supervisor will have completed an additional 8 hours of training in site management in accordance with 29 CFR 1910.120(e). Personnel must keep their health and safety training current by attending an 8 hour refresher training course annually. OSHA 24-hour or 40-hour training and 8-hour refresher certificates must be presented to the site manager before site work may begin. In addition, personnel must provide proof of participation in an annual medical surveillance program (medical surveillance records for EnSafe personnel are maintained in the Memphis office).

9.0 EXPOSURE EVALUATION

Personnel scheduled for site activities will have a baseline physical examination which includes a physical examination, stressing examination of the neurologic, cardiopulmonary, musculoskeletal and dermatological systems, pulmonary function testing, multi-chemistry panel and urinalysis, and be declared fit for duty. An exposure history form will be completed for each worker participating in site activities. An examination and updated occupational history will be repeated on an annual basis and upon termination of employment as required by 29 CFR 1910.120(f). The content of the annual or termination examination will be the same as the baseline physical. A qualified physician will review the results of the annual examination and exposure data and request further tests or issue medical clearances as appropriate.

After any job-related injury or illness, there will be a medical examination to determine fitness for duty or for the need for any job restrictions. The site health and safety manager will review the results with the examining physician before releasing the employee for work. A similar examination will be performed if an employee has missed at least 3 days of work due to a non-job related injury or illness requiring medical attention. Medical records shall be maintained by the employer or the physician for at least 30 years following termination of employment. The symptoms of exposure to site contaminants are contained in the respective site contaminant Material Safety Data Sheets that are attached to this plan.

10.0 MONITORING REQUIREMENTS

Air monitoring will not be conducted during this work. It is assumed that MCB Base Safety has cleared these sites for working conditions without the need for upgraded personnel protection.

11.0 AUTHORIZED PERSONNEL

It is anticipated that the following EnSafe personnel will be onsite at various times during the SWMU Assessment:

- EnSafe Principal-in-Charge — Dr. James Speakman
- EnSafe Project Manager — Mr. Robert Moser
- EnSafe Project Scientist — Mr. Chris McKeeman
- EnSafe Site Personnel — To be assigned

11.1 Responsibilities of the EnSafe Project Manager

The project manager will direct the site activities and operation, and are responsible for the following:

- Ensuring that personnel are aware of:
  - — Names of personnel and alternates responsible for site safety and health
  - — Safety, health and other hazards present on the site
  - — Use of PPE and assuring that the equipment is available
  - — Work practices which can minimize hazard risks
  - — Safe use of engineering controls and equipment on the site
  - — Medical surveillance requirements including recognition of symptoms and signs which might indicate over-exposure to hazards
  - — Site control measures, decontamination procedures, site standard operating procedures, the contingency plan, and responses to emergencies including the necessary PPE.

- The project manager will also ensure that employees have received at least 40 hours of health and safety instruction off the site, and actual field experience under the direct supervision of a trained experienced supervisor. Workers who may be exposed to unique or special hazards will receive additional training.

- Monitoring personnel performance to ensure that mandatory health and safety procedures are being performed and correcting any performances that do not comply with the health and safety plan.

- Ensuring that field personnel employed on the site are covered by a medical surveillance program as required by 29 CFR 1910.120(f):
  - — Consulting with the health and safety coordinator and/or other personnel.
  - — Preparing and submitting project reports, including progress, accident, incident, contractual, etc.
  - — Monitoring personnel decontamination to ensure that personnel are complying with the established decontamination procedures.

11.2 Responsibilities of the EnSafe Health and Safety Coordinator

It is the responsibility of the EnSafe health and safety coordinator to:

- Assure that a copy of the health and safety plan is maintained onsite during field activities.
- Advise the project manager on health and safety related matters involved at the site.
- Direct and ensure that the safety program is being correctly followed in the field, including the proper use of personal protective and site monitoring equipment.
- Ensure that field personnel observe the appropriate work zones and decontamination procedures.
- Report any safety violations to the project manager.

- Ensure safety briefings are schedule during field activities. The items listed in Table 11-1 will be discussed by a qualified individual at the site pre-entry briefings, as well as at daily or periodic site briefings.

Table 11-1
Items to be Discussed at Safety Briefings

Frequency	Items to be discussed
Initial at each site	• Site Chemical Hazards • Types and use of PPE • Location and Use of Emergency Equipment • Decontamination Procedures
Seasonal	• Heat Stress • Cold Exposure

The person responsible for daily health and safety will be trained to use the air monitoring equipment, and interpret the data collected with the instruments, and be familiar with symptoms of heat stress and cold exposure and the location and use of safety equipment onsite. He will also be familiar with this health and safety plan.

11.3 Responsibilities of Onsite Field Personnel

- Personnel going on the site must be thoroughly briefed on anticipated hazards and trained in equipment to be worn, safety procedures to be followed, and emergency procedures and communications.
- Field personnel should be aware of and avoid potentially dangerous situations such as the presence of strong, irritating or nauseating odors.
- Field personnel will be familiar with the physical characteristics of the site:
  - wind direction relative to contamination zones
  - accessibility to associates, equipment and vehicles
  - communications
  - operation zones

- site access
- nearest water sources

- Procedures for leaving a contaminated area must be planned and implemented before going onsite according to the health and safety plan.

12.0 SITE CONTINGENCY PLAN

Site assessment activities present a potential risk to onsite personnel. During routine operations, risk is minimized by establishing good work practices, staying alert, and using proper PPE. Unpredictable events such as physical injury, chemical exposure, or fire may occur.

12.1 Emergency Contacts

In the event of such an incident, the EnSafe Health and Safety Coordinator, Mr. Doug Petty, must be contacted immediately by phone or messenger at (919) 372-7962.

Local agencies to be contacted:

MCB Fire Department	—	(911)
MCB Police	—	(911)
Paramedics (Fire Rescue)	—	(911)
Hospital	—	_____ (to be completed)
(Emergency Room)		
Onslow County	—	_____ (to be completed)
Emergency Management Agency		

12.2 Directions from the site to Jacksonville Hospital

(To be completed)

Figure 12-1 is a map showing the route from the site to Hospital.

Figure 12-1 Route from site to Hospital

12.3 Site Resources

Telephones for communication offsite are located in the main office of Valley Products Company.

Response Resources

EnSafe personnel are trained for response to onsite emergency situations involving assessment accidents. The response equipment outlined below will be available during this assessment.

Emergency Supplies:

Goggles (plastic)
1 First aid kit
Latex gloves

12.4 Pre-Emergency Planning

During the daily site briefing, site workers will be trained in and reminded of the provisions of the emergency response plan, communications systems, and evacuation routes.

12.5 Personnel Roles and Lines of Authority

The EnSafe Health and Safety Coordinator has the primary responsibility for responding to and correcting emergency situations that EnSafe personnel are associated with. This includes taking appropriate measures to ensure the safety of site personnel, facility personnel, and the public. Possible actions may involve evacuation of personnel from the site and/or site area, and the evacuation of personnel from adjacent facilities or residences.

12.6 Emergency Recognition/Prevention

Table 5-3 lists the mechanical, electrical, physical and temperature hazards associated with activities on this site. The MSDS for some of the suspected site contaminant have been included in the appendix to this plan and list the chemical and toxicological hazards.

12.7 Evacuation Routes/Procedures

In the event of an emergency which necessitates an evacuation:

STOP WORK. Keep calm, think, avoid panic and confusion. Move to the nearest safe exit in your area.

WALK to the nearest safe exit. **DO NOT RUN,** nor **LINGER** in entry ways or roadways.

LEAVE the area and report to your designated assembly area (**IF SAFE**), or to a safe area away from the incident.

REPORT to your supervisor as soon as you arrive at the assembly area and follow his/her instructions.

STAY outside the affected area until notified that it is safe to reenter.

Each supervisor must conduct a **HEAD COUNT** and report to the Health and Safety Coordinator when his/her employees have cleared the area or if any employees are missing.

Personnel will **REMAIN** at the assembly area until directed to another location by the Health and Safety Coordinator.

12.8 Emergency Medical Treatment Procedures

Any person who becomes ill or injured must be decontaminated to the maximum extent possible. If the injury or illness is minor, full decontamination should be performed. First aid should be administered while awaiting an ambulance or paramedics. Injuries and illnesses must be immediately reported to the EnSafe Project Manager. When it is necessary to transport an

employee to a hospital, the employee must be accompanied by an individual who is familiar with the suspected site contaminants. A copy of the suspected site contaminant MSDS should accompany any employee to the hospital.

12.9 Emergency Incident Procedures

In an actual emergency, EnSafe's Health and Safety Coordinator or his designee must immediately notify site/facility personnel by proper communication. In an actual emergency (spill, release, fire, or explosion), the Health and Safety Coordinator must immediately identify the character, exact source, amount, and the real extent of any released material. Assessment of possible hazards to human health or environment due to release, fire, or explosion must be made. Effects of response methods must be understood to control the hazard area.

If the Health and Safety Coordinator determines that the facility has had a release, fire, or explosion which could threaten human health or the environment outside the facility, he must report his finding as follows:

- If his assessment indicates that evacuation of local areas may be advisable, he must immediately notify appropriate MCB emergency response authorities.
- He must immediately notify the National Response Center at (800) 424-8802.
- His report must include the following information:
  - Name and telephone number of reporter
  - Name and address of facility
  - Time and type of incident (spill, fire, explosion)
  - Name and quantity of material(s) involved, to the extent known
  - Extent of injuries, if any
  - Possible hazards to human health or environment

Emergency equipment must be decontaminated, cleaned, and placed in proper condition for its intended use after each response action.

13.0 FORMS

The following forms will be used in implementing this health and safety plan:

Plan Acceptance Form

Plan Feedback Form

Accident Report Form

Exposure History Form

The Plan Acceptance Form will be filled out by employees working on the site before site activities begin. The Plan Feedback Form will be filled out by the site safety officer and any other onsite employee who wishes to fill one out. The Accident Report Form will be filled out by the Project Manager if an accident occurs. The Exposure History Form will be completed by both the project manager and the individual(s) for whom the form is intended.

Completed forms must be returned to the Principal-in-Charge at EnSafe, Memphis, Tennessee.

PLAN ACCEPTANCE FORM

PROJECT HEALTH AND SAFETY PLAN

SUPPLEMENT

INSTRUCTIONS: This form is to be completed by each person involved in the field assessment at Marine Corps Base Camp Lejeune and returned to the EnSafe Site Project Manager.

Job No. 2284-004

Project: SOLID WASTE MANAGEMENT UNIT ASSESSMENT

I have read and understand the contents of the above plan and agree to perform my work in accordance with said plan.

_____

Signature

_____

Print Name

_____

Company

_____

Date

PLAN FEEDBACK FORM

Problems with plan requirements:

Unexpected situations encountered:

Recommendations for revisions:

ACCIDENT REPORT FORM GOES HERE

EMPLOYEE EXPOSURE HISTORY FORM

EMPLOYEE NAME: _____

JOB NAME: _____

JOB NUMBER: _____

DATE(S) ON SITE (FROM/TO): _____

HOURS ON SITE: _____

CONTAMINANTS (SUSPECTED/REPORTED)

_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____
_____	_____

(SEE ATTACHED LABORATORY ANALYSIS)

APPENDIX A

MSDS

COMMON SYNONYMS:	CAS Reg. No.	NIOSH No.
Fuel oil (unspecified)		LS8950000
Fuel oil No. 1		
Kerosene	8008-20-6	OA5500000
Range oil		
JP-1		
Coal oil		
Fuel oil No. 2	68476-30-2	HZ1800000
Home heating oil		
Diesel oil		
Fuel oil No. 4	68476-31-3	
Residual fuel oil No. 4		
Fuel oil No. 5		
Residual fuel oil No. 5		
Navy special fuel oil		
Fuel oil No. 6	68553-00-4	
Residual fuel oil No. 6		
Bunker C oil		
Fuel oil 1-D		
Diesel oil (light)		
Fuel oil 2-D		
Diesel oil (medium)		

COMPOSITION:
Aliphatic hydrocarbons 64%
Olefinic hydrocarbons 1-2%
Aromatic hydrocarbons 35%

REACTIVITY	Various sources typically report that fuel oils are incompatible with strong acids, alkalies, and strong oxidizers such as liquid chlorine and oxygen. The NFPA reports vigorous reactions, ignition, or explosions involving chlorine, fluorine, or magnesium perchlorate. Fuel oils are considered to be miscellaneous combustible or flammable materials for compatibility classification purposes. Such substances typically evolve heat, fire, and toxic or flammable gases in reactions with oxidizing mineral acids, alkali or alkaline earth elemental metals, nitrides, organic peroxides or hydroperoxides, or strong oxidizing agents. Reactions with explosive materials may result in an explosion, while those with strong reducing agents may evolve heat and flammable gases. Non-oxidizing mineral acids generally evolve heat and innocuous gases (505,507,511).

PHYSICO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(60)
	• Color: colorless to brown	(60)
	• Odor: characteristic kerosene like	(60)
	• Odor Threshold: no data	( )
	• Liquid Density (g/ml at 20°C): fuel oil nos. 2, 6, 2-D - .87-.95; fuel oil nos. 1, 4, 5, 1-D (at 15°C) - .81-.936	(60)
	• Freezing/Melting Point (°C): -48-18	(60)
	• Boiling Point (°C): 151- >588	(60)
	• Flash Point (°C): ranges from 38-74 for various grades of fuel oil no. 1 to 69-169 for grades of fuel oil no. 5	(12,51,60, 504,506, 507)
	• Flammable Limits in Air, % by Volume: 0.6-1.3% to 5.0-7.5% for fuel oil nos. 1 - 5	(51,60 506,507)
	• Autoignition Temperature (°C): 177-329 depending on grade for fuel oil nos. 1 - 5	(51,60,506, 507,513)
	• Vapor Pressure (mm Hg at 21°C): 2.12-26.4	(60)
	• Saturated Concentration in Air (mg/m^3 at 20°C): not available	( )
	• Solubility in Water (mg/L at 20°C): -5	(2297)
	• Viscosity: fuel oil nos. 1, 2 1-D, 2-D (cp at 21°C) - 1.152-1.965; fuel oil nos. 4, 5, 6 (cp at 38°C) - 14.5-493.5	(60)
	• Surface Tension (dyne/cm at 20°C): 21-32	(60)
	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 3.3-7.06	(*)
	• Soil Adsorption Coefficient, K_{oc}: 962-5.5 x 10^6	(*)
	• Henry's Law Constant ($atm \cdot m^3/mol$ at 20°C): 5.9 x 10^{-5} - 7.4	(*)
	• Bioconcentration Factor: not available	( )

PERSISTENCE IN THE SOIL-WATER SYSTEM	Diesel oil hydrocarbons are expected to have moderate mobility and moderate persistence in most surface soils; persistence in deep soils and ground water may be higher. Volatilization, sorption, photooxidation, and biodegradation are all potential fate processes. Surface spills may be weathered to a limited extent by evaporation; downward migration of weathered surface spills and sub-surface discharges represent a potential threat to underlying ground water. Biodegradation of fuel oil hydrocarbons is expected to occur under environmental conditions favorable to microbial oxidation; naturally-occurring, hydrocarbon-degrading microorganisms have been isolated from polluted soils and, to a lesser extent, non-polluted soils. The hydrocarbons of residual fuel oils are expected to be less mobile (lower aqueous solubility, higher sorption and lower volatility) and more persistent (slower biodegradation) than the lighter diesel oil hydrocarbons.

*Range of values for representative hydrocarbons from major component classes (see Table 66-3).

PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water system is the migration of fuel oils to ground water drinking water supplies from leaking underground storage tanks or large spills. Vapors from leaked or spilled fuels may diffuse through soil and migrate into structures resulting in inhalation exposures.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (54,17): The effects of exposure to fuel oils are expected to resemble those of kerosene. Inhalation of high concentrations may cause headache, nausea, confusion, drowsiness, convulsions and coma. Ingestion may cause nausea, vomiting and in severe cases, drowsiness progressing to coma. Aspiration may cause extensive pulmonary injury. The liquid may produce primary skin irritation.
	Toxicity Based on Animal Studies: LD_{50} (g/kg) oral [rat] 5.1->24 (1924) skin -- no data LC_{50} (mg/m^3) inhalation -- no data
	Long-Term Effects: Kidney damage
	Pregnancy/Neonate Data: Negative
	Mutation Data: Limited evidence
	Carcinogenicity Classification: IARC - none assigned; NTP - none assigned

HANDLING PRECAUTIONS (1967)	No specific respirator guidelines were found for fuel oils. The following guidelines are for kerosene with a boiling range of 175-325°C • Less than or equal to 1000 mg/m^3: chemical cartridge respirator with half-mask facepiece and organic vapor cartridge or supplied air respirator with half-mask facepiece operated in demand mode • 1000-5000 mg/m^3: gas mask with full facepiece and organic canister, supplied-air respirator with full facepiece or self-contained breathing apparatus with full facepiece operated in demand mode • Appropriate protective clothing including gloves, aprons and boots • Chemical goggles if there is probability of eye contact.

EMERGENCY FIRST AID TREATMENT (1932)	Ingestion: Do not induce vomiting. Get medical attention • Inhalation: Move victim to fresh air. Give artificial respiration if necessary. Get medical attention • Skin: Wash contaminated skin with soap and water. If blistering or skin loss has occurred, wash remaining fuel off with sterile water only and treat as a thermal burn. Get medical attention • Eye: Irrigate with large amounts of water. Get medical attention.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): petroleum distillates (naphtha) 500 ppm
- AFOSH PEL (8-hr TWA): petroleum distillates (naphtha) 500 ppm

Criteria
- NIOSH IDLH (30-min): petroleum distillates (naphtha) 10,000 ppm
- ACGIH TLV® (8-hr TWA): petroleum distillates (naphtha) none established
- ACGIH STEL (15-min): petroleum distillates (naphtha) none established

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; fuel oils are not priority pollutants
- Aquatic Life
  No criterion established; fuel oils are not priority pollutants

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;
- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;
- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Toxic Substances Control Act (TSCA)
  Manufacturers and processors of the C9 aromatic hydrocarbon fraction must test it for neurotoxicity, mutagenicity, developmental toxicity, reproductive effects and oncogenicity. The C9 fraction is obtained from the reforming of crude petroleum. It consists of ethyltoluenes and trimethylbenzenes (1988). Testing will be conducted by the American Petroleum Institute. Interim reports must be submitted at 6-month intervals (1987).

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Occupational Safety and Health Act (OSHA)
  Employee exposure to petroleum distillates (naphtha) shall not exceed an 8-hour time-weighted-average of 500 ppm (298).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated fuel oils as hazardous materials which are subject to requirements for packaging, labeling and transportation (305).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs
  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Petroleum and coal tar distillates with flash points below 21°C are classified as flammable substances and are subject to packaging and

labeling regulations. Because of the variable composition of other petroleum and coal tar distillates (excluding those used as motor fuels), they are considered preparations and their labeling shall be done in accordance with the procedures outlined in the Directive Relating to the Classification Packaging and Labeling of Dangerous Preparations (solvent).

Directive on Disposal of Waste Oils (1986)
Establishments collecting and/or disposing of waste oils must carry out these operations so that there will be no avoidable risk of water, air or soil pollution.

EEC Directives - Proposed

Proposal for a Council Directive on the Dumping of Waste at Sea (1793)
EEC has proposed that the dumping of crude oil, petroleum hydrocarbons, lubricants and hydraulic fluids at sea be prohibited.

66.1 MAJOR USES

Fuel oils have various uses for which they are specifically formulated. Fuel oil number 1 is used almost exclusively for domestic heating. Fuel oil number 2 is used as a general purpose domestic or commercial fuel in atomizing type burners. Number 4 oil is used in commercial or industrial burner installations not equipped with preheating facilities. Numbers 5 and 6 are used in furnaces and boilers of utility power plants, ships, locomotives, metallurgical operations and industrial power plants (23).

Diesel fuel is available in different grades. Number 1-D is used for engines in service requiring frequent speed and load changes. Number 2-D is used for engines in industrial and heavy mobile service while number 4-D is used in low and medium speed engines (2342).

66.1.2 Composition

The discussion of fuel oil in this chapter largely focuses on diesel fuel. Limited information on residual fuel oils, which are generally defined as the product remaining after removal of the appreciable quantities of the more volatile components, is included but environmental fate data are not specifically addressed. Residual fuel oils are expected to be extremely complex in composition, with higher concentrations of the many high molecular weight asphaltic compounds and impurities present in the original crude oils. Available data suggest sulfur values ranging from 0.18 to 4.36% by weight; trace element data indicate that concentrations of many elements vary by one or more orders of magnitude, as shown in Table 66-1 (1843). The environmental transport and transformation of the high molecular weight organics is expected to be minimal and is not addressed in detail.

Diesel fuel is usually that fraction of petroleum that distills after kerosene in the 200°C to 400°C range. Several commercial grades of diesel fuels are obtained by blending various feedstocks to achieve established specifications. Due to differences in feed stocks, refining methods, and blending practices, the composition of diesel fuel samples is expected to be highly variable. Sulfur content has been reported to vary by several orders of magnitude (0-0.57% by weight); similar variations have been documented for a number of trace elements, as shown in Table 66-1 (1843).

Diesel fuel is predominantly a mixture of C_{10} through C_{19} hydrocarbons. Composition by chemical class has been reported to be approximately 64% aliphatic hydrocarbons (straight chain alkanes and cycloalkanes), 1-2% olefinic hydrocarbons and 35% aromatic hydrocarbons, including alkylbenzenes and 2-3 ring aromatics (1847). Other authors have reported a somewhat lower aliphatic content (1849). As discussed in Chapter 64 (JP-4), petroleum distillates may contain many non-hydrocarbon components in varying concentrations.

TABLE 66-1

TRACE ELEMENT CONTENT IN PETROLEUM-DERIVED FUEL OILS[a]

	Range of Eleven Residual Oils	Range of Six Domestic Diesel Fuels[b]
Arsenic	<0.01-2.0	0.012-0.13
Beryllium	<0.0023-0.22	
Cadmium	<0.01-0.83	0.089-0.89
Chromium	0.09-1.9	0.55-2.8
Iron		3.8-71.0
Lead		<0.49-2.0
Manganese	<0.0095-27	0.29-6.2
Mercury	0.007-0.17	
Molybdenum	<0.01-1.1	0.018-0.27
Nickel	6.0-51	<6.1-23.0
Selenium	0.02-4.2	
Vanadium	1.0-110	<0.06-0.16
Zinc		1.3-4.8

[a]Reference 1843

[b]ppm by weight

Fuel oils also contain a number of additives used as ignition improvers, combustion catalysts, antioxidants, flow improvers, metal deactivators, detergents and demulsifiers. Many compounds added to fuel oils are similar to those added to gasoline (Chapter 65). A list of some of the chemical classes and specific chemicals that may be added to diesel fuel is provided in Table 66-2.

TABLE 66-2

COMMON ADDITIVES IN DIESEL FUELS

Ignition Improvers (Cetane Improvers)

Alkyl nitrate and nitrites (C_3-C_8), primarily octyl nitrate
Nitro and nitroso compounds
Peroxides

Combustion Catalysts/Deposit Modifiers

Organometallics of barium, calcium, manganese, and iron
Mn, MnO
Mg, MgO, MgO_2
Al_2O_3

Antioxidants

N,N'-Dialkylphenylenediamines
2,6-Dialkyl and 2,4,6-trialkylphenols

Cold Flow Improvers

Ethylene vinyl acetate copolymers
Ethylene vinyl chloride copolymers
Polyolefins
Chlorinated hydrocarbons

Metal Deactivators

N,N'-Disalicylidene-alkyldiamines

Detergents/Dispersants

Long chain alcohols
Long chain amines
Long chain alkyl phenols
Long chain carboxylic acids
Sulfonates
Succinimides

Source: References 2326,2327,2335,2336

66.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

A discussion of the environmental behavior of fuel oil is limited by the lack of data defining its major components. The environmental behavior of hydrocarbons selected from the major classes will be addressed; however, trace elements and the many diverse additives will not be specifically addressed. Many of the hydrocarbons characteristic of diesel fuel have been addressed previously in the more extensive environmental fate section of the JP-4 chapter since these hydrocarbons are common to both petroleum fuels. The general discussions of aliphatic and aromatic hydrocarbons and their behavior in soil/ground-water systems will not be repeated here; the reader is referred to the relevant sections of Chapter 64.

66.2.1 Equilibrium Partitioning Model

In general, soil/ground-water transport pathways for low concentrations of pollutants in soil can be assessed by using an equilibrium partitioning model. For the purposes of assessing the environmental transport of diesel fuel, a group of specific hydrocarbons was selected from the dominant hydrocarbon classes, i.e., alkanes, cycloalkanes, and aromatics; there were no available data to confirm the presence of the selected compounds in a typical diesel fuel sample. Table 66-3 identifies the selected hydrocarbons and presents the predicted partitioning of low soil concentrations of those hydrocarbons among soil particles, soil water, and soil air. The portions associated with the water and air phases of the soil are expected to have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil indicate that sorption is expected to be an important process for all the dominant hydrocarbon categories. Partitioning to the soil-vapor phase is much less important than for other petroleum distillates since many of the lower molecular weight aliphatic hydrocarbons (C_4-C_8) characterized by high vapor pressure and low water solubility are not expected to be major components of diesel fuel. The aromatics have slightly higher water solubilities and transport with infiltrating water may be more important for these compounds; volatilization, on the other hand, is not expected to be important. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a significant percent of the aromatic hydrocarbons is predicted to be present in the soil-water phase and available for transport with flowing ground water. Partitioning to the air and water phases is expected to be even less important for the organic components of residual fuel oils compared to components of diesel oil; sorption to soil particles is expected to be significant.

In interpreting these results, it must be remembered that this model is valid only for low soil concentrations (below aqueous solubility) of the components. Large releases of diesel fuel (spills, leaking underground storage tanks) may exceed the sorptive capacity of the soil, thereby filling the pore spaces of the soil with the fuel.

TABLE 66-3

EQUILIBRIUM PARTIONING OF POTENTIAL
DIESEL FUEL HYDROCARBONS IN MODEL ENVIRONMENTS[a]

COMPOUND	Log K_{ow}	K_{oc}[b]	H[c]	UNSATURATED TOPSOIL (%)			SATURATED DEEP SOIL[d] (%)	
				Soil	Water	Air	Soil	Water
Octane	5.18 (e)	73,000	2.96	97.4	0.01	2.6	99.7	0.3
Dodecane	7.06 (f)	5.5×10^6	7.4	99.9	0.0001	0.09	99.9	0.004
Trimethylpentane	4.87 (f)	36,000	1.9-3.3	94.7	0.01	5.3	99.3	0.7
Trimethylcyclohexane	5.02 (h)	50,500	1.6	98.0	0.01	2.0	99.5	0.5
Trimethylbenzenes	3.65 (h)	2,150	5×10^{-3}	99.6	0.2	0.2	90.0	10.0
Naphthalene	3.30 (e)	962	4.82×10^{-4}	99.4	0.5	0.03	80.2	19.8
Methylnaphthalenes	3.87 (e)	3,570	4.4×10^{-4}	99.8	0.1	0.01	93.7	6.3
Anthracene	4.45 (g)	13,500	5.9×10^{-5}	99.9	0.04	0.0003	98.3	1.7

[a]Calculations based on Mackay's equilibrium partioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

[b]Reference 652.

[c]Taken from Reference 74 unless otherwise specified. Units equal $atm\text{-}m^3/mol$.

[d]Used sorption coefficient $K_p = 0.001 \times K_{oc}$.

[e]Reference 29.

[f]Arthur D. Little, Inc., estimate according to equations provided in Reference 31.

[g]Reference 10.

[h]Reference 31.

In this situation, the hydrocarbon mixture would move as a bulk fluid and the equilibrium partitioning model would not be applicable.

66.2.2 Transport and Transformation Processes

Transport and transformation of individual fuel oil constituents will depend on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly in the percolating ground waters, be sorbed less strongly on the soils thus being transported more rapidly, and may be more or less susceptible to degradation by chemical or biological action. Thus, as was shown in Figure 65-1, the relative concentrations of the constituents of the fuel will vary with time and distance from the site of contamination. This effect is called "weathering". (This term is also used to describe the changes to oil following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation are all involved.)

Transport processes have been shown to be more significant than transformation processes in determining the initial fate of lower molecular weight petroleum hydrocarbons released to soil/ground-water systems. However, due to the lower water solubilities and lower vapor pressures of the higher molecular weight hydrocarbons, environmental transformation processes may be increasingly significant for hydrocarbons in the C_{10}-C_{19} range characteristic of diesel fuel and in the > C_{19} range expected in residual fuel oils. Spain et al. (1846) demonstrated that compounds having up to nine carbons are weathered almost exclusively by evaporation; larger compounds were weathered by evaporation and biodegradation.

Under conditions of limited volatilization (low temperatures, subsurface release or concentrated spill) other transport processes including downward migration into the soil, sorption to soils, and transport to ground water may be important. Several authors (1811, 2243,2252,2329) have reported that oil substances released in significant quantities to soils result in a separate organic phase which moves downward through the unsaturated zone to the less permeable layer, the soil/ground-water boundary, where they tend to accumulate and spread horizontally.

The organic layer floating on the ground water is carried in the general direction of ground water flow. At the oil-water interface, some hydrocarbons are leached according to their aqueous solubility. As discussed in Chapter 64, the pollution caused by the hydrocarbon phase is much less extensive (10s-100s of meters) than pollution caused by hydrocarbons dissolved in ground water (100s-1000s of meters) (1811). Furthermore, the pattern of migration of the hydrocarbon phase may be very different from that of the ground water. Due to fluctuations in ground-water elevation over time, the organic layer on top of the aquifer may be transported into several zones where the components occur in the gaseous phase (able to diffuse in all directions, including upward), liquid phase (adsorbed onto rock particles or sealed under water) or dissolved/ emulsified in water (1811,2329).

Migration through soils may be retarded by sorption. Sorption is expected to be significant for high molecular weight aliphatics, particularly > C_{20}. Migration is expected to be fastest through previously contaminated soils where the sorptive sites may be unavailable; on the other hand, soil-water content increases sorption and slows migration of hydrocarbons. In fissured rock, the migration of hydrocarbons is much less uniform than in porous soils. Preferential spreading through crevices, sometimes changing the direction of flow, may occur. Determination of the potential ground-water contamination in fissured rock is thus very difficult (1811).

The water-soluble portion of No. 2 fuel oil (a higher temperature distilling fraction than diesel oil) was shown to be almost entirely aromatic (99%) even though the product itself was 48% aliphatic; the aliphatic fuel oil hydrocarbons have very low water solubility compared with the aromatics (1849,2238). The largest percentage (40%) of the water-soluble fraction of fuel oil was represented by C_{11}-aromatics (1849). In deep, saturated soils with no soil air, the aromatics represent the greatest threat of contamination to ground-water supplies. Solubility in aqueous solution of polar, non-hydrocarbon components of some higher boiling petroleum fractions such as diesel oil and other fuel oils has also been reported (2238).

In summary, the physical distribution of fuel oil contamination affects its impact on, and removal from, the soil environment. Lateral spreading along the surface increases the initial contaminated area while facilitating evaporative removal or sorption of different hydrocarbons. Subsurface release or vertical penetration mediated by gravitation and capillary forces decreases evaporation, reduces the importance of some transformation pathways (see below), and may lead to ground-water contamination.

Photooxidation has been reported to play a significant role in the chemical degradation of petroleum hydrocarbons in the sunlit environment (1845,1848,2252,2259,2337). Alkanes, benzenes, and mono-substituted benzenes have been shown to be relatively resistant to photolysis in aqueous systems; xylenes photolyzed slowly while trisubstituted benzenes and naphthalenes photolyzed at rates competitive with volatilization (1845). Lee *et al.* report that anthracene and other polycyclic aromatic hydrocarbons (PAH) in the carbon range of diesel fuel are subject to photochemical oxidation; benzo(a)pyrene is the most susceptible of the PAH compounds, suggesting that the residual fuel oils may be even more affected by photodegradation than diesel oil. Penetration of oil below the soil surface limits exposure to solar radiation while extensive lateral spreading of oil over impermeable or rocky surfaces may promote substantial photooxidative degradation. The oxygenated products of photooxidation are generally more water-soluble than the parent hydrocarbons and are thus more likely to be leached from soil; enhanced toxicity of the oxygenated hydrocarbons has also been observed (2248, 2252).

Natural ecosystems have considerable exposure to petroleum hydrocarbons from natural emissions, accidental contamination through oil spills and storage tank leaks, and deliberate application to land in waste disposal activities such as land-farming; therefore, their biodegradation is of environmental importance. Numerous authors have observed the biodegradation of petroleum hydrocarbons, and several extensive reviews and reports are available (1846,2252,2255,2249, 2253). An extensive and diverse group of petroleum hydrocarbon-degrading bacteria and fungi are widely distributed in the environment. Although the microbiota of most non-contaminated soils include many naturally occurring hydrocarbon-degrading populations, the addition of petroleum selectively enriches that sector able to adapt and utilize the new substrate. Other environmental factors shown to have a major effect on biodegradability are availability of oxygen and moderate temperatures. The reader is referred to Chapter 64 for a more detailed summary of the biodegradation of petroleum hydrocarbons.

The qualitative hydrocarbon content of petroleum mixtures largely determines their degradability. In general, microorganisms exhibit decreasing ability to degrade aliphatic hydrocarbons with increasing chain length; aromatics are generally more rapidly biodegraded than alkanes. The composition of diesel oil suggests that some of the aromatic species will be biodegradable; biodegradation of the high molecular weight aromatics expected to be present in residual oils will be slower (2339).

In summary, biodegradation of the petroleum hydrocarbons comprising diesel and fuel oils may occur under conditions favorable for microbial activity and when fuel components are freely available to the microorganisms. Degradation may be limited and/or slow in environments with few degrading organisms, low pH, low temperatures, and high salinity (e.g., arctic environments). It should be mentioned that Walker et al. (2257) state that even under optimum conditions, total and complete biodegradation is not expected to occur except possibly over an extremely long time period.

66.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that pure fuel oils have low vapor pressure but that their components vary in their volatility from water. The components are strongly or very strongly sorbed to soil. The polycyclic aromatic hydrocarbons in fuel oils have a moderate or high potential for bioaccumulation, while the longer-chain aliphatic compounds have low potential for bioaccumulation. These fate characteristics suggest that the various components may have somewhat different potential exposure pathways.

Volatilization of fuel oils from a disposal site or spill would not be expected to result in significant inhalation exposures to workers or residents in the area. Gravity would tend to carry bulk quantities of the oil down towards the water table leaving only a

relatively small fraction on the soil surface to volatilize. Volatilization of the remaining oil would occur very slowly because of its low vapor pressure, especially for the heavier weight fuel oils, and because of strong sorption to soil.

Ground-water contamination may result from large spills that reach the water table. There, the more soluble components will dissolve in the ground-water or form emulsions with it. The soluble fraction is mainly aromatic and lower molecular weight aliphatic compounds. In one study using No. 2 fuel oil, 40% of the water soluble fraction was made up of aromatic compounds composed of 11 carbon atoms and 25% each of compounds containing 10 and 12 carbon atoms (2318). The hydrocarbons dissolved in the ground water may move hundreds to thousands of meters. By comparison, the undissolved fraction, which floats on the surface of the water table as a separate phase, would be expected to move only tens of meters, unless cracks or fissures were present.

The movement of fuel oil components in ground water may contaminate drinking water supplies, resulting in ingestion exposures. Ground-water discharges to surface water or the movement of contaminated soil particles to surface water drinking water supplies may also result in ingestion exposures and in dermal exposures from the recreational use of these waters. The potential also exists for the uptake of polynuclear aromatic compounds in fuel oil (e.g., naphthalene, methylnaphthalene and higher weight PAH's) by fish and domestic animals, which may also result in human exposures. Exposures to high concentrations of fuel oil components in drinking water and food are expected to be rare because tainting becomes apparent at relatively low concentrations (982).

Volatilization of fuel oil hydrocarbons in soil is another potential source of human exposure. Despite their relatively low vapor pressure, the more volatile components of fuel oil in soil evaporate, saturating the air in the soil pores, and diffusing in all directions including upward to the surface. The vapors may diffuse into basements of homes or other structures in the area, resulting in inhalation exposures to the building's occupants. Exposures may be more intensive when the soil is contaminated from leaking underground storage tanks and pipes, rather than from surface spills, because the more volatile components do not have an opportunity to evaporate before penetrating the soil. Even then, this exposure pathway is expected to be much less important for fuel oils than for more volatile petroleum products like gasoline.

66.2.4 Other Sources of Human Exposure

Data on ambient concentrations of fuel oil in air and water as well as in food and drinking water are not readily available in the literature. Exposure information on specific components may be found in other chapters of this Guide. Several population groups susceptible to exposure to fuel oil may be identified. Personnel involved in fuel

handling operations may experience direct dermal contact if protective gloves and clothing are not worn. They may also receive small inhalation exposures from the more volatile components.

66.3 HUMAN HEALTH CONSIDERATIONS

66.3.1 Animal Studies

66.3.1.1 Carcinogenicity

Generally, number 1 and number 2 fuel oils are not carcinogenic even though they contain aromatic hydrocarbons (2219). In contrast, industrial fuels such as number 6 oil are residual oils which often contain highly condensed aromatic products from severe cracking processes. They may be carcinogenic to animals if they contain PAH components which boil above 370°C (2219).

Certain currently available fuel oils may be carcinogenic because they are derived from the blending of fractions boiling below 370°C with those boiling at higher temperatures. Some of these high-boiling fuels which are derived from catalytic cracking processes may contain carcinogens (2219).

Studies have demonstrated a direct relationship between tumor potency and the concentration of high-boiling fractions which are added to form blends. It was determined that when not more than 10 volume % of 700°F$^+$ catalytic gas oil or clarified oil is present, the tumor potency values of the blends are less than 20 and therefore have borderline significance. The tumor potency value is a representation of the tumor formation rate in response to application to mouse skin. For a value of 20, 500 days would be required for a 50% tumor response (1818) (see Table 66-4). Examination of boiling ranges of blended petroleum products may not provide an accurate assessment of their carcinogenic potential. In the opinion of Bingham et al. (2219), these materials are probably carcinogenic and their potency may be underestimated or overestimated if the diluent contains cocarcinogens or inhibitors.

Frazier and Mahlum (1819) tested the initiation activity of a fuel oil blend (FOB) which contained part of a heavy molecular weight distillate boiling at 288-454°C and 2.9 parts of a distillate boiling between 176 and 288°C. The FOB (25 mg) was applied to the clipped backs of CD-1 mice in a 50 μL volume. Two weeks after initiation, the animals received doses of 5 μg phorbol myristate acetate in 50 μL acetone twice weekly for 24 weeks. Negative controls were treated with acetone. Positive controls were initiated with 50 μg benzo[a]pyrene (BAP) or dimethylbenzanthracene (DMBA). The FOB showed significant initiating activity. Approximately 60 tumors were seen after ~170 days. Greater than 200 tumors were observed in the BAP positive controls. Hydrotreated FOB was also tested in the same manner. Hydrotreatment has been suggested as a possible method for reducing biological activity of coal-derived materials. In this group about 17%

TABLE 66-4

POTENCIES OF TWO BLENDED FUEL OILS FOR THE SKIN OF C3H MICE

Base Blending Stock	Cracked Residuum Added[a] (%)	Content of BaP (%)	Dosage[b] (mg/ mouse)	Number of Mice	Final Effective Number[c]	Number of Mice Developing Tumors Malignant	Number of Mice Developing Tumors Benign	Average Time of Appearance of Papillomas (Weeks)
A[d]	0	0.01	20	19	17	1	1	--
			50	20	17	3	7	58.8 ± 1.7[e]
B[d]	0	0.00	20	40	23	0	1	--
A	5	0.05	20	30	27	15	8	41.5 ± 3.6
			50	30	27	13	8	28.3 ± 3.3
B	5	0.04	20	40	31	9	11	49.1 ± 5.5
			50	28	27	9	9	36.9 ± 3.3
A	10	0.08	20	30	26	19	7	40.4 ± 3.2
			50	30	25	22	3	32.2 ± 2.5
B	10	0.075	20	40	35	22	13	40.5 ± 1.9
			50	30	30	9	18	26.7 ± 1.6
A	20	0.16	20	25	23	12	9	25.2 ± 2.8
B	20	0.15	20	29	28	11	16	23.4 ± 1.7

[a] Residuum (> 700°F) from thermal cracking of FCC clarified oil.
[b] Applied twice weekly
[c] Number alive at time of appearance of median tumor plus number of tumor-bearing mice which died earlier.
[d] Base stock A is cracked Bunker fuel; Base stock B is West Texas uncracked residuum.
[e] Limits of confidence (P = 0.05).

Reference 1820

of the animals developed a total of 12 tumors. Each tumor-bearing mouse had an average of 2.4 tumors. The hydrotreated FOB was also tested for promoting activity. In these studies, mice were initiated with 50 μg DMBA. After 2 weeks, they were promoted twice weekly for 24 weeks with 50 μL of a 1:3 solution of hydrotreated FOB in acetone. The control group was treated with acetone for 2 weeks and similarly promoted with the hydrotreated FOB. The hydrotreated FOB possessed measurable tumor promoting activity. When DMBA was used as the initiator, 41% of the mice had tumors after 6 months. Each mouse had an average of 2.5 tumors. No tumors were reported in mice treated with hydrotreated FOB on noninitiated (acetone-treated) skin.

66.3.1.2 Mutagenicity

API has conducted a battery of 3 tests to evaluate the mutagenicity of diesel fuel and number 2 fuel oil (1914).

Number 2 fuel oil (50% catalytically cracked stock) gave positive results in each test. In the Ames assay, it was judged to be equivocal rather than negative because the relatively high mutant frequencies in S. typhimurium strain TA98 were observed at 4 concentrations. In a mouse lymphoma assay, it was mutagenic under activation and non-activation conditions. At a test concentration of 1200 μg/mL, the mutation frequency was 17 times the solvent control without metabolic activation. In a rat bone marrow cytogenetic study, Sprague-Dawley rats were administered number 2 fuel oil dissolved in corn oil by gavage at dosages ranging from 0.125 to 1.25 g/kg/day for 5 days. The percentage of aberrant cells ranged from 7.5 to 12.5%. A high percentage of cells with chromatid breaks was seen at all treatment levels. In both cases, the increases were statistically significant only at the low and high dose levels.

Diesel fuel gave negative results in both the Ames and mouse lymphoma assay. Positive results were obtained in the rat bone marrow cytogenetic assay. The diesel fuel was administered undiluted by intraperitoneal injection. Dose was 0.6, 2.0 or 6.0 mL/kg/day for 1 or 5 days. Single injections at the mid- or high-dose, as well as the high-dose in the 5 day protocol caused statistically significant increases in chromosome abnormalities (1914).

66.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

One teratology study of fuel oil was found. The study was sponsored by API. The material used was labeled "fuel oil". No specifications were provided. From days 6 through 15 of gestation, pregnant CRL:COBS CD(SD)BR rats were exposed to airborne concentrations of 0, 86.9 or 408.4 ppm for 6 hours daily. There was no evidence of teratogenicity, embryotoxicity or inhibition of fetal growth and development (1915).

66.3.1.4 Other Toxicologic Effects

66.3.1.4.1 Short-term Toxicity

The following fuel oils were evaluated for acute toxicity in 6 tests:

Diesel Fuel (marketplace sample)
Number 2 fuel oil
low-catalytic cracked (10%)
medium-catalytic cracked (30%)
high-catalytic cracked (50%)

Number 6 fuel oil
API gravity 11.7/2.7% Sulfur content
API gravity 17.1/0.8% Sulfur content
API gravity 23.1/0.2% Sulfur content
API gravity 5.2/1.2% Sulfur content

The 6 tests which were conducted were:

- Primary eye and dermal irritation in rabbits
- Acute dermal and subacute dermal toxicity in rabbits
- Dermal sensitization in guinea pigs
- Acute oral toxicity in rats

Results of these tests are discussed below.

The acute oral toxicity was evaluated in Sprague-Dawley rats. The number 2 oils caused 70 to 100% mortality with doses of 16.5 to 21 g/kg. LD_{50} values ranged from 12.0 to 17.5 g/kg. Toxic signs included alopecia, dermal irritation and open sores around the genital area. The number 6 fuel oil with an API specific gravity of 5.2 and 1.2% sulfur content was the most toxic material tested. The LD_{50} was 5.1 g/kg. A dose of 25 g/kg caused 100% mortality. None of the other number 6 fuel oils caused mortality at 22-24 g/kg. A significant degree of gastrointestinal distress was observed at doses greater than 15-20 g/kg until the material cleared the gastrointestinal tract. This was thought to be due to volume overload. Mortality generally occurred 2-3 days after dosing. Necropsy revealed evidence of hemorrhagic gastroenteritis and pneumonia with abscess formation (1929).

A marketplace sample of diesel fuel had an LD_{50} of 7.5 g/kg and caused 90% mortality at a dose of 16.6 g/kg. Toxic signs were the same as those seen with the number 2 oils (1924).

Male CD-1 mice subjected to nose only exposure of 0.065, 0.135 or 0.204 mg/L uncombusted diesel fuel vapor for 8 hours per day on 5 consecutive days developed vasodilation, ataxia, poor grooming habits, and in some cases, tremors. All signs varied with the dose and

duration of exposure. Dose-related effects in neurological testing indicate that the uncombusted diesel vapors may also act as a neurodepressant (2334).

In acute dermal studies conducted in rabbits, number 6 heavy fuel oil (API gravity 5.2/1.2% sulfur) induced significant signs of toxicity at 5 g/kg. It caused severe dermal irritation, weight loss, anorexia, ataxia and lethargy. Mortality was 37.5%. Necropsy revealed acute toxic hepatitis, gastrointestinal irritation and congested lungs. Other grades of number 6 and number 2 oils as well as diesel oil produced mild to moderate dermal irritation but no systemic signs of toxicity (1924).

In the subacute dermal study, doses ranging from 1 to 10 mL/kg were applied to rabbits clipped free of hair. The area remained bandaged for 24 hours at which time the patches were removed and a new dose applied. This continued for 5 consecutive days followed by a 2 day rest period and a repeat application for 5 consecutive days. In this test, the number 6 fuel oil (API gravity 5.1/1.2% sulfur) produced the greatest degree of toxicity at the lowest dose (75% mortality at 2.5 mL/kg). Clinical signs included severe weight loss, anorexia and signs of dermal irritation. Gross necropsy revealed hemorrhagic gastroenteritis, and congested, mottled livers with multifocal necrosis and centrilobular vacuolar degeneration. In all cases, the number 6 oils caused inflammation, dermal congestion and edema, dermal necrosis, acanthosis and parakeratosis. Liver necrosis and degeneration were also seen but the severity was not as great as that with 5.1/1.2% sulfur. All 3 number 2 oils caused weight loss, anorexia and various degrees of dermal irritation. At a dose of 10 mL/kg, mortality ranged from 75 to 100%. Gross necropsy lesions at all dosage levels included renal and hepatic congestion, At the 10 mL/kg level, multifocal hepatic necrosis was observed (1924).

In primary dermal irritation tests, the number 2 oils were moderately irritating while the number 6 oils were minimally to slightly irritating. Diesel fuel was extremely irritating. Signs included severe erythema and edema with blistering and open sores. The test was conducted by applying 0.5 mL of undiluted material to abraded rabbit skin. The test was then covered for 24 hours at which point the bandage was removed and the animals scored according to the Draize technique (1924).

The number 6 oils were minimally to moderately irritating when 0.1 mL was applied to rabbit eyes. These materials produced conjunctival redness, swelling and discharge. Few corneal opacities were produced but eyes returned to normal within 72 hours. Diesel fuel was non-irritating and number 2 oils ranged from practically non-irritating to mildly irritating (1924).

66.3.1.4.2 Chronic Toxicity

No studies were found regarding the chronic toxicity of fuel oils in animals.

66.3.2 Human and Epidemiologic Studies

66.3.2.1 Short-term Toxicologic Effects

The chief systemic reaction to petroleum hydrocarbons, such as fuel oils, is central nervous system depression (17). Toxicological effects are expected to resemble those of kerosene; i.e., a low oral, moderate dermal and high aspiration hazard (12). Provided that aspiration does not occur, the mean oral lethal dose of kerosene for an adult is estimated to be 4 to 6 ounces. However, twice this amount has been tolerated and less than ½ ounce has caused death (17). This estimate may be low since oral LD_{50} values in rats, rabbits and guinea pigs exceed 20 mL/kg. In fatal poisonings, death may occur within 2 to 24 hours after ingestion. The difference between cases of uncomplicated ingestion and the lethal dose where aspiration occurs may be as great as a pint and a teaspoonful. The characteristic lesion resulting from aspiration is an acute and often fatal bronchopneumonia. Kerosene and related hydrocarbons are also irritating to the skin and mucous membranes. Percutaneous absorption may be significant (17).

Dermal exposure to diesel oil has caused nephrotoxicity. A man who cleaned his hands and arms with diesel oil over several weeks experienced symptoms of epigastric and loin pain, thirst, nocturia, nausea, anorexia, scrotal swelling, severe exhaustion and pitting ankle edema. Renal biopsy revealed acute tubular necrosis with patchy degeneration and necrosis of the proximal and distal tubular epithelium (1814). Another case was described by Barrientos et al. (1815) who reported acute oliguric failure in a patient who had washed his hair with diesel oil. A renal biopsy performed the next day showed tubular dilation and a proliferation of cells in the glomeruli. Similar nephrotoxic effects were reported as a result of inhalation of diesel oil vapors in a truck cab over a 10 day period (1816).

Liquid petroleum hydrocarbons cause little or no injury on direct eye contact. Kerosene and petroleum oil on rabbit and human corneas are essentially innocuous (19).

66.3.2.2 Chronic Toxicologic Effects

No studies were found which evaluated the effects of long-term exposure to fuel oils. However, numerous epidemiology studies evaluating the effects of petroleum exposure have been conducted. While most have shown overall standardized mortality ratios to be lower than those of the general population, elevated numbers of deaths have been observed for cancers at several sites. However, these elevations are not found consistently in all of the studies. Cancers have been

observed in the lung, nasal cavity and sinuses, digestive system, brain, skin, pancreas and kidney. Leukemias and lymphomas have also been reported (1817).

66.3.3 Toxicology of Fuel Oil Components

A brief overview of the toxicology of the major hydrocarbon components of fuel oils are summarized below (see Table 66-5).

n-Hexane

Hexane may be the most highly toxic member of the alkanes. When ingested, it causes nausea, vertigo, bronchial and general intestinal irritation and CNS effects. It also presents an acute aspiration hazard. Acute exposure occurs primarily through inhalation. Non-specific symptoms such as vertigo, headache, nausea and vomiting are the first to be manifested. At high concentrations, a narcosis-like state appears as a result of CNS depression. Pre-narcotic symptoms occur at vapor concentrations ranging from 1500-2500 ppm. n-Hexane irritates the eyes and mucous membranes. These effects can be seen after an exposure of 880 ppm for 15 minutes. Skin contact primarily causes fat removal and cutaneous irritation.

Chronic exposure to n-hexane vapors causes peripheral neuropathy. The first clinical sign of neural damage is a feeling of numbness in the toes and fingers. Progression leads to further symmetrical sensory impairment in the distal portions of the extremities and to loss of muscular stretching reflexes. Ultimately, symmetrical muscular weakness develops, chiefly in the distal portion of the extremities. Paralysis develops with varying degrees of impaired grasping and walking. This may include muscular atrophy (sensorimotor polyneuropathy). The development of electrophysiological changes parallels the severity of the clinical picture. In the most severe cases, nerve conductivity is neutralized. In some cases, cranial nerve involvement is also observed. After exposure ceases, recovery begins within 6 to 10 months in mild to moderate cases, but may take up to 3 years in serious cases. The threshold level at which neuropathy occurs has not been firmly established but symptoms have been observed in people exposed to concentrations ranging from 10 to 200 ppm for 9-12 months.

In animals, signs of narcosis are seen after mice are exposed to vapor levels of 16,000 ppm for 5 minutes. Death generally occurred at concentrations between 43,800 and 52,000 ppm after 9-119 minutes. The oral LD_{50} is cited as 24 mL/kg for 14-day-old rats and 49 mL/kg for young adult rats.

Long-term inhalation experiments in rats suggest that the first signs of neurotoxicity appear after they are exposed to levels of 200 ppm for 24 weeks. This higher threshold to induce neurotoxicity in animals may be due to differences in metabolism. Specifically, 2-hexanol is the chief metabolite in animals, while 2,5-hexanedione

TABLE 66-5

ACUTE TOXICITY OF COMPONENTS OF FUEL OILS

Component	Oral LD_{50}	Dermal LD_{50}	LC_{50}
n-hexane	24-49 mL/kg [rat] (1935) 28,710 mg/kg [rat] (1937)	no data	33,000 ppm •4 hr [rat] (1935)
octane	<— no data —>		
dodecane	<— no data —>		
isopentane	no data	no data	1000 mg/L [mouse] (12)
isooctane	<— no data —>		
methylcyclopentane	<— no data —>		
methycyclohexane	2250 mg/kg [rat] (47)	no data	no data
cyclohexane	29,820 mg/kg [rat] (1935)	no data	no data
benzene	3800 mg/kg [rat] (59) 4700 mg/kg [mouse] (47)	no data	10,000 ppm •7 hr [rat] (47)
toluene	5000 mg/kg [rat] (47)	12,124 mg/kg [rabbit] (47)	5320 ppm •8 hr [mouse] (47)
xylenes	4300 mg/kg [rat] (47)	no data	5000 ppm •4 hr [rat] (47)
ethyl benzene	3500 mg/kg [rat] (47)	5000 mg/kg [rabbit] (59)	no data
trimethylbenzenes	no data	no data	18 mg/m^3 •4 hr [rat] (47)
1-methylnaphthalene	1840 mg/kg [rat] (47)	no data	no data
2-methylnaphthalene	1630 mg/kg [rat] (47)	no data	no data

which is neurotoxic, predominates in man. Chronic topical application of a solvent containing 35.2% n-hexane caused axonal swelling and myelin degeneration in chicks. No clinical signs were seen. Dosage was 1 g/kg/day for 64 days. In rabbits, topical application of 0.5 mL/day for up to 10 days caused redness, irritation and scab formation. N-hexane is neither carcinogenic or teratogenic. One in vivo study in rats that inhaled 150 ppm for 5 days found an increased number of chromosome aberrations in the bone marrow cells. No studies on mutagenicity, reproductive toxicity or carcinogenicity in man were found (12,1930,1935).

Octane

By the oral route, octane may be more toxic than its lower homologues. If it is aspirated into the lungs, it may cause rapid death due to cardiac arrest, respiratory paralysis and asphyxia. The narcotic potency of octane is approximately that of heptane but it does not exhibit the CNS effects seen with hexane or heptane.

In humans, the only reported effects are blistering and burning due to prolonged skin contact.

In animals, octane is a mucous membrane irritant. At high concentrations, it causes narcosis. It is expected that severe exposure in humans will produce the same effects. Mice exposed to vapor levels of 32,000 ppm suffered respiratory arrest after 4 minutes of exposure. Exposure to 12,840 ppm for 185 minutes caused a decreased respiratory rate, followed by death within 24 hours. No narcosis was seen after 48 minutes of exposure to 5350 ppm (12,46,1938).

Dodecane

Dodecane is not highly toxic. The lowest toxic dose for mice is 11 g/kg when administered percutaneously for 22 weeks. Dodecane is a potentiator of skin tumorigenesis by benzo(a)pyrene. It decreased the effective threshold dose by a factor of 10. Dodecane and phenyldodecane applied topically to the progeny of rats treated with benzo(a)pyrene, chrysene or benzo(b)triphenylene on the seventeenth day of gestation produced tumors in offspring. No additional information is available (12,1937).

Isopentane

Isopentane is a CNS depressant. Effects may include exhilaration, dizziness, headache, loss of appetite, nausea, confusion, inability to do fine work, a persistent taste of gasoline and in extreme cases, loss of consciousness. Inhalation of up to 500 ppm appears to have no effect on humans. "Very high" vapor concentrations are irritating to the skin and eyes. Repeated or prolonged skin contact will dry and defat skin resulting in irritation and dermatitis. The LC_{50} in the mouse is estimated to be 1000 mg/L (12).

Iso-octane (2,2,4-trimethylpentane)

The iso-octanes are moderately toxic by the oral route. If aspirated into the lungs of rats, they will cause pulmonary lesions. When injected intramuscularly into rabbits, iso-octane produced hemorrhage, edema, interstitial pneumonitis, abscess formation, thrombosis and fibrosis. Inhalation of 16,000 ppm caused respiratory arrest in mice and 5 minutes exposure to 1000 ppm was highly irritating (1937).

Methylcyclopentane

Methylcyclopentane resembles cyclopentane in its toxicity. Cyclopentane is a CNS depressant. Humans can tolerate 10-15 ppm. In mice, 38 ppm causes loss of reflexes, narcosis and death demonstrating that no safety margin exists. Methylcyclopentane also exhibits no safety margin between the onset of narcosis and death. When applied to guinea pig skin, cyclopentane produced dryness and slight erythema. Methylcyclopentane would be expected to have the same effect (12).

Methylcyclohexane

No systemic poisonings by methylcyclohexane have been reported in man. At high vapor concentrations it causes narcosis in animals and it is expected that it would produce the same effect in humans. The no-effect level is about 300 ppm in primates and 1200 ppm in rabbits. Rabbits did not survive 70 minutes of exposure to 15,227 ppm. Death was preceded by conjunctival congestion, dyspnea, severe convulsions and rapid narcosis. There were no signs of intoxication in rabbits exposed to 2880 ppm for a total of 90 hours, but slight cellular injury was observed in the liver and kidneys. In primates, lethal concentrations caused mucous secretion, lacrimation, salivation, labored breathing and diarrhea.

In chronic inhalation studies, exposure to 2000 ppm, 6 hours per day, 5 days per week for 2 years produced no tumors in rats, mice, hamsters or dogs. The only significant toxic effect found was renal changes in male rats. These included renal tubular dilation, papillary hyperplasia and medullary mineralization.

Dermal application of the liquid produced local irritation, thickening and ulceration (12,46,54,17,1936).

Cyclohexane

Cyclohexane is a CNS depressant of low toxicity. Symptoms of acute exposure are excitement, loss of equilibrium, stupor and coma. Rarely, death results due to respiratory failure. The anesthesia which is induced is weak and of brief duration but more potent than that caused by hexane. The oral LDLo in rabbits ranges from 5.5 to 6.0 g/kg. Within 1.5 hours the animals exhibited severe diarrhea, widespread vascular damage and collapse. Degenerative lesions were

seen in the heart, lung, liver, kidney and brain. A one-hour vapor exposure to 26,752 ppm caused rapid narcosis and tremor and was lethal to all exposed rabbits. In mice, concentrations causing narcosis vary from 14,600 to 122,000 ppm.

Cyclohexane is nominally absorbed through the skin although massive applications (> 180.2 g/kg) to rabbit skin resulted in microscopic changes in the liver and kidneys and caused the death of all animals.

The danger of chronic poisoning is relatively slight because this compound is almost completely eliminated from the body. No toxic changes were seen in rabbits exposed to vapor levels of 434 ppm, 6 hours daily for 50 exposures, but some microscopic changes were seen in the liver and kidneys when the exposure was to 786 ppm for the same period.

In man, no systemic poisonings by cyclohexane have been reported. A vapor level of 300 ppm is somewhat irritating to the eyes and mucous membranes. It has been reported that cyclohexane may potentiate the toxic effects of TOCP but no additional details of this interaction are available (12,17,46,54,1937).

Benzene

The primary effects of benzene inhalation and ingestion are on the central nervous system (54). Benzene is carcinogenic in both animals and man. Several reports have established a relationship between benzene exposure and leukemia. For more information, refer to Chapter 18 of the Installation Restoration Program Toxicology Guide, Volume 1.

Toluene

Toluene is a CNS depressant with a low toxicity. For more information, refer to Chapter 19 of the Installation Restoration Program Toxicology Guide, Volume 1.

Xylenes

Acute exposure to high concentrations of xylene vapors may cause CNS depression. Both the liquid and the vapor are irritating to the eyes, mucous membranes and skin (46). The National Toxicology Program recently reported that there was no evidence of carcinogenicity of mixed xylenes in either mice or rats given daily doses ranging from 250 to 1000 mg/kg by gavage for 2 years (1939).

For more information, refer to Chapter 21 of the Installation Restoration Program Toxicology Guide, Volume 1.

Ethyl Benzene

Ethyl benzene is primarily an irritant to the skin, eyes and upper respiratory tract. Systemic absorption causes CNS depression (46).

For more information, refer to Chapter 20 of the Installation Restoration Program Toxicology Guide, Volume 1.

Trimethylbenzenes

The trimethylbenzenes occur in 3 isomeric forms. The 1,3,5-isomer (mesitylene) and the 1,2,4-isomer (pseudocumene) are toxicologically similar. High vapor concentrations (5000-9000 ppm) cause CNS depression in animals. Loss of reflexes was seen in mice exposed to 8130-9140 ppm of the 1,2,4-isomer or 8130 ppm of the 1,3,5-isomer. Rats exposed to 1700 ppm of an isomeric mixture for 10-21 days had no adverse effects or fatalities.

The fatal intraperitoneal dose of the 1,2,4-isomer for the guinea pig is 1.788 g/kg, while the fatal dose of the 1,3,5-isomer by the same route is 1.5-2 g/kg for the rat. For the 1,2,3-isomer, an oral LDLo of 5000 mg/kg has been reported in the rat. Trimethylbenzene liquid is a primary skin irritant. Deposition into the lungs causes pneumonitis at the site of contact.

The only report of human exposure described symptoms of nervousness, tension, anxiety, asthmatic bronchitis, hypochromic anemia and changes in the coagulability of the blood. Vapor concentrations ranged from 10-60 ppm. Exposure was to a mixture containing 30% of the 1,3,5-isomer and 50% of the 1,2,4-isomer (2,12).

Methylnaphthalene

The only adverse effects of methylnaphthalene reported in man are skin irritation and photosensitization (17). Oral LD_{50} values of 1840 mg/kg and 1630 mg/kg have been reported for 1-methylnaphthalene and 2-methylnaphthalene, respectively, in the rat (47).

Naphthalene

Ingestion or prolonged inhalation of naphthalene produces nausea, vomiting and disorientation. It is irritating to the skin and eyes and prolonged vapor exposure has led to cataract formation in humans (17). Hemolytic anemia is the most severe effect associated with naphthalene exposure, but this effect is seen predominantly in individuals with an enzyme deficiency (54).

For more information, refer to Chapter 32 of the Installation Restoration Toxicology Guide, Volume 1.

Anthracene

Anthracene asserts phototoxic and photoallergic action on the human skin. It is carcinogenically inactive (12). Various mutagenicity studies have produced negative responses (2315). The lowest toxic oral dose in the rat is 20 g/kg (47).

66.3.4 Toxicology of Fuel Oil Additives

The toxicity of selected fuel oil additives is outlined below.

Manganese Compounds

Manganese affects the CNS. Intoxication occurs mostly in the chronic form known as manganism which is similar to Parkinsonism. Usually manganism occurs after 1-2 years exposure to manganese oxides although it may develop after only a few months. Initial symptoms include headache, asthenia (loss of strength and energy), restless sleep and personality change. This is followed by an intermediate phase with visual hallucinations, double vision, impaired hearing, uncontrollable impulses, mental confusion and euphoria. In advanced stages, the patient experiences excessive salivation, muscle weakness, muscle rigidity, tremor of the upper extremities and head, and impaired gait. In manganism with neurologic symptoms, the course is frequently progressive although some cases are stationary and others recover (2,46).

Inhalation of high concentrations of manganese oxide causes metal fume fever - a 24-48 hour illness characterized by chills, fever, aching muscles, dry mouth and throat, and headache (46).

Magnesium Oxide

Magnesium oxide fumes are irritating to the eyes and nose. It also causes metal fume fever which is a 24-48 hour influenza-type illness (46).

Aluminum Oxide

Aluminum oxide is a nuisance dust which has little adverse effect on the lungs at low exposure levels. Excessive concentrations may cause deposits in the eyes, ears and nasal passages or may cause mild injury to the skin and mucous membranes (46).

Peroxides

In general, peroxides are strong oxidizing agents capable of skin irritation, burns or eye damage (200).

Alkyl Nitrate and Nitrites / Nitro and Nitroso Compounds

Methemoglobinemia (a loss of the oxygen carrying capacity of the blood), is the main toxic effect of nitrite and nitrate ingestion.

Early symptoms include headache, fatigue, nausea, vomiting, chest pain and cyanosis. With increasing methemoglobin concentrations, there may be weakness, dizziness, incoordination, joint pain and muscular tremors (200,480).

Various N-nitroso derivatives have caused malignant tumors in various organ systems in laboratory animals. Generally, as the molecule increases in size, carcinogenic activity decreases (12). Exposure to these compounds should be avoided. Specific information on 2 nitroso compounds - N-nitrosodimethylamine and N-nitrosodiphenylamine - may be found in Volume 1 of the Guide.

66.3.5 Levels of Concern

There are no criteria or standards for fuel oils. OSHA (298) has set a time-weighted-average exposure limit for kerosene at 500 ppm.

66.3.6 Hazard Assessment

Fuel oils themselves do not appear to be carcinogens but they do contain several polycyclic aromatic hydrocarbons which are carcinogens and/or cocarcinogens (2219). A fuel oil blend was highly active in both cellular assays and skin painting studies (1819). Positive mutagenic findings were observed in an Ames test, a mouse lymphoma assay and a rat bone marrow study for fuel oil number 2 (1914). Diesel fuel gave negative results in both the Ames and mouse lymphoma assay but positive results in the rat bone marrow assay (1914).

A reproductive study with rats exposed by inhalation at levels up to 408 ppm suggested no adverse effects (1915).

Acute toxic effects of ingested fuel oils included alopecia, dermal irritation and open sores in the genital area of exposed rats. The oral LD_{50} values ranged from 5 to 17.5 g/kg for rats (1924).

Dermal studies in rabbits indicated severe dermal irritation, weight loss, anorexia, ataxia and lethargy following a dose of 5 g/kg (1924). Fuel oils are also minimally to moderately irritating to rabbit eyes (1924). No chronic animal data were found.

In humans, CNS depression is the chief systemic reaction to fuel oils (17). Ingestion of less than ½ ounce has been fatal (17). Dermal and inhalation exposures to diesel fuel have induced nephropathy in humans (1814,1815,1816).

66.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of fuel oils in soil and water requires collection of a representative field sample and laboratory analysis for the specific major components attributed to fuel oil; however, the relative concentrations of the constituents, and even the constituents themselves, will vary with time and distance from the site of initial contamination due to weathering. The major component categories in fuel oil have been identified as the following:

n-alkanes
branched alkanes
benzene and alkylbenzenes
naphthalenes
polynuclear aromatic hydrocarbons

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques may be used to identify the principal components in fuel oils. Fuel samples, and probably any samples collected in the field which are primarily organic in nature, may require the separation (prior to GC or GC/MS analysis) of the aliphatic, monoaromatic and polycyclic aromatic hydrocarbon fractions using liquid solid column chromatography; the various column eluates, with or without dilution in carbon disulfide, can then be analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (i.e., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet extraction or sonication methods (1422). An aliquot of the sample extract, with or without concentration, is then analyzed by GC or GC/MS. Sampling and analysis considerations for some specific components in fuel oil, i.e., benzene, toluene, xylenes, ethyl benzene and naphthalene, have been addressed in Volume 1.

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component, but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorotrifluoroethane). The "oil and grease" content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; extraction methods are those described above for aqueous and soil samples.

A detection limit for fuel oils was not determined; the detection limit for specific components is expected to be in the range of $\mu g/L$ for aqueous samples and $\mu g/g$ for non-aqueous samples.

APPROXIMATE COMPOSITION:	
alkanes	61%
cycloalkanes	29%
alkylbenzenes	8%
indans/tetralins	1.1%
naphthalenes	<1%

REACTIVITY	Various sources typically report that hydrocarbon mixtures are incompatible with strong acids, alkalis, and strong oxidizers such as liquid chlorine and oxygen. The NFPA reports vigorous reactions, ignition, or explosions involving chlorine, fluorine, or magnesium perchlorate. Jet fuels are considered to be miscellaneous combustible or flammable materials for compatibility classification purposes. Such substances typically evolve heat, fire, and toxic or flammable gases in reactions with oxidizing mineral acids, alkali or alkaline earth metals, nitrides, organic peroxides or hydroperoxides, or strong oxidizing agents. Reactions with explosive materials may result in an explosion, while those with strong reducing agents may evolve heat and flammable gases. Non-oxidizing mineral acids generally evolve heat and innocuous gases (505,507,511).

PHYSICO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(60)
	• Color: colorless to light brown	(60)
	• Odor: fuel-oil	(60)
	• Odor Threshold: 1 ppm	(60)
	• Liquid Density (g/ml at 20°C): 0.75	(1934)
	• Freezing/Melting Point (°C): -72	(1933)
	• Boiling Point (°C): 60-270	(1933)
	• Flash Point (°C): -23 to -1, closed cup; -29	(23,51,60, 1934)
	• Flammable Limits in Air, % by Volume: 1.3-8%	(60,506)
	• Autoignition Temperature (°C): 240-242	(23,51,60, 506)
	• Vapor Pressure (mm Hg at 20°C): 91	(1934)
	• Saturated Concentration in Air (mg/m^3 at 20°C): 660,000	(ADL estim)
	• Solubility in Water (mg/L at 20°C): 300 (max)	(2251)
	• Viscosity (cp at 21°C): 0.829	(60)
	• Surface Tension (dyne/cm at 20°C): 25 (estim)	(60)
	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 3-7	(*)
	• Soil Adsorption Coefficient, K_{oc}: 240 - 5×10^{-6}	(*)
	• Henry's Law Constant (atm•m^3/mol at 20°C): 10^{-4} - 10	(*)
	• Bioconcentration Factor: 50-500,000*	(ADL estim)

*Range for typical components (see Table 64-4).

PERSISTENCE IN THE SOIL-WATER SYSTEM	JP-4 hydrocarbons are expected to be relatively mobile and non-persistent in most soil systems. Persistence in deep soils and ground water may be higher. Volatilization, photooxidation and biodegradation are important fate processes. Surface spills are expected to be weathered by evaporation and photooxidation. Downward migration of weathered JP-4 surface spills and sub-surface discharges represent a potential threat to underlying ground water. Biodegradation of JP-4 hydrocarbons is expected to be significant under environmental conditions favorable to microbial oxidation; naturally-occcurring, hydrocarbon-degrading microorganisms have been isolated from polluted soils and, to a lesser extent, non-polluted soils.

PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water system is the contamination of ground-water drinking water supplies by JP-4 from leaking storage tanks or large spills. Vapors from leaked or spilled fuel may diffuse through soils and migrate into structures, resulting in inhalation exposures. Inhalation exposure may also occur from the direct volatilization of spills, and in some instances, aircraft fuel jettisoning may result in the contamination of surface water and agricultural land, leading to ingestion with water or food.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (60.1932): Short-term exposure to high vapor levels can cause irritation of the respiratory tract, headaches, nausea, and mental confusion. In extreme cases, loss of consciousness can occur. Ingestion is irritating to the stomach. Aspiration of the liquid into the lungs can give rise to chemical pneumonitis. The liquid may cause defatting, drying and irritation of the skin. Both the vapor and the liquid are irritating to the eyes.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral -- no data skin -- no data LC_{50} (mg/m^3) inhalation -- no data
	Long-Term Effects: Liver, kidney and neurological damage
	Pregnancy/Neonate Data: No data
	Mutation Data: Negative
	Carcinogenicity: No data

HANDLING PRECAUTIONS (1967)	No specific respirator guidelines were found for JP-4. The following guidelines are for kerosene with a boiling range of 175-325°C • Less than or equal to 1000 mg/m^3: chemical cartridge respirator with half-mask facepiece and organic vapor cartridge or supplied-air respirator with half-mask facepiece operated in demand mode • 1000-5000 mg/m^3: gas mask with full facepiece and organic canister; supplied-air respirator with full facepiece or self-contained breathing apparatus with full facepiece operated in demand mode • Appropriate protective clothing including gloves, aprons and boots • Chemical goggles if there is probability of eye contact.

EMERGENCY FIRST AID TREATMENT (1932)	Ingestion: Do not induce vomiting. Get medical attention • Inhalation: Move victim to fresh air. Give artificial respiration if necessary. Get medical attention • Skin: Remove contaminated clothing. Wash skin with soap and water. If blistering or skin loss has occurred, wash remaining fuel off with sterile water only and treat as a thermal burn. Get medical attention • Eye: Irrigate with large amounts of water. Get medical attention.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards

- OSHA PEL (8-hr TWA): petroleum distillates (naphtha) 500 ppm
- AFOSH PEL (8-hr TWA): petroleum distillates (naphtha) 500 ppm

Criteria

- NIOSH IDLH (30-min): petroleum distillates (naphtha) - 10,000 ppm; gasoline - none established
- ACGIH TLV® (8-hr TWA): petroleum distillates (naphtha) - none established; gasoline - 300 ppm
- ACGIH STEL (15-min): petroleum distillates (naphtha) - none established; gasoline - 500 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)

- Human Health
  None established; JP-4 is not a priority pollutant.

- Aquatic Life
  None established; JP-4 is not a priority pollutant.

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;

- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;

- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Occupational Safety and Health Act (OSHA)
  Employee exposure to petroleum distillates (naphtha) shall not exceed an 8-hour time-weighted-average (TWA) of 500 ppm (298).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated aviation fuel as a hazardous material which is subject to requirements for packaging, labeling and transportation (306).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

64.1 MAJOR USES AND COMPOSITION

64.1.1 Major Uses

Jet Fuel 4 (JP-4) is a turbine engine fuel used exclusively by the U.S. Air Force; it constitutes 85% of the turbine fuels used by the Department of Defense (1933).

64.1.2 Composition

Jet fuel petroleum products are made by blending various proportions of distillate stocks such as naphtha, gasoline and kerosene to meet military and commercial specifications. Most of the available characterization data (e.g., military specifications) address gross performance properties. There is considerable variability in the concentration of major components, as well as in the performance characteristics, of JP-4 fuel derived from different crude oil supplies (1843,2246,2247,2251). In general, the reported distillation range for JP-4 fuel is approximately 140°C-270°C (1844); most of the hydrocarbons fall in the range of C_4 to C_{14}. A typical JP-4 composition expressed as percent volume by compound category has been reported (1845) to be: paraffins (61%), monocycloparaffins (24%), dicycloparaffins (5%), alkylbenzenes (8%), indans and tetralins (1%) and naphthalenes (<1%). JP-4 fuel may contain olefinic hydrocarbons up to 5% (volume) and total sulfur up to 0.4% (weight) (1844).

The individual major components of JP-4 representing at least 0.1% by weight have been characterized by several authors (1822,1845) and account for approximately 70-75% by weight of the fuel. The approximate distribution of the major components by compound category is: n-alkanes, 32%; branched alkanes, 31%; cycloalkanes, 16%; benzenes and alkylbenzenes, 18%; and naphthalenes, 3% (1846). Table 64-1 presents detailed data on the specific hydrocarbon composition of one JP-4 fuel.

Although they are generally considered minor components, there are many non-hydrocarbons present in petroleum-derived distillates. In general, these become major concerns in the heavy distillates and residues (almost 70% of total composition in heavy oils) and are much less important components in middle distillates such as JP-4. Sulfur compounds represent the largest class of non-hydrocarbons found in petroleum; this group might include aliphatic and aromatic compounds such as thiols, sulfides, disulfides, and thiophenes, as well as elemental sulfur, hydrogen sulfide, and carbon sulfide. The majority of crude oils have low oxygen content. Most of the oxygen is in the form of fatty acids and acids with aromatic functional groups; smaller contributions come from alcohols, ketones, esters, fluorenones, furans, dibenzofurans, and benzonaphthofurans. The level of nitrogen compounds is generally less than 0.1% but may be higher (0.5-15%) in heavy distillates and residues. Nitrogen compounds that may be present in petroleum fuels, particularly in heavier distillates than JP-4, include pyridines, quinolines, acridines, amines, pyrroles, indoles and carbazoles (1848).

TABLE 64-1

MAJOR COMPONENTS OF ONE JP-4 SAMPLE

Fuel Component	Percent by Weight
n-Butane	0.12
Isobutane	0.66
n-Pentane	1.06
2,2-Dimethylbutane	0.10
2-Methylpentane	1.28
3-Methylpentane	0.89
n-Hexane	2.21
Methylcyclopentane	1.16
2,2-Dimethylpentane	0.25
Benzene	0.50
Cyclohexane	1.24
2-Methylhexane	2.35
3-Methylhexane	1.97
trans-2,3-Dimethylcyclopentane	0.36
cis-1,3-Dimethylcyclopentane	0.34
cis-1,2-Dimethylcyclopentane	0.54
n-Heptane	3.67
Methylcyclohexane	2.27
2,2,3,3-Tetramethylbutane	0.24
Ethylcyclopentane	0.26
2,5-Dimethylhexane	0.37
2,4-Dimethylhexane	0.58
1,2,4-Trimethylcyclopentane	0.25
3,3-Dimethylhexane	0.26
1,2,3-Trimethylcyclopentane	0.25
Toluene	1.33
2,2,-Dimethylhexane	0.71
2-Methylheptane	2.70
4-Methylheptane	0.92
cis-1,3-Dimethylcyclohexane	0.42
3-Methylheptane	3.04
1-Methyl-3-ethylcyclohexane	0.17
1-Methyl-2-ethylcyclohexane	0.39
Dimethylcyclohexane	0.43
n-Octane	3.80
1,3,5-Trimethylcyclohexane	0.99
1,1,3-Trimethylcyclohexane	0.48
2,5-Dimethylheptane	0.52
Ethylbenzene	0.37
m-Xylene	0.96
p-Xylene	0.35
3,4-Dimethylheptane	0.43

Source: 1845

(Continued)

TABLE 64-1 - Continued

MAJOR COMPONENTS OF ONE JP-4 SAMPLE

Fuel Component	Percent by Weight
4-Ethylheptane	0.18
4-Methyloctane	0.86
2-Methyloctane	0.88
3-Methyloctane	0.79
o-Xylene	1.01
1-Methyl-4-ethylcyclohexane	0.48
n-Nonane	2.25
Isopropylbenzene	0.30
n-Propylbenzene	0.71
1-Methyl-3-ethylbenzene	0.49
1-Methyl-4-ethylbenzene	0.43
1,3,5-Trimethylbenzene	0.42
1-Methyl-2-ethylbenzene	0.23
1,2,4-Trimethylbenzene	1.01
n-Decane	2.16
n-Butylcyclohexane	0.70
1,3-Diethylbenzene	0.46
1-Methyl-4-propylbenzene	0.40
1,3-Dimethyl-5-ethylbenzene	0.61
1-Methyl-2-isopropylbenzene	0.29
1,4-Dimethyl-2-ethylbenzene	0.70
1,2-Dimethyl-4-ethylbenzene	0.77
n-Undecane	2.32
1,2,3,4-Tetramethylbenzene	0.75
Naphthalene	0.50
2-Methylundecane	0.64
n-Dodecane	2.00
2,6-Dimethylundecane	0.71
2-Methylnaphthalene	0.56
1-Methylnaphthalene	0.78
n-Tridecane	1.52
2,6-Dimethylnaphthalene	0.25
n-Tetradecane	0.73

Source: 1845

In addition to the aliphatic/aromatic hydrocarbon content and trace N-containing, O-containing and S-containing species, JP-4 distillate fuel may also contain trace inorganic elements. All metals through atomic number 42, except rubidium and niobium, have been found in petroleum. Generally, the concentrations are quite low; the most prevalent metals are nickel and vanadium (1848). Table 64-2 presents the results of an analysis of the trace elements in one JP-4 fuel sample. The JP-4 concentration of these elements is expected to vary from one crude oil source to another.

Actual stocks of JP-4 fuel may also contain a number of additives used as anti-oxidants, metal deactivators, corrosion or icing inhibitors, or electrical conductivity agents. A list of some of the chemicals that may be used for these purposes is provided in Table 64-3. The composition of JP-4, particularly older stocks, may also vary due to contaminants from the storage container. In addition, microbes can be anticipated to grow well on these hydrocarbons; bacterial and/or fungal contamination may also affect the composition of JP-4 stocks.

64.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

For the purposes of this chapter, the discussions of the environmental behavior of JP-4 will be limited to a discussion of the major components; the environmental behavior of the trace elements and the many diverse additives will not specifically be addressed.

Transport and transformation of individual JP-4 constituents will vary depending on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly (in the percolating ground waters), be sorbed less strongly on the soils (thus being transported more rapidly), and may be more or less susceptible to degradation by chemical or biological action. Thus, the relative concentrations of the constituents of the fuel will vary with time and distance from the site of initial contamination. This effect is called "weathering." (This term is also used to describe the changes to oil following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation all are involved.)

64.2.1 Transport in Soil/Ground-water Systems

64.2.1.1 Equilibrium Partitioning Model

In general, soil/ground-water transport pathways for low concentrations of pollutants in soil can be assessed by using an equilibrium partitioning model. For the purposes of assessing the environmental transport of JP-4 fuel, a group of specific hydrocarbons was selected from the dominant JP-4 hydrocarbon classes, i.e., alkanes, cycloalkanes, and alkylbenzenes. These specific compounds were chosen on the basis of their relatively high concentrations in JP-4 and span the boiling point range of the JP-4 hydrocarbons. Table 64-4 lists the hydrocarbons which were selected and presents the predicted

TABLE 64-2

CONTENT OF TRACE ELEMENTS IN ONE SAMPLE OF PETROLEUM-DERIVED JP-4

Trace Element	Parts per Million By Weight
Al	NA[a]
Sb	<0.5
As	0.5
Be	NA
Cd	<0.03
Ca	NA
Cl	NA
Cr	<0.05
Co	NA
Cu	<0.05
Fe	<0.05
Pb	0.09
Mg	NA
Mn	NA
Hg	<1
Mo	NA
Ni	<0.05
Se	<0.3
Si	NA
Ag	NA
Na	NA
Sr	NA
Th	NA
Sn	NA
Ti	NA
V	<0.05
Zn	<0.05

Source: 1843

[a]NA indicates data not available

partitioning of low soil concentrations of those hydrocarbons among soil particles, soil water, and soil air. The portions associated with the water and air phases of the soil are expected to have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil indicate that sorption is expected to be an important process for all the dominant hydrocarbon categories. Partitioning to the soil-vapor phase is also expected to be important for the lower molecular weight aliphatic hydrocarbons (C_4-C_8) which are characterized by high vapor pressures and low water

TABLE 64-3

ADDITIVE COMPOUNDS APPROVED FOR USE IN MILITARY JP-4 FUEL

Antioxidants (≤24 mg/L)[a]

2-6-di-t-butylphenol
2-6-di-t-butyl-4-methylphenol
6-t-butyl-2,4-dimethylphenol
Other alkyl phenols (mono,di,tri; methyl, ethyl, isopropyl, t-butyl)
N,N'-di-sec-butyl-p-phenylenediamine[c]

Metal Deactivators (≤ 5.8 mg/L)[a]

N,N'-disalicylidene-1,2-propanediamine
N,N'-disalicylidene-1,2-cyclohexanediamine
N,N'-disalicylidene-1,2-ethanediamine[c]

Corrosion Inhibitors

MIL-I-25017/QPL-25017[a]
Amine carboxylates (5-20 ppm)[b]: $(RCOO^-)(NH_3R'^+)$, R = C_{16}-C_{18}
Ethylene diamine dinonyl naphthalene sulfonates[c]

Icing Inhibitors

MIL-I-27686[a]
Carboxylates (40-150 ppm)[b]: $RCOO^-$, R = C_{16}-C_{18}
C_1-C_3 alcohols[c]
Dimethylformamide[c]
Ammonium dinonylnaphthalene[c]

Electrical Conductivity Additive[a]

ASA-3 (Shell Chemical Co., Houston, TX)

---

[a] Reference 1844

[b] Reference 1847

[c] Reference 1824

solubility. The alkyl benzenes have higher water solubilities and transport with infiltrating water may be important for these compounds; volatilization, on the other hand, may be less important. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a significant percent of both aliphatic (particularly less than C_7) and aromatic hydrocarbons is predicted to be present in the soil-water phase and available for transport with flowing ground water.

In interpreting these results, it must be remembered that this model is valid only for low soil concentrations (below aqueous solubility) of the components. Large releases of JP-4 (spills, leaking underground storage tanks) may exceed the sorptive capacity of the soil, thereby filling the pore spaces of the soil with the fuel. In this situation, the hydrocarbon mixture would move as a bulk fluid and the equilibrium partitioning model would not be applicable.

Overall, ground water underlying soil contaminated with JP-4 hydrocarbons is expected to be vulnerable to contamination by at least some of these components. The type of spill (surface vs. sub-surface) is of particular importance, since volatilization from the surface is expected to be a significant removal process for low molecular weight aliphatics. At this point, it should be mentioned that environmental fate/exposure/toxicology chapters for several of the components in Table 64-4 were included in Volume 1 of the IRP Toxicology Guide. The JP-4 components addressed in Volume 1 include: benzene, toluene, xylenes, ethyl benzene, and naphthalene.

64.2.1.2 Transport Studies

Due to the extensive use of JP-4 and other aviation fuels and their potential for environmental release during use, storage or transport, several groups have addressed its fate. The fate of JP-4 in the soil environment is basically a function of the solubility, volatility, sorption, and degradation of its major components. The relative importance of each of these processes is influenced by the type of contamination (e.g., surface spill *vs*. underground release, major vs. minor quantity), soil type (e.g., organic content, previous history of soil contamination), and environmental conditions (e.g., pH, temperature, oxygen content).

Transport processes have been shown to be more significant than transformation processes in determining the initial fate of petroleum hydrocarbons released to soil/ground-water systems (1845,1848,1846). For JP-4 released to surface soils or waters, transport to the atmosphere through volatilization has been shown to be the primary fate pathway for most of the JP-4 hydrocarbons; subsequent atmospheric photolysis is expected to be rapid (1845). Using a model fuel mixture containing approximately fifteen compounds representative of major JP-4 hydrocarbons, Spain *et al*. (1846) demonstrated that compounds having up to nine carbons are weathered almost exclusively by evaporation; larger compounds were weathered primarily by evaporation and biodegradation; dimethylnaphthalene and highly substituted aromatics ($>C_{14}$) were shown to be persistent in these tests. Reduced temperatures tend to increase JP-4 persistence by retarding the rates of volatilization and biodegradation (1846,1822).

TABLE 64-4

EQUILIBRIUM PARTITIONING OF SELECT JP-4 HYDROCARBONS IN MODEL ENVIRONMENTS[a]

COMPOUND	log K_{ow}	K_{oc}[b]	H[c]	UNSATURATED TOPSOIL (%) Soil	Water	Air	SATURATED DEEP SOIL[d] (%) Soil	Water
Hexane	3.90 (e)	3,830	1.68	77.5	0.1	22.4	94.1	5.9
Octane	5.18 (e)	73,000	2.96	97.4	0.01	2.6	99.7	0.3
Dodecane	7.06 (f)	5.5×10^6	7.4	99.9	0.0001	0.09		
Isopentane	3.37 (f)	900	1.36	50.3	0.3	49.4	79.1	20.9
Trimethylpentane	4.87 (f)	36,000	1.9-3.3	94.7	0.01	5.3	99.3	0.7
Methylcyclopentane	3.47 (f)	1,400	0.36	85.4	0.3	14.3	85.5	14.5
Cyclohexane	3.44 (e)	1,330	0.18	91.8	0.4	8.0	84.8	15.2
Methylcyclohexane	4.10 (f)	6,070	0.39	95.9	0.08	4.0	96.2	3.8
Toluene	2.69 (g)	240	6.6×10^{-3}	96.5	1.9	1.6	52.1	47.9
Xylenes	3.15 (e)	700	7×10^{-3}	98.8	0.7	0.5	74.4	25.6
Trimethylbenzenes	3.65 (h)	2,150	5×10^{-3}	99.6	0.2	0.2	90.0	10.0
Methylnaphthalenes	3.87 (e)	3,570	4.4×10^{-4}	99.8	0.1	0.01	93.7	6.3

[a] Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

[b] Reference 652.

[c] Taken from Reference 74 unless otherwise specified. Units equal $atm \cdot m^3/mol$.

[d] Used sorption coefficient $K_p = 0.001 \times K_{oc}$.

[e] Reference 29.

[f] Arthur D. Little, Inc., estimate according to equations provided in Reference 31.

[g] Reference 10.

[h] Reference 31.

Compared with the marine environment, infiltration into porous soils slows the evaporative loss of volatile hydrocarbons. McGill et al. (2267) concluded that up to 20-40% of crude oils may volatilize from soils; elevated temperatures, lateral spreading and adsorption onto surface vegetation may facilitate evaporation at such levels. Volatilization of JP-4 components is expected to be more extensive than volatilization of crude oils. Purging of the water soluble fraction of JP-4 fuel with nitrogen and air demonstrated a rapid loss of JP-4 hydrocarbons (80% loss in 2 minutes) (2250).

Under conditions of limited volatilization (low temperatures, subsurface release or concentrated spill) downward migration into the soil and to the ground water may be important. Several authors (1811,2243,2252) have reported that oil substances released in significant quantities to soils result in a separate organic phase which moves downward through the unsaturated zone to the less permeable layer, the soil/ground-water boundary, where they tend to accumulate and spread horizontally. The organic layer floats on the ground water and is carried in the general direction of ground water flow. At the oil-water interface, some hydrocarbons are leached according to their aqueous solubility. The pattern of migration of the hydrocarbon phase may be very different from that of the ground water. Due to fluctuations in ground-water elevation, over time the organic layer on top of the aquifer may be transported into several zones where the components occur in the gaseous phase (able to diffuse in all directions, including upward), liquid phase (adsorbed onto rock particles or sealed under water) or dissolved/emulsified in water (1811).

Migration through soils may be retarded to some extent by sorption. In general, sorption of aviation kerosene on soils has been reported to be weak. Migration is expected to be fastest through previously contaminated soils where the sorptive sites may be unavailable; on the other hand, soil-water content increases sorption and slows migration of JP-4 hydrocarbons. Sorption may also alter the availability of hydrocarbons for biodegradation and other weathering processes (1846,1811,2248).

In fissured rock the migration of JP-4 hydrocarbons is much less uniform than in porous soils. Preferential spreading through crevices, sometimes changed the direction of flow, may occur. Determination of the potential ground-water contamination in fissures rock is thus very difficult (1811).

Sediment-water sorption studies (2248) were performed on jet fuel dissolved in water; 3 sediments and 3 clays were utilized. The observed adsorption constants were small compared to those of other non-polar organics. For the individual JP-4 components the magnitude of the adsorption constant is dependent on the size and complexity of the hydrocarbon, and bears an inverse relationship to its aqueous solubility. The nature of adsorbent was important (non-swelling clays were reported to be poor adsorbents compared to sediments) but the organic carbon content exhibited only a casual relationship to

adsorbent ability. Temperature and pH did not have an important effect over naturally occurring ranges; increasing salinity produced a small increase in hydrocarbon adsorption. Reversible adsorption was observed in experiments with benzenes and naphthalenes; strong sorbent-sorbate bonding (chemisorption) does not occur with light hydrocarbons found in JP-4 fuel.

In the vicinity of Prague airport (1811), release of aviation kerosene (similar to JP-4) resulted in extensive soil/ground-water contamination. The petroleum hydrocarbons spread as a separate organic phase as well as dissolved contaminants in the aquifer. In porous formations, pollution caused by the oil phase extended tens of hundreds of meters, while the contamination from dissolved hydrocarbons extended hundreds to thousands of meters. Within five months, a 1-m thick layer of oil extended 700 m by 200 m on the surface of the ground-water aquifer; an area of 15 km^2 was polluted by the dissolved hydrocarbons.

The transport of JP-4 contamination to ground-water aquifers and subsequent dissolution of JP-4 hydrocarbons in ground water have been discussed in several papers (1811,1845,2245,2241,1849). Crude oil and petroleum products have been shown to produce qualitatively similar water-soluble fractions (1849,2241,2248,2250). The water-soluble portion of JP-4 distillate fractions were shown to be almost entirely aromatic (87-99%) even though the distillate fuels themselves were higly aliphatic in nature; the aliphatic hydrocarbons either volatilized ($<C_{12}$) or were essentially not water soluble ($>C_{12}$). In deep, saturated soils with no soil-air, some low molecular weight aliphatics may be transported and dissolved in ground water. Table 64-5 presents fuel-water partition coefficients for some JP-4 hydrocarbons; the data support the observation that the light aromatics represent the greatest threat to contamination of ground-water supplies. In general, a decreasing degree of petroleum contamination has been observed over time in the absence of further aquifer pollution; some removal due to sorption onto rock particles and degradation by microorganisms is suspected (2244,2243,2255).

In summary, the physical distribution of JP-4 contamination affects its impact on, and removal from, the soil environment. Lateral spreading along the surface increases the contaminated area while facilitating evaporative removal of the low molecular weight hydrocarbons. Subsurface release or vertical penetration mediated by gravitation and capillary forces decreases evaporation, reduces the importance of transformation pathways (see below), and may lead to ground-water contamination.

TABLE 64-5

JP-4 FUEL-WATER PARTITION COEFFIENTS (K_{fw}) FOR SELECTED HYDROCARBONS[a]

Compound	Log K_{fw}[b]
Methylcyclopentane	4.97
Benzene	3.39
Cyclohexane	4.69
2-Methylhexane	5.57
3-Methylhexane	5.56
n-Heptane	5.50
Methylcyclohexane	4.87
Toluene	3.44
n-Octane	5.98
Ethylbenzene	3.68
m-Xylene	3.57
p-Xylene	3.88
o-Xylene	3.85
1,2,4-Trimethylbenzene	3.95
Isopropylbenzene	4.25
Naphthalene	3.88
2-Methylnaphthalene	4.35
1-Methylnaphthalene	4.67

[a] Reference 1845

[b] K_{fw} = (concentration of chemical in fuel) ÷ concentration of chemical in water) at equilibrium, T = 20°C. Fuel-water ratio = 1:1000.

64.2.2 Transformation Processes in Soil/Ground-water Systems

64.2.2.1 Chemical Transformation

Photooxidation has been reported to play a significant role in the chemical degradation of petroleum hydrocarbons in the sunlit environment (1845,1848,2252,2259). Alkanes, benzenes, and mono-substituted benzenes have been shown to be relatively resistant to photolysis in aqueous systems; xylenes photolyzed slowly while trisubstituted benzenes and naphthalenes photolyzed at rates competitive with volatilization (1845). Penetration of oil below the soil surface limits exposure to solar radiation while extensive lateral spreading of oil over impermeable or rocky surfaces may promote substantial photooxidative degradation.

The oxygenated products of photooxidation are generally more water-soluble than the parent hydrocarbons and are thus more likely to be leached from soil; enhanced toxicity of the oxygenated hydrocarbons has also been observed (2248,2252). Larson et al. (2260) have reported that in marine environments weathering of crude oils resulted in decreased growth of algae.

64.2.2.2 Biological Degradation

Natural ecosystems have considerable exposure to petroleum hydrocarbons from natural emissions, accidental contamination through oil spills and storage tank leaks, and deliberate application to land in land-farming waste disposal activities; therefore, their biodegradation is of environmental importance. Numerous authors have observed the biodegradation of petroleum hydrocarbons, and several extensive reviews and reports are available (1846,2252,2255,2249,2253). Hydrocarbon-degrading bacteria and fungi are widely distributed in marine, freshwater, and soil environments. As reported in the review by Atlas (2255), an extensive and diverse group of bacteria and fungi have been shown to have the ability to degrade petroleum hydrocarbons.

The qualitative hydrocarbon content of petroleum mixtures largely determines their degradability. In general, microorganisms exhibit decreasing ability to degrade aliphatic hydrocarbons with increasing chain length; however, Haines and Alexander (2254) showed that n-alkanes up to C_{44} were metabolized. n-Alkanes are considered more easily biodegraded than branched or cyclic alkanes; aromatics are generally more rapidly biodegraded than alkanes.

The relative biodegradation susceptibility of petroleum hydrocarbons has been summarized in a review by Bossert and Bartha (2252): n-alkanes, n-alkylaromatics, and aromatics of the C_{10}-C_{22} range are the most readily biodegradable; n-alkanes, alkylaromatics, and aromatics in the C_5-C_9 range are biodegradable at low concentrations by some microorganisms but are removed by volatilization and unavailable for biodegradation in most environments; n-alkanes in the C_1-C_4 range are biodegradable only by a narrow range of specialized hydrocarbon degraders; and n-alkanes, alkylaromatics, and aromatics above C_{22} are generally not available to degrading microorganisms. Hydrocarbons with condensed ring structures, such as polycyclic aromatic hydrocarbons, have been shown to be relatively resistant to biodegradation. The biodegradability of some hydrocarbons may be enhanced when present in petroleum mixtures.

Fatty acids and long chain n-alkanes not originally present in weathered petroleum samples have been observed after biodegradation; generation of tar balls which are quite resistant to microbial degradation has also been reported (2252,2255,2257,2258). Therefore, enhanced solubilization or sorption of some metabolic intermediates (some of which may be more toxic than the original hydrocarbons) may be significant in the soil environment (2249).

Although the microbiota of most non-contaminated soils include many naturally occurring hydrocarbon-degrading populations, the addition of petroleum selectively enriches that sector able to adapt and utilize the new substrate. The available review articles (citing laboratory studies and field studies) confirm that the distribution of hydrocarbon-utilizing microorganisms reflects the historical exposure of the environment to hydrocarbons (2252,2255,2257,2249). In unpolluted ecosystems, hydrocarbon utilizers generally constitute less than 0.1% of the microbial community; in oil-polluted ecosystems, they can constitute up to 100% of the viable microorganisms (2255). Walker et al. (2257) reported that all classes of petroleum hydrocarbons were degraded by microorganisms in an oil-exposed sediment but not in a similar unexposed sediment.

Biodegradability has been shown to be related to JP-4 hydrocarbon concentrations. When concentrations are too low, biodegradation may cease. However, at high concentrations the components or their metabolic intermediates may be toxic and inhibit degradation (2249).

Biodegradation of petroleum hydrocarbons has also been shown to be dependent on other environmental factors including: temperature, oxygen and moisture, nutrients, salinity, and pH (2252,2249,2255,1846). Petroleum biodegradation has been reported to occur over a wide range of temperatures: Huddleston and Cresswell (2261) reported biodegradation at -1.1°C; Dibble and Bartha (2262) reported that the highest rates occurred between 30°C and 40°C with no increase observed above 37°C; and Atlas and Bartha (2263) reported that the degradation rate roughly doubles with each 5°C increase in the 5° to 20°C range; degradation in arctic environments has been reported to be dramatically reduced (2255,2266).

Oxygen has been reported to be necessary for the initial steps of hydrocarbon degradation; reports of anaerobic degradation have been sporadic and controversial (2252,2255,2249). Oxygen depletion has been shown to lead to sharply reduced hydrocarbon utilization in soils (2261). Tilling of soil has been shown to have a positive effect on petroleum degradation (1811,2256).

In the presence of large quantities of hydrocarbon substrates, the availability of nutrients, particularly nitrogen and phosphorus, becomes increasingly important and the addition of fertilizers has a notable positive effect on biodegradation (2249,2252,2255); in subsoil treated with 1-10% oil, the addition of fertilizer had little effect (2256).

There are limited data available on the effects of pH and salinity on biodegradation of petroleum. In general, degradation was reported to decrease with increasing salinity (2249) although the effect of different microbial populations in the experiment was not determined. Hydrocarbon degradation was reported to be low in naturally acidic soils and increased up to pH 7.8 (2262).

The fate of petroleum hydrocarbons from various actual environmental incidents has been summarized by Atlas (2255). Microbial degradation of JP-4 residues in cold anoxic marine sediments was essentially zero following a release in Searsport, ME (2266); however, microbial degradation did apparently occur during transport from the spill location to the marine sediment. Microbial degradation of petroleum hydrocarbons in ground water, river water and soils has also been reported (2255).

In summary, biodegradation of the petroleum hydrocarbons comprising JP-4 fuel is expected to be rapid under conditions favorable for microbial activity and when fuel components are freely available to the microorganisms. Degradation may be limited and/or slow in environments with few degrading organisms, low pH, low temperatures, and high salinity (e.g., arctic environments). It should be mentioned that Walker et al. (2257) state that even under optimum conditions, total and complete biodegradation is not expected to occur except possibly over an extremely long time period.

64.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that the major components of JP-4 fuel are highly volatile but vary in their potential for bioaccumulation and tendency to sorb to soil. They range from moderately to strongly sorbed to soil, and their bioaccumulation potential ranges from low to high. The variability in the properties of the components suggest they may have somewhat different potential exposure pathways.

Spills of JP-4 would result in the evaporative loss of the more highly volatile components leaving those of lesser volatility in the soil. The fraction remaining in the soil is expected to be relatively mobile and will be carried by gravity to the saturated zone of the soil. There, the more soluble components (aromatic and lower molecular weight aliphatic compounds) will dissolve into the ground water or form emulsions with ground water, while the insoluble fraction will float as a separate phase on top of the water table. The movement of dissolved hydrocarbons in ground water is much greater than the separate liquid phase, reaching distances of hundred to thousands of meters compared to tens of meters for the movement of the separate phase. In the presence of cracks and fissures, however, the flow of the separate hydrocarbon phase is greatly enhanced.

The movement of JP-4 fuel in ground water may contaminate drinking water supplies, resulting in ingestion exposures. Ground-water discharges to surface water or the movement of contaminated soil particles to surface water drinking water supplies may also result in ingestion exposures and in dermal exposures from the recreational use of these waters. The potential also exists for uptake by fish and domestic animals, which may also result in human exposures due to the bioconcentration of various fuel components.

Volatilization of JP-4 hydrocarbons in soil is another potential source of human exposure. Exposures may be more intensive when the soil is contaminated directly from leaking underground storage tanks and pipes, rather than from spills. In such cases, the more volatile components do not have an opportunity to evaporate before penetrating the soil. Once in the soil, the hydrocarbons evaporate, saturating the air in the soil pores, and the vapors diffuse in all directions including upward to the surface. The vapors may diffuse into the basements of homes or other structures in the area, resulting in inhalation exposures to the buildings' occupants.

64.2.4 Other Sources of Human Exposure

The volatile nature of JP-4 fuel suggests that inhalation exposures to residents in the vicinity of air fields may occur during large spills. Volatilization also occurs during routine fuel handling operations and from fuel losses during the cooling of jet engines (1811), but these sources are expected to result in negligible exposures to residents in the area. Workers in the immediate area could receive much greater exposures, however.

Human exposure to JP-4 fuel may result from fuel-jettisoning by aircraft. The effect of the evaporated fuel vapors is considered negligible (1912), but several exposure pathways exist for the fraction reaching the ground.

The composition and fraction of the jettisoned JP-4 fuel that reaches the ground depends upon the altitude of its release and the temperature at ground level. For example, at a ground temperature of -20°C, over 20% of the JP-4 released below 400 meters may reach the ground but at a ground temperature of 20°C, less that 1% of the JP-4 fraction will reach the ground regardless of the altitude of release. At altitudes above 3000 meters, release height has almost no effect on the JP-4 fraction reaching the ground; however the surface area of fuel distribution will be affected (1913).

Because the volume of fuel released in a jettison may range from a few thousand to over 50,000 liters (1912), the amount reaching the ground may lead to significant human exposure if released at a low altitude. Contamination of surface water, crops and pasture land may result in human ingestion. However, significant human exposure is expected to be rare since Air Force directives specify that, whenever possible, release be made over unpopulated areas and at altitudes above 1500 meters (6000 meters in some aircrafts) (1912).

64.3 HUMAN HEALTH CONSIDERATIONS

64.3.1 Animal Studies

64.3.1.1 Carcinogenicity

The carcinogenicity of petroleum-derived and shale-derived JP-4 was evaluated in Fischer 344 rats and C57BL/6 mice. The animals were exposed to vapor concentrations of 5000 or 1000 mg/m^3 in whole-body inhalation chambers. Exposure was for 6 hours daily, 5 days per week for 1 year. The animals were held for an additional year. The results of histopathologic evaluation of the tissues are in progress (1936).

No other studies dealing with the carcinogenicity of JP-4 were located.

64.3.1.2 Mutagenicity

The mutagenicity of JP-4 H-Farm B-42 was evaluated in a number of *in vitro* and *in vivo* assays. Other than an increase in unscheduled DNA synthesis, test results were negative.

In the Ames test, negative results were obtained in *Salmonella typhimurium* strains TA98, 100, 1535, 1537 and 1538 both with and without microsomal activation. Test concentrations ranged from 0.001 to 5.1 μL per plate. Concentrations above 1 μL per plate were toxic to most of the bacterial strains. Negative results were also seen in *Saccharomyces cerevisiae* strain D4 and in the TK mouse lymphoma cell assay (1813).

JP-4 H-Farm B-42 produced a dose-related increase in unscheduled DNA synthesis in WI-38 cells both with and without activation, although the results from tests with activation were of greater magnitude than those without (1813).

In the dominant lethal assay, negative results were obtained in mice given doses of 0.01, 0.03 or 0.09 mL/kg/day for 5 days (1813). In rats, results were negative overall but significant preimplantation loss was observed after the fourth mating. Rats received ip doses of 0.09, 0.3 or 0.9 mL/kg/day for 5 days (1813).

64.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No studies were found in this area for JP-4.

64.3.1.4 Other Toxicologic Effects

64.3.1.4.1 Short-term Toxicity

No information was found regarding the acute toxic effects of JP-4 in animals. Due to the nature of its components, CNS effects would be expected. Vapors would be irritating to the eyes and mucous membranes and the liquid would cause irritation and defatting of the skin (200).

In tests conducted in New Zealand white rabbits, both petroleum- and shale-derived JP-4 produced no signs of irritation when 0.1 mL of undiluted material was applied to eyes. Both fuel types were also tested for primary skin irritation on intact and abraded rabbit skin. Undiluted material (0.5 mL) was applied and covered for 24 hours. Neither fuel type caused any irritation after 24 hours. By 72 hours, moderate erythema was seen in both instances. Shale-derived JP-4 caused mild edema compared to none in the petroleum-derived JP-4 group. After one week, mild edema and erythema were seen in the shale-derived JP-4 group, in the petroleum-derived group there was mild exfoliation and erythema but no edema. In skin sensitization tests conducted in guinea pigs, petroleum-derived JP-4 exhibited no sensitization response. In contrast, shale-derived JP-4 demonstrated responses indicative of a mild to moderate sensitizer (1930).

No LD_{50} data were found for JP-4. An oral LD_{50} of 20 g/kg has been reported for kerosene in guinea pigs (47).

64.3.1.4.2 Chronic Toxicity

Chronic inhalation studies have been conducted with JP-4 in various species.

Whole body vapor exposures to petroleum-derived JP-4 were conducted in beagle dogs, Fischer 344 rats and C57BL/6 mice for 90 days. The animals were exposed to 500 or 1000 mg/m^3 continuously. Animals were sacrificed immediately following the exposure period. Histopathology revealed significant exposure-related lesions in both rodent species. In female mice, the incidence of centrilobular hepatocellular fatty change was 88% in the low-dose group and 89% in the high-dose group. These lesions were absent in the control group and were thought to be the result of mild reversible toxic insult. In male rats, 100% of the kidneys in both groups exhibited hyaline droplet formation in the proximal tubular epithelium. In 96% and 100% of the low- and high-dose males, respectively, the renal tubules near the corticomedullary junction were dilated and plugged with necrotic cell debris. All lesions found in exposed and control dogs were changes consistent with aging and not due to JP-4 exposure (1933).

In a 90-day study on shale-derived JP-4, Fischer 344 rats and C57BL/6 mice were given whole body vapor exposures to 400 or 1000 mg/m^3 continuously. Groups of animals were sacrificed immediately after exposure and at 2 weeks, 2 months and 9 months post-exposure. Blood values at all post-exposure periods were all within normal limits. In the male rats, there was a significant difference in kidney and liver weights in the animals sacrificed immediately after exposure. This difference was no longer present 9 months post-exposure (1936).

Intermittent whole body vapor exposures at higher levels for 8 months failed to show any treatment-related histopathologic effects in dogs, rats, mice or monkeys; vapor level exposures were 2500 or 5000 mg/m^3, 6 hours per day, 5 days per week. The only abnormalities

observed in high-dose animals were increases in organ weight and in the organ to body weight ratios for the male rat kidney, liver, lung and spleen. There was also a 27% incidence of rat murine bronchitis (1933).

64.3.2 Human and Epidemiologic Studies

64.3.2.1 Short-term Toxicologic Effects

Acute exposure to petroleum distillates is known to cause CNS depression in man. For fuels with high vapor pressures such as JP-4, there is the possibility of significant vapor exposures, particularly in poorly ventilated or closed handling areas. Short-term exposure to high concentrations can lead to headache, nausea, mental confusion, and irritation of the respiratory system. In extreme cases, loss of consciousness can occur (1932). One case of jet fuel intoxication by the inhalation route was reported by Davies (1931). In this instance, a pilot was exposed to vapor levels of 3000-7000 ppm in the cockpit of his aircraft for approximately 7 minutes. He complained of feeling sleepy and groggy and his speech was slurred but he managed to land the aircraft safely. Neurological examination revealed a staggering gait, a positive Romberg test (indicates peripheral ataxia) generalized muscular weakness and possibly decreased sensation to painful stimuli over the dorsal surface of the right forearm. The pilot did not feel "normal" for 36 hours. He was observed during the next few days and appeared in good condition. He was examined 5 months after the incident at which time he felt fine.

Petroleum fuels generally have a low oral toxicity. Ingestion is likely to occur only through accidents and the taste and smell will usually limit the amount swallowed. Aspiration of the liquid into the lungs can cause pneumonitis (1932).

The lower boiling point hydrocarbons which are present in most liquid fuels defat the skin and cause dryness and irritation. Prolonged or repeated skin contact may result in oil acne or oil folliculitis (1832).

Eye irritation can be caused by exposure to high vapor concentrations or if the liquid is splashed into the eyes (1932).

64.3.2.2 Chronic Toxicologic Effects

Long-term exposure to jet fuel causes neurological effects. Knave et al.. (1929) conducted a cross-sectional epidemiologic study in 30 Swedish aircraft factory workers exposed to jet fuel with the following composition: aromatic hydrocarbons 12 vol %; olefin hydrocarbons 0.5 vol %; saturated hydrocarbons 87.5 vol%. Duration of exposure ranged from 2 to 32 years with a mean of 17.1 years. Exposure levels ranged from 128-432 mg/m^3. Controls were age-matched and were employed for a similar time period but had no exposure. Twenty-one of the 30 exposed workers experienced recurrent acute symptoms such as dizziness,

headache, nausea, pain upon inhalation, feelings of suffocation, slight cough and palpitations. Thirteen subjects also reported fatigue during and after work. No significant differences were seen at different exposure levels. Among chronic neurasthenic symptoms, the most obvious differences between control and exposed workers were fatigue, depressed mood, lack of initiative, dizziness, palpitations, thoracic oppression, sleep disturbances and headaches. In psychological tests, the exposed subjects had a greater irregularity of performance on a test of complex reaction time; a greater performance decrement over time in a simple reaction time task and poorer performance in a task of perceptual speed when compared to the non-exposed subjects. There was also a significant difference between the groups when EEG's were ranked as to configuration of alpha activity. Symptoms indicative of polyneuropathy (e.g., restless legs, muscle cramps, diffuse pain in the extremities, paresthesia, numbness) occurred with a higher prevalence in exposed workers. Measurements of peripheral nerve functions indicated differences in exposed workers vs. non-exposed groups. The same group of investigators conducted similar studies in other jet fuel workers and obtained similar results (1926-1928).

In a study on the effects of jet fuel on liver function, Dossing et al. (1925) found no changes in the biochemical indices of liver injury in 91 fuel-filling attendants employed on Danish air force bases for periods up to 31 years (median = 7.6 years). The median jet fuel concentration was 31 mg/m^3 with a range of 1 to 1020 mg/m^3.

64.3.3 Toxicology of JP-4 Components

A brief overview of the toxicology of the major hydrocarbon components of JP-4 (see Table 64-4) are summarized below. The acute toxicity values for these components are presented in Table 64-6.

n-Hexane

Hexane may be the most highly toxic member of the alkanes. When ingested, it causes nausea, vertigo, bronchial and general intestinal irritation and CNS effects. It also presents an acute aspiration hazard. Acute exposure occurs primarily through inhalation. Non-specific symptoms such as vertigo, headache, nausea and vomiting are the first to be manifested. At high concentrations, a narcosis-like state appears as a result of CNS depression. Pre-narcotic symptoms occur at vapor concentrations ranging from 1500-2500 ppm. n-Hexane irritates the eyes and mucous membranes. These effects can be seen after an exposure of 880 ppm for 15 minutes. Skin contact primarily causes fat removal and cutaneous irritation.

Chronic exposure to n-hexane vapors causes peripheral neuropathy. The first clinical sign of neural damage is a feeling of numbness in the toes and fingers. Progression leads to further symmetrical sensory impairment in the distal portions of the extremities and to loss of muscular stretching reflexes. Ultimately, symmetrical muscular weakness develops, chiefly in the distal portion of the extremities.

TABLE 64-6

ACUTE TOXICITY OF COMPONENTS OF JP-4

Component*	Oral LD_{50}	Dermal LD_{50}	LC_{50}
n-hexane	24-49 mL/kg [rat] (1935) 28,710 mg/kg [rat] (1937)	no data	33,000 ppm •4 hr [rat] (1935)
octane	<— no data —>		
dodecane	<— no data —>		
isopentane	no data	no data	1000 mg/L [mouse] (12)
isooctane	<— no data —>		
methylcyclopentane	<— no data —>		
methycyclohexane	2250 mg/kg [rat] (47)	no data	no data
cyclohexane	29,820 mg/kg [rat] (1935)	no data	no data
benzene	3800 mg/kg [rat] (59) 4700 mg/kg [mouse] (47)	no data	10,000 ppm •7 hr [rat] (47)
toluene	5000 mg/kg [rat] (47)	12,124 mg/kg [rabbit] (47)	5320 ppm •8 hr [mouse] (47)
xylenes	4300 mg/kg [rat] (47)	no data	5000 ppm •4 hr [rat] (47)
ethyl benzene	3500 mg/kg [rat] (47)	5000 mg/kg [rabbit] (59)	no data
trimethylbenzenes	no data	no data	18 mg/m^3 •4 hr [rat] (47)
1-methylnaphthalene	1840 mg/kg [rat] (47)	no data	no data
2-methylnaphthalene	1630 mg/kg [rat] (47)	no data	no data

* See Table 64-1 for component concentrations in sample JP-4 fuel.

Paralysis develops with varying degrees of impaired grasping and walking. This may include muscular atrophy (sensorimotor polyneuropathy). The development of electrophysiological changes parallels the severity of the clinical picture. In the most severe cases, nerve conductivity is neutralized. In some cases, cranial nerve involvement is also observed. After exposure ceases, recovery begins within 6 to 10 months in mild to moderate cases, but may take up to 3 years in serious cases. The threshold level at which neuropathy occurs has not been firmly established but symptoms have been observed in people exposed to concentrations ranging from 10 to 200 ppm for 9-12 months.

In animals, signs of narcosis are seen after mice are exposed to vapor levels of 16,000 ppm for 5 minutes. Death generally occurred at concentrations between 43,800 and 52,000 ppm after 9-119 minutes. The oral LD_{50} is cited as 24 mL/kg for 14-day-old rats and 49 mL/kg for young adult rats.

Long-term inhalation experiments in rats suggest that the first signs of neurotoxicity appear after they are exposed to levels of 200 ppm for 24 weeks. This higher threshold to induce neurotoxicity in animals may be due to differences in metabolism. Specifically, 2-hexanol is the chief metabolite in animals, while 2,5-hexanedione which is neurotoxic, predominates in man. Chronic topical application of a solvent containing 35.2% n-hexane caused axonal swelling and myelin degeneration in chicks. No clinical signs were seen. Dosage was 1 g/kg/day for 64 days. In rabbits, topical application of 0.5 mL/day for up to 10 days caused redness, irritation and scab formation. N-hexane is neither carcinogenic or teratogenic. One in vivo study in rats that inhaled 150 ppm for 5 days found an increased number of chromosome aberrations in the bone marrow cells. No studies on mutagenicity, reproductive toxicity or carcinogenicity in man were found (12,1930,1935).

Octane

By the oral route, octane may be more toxic than its lower homologues. If it is aspirated into the lungs, it may cause rapid death due to cardiac arrest, respiratory paralysis and asphyxia. The narcotic potency of octane is approximately that of heptane but it does not exhibit the CNS effects seen with hexane or heptane.

In humans, the only reported effects are blistering and burning due to prolonged skin contact.

In animals, octane is a mucous membrane irritant. At high concentrations, it causes narcosis. It is expected that severe exposure in humans will produce the same effects. Mice exposed to vapor levels of 32,000 ppm suffered respiratory arrest after 4 minutes of exposure. Exposure to 12,840 ppm for 185 minutes caused a decreased respiratory rate, followed by death within 24 hours. No narcosis was seen after 48 minutes of exposure to 5350 ppm (12,46,1938).

Dodecane

Dodecane is not highly toxic. The lowest toxic dose for mice is 11 g/kg when administered percutaneously for 22 weeks. Dodecane is a potentiator of skin tumorigenesis by benzo(a)pyrene. It decreased the effective threshold dose by a factor of 10. Dodecane and phenyldodecane applied topically to the progeny of rats treated with benzo(a)pyrene, chrysene or benzo(b)triphenylene on the seventeenth day of gestation produced tumors in offspring. No additional information is available (12,1937).

Isopentane

Isopentane is a CNS depressant. Effects may include exhilaration, dizziness, headache, loss of appetite, nausea, confusion, inability to do fine work, a persistent taste of gasoline and in extreme cases, loss of consciousness. Inhalation of up to 500 ppm appears to have no effect on humans. "Very high" vapor concentrations are irritating to the skin and eyes. Repeated or prolonged skin contact will dry and defat skin resulting in irritation and dermatitis. The LC_{50} in the mouse is estimated to be 1000 mg/L (12).

Iso-octane (2,2,4-trimethylpentane)

The iso-octanes are moderately toxic by the oral route. If aspirated into the lungs of rats, they will cause pulmonary lesions. When injected intramuscularly into rabbits, iso-octane produced hemorrhage, edema, interstitial pneumonitis, abscess formation, thrombosis and fibrosis. Inhalation of 16,000 ppm caused respiratory arrest in mice and 5 minutes exposure to 1000 ppm was highly irritating (1937).

Methylcyclopentane

Methylcyclopentane resembles cyclopentane in its toxicity. Cyclopentane is a CNS depressant. Humans can tolerate 10-15 ppm. In mice, 38 ppm causes loss of reflexes, narcosis and death demonstrating that no safety margin exists. Methylcyclopentane also exhibits no safety margin between the onset of narcosis and death. When applied to guinea pig skin, cyclopentane produced dryness and slight erythema. Methylcyclopentane would be expected to have the same effect (12).

Methylcyclohexane

No systemic poisonings by methylcyclohexane have been reported in man. At high vapor concentrations it causes narcosis in animals and it is expected that it would produce the same effect in humans. The no-effect level is about 300 ppm in primates and 1200 ppm in rabbits. Rabbits did not survive 70 minutes of exposure to 15,227 ppm. Death was preceded by conjunctival congestion, dyspnea, severe convulsions and rapid narcosis. There were no signs of intoxication in rabbits

exposed to 2880 ppm for a total of 90 hours, but slight cellular injury was observed in the liver and kidneys. In primates, lethal concentrations caused mucous secretion, lacrimation, salivation, labored breathing and diarrhea.

In chronic inhalation studies, exposure to 2000 ppm, 6 hours per day, 5 days per week for 2 years produced no tumors in rats, mice, hamsters or dogs. The only significant toxic effect found was renal changes in male rats. These included renal tubular dilation, papillary hyperplasia and medullary mineralization.

Dermal application of the liquid produced local irritation, thickening and ulceration (12,46,54,17,1936).

Cyclohexane

Cyclohexane is a CNS depressant of low toxicity. Symptoms of acute exposure are excitement, loss of equilibrium, stupor and coma. Rarely, death results due to respiratory failure. The anesthesia which is induced is weak and of brief duration but more potent than that caused by hexane. The oral LDLo in rabbits ranges from 5.5 to 6.0 g/kg. Within 1.5 hours the animals exhibited severe diarrhea, widespread vascular damage and collapse. Degenerative lesions were seen in the heart, lung, liver, kidney and brain. A one-hour vapor exposure to 26,752 ppm caused rapid narcosis and tremor and was lethal to all exposed rabbits. In mice, concentrations causing narcosis vary from 14,600 to 122,000 ppm.

Cyclohexane is nominally absorbed through the skin although massive applications (> 180.2 g/kg) to rabbit skin resulted in microscopic changes in the liver and kidneys and caused the death of all animals.

The danger of chronic poisoning is relatively slight because this compound is almost completely eliminated from the body. No toxic changes were seen in rabbits exposed to vapor levels of 434 ppm, 6 hours daily for 50 exposures, but some microscopic changes were seen in the liver and kidneys when the exposure was to 786 ppm for the same period.

In man, no systemic poisonings by cyclohexane have been reported. A vapor level of 300 ppm is somewhat irritating to the eyes and mucous membranes. It has been reported that cyclohexane may potentiate the toxic effects of TOCP but no additional details of this interaction are available (12,17,46,54,1937).

Benzene

The primary effects of benzene inhalation and ingestion are on the central nervous system (54). Benzene is carcinogenic in both animals and man. Several reports have established a relationship between benzene exposure and leukemia. For more information, refer to Chapter 18 of the Installation Restoration Program Toxicology Guide, Volume 1.

Toluene

Toluene is a CNS depressant with a low toxicity. For more information, refer to Chapter 19 of the Installation Restoration Program Toxicology Guide, Volume 1.

Xylenes

Acute exposure to high concentrations of xylene vapors may cause CNS depression. Both the liquid and the vapor are irritating to the eyes, mucous membranes and skin (46). The National Toxicology Program recently reported that there was no evidence of carcinogenicity of mixed xylenes in either mice or rats given daily doses ranging from 250 to 1000 mg/kg by gavage for 2 years (1939).

For more information, refer to Chapter 21 of the Installation Restoration Program Toxicology Guide, Volume 1.

Ethyl Benzene

Ethyl benzene is primarily an irritant to the skin, eyes and upper respiratory tract. Systemic absorption causes CNS depression (46).

For more information, refer to Chapter 20 of the Installation Restoration Program Toxicology Guide, Volume 1.

Trimethylbenzenes

The trimethylbenzenes occur in 3 isomeric forms. The 1,3,5-isomer (mesitylene) and the 1,2,4-isomer (pseudocumene) are toxicologically similar. High vapor concentrations (5000-9000 ppm) cause CNS depression in animals. Loss of reflexes was seen in mice exposed to 8130-9140 ppm of the 1,2,4-isomer or 8130 ppm of the 1,3,5-isomer. Rats exposed to 1700 ppm of an isomeric mixture for 10-21 days had no adverse effects or fatalities.

The fatal intraperitoneal dose of the 1,2,4-isomer for the guinea pig is 1.788 g/kg, while the fatal dose of the 1,3,5-isomer by the same route is 1.5-2 g/kg for the rat. For the 1,2,3-isomer, an oral LDLo of 5000 mg/kg has been reported in the rat. Trimethylbenzene liquid is a primary skin irritant. Deposition into the lungs causes pneumonitis at the site of contact.

The only report of human exposure described symptoms of nervousness, tension, anxiety, asthmatic bronchitis, hypochromic anemia and changes in the coagulability of the blood. Vapor concentrations ranged from 10-60 ppm. Exposure was to a mixture containing 30% of the 1,3,5-isomer and 50% of the 1,2,4-isomer (2,12).

Methylnaphthalene

The only adverse effects of methylnaphthalene reported in man are skin irritation and photosensitization (17). Oral LD_{50} values of 1840

mg/kg and 1630 mg/kg have been reported for 1-methylnaphthalene and 2-methylnaphthalene, respectively, in the rat (47).

JP-4 Additives

Additives used in JP-4 are listed in Table 64-3. Little toxicological data were found regarding these compounds. The information which was available is outlined below:

6-t-butyl-2,4-dimethylphenol
An oral LD_{50} of 530 mg/kg in the rat was reported (47).

N,N'-di-sec-butyl-p-phenylenediamine
A percutaneous LD_{50} of 5000 mg/kg was reported in guinea pigs. The lowest lethal oral dose reported in rats is 200 mg/kg. The LDLo in rats is 600 mg/m^3 for 6 hours (1937).

N,N-dimethylformamide
An oral LD_{50} of 2800 mg/kg in the rat and 3750 mg/kg in the mouse have been reported. In humans, N,N-dimethylformamide is irritating to the eyes, skin and mucous membranes. Case reports have indicated that the liver is the main target organ following acute and chronic exposure to dimethylformamide. One of the earliest manifestations of excessive exposure is ethanol intolerance followed at higher exposure levels by symptoms of nausea, vomiting and abdominal pain (1937,2316).

64.3.4 Levels of Concern

No criteria or standards specific for JP-4 were located. EPA (2012) does list a criterion for oil and grease which requires domestic water supplies to be virtually free from oil and grease, particularly with regard to taste and odor.

OSHA (298) has set a standard of 500 ppm for petroleum distillates (naphtha). The ACGIH (3) recommends a threshold limit value of 300 ppm for gasoline, with a short-term exposure limit of 500 ppm.

64.3.5 Hazard Assessment

Toxicological data located for JP-4 are limited. No data are currently available regarding the carcinogenicity of JP-4 but a study is in progress (1936). Shale- and petroleum-derived JP-4 have been tested in F344 rats and C57BL/6 mice at vapor concentrations of 5000 or 10,000 mg/m^3. Histopathology is now in progress.

Mutagenicity tests in bacterial and mammalian test systems are negative as are dominant-lethal tests for both rats and mice (1813). No data on reproductive toxicity were located.

Acute eye irritation studies with undiluted shale- or petroleum-derived JP-4 have produced negative responses in rabbits (1930). Skin irritation studies with the same test samples indicated no effect at 24 hours; by 72 hours, mild erythema was induced with both samples and mild edema with the shale-derived sample (1930). Skin sensitization studies in guinea pigs were negative for the petroleum-derived sample but suggested mild to moderate sensitization with the shale-derived JP-4 sample (1930).

Long-term, continuous inhalation exposure to petroleum-derived JP-4 at levels up to 1000 mg/m^3 for 90 days produced fatty changes in the liver of mice and rats and kidney damage in rats; no significant effects were noted in dogs (1933). Intermittent exposure to higher concentrations (5000 mg/m^3) produced increases in organ weights of the kidney, liver, lung and spleen of rats but no histopathologic changes (1933).

Human exposure to petroleum distillates is known to cause headache, nausea, mental confusion, CNS depression and respiratory tract irritation. Aspiration can produce chemical pneumonitis (1932).

Neurological effects have been linked to chronic exposure to jet fuels in aircraft factory workers. Average exposure concentrations ranged from 128 to 432 mg/m^3 for 2 to 32 years. Performance decrement and polyneuropathy correlated with exposure. Other symptoms included depression, irritability, lassitude, disturbed sleep rhythm and changes in conduction velocities in peripheral motor nerves (1929).

64.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of JP-4 fuel in soil and water requires collection of a representative field sample and laboratory analysis for the specific major components attributed to JP-4; however, the relative concentrations of the constituents, and even the constituents themselves, will vary with time and distance from the site of initial contamination due to weathering. The major component categories in JP-4 fuel have been identified as the following:

n-alkanes
branched alkanes
cycloalkanes
benzenes and alkylbenzenes
naphthalenes

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques has been used to identify the principal components in JP-4 fuel (ESL-TR-81-54, SRI). Fuel samples, and probably any samples collected in the field which are primarily organic in nature, require the separation (prior to GC or GC/MS analysis) of the aliphatic, monoaromatic and polycyclic aromatic hydrocarbons fractions using liquid solid column chromatography; the

various column eluates, with or without dilution in carbon disulfide, are then analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (i.e., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet extraction or sonication methods (Standard Methods). An aliquot of the sample extract, with or without concentration, is then analyzed by GC or GC/MS. (Sampling and Analysis Considerations for some specific components in JP-4, i.e., benzene, toluene, xylenes, ethyl benzene and naphthalene, have been addressed in Volume 1.)

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component, but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorotricluoroethane). The "oil and grease" content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; extraction methods are those described above for aqueous and soil samples.

A detection limit for JP-4 was not determined; the detection limit for specific components is expected to be in the range of µg/L for aqueous samples and µg/g for non-aqueous samples.

COMMON SYNONYMS:	CAS REG. NO.:	AIR W/V CONVERSION FACTORS at 25°C (2228)
Petrol Motor spirits Benzin	8006-61-9 NIOSH NO.: LX3300000	4.5 mg/m³ = 1 ppm 0.222 ppm = 1 mg/m³
APPROXIMATE COMPOSITION: n-alkanes 15-17% cycloalkanes 3-5% benzenes and alkylbenzenes 20-49% branched alkanes 28-36% olefins 1-11% naphthalenes ≤1%		

REACTIVITY	Several sources indicate that strong acids or strong oxidizers are incompatible with gasoline and that vigorous reactions, ignition, and/or explosion may be expected. The NFPA specifically notes such events when chlorine, fluorine, or magnesium perchlorate are mixed with hydrocarbons. Gasoline is considered a miscellaneous combustible or flammable material for compatibility classification purposes. Such substances typically evolve heat, fire, and toxic or flammable gases in reactions with oxidizing mineral acids, alkali or alkaline earth elemental metals, nitrides, organic peroxides or hydroperoxides, or strong oxidizing agents. Reactions with explosive materials may result in an explosion, while those with strong reducing agents may evolve heat and flammable gases. Non-oxidizing mineral acids generally evolve heat and innocuous gases (51,505,507,511).

PHYSICO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(60)
	• Color: colorless to pale brown or pink	(60)
	• Odor: characteristic	(54,60)
	• Odor Threshold: 0.25 ppm	(60)
	• Liquid Density (g/ml at 20°C): 0.7321	(60)
	• Freezing/Melting Point (°C): no data	(60)
	• Boiling Point (°C): 60-199, 38-204	(60,39)
	• Flash Point (°C): -38 to -46, closed cup, depending on grade	(60,506, 507)
	• Flammable Limits in Air, % by Volume: 1.2-1.5% to 7.1-7.6% depending on grade	(60,504, 506,507)
	• Autoignition Temperature (°C): 257-471, varies with grade	(51,60,507, 510,513)
	• Vapor Pressure (mm Hg at 38°C): 263-675	(1932)
	• Saturated Concentration in Air (mg/m³ at 20°C): no data	( )
	• Solubility in Water (mg/L at 20°C): insoluble	(60)
	• Viscosity (cp at 20°C): 0.451	(60)
	• Surface Tension (dyne/cm at 20°C): 19-23	(60)

PHYSICO-CHEMICAL DATA (continued)	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 2.13-4.87 (*) • Soil Adsorption Coefficient, K_{oc}: 65-36,000 (*) • Henry's Law Constant ($atm \cdot m^3/mol$ at 20°C): 4.8×10^{-4} - 3.3 (*) • Bioconcentration Factor: no data ( )

PERSISTENCE IN THE SOIL-WATER SYSTEM	Gasoline hydrocarbons are expected to be relatively mobile and moderately persistent in most soil systems. Persistence in deep soils and ground water may be higher. Volatilization, photooxidation and biodegradation are important fate processes. Surface spills are expected to be weathered by evaporation and photooxidation. Downward migration of weathered surface spills and sub-surface discharges represent a potential threat to underlying ground water. Biodegradation of gasoline hydrocarbons is expected to be significant under environmental conditions favorable to microbial oxidation; naturally-occurring, hydrocarbon-degrading microorganisms have been isolated from polluted soils and, to a lesser extent, non-polluted soils.

PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water system is the migration of gasoline to ground water drinking water supplies from leaking underground storage tanks or large spills. The use of this water may cause inhalation exposures as well as ingestion and dermal exposures. Vapors from leaked or spilled gasoline may diffuse through soil and migrate into structures, resulting in inhalation exposures.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (54): Gasoline vapor is a CNS depressant. Low vapor levels may produce flushing, staggering gait, slurred speech and mental confusion. High vapor levels may cause coma and death from respiratory failure. Ingestion and aspiration may cause chemical pneumonitis, pulmonary edema and hemorrhage. Gasoline is irritating to the skin, conjunctiva and mucous membranes. Prolonged contact may defat the skin and cause dermatitis. Certain individuals may develop hypersensitivity.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral 13,600 [rat] (1924) skin -- no data LCLo (mg/m^3) inhalation [mammal] (51) 135,000•5 min

*Range of values for representative hydrocarbons from major component classes (See Table 65-3).

HEALTH HAZARD DATA (continued)	Long-Term Effects: Kidney injury; lead toxicity with leaded gas
	Pregnancy/Neonate Data: Negative
	Mutation Data: Negative
	Carcinogenicity Classification: IARC-none assigned; NTP - none assigned

HANDLING PRECAUTIONS (45,52)	Handle only with adequate ventilation. There are no specific respirator guidelines for gasolines. A chemical cartridge respirator is recommended • Chemical goggles if there is probability of eye contact • Nitrile, PVA or other protective clothing to prevent prolonged or repeated skin contact with the liquid.

EMERGENCY FIRST AID TREATMENT (1311,60, 1932)	Ingestion: Do not induce vomiting. Get medical attention • Inhalation: Move victim to fresh air. Give artificial respiration if necessary. Get medical attention • Skin: Remove contaminated clothing. Wash with water for one hour. Get medical attention • Eye: Wash with copious amounts of water. Get medical attention.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): none established
- AFOSH PEL (8-hr TWA): none established

Criteria
- NIOSH IDLH (30-min): none established
- ACGIH TLV® (8-hr TWA): 300 ppm
- ACGIH STEL (15-min): 500 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; automotive gasoline is not a priority pollutant.
- Aquatic Life
  No criterion established; automotive gasoline is not a priority pollutant.

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;
- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;
- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Toxic Substances Control Act (TSCA)
  Manufacturers and processors of the C9 aromatic hydrocarbon fraction must test it for neurotoxicity, mutagenicity, developmental toxicity, reproductive effects and oncogenicity. The C9 fraction is obtained from the reforming of crude petroleum. It consists of ethyltoluenes and trimethylbenzenes (1988). Testing will be conducted by the American Petroleum Institute. Interim reports must be submitted at 6-month intervals (1987).

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated gasoline as a hazardous material which is subject to requirements for packaging, labeling and transportation (305).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Petroleum and coal tar distillates with flash points below 21°C are classified as flammable substances and are subject to packaging and labeling regulations. Because of the variable composition of other petroleum and coal tar distillates (excluding those used as motor fuels) they are considered preparations and their labeling shall be done in accordance with the procedures outlined in the Directive Relating to the Classification Packaging and Labeling of Dangerous Preparations (solvents).

EEC Directives - Proposed

Proposal for a Council Directive on the Dumping of Waste at Sea (1793)
EEC has proposed that the dumping of crude oil, petroleum hydrocarbons, lubricants and hydraulic fluids at sea be prohibited.

65.1 MAJOR USES AND COMPOSITION

65.1.1 Major Uses

Gasoline is a volatile mixture of flammable liquid hydrocarbons derived chiefly from crude petroleum and used principally as a fuel for internal combustion engines. Consumption of gasoline by motor vehicles in this country was approximately 103 billion gallons in 1983, down from a peak consumption of 116 billion gallons in 1978 (1409).

65.1.2 Composition

Automotive gasoline is composed of several hundred hydrocarbons in the range of C_4 to C_{11} and with boiling points from approximately 30°C to 210°C. General composition expressed as percent weight by compound category has been reported to be: 49% to 62% aliphatic hydrocarbons (28-36% branched alkanes, 15-17% n-alkanes, and 3-5% cycloalkanes), 1% to 11% olefinic hydrocarbons, 20% to 49% benzenes and alkylbenzenes and up to to 1% naphthalenes (2320,1843,1849).

As noted with JP-4 (Chapter 64), the concentrations of specific hydrocarbons in different gasoline samples are highly variable and are expected to become even more variable as the availability of leaded gasoline is reduced. For example, as reforming severity was adjusted to achieve the required increase in octane levels of unleaded gasoline pools, average aromatic content increased from 22% in 1970 to 27% in 1980 and 1984; as leaded gasoline is phased out, the aromatic content will increase further to 35%. Olefin content also increased from 8% in 1980 to 11% in 1984 (2319).

The individual components of gasoline have been characterized by several authors (2320,2311,1843). Table 65-1 summarizes the available hydrocarbon composition data for various gasolines. As discussed in Chapter 64 (JP-4), petroleum-derived distillates may also contain many non-hydrocarbon components. These may become major concerns in heavy distillates and residues but are much less important in light distillates such as automotive gasoline where only trace quantities of sulfur-, nitrogen-, and oxygen-containing compounds have been detected. Large variations in trace element concentrations were reported but no quantitative data were available (1843).

Automotive gasoline also contains a number of additives used as octane improvers, antioxidants, metal deactivators, corrosions or icing inhibitors, detergents or demulsifiers. A list of some of the chemical classes and specific chemicals that may be used for these purposes is provided in Table 65-2.

TABLE 65-1

COMPOSITION DATA (% W/W) FOR VARIOUS GASOLINES

Hydrocarbon	Leaded[a]	Unleaded[a]	Super Unleaded[a]	(Vol. %) Gasoline[b]	Premium Gasoline[c]	Regular Gasoline[c]
• n-Alkanes				11.4		
C_4					4.8	7.0
C_5	2.2	3.0	1.9		3.4	4.5
C_6	11.0	11.6	12.9		2.0	3.3
C_7	2.3	1.2	0.2		1.2	2.0
C_8					1.3	
C_9	0.8	0.7	0.4			
C_{10}-C_{13}	0.6	0.8	0.2			
• Branched Alkanes						
C_4	1.6	2.2	1.2	1.1	0.7	
C_5	17.3	15.1	8.6	10.3	8.5	9.3
C_6	9.7	8.0	6.2	9.0	4.6	7.6
C_7	2.7	1.9	1.4	4.8	8.3	7.7
C_8	2.0	1.8	8.7	16.7	15.1	11.4
C_9	2.7	2.1	1.2	2.0	1.4	
C_{10}-C_{13}	0.5	1.0	1.1	2.6		
• Cycloalkanes						
C_5				0.2		
C_6	3.9	3.0	3.0	1.0	2.9	1.8
C_7	1.0	1.4	0.2	1.1	1.2	1.0
C_8	0.6	0.6	0.2	0.7		
Others				1.6	7.5	
• Olefins						
C_4				0.9		
C_5				1.3	3.3	3.3
C_6	1.1	1.8	1.0	1.4	0.8	1.5
Others				5.3	7.5	2.5
• Aromatics						
Benzene	3.9	3.2	4.4	1.7	0.9	1.5
Toluene	4.5	4.8	6.0	4.0	6.5	5.9
Xylenes	5.6	6.6	7.4	9.8	8.8	5.9
Ethylbenzene	1.2	1.4	1.4		1.3	1.3
C_9-benzenes	3.4	4.2	5.7	7.7	11.3	3.2
C_4-benzenes	5.6	7.6	5.8	2.1		
Naphthalenes				0.7		
Others	2.0	2.7	1.6	2.27	5.2	
• Unknowns	7.8	6.6	13.8			

[a] Reference 2320
[b] Reference 2311
[c] Reference 1843; average data for 15 premium and 36 regular gasoline samples.

TABLE 65-2

GASOLINE ADDITIVES[a]

Anti-Knock Compounds (leaded gasoline)

- Tetraethyl lead (TEL)[b]
- Tetramethyl lead (TML)
- Methylcyclopentadienyl manganese tricarbonyl (MMT)

Lead Scavenging Agents

- Ethylene dibromide (EDB)[b]
- 1,2-Dichloroethane

Octane Enhancers (unleaded gasoline)

- Methyl t-butyl ether (MTBE)
- t-Butyl alcohol (TBA)
- Ethanol
- Methanol

Antioxidants

- N,N'-Dialkylphenylenediamines
- 2,6-Dialkyl and 2,4,6-trialkylphenols
- Butylated methyl, ethyl and dimethyl phenols
- Triethylene tetramine di(monononylphenolate)

Metal Deactivators

- N,N'-Disalicylidene-1,2-ethanediamine
- N,N'-Disalicylidene-propanediamine
- N,N'-Disalicylidene-cyclohexanediamine
- Disalicylidene-N-methyl-dipropylene-triamine

Ignition Controllers

- Tri-o-cresylphosphate (TOCP)[b]

Icing Inhibitors

- Isopropyl alcohol

Detergents/Dispersants

- Alkylamine phosphates
- Poly-isobutene amines
- Long chain alkyl phenols
- Long chain alcohols
- Long chain carboxylic acids
- Long chain amines

Continued

TABLE 65-2 - Continued

GASOLINE ADDITIVES[a]

Corrosion Inhibitors

MIL-I-25017/QPL-25017[c]
Carboxylic acids
Phosphoric acids
Sulfonic acids

[a] References 1409,2325,2326,2327,2328,1847
[b] Compounds addressed in other chapters of IRP Toxicology Guide
[c] As cited in 2328

65.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

In this chapter, the discussions of the environmental behavior of gasoline will be limited to a discussion of its major components; the environmental behavior of the trace elements and the many diverse additives will not specifically be addressed. Many of the hydrocarbons characteristics of gasoline have been addressed previously in the more extensive environmental fate section of the JP-4 chapter since these hydrocarbons are common to both petroleum fuels. The general discussions of aliphatic and aromatic hydrocarbons and their behavior in soil/ground-water systems will not be repeated here; the reader is referred to the relevant sections of Chapter 64.

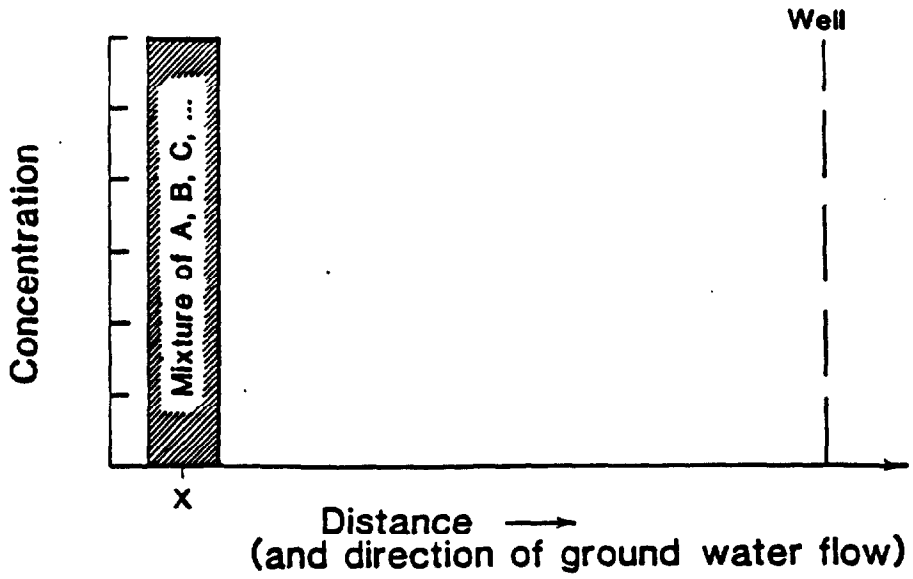
Transport and transformation of individual gasoline constituents will depend on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly (in the percolating ground waters), be sorbed less strongly on the soils (thus being transported more rapidly), and may be more or less susceptible to degradation by chemical or biological action. Thus, as shown in Figure 65-1, the relative concentrations of the constituents of the fuel will vary with time and distance from the site of contamination. This effect is called "weathering." (This term is also used to describe the changes to oil following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation are all involved.)

65.2.1 Transport in Soil/Ground-water Systems

65.2.1.1 Equilibrium Partitioning Model

In general, soil/ground-water transport pathways for low concentrations of pollutants in soil can be assessed by using an equilibrium partitioning model. For the purposes of assessing the environmental transport of automotive gasoline, a group of specific

a. Initial "Spike" profile after release to environment at Point x. A, B, and C are chemicals in the mixture (e.g., gasoline).



b. Profiles of A, B, C after transport with flowing ground water for some time.

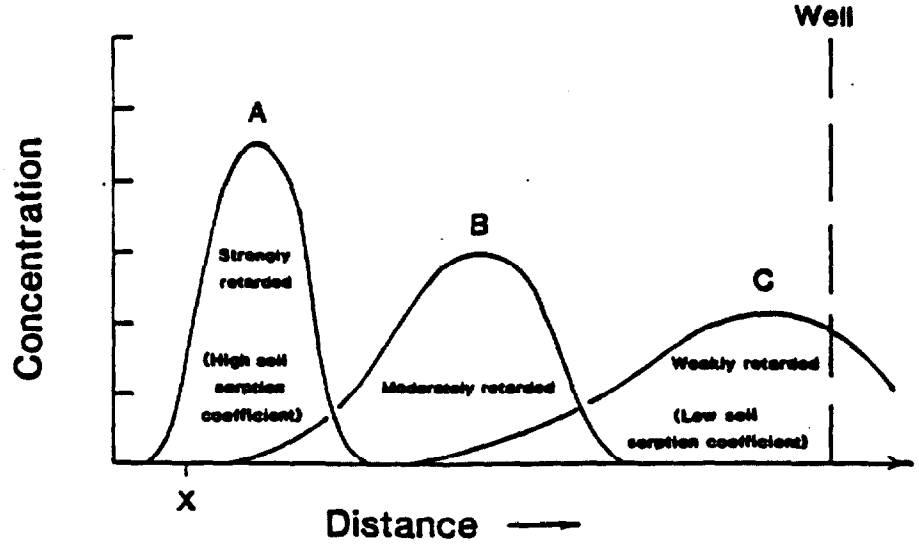


FIGURE 65-1

SCHEMATIC SHOWING DIFFERING DEGREES OF RETARDATION OF MIXTURE CHEMICALS TRANSPORTED BY FLOWING GROUND WATER

NOTE: Effects of degradation are not shown; but they would have the effect of lowering peak heights and area under each curve.

hydrocarbons was selected from the dominant hydrocarbon classes, i.e., alkanes, cycloalkanes, and aromatics. These specific compounds were chosen on the basis of their relative concentrations, and were intended to span the boiling point average of the gasoline hydrocarbons. Table 65-3 identifies the selected hydrocarbons and presents the predicted partitioning of low soil concentrations of those hydrocarbons among soil particles, soil water, and soil air. The portions associated with the water and air phases of the soil are expected to have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil indicate that sorption is expected to be an important process for all the dominant hydrocarbon categories. Partitioning to the soil-vapor phase is also expected to be important for the lower molecular weight aliphatic hydrocarbons (C_4-C_8) which are characterized by high vapor pressure and low water solubility. The alkyl benzenes have higher water solubilities and transport with infiltrating water may be important for these compounds; volatilization, on the other hand, may be less important. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a significant percent of both aliphatic (particularly less than C_7) and aromatic hydrocarbons is predicted to be present in the soil-water phase and available for transport with flowing ground water.

In interpreting these results, it must be remembered that this model is valid only for low soil concentrations (below aqueous solubility) of the components. Large releases of gasoline (spills, leaking underground storage tanks) may exceed the sorptive capacity of the soil, thereby filling the pore spaces of the soil with the fuel. In this situation, the hydrocarbon mixture would move as a bulk fluid and the equilibrium partitioning model would not be applicable.

Overall, ground water underlying soil contaminated with gasoline hydrocarbons is expected to be vulnerable to contamination by at least some of these components. The type of spill (surface vs. sub-surface) is of particular importance, since volatilization from the surface is expected to be a significant removal process for low molecular weight aliphatics. At this point, it should be mentioned that environmental fate/exposure/toxicology chapters for several of the components in Table 65-3 were included in Volume 1 of the IRP Toxicology Guide. The gasoline components addressed in Volume 1 include: benzene, toluene, xylenes, ethyl benzene, and naphthalene. Three major gasoline additives - TOCP, tetraethyl lead and ethylene dibromide were addressed in Volume 2 of the IRP Toxicology Guide, while ethylene dichloride was addressed in Volume 1.

65.2.1.2 Transport Studies

Hundreds of thousands of underground gasoline storage tanks are currently used at service stations, commercial locations, residences, and petroleum depots; and almost all the gasoline used for transportation purposes in the U.S. is stored underground at least once

TABLE 65-3

EQUILIBRIUM PARTIONING OF SELECT GASOLINE HYDROCARBONS IN MODEL ENVIRONMENTS[a]

COMPOUND	Log K_{ow}	K_{oc}[b]	H[c]	UNSATURATED TOPSOIL (%)			SATURATED DEEP SOIL[d] (%)	
				Soil	Water	Air	Soil	Water
Hexane	3.90 (e)	3,830	1.68	77.5	0.1	22.4	94.1	5.9
Isopentane	3.37 (f)	900	1.36	50.3	0.3	49.4	79.1	20.9
Methylpentane	3.90 (e)	3,830	1.69	77.6	0.1	22.3	94.1	5.9
Trimethylpentane	4.87 (f)	36,000	1.9-3.3	94.7	0.01	5.3	99.3	0.7
Cyclohexane	3.44 (e)	1,330	0.18	91.6	0.4	8.0	84.8	15.2
Benzene	2.13 (e)	65	5.43×10^{-3}	88.1	7.1	4.8	21.4	78.6
Toluene	2.69 (g)	240	6.6×10^{-3}	96.5	1.9	1.6	52.1	47.9
Xylenes	3.16 (e)	700	7×10^{-3}	98.8	0.7	0.5	74.4	25.6
Trimethylbenzenes	3.65 (h)	2,150	5×10^{-3}	99.6	0.2	0.2	90.0	10.0
Naphthalene	3.30 (e)	962	4.82×10^{-4}	99.4	0.5	0.03	80.2	19.8

[a]Calculations based on Mackay's equilibrium partioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

[b]Reference 652.

[c]Taken from Reference 74 unless otherwise specified. Units equal $atm \cdot m^3/mol$.

[d]Used sorption coefficient $K_p = 0.001 \times K_{oc}$.

[e]Reference 29.

[f]Arthur D. Little, Inc., estimate according to equations provided in Reference 31.

[g]Reference 10.

[h]Reference 31.

before its intended use. Since it is possible for one gallon of gasoline containing 1% benzene by volume to contaminate 10 million liters (2.69 million gallons) of water to the drinking standard of 1 ppb, underground gasoline storage tanks are a major environmental concern (2320).

Many authors have documented ground-water contamination as a result of hydrocarbon spills. For example, Osgood (2322) reported over 200 hydrocarbon spills in Pennsylvania in a 2.5-year period; in that time, 14 public water supplies were polluted or threatened, 104 wells seriously damaged, and one spill resulted in the subsurface discharge of over 270,000 gallons of gasoline. Matis (2323) reported over 60 cases of ground-water contamination in Maryland from 1969 to 1970. Drinking water contamination caused by gasoline migration and subsequent penetration of a subsurface water supply line has also been reported (2321); the most serious contaminant was ethylene dibromide (EDB), a gasoline additive. EDB has been reported to be present in leaded gasoline in sufficient quantities to constitute a threat to ground water following a gasoline discharge to the environment (2320).

Due to the extensive use of gasoline and its potential for environmental release during use, storage or transport, several groups have addressed its fate. The fate of gasoline in the soil environment is basically a function of the solubility, volatility, sorption, and degradation of its major components. The relative importance of each of these processes is influenced by the type of contamination (e.g., surface spill *vs*. underground release, major *vs*. minor quantity), soil type (e.g., organic content, previous history of contamination), and environmental conditions (e.g., pH, temperature, oxygen content).

Transport processes have been shown to be more significant than transformation processes in determining the initial fate of petroleum hydrocarbons released to soil/ground-water systems (1845,1848,1846). For gasoline released to surface soils or waters, transport to the atmosphere through volatilization is expected to be the primary fate pathway; subsequent atmospheric photolysis is expected to be rapid (1845). Spain *et al*. (1846) demonstrated that compounds having up to nine carbons are weathered almost exclusively by evaporation; larger compounds were weathered primarily by evaporation and biodegradation. Composition data for gasoline vapor indicate that C_4-C_6 aliphatic hydrocarbons are rapidly volatilized (2324).

Under conditions of limited volatilization (low temperatures, subsurface release or concentrated spill) downward migration into the soil and to the ground water may be important. Several authors (1811,2243,2252,2329) have reported that oil substances released in significant quantities to soils result in a separate organic phase which moves downward through the unsaturated zone to the less permeable layer, the soil/ground-water boundary, where they tend to accumulate and spread horizontally.

Some residual gasoline is left behind in the area through which the gasoline has percolated; the residue tends to be more concentrated in fine sand than in the coarser materials (2329). Solubilized gasoline components may leach from residually contaminated soils for long periods of time. Induced soil venting has been demonstrated to be a rapid and efficient method for removal of gasoline trapped in soils following a spill or leak (2320). The importance of subsurface volatilization of gasoline components has also been demonstrated in an article by Yaniga (2330). Volatilization of gasoline components from residual contamination and contamination accumulated at the ground-water interface resulted in detection of gasoline vapors in nearby basements.

The organic layer floating on the ground water is carried in the general direction of ground water flow. At the oil-water interface, some hydrocarbons are leached according to their aqueous solubility. As discussed in Chapter 64, the pollution caused by the hydrocarbon phase is much less extensive (10s-100s of meters) than pollution caused by hydrocarbons dissolved in ground water (100s-1000s of meters) (1811). Furthermore, the pattern of migration of the hydrocarbon phase may be very different from that of the ground water. Due to fluctuations in ground-water elevation over time, the organic layer on top of the aquifer may be transported into several zones where the components occur in the gaseous phase (able to diffuse in all directions, including upward), liquid phase (adsorbed onto rock particles or sealed under water) or dissolved/emulsified in water (1811,2329).

Migration through soils may be retarded to a minor extent by sorption. Migration is expected to be fastest through previously contaminated soils where the sorptive sites may be unavailable; on the other hand, soil-water content increases sorption and slows migration of hydrocarbons. In fissured rock, the migration of hydrocarbons is much less uniform than in porous soils. Preferential spreading through crevices, sometimes changing the direction of flow, may occur. Determination of the potential ground-water contamination in fissured rock is thus very difficult (1811).

The water-soluble portion of gasoline was shown to be almost entirely aromatic (87-94%) even though the product itself was almost 50% aliphatic; the aliphatic hydrocarbons either volatilized or were essentially not water-soluble (1849). In deep, saturated soils with no soil air, some low molecular weight aliphatics may be dissolved in and transported with ground water; however, the light aromatics represent the greatest threat of contamination to ground-water supplies.

In summary, the physical distribution of gasoline contamination affects its impact on, and removal from, the soil environment. Lateral spreading along the surface increases the initial contaminated area while facilitating evaporative removal of the low molecular weight hydrocarbons. Subsurface release or vertical penetration mediated by

gravitation and capillary forces decreases evaporation, reduces the importance of some transformation pathways (see below), and may lead to ground-water contamination.

65.2.2 Transformation Processes in Soil/Ground-water Systems

65.2.2.1 Chemical Transformation

No data were available on chemical transformation of gasoline in the environment. However, as discussed in Chapter 64, photooxidation has been reported to play a significant role in the chemical degradation of some petroleum hydrocarbons in the sunlit environment (1845,1848,2252,2259). Alkanes, benzenes, and mono-substituted benzenes have been shown to be relatively resistant to photolysis in aqueous systems; xylenes photolyzed slowly while trisubstituted benzenes and naphthalenes photolyzed at rates competitive with volatilization (1845). Penetration of oil below the soil surface limits exposure to solar radiation while extensive lateral spreading of oil over impermeable or rocky surfaces may promote substantial photooxidative degradation. The oxygenated products of photooxidation are generally more water-soluble than the parent hydrocarbons and are thus more likely to be leached from soil; enhanced toxicity of the oxygenated hydrocarbons has also been observed (2248,2252).

65.2.2.2 Biological Degradation

Natural ecosystems have considerable exposure to petroleum hydrocarbons from natural emissions, accidental contamination through oil spills and storage tank leaks, and deliberate application to land in disposal activities such as land-farming waste; therefore, their biodegradation is of environmental importance. Numerous authors have observed the biodegradation of petroleum hydrocarbons, and several extensive reviews and reports are available (1846,2252,2255,2249,2253). An extensive and diverse group of petroleum hydrocarbon-degrading bacteria and fungi are widely distributed in the environment. The reader is referred to Chapter 64 for a more detailed summary of the biodegradation of petroleum hydrocarbons.

The qualitative hydrocarbon content of petroleum mixtures largely determines their degradability. In general, microorganisms exhibit decreasing ability to degrade aliphatic hydrocarbons with increasing chain length. n-Alkanes are considered more easily biodegraded than branched or cyclic alkanes; aromatics are generally more rapidly biodegraded than alkanes. The composition of gasoline suggests that most of the aromatic species will be highly biodegradable, and many of the aliphatic species that are not volatilized will be moderately biodegradable. In a study of the biodegradation of individual components of gasoline using microorganisms isolated from ground water, the aliphatics and aromatics were shown to be sources of carbon for Nocardia and Pseudomonas cultures, respectively (2331). Very few of the remaining components supported bacterial growth; co-oxidation was suggested as a possible mechanism for removal of non-growth components.

Although the microbiota of most non-contaminated soils include many naturally occurring hydrocarbon-degrading populations, the addition of petroleum selectively enriches that sector able to adapt and utilize the new substrate. Other environmental factors shown to have a major effect on biodegradability are availability of oxygen and moderate temperatures.

In summary, biodegradation of the petroleum hydrocarbons comprising automotive gasolines is expected to be rapid under conditions favorable for microbial activity and when fuel components are freely available to the microorganisms. Degradation may be limited and/or slow in environments with few degrading organisms, low pH, low temperatures, and high salinity (e.g., arctic environments). It should be mentioned that Walker et al. (2257) state that even under optimum conditions, total and complete biodegradation is not expected to occur except possibly over an extremely long time period.

65.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that the major components of gasoline are highly volatile but vary in their potential for bioaccumulation and tendency to sorb to soil. They range from moderately to strongly sorbed to soil, and their bioaccumulation potential ranges from low to high. The variability in the properties of the components suggests they may have somewhat different potential exposure pathways.

Spills of gasoline would result in the evaporative loss of the more highly volatile components leaving those of lesser volatility in the soil. The fraction remaining in the soil is expected to be relatively mobile and will be carried by gravity to the saturated zone of the soil. There, the more soluble components will dissolve into the ground water or form emulsions with it. These components are primarily aromatic and lower molecular weight aliphatic compounds; in one study using unleaded gasoline, approximately 95% of the water soluble fraction was benzene and substituted benzenes (2318). The insoluble fraction of gasoline floats as a separate phase on top of the water table. The movement of gasoline dissolved in ground water is especially important because of its relatively high solubility (173-200 mg/L (2287,2297)). Furthermore, the movement of dissolved hydrocarbons in ground water is much greater than that of the separate liquid phase, reaching distances of hundreds to thousands of meters compared to tens of meters for the movement of the separate phase. In the presence of cracks and fissures, however, the flow of the separate hydrocarbon phase is greatly enhanced.

The movement of gasoline in ground water may contaminate drinking water supplies, resulting in ingestion exposures. Ground-water discharges to surface water or the movement of contaminated soil particles to surface water drinking water supplies may also result in ingestion exposure and in dermal exposures from the recreational use of these waters. The potential also exists for the uptake of some gasoline

components (e.g., naphthalene and methylnaphthalene) by fish and domestic animals, which may also result in human exposures due to the bioconcentration of these components.

Ground water contaminated with gasoline can lead to inhalation exposures in homes using this water. In one study of homes in Maine (2313), concentrations of total benzene, toluene and xylene measured in air of the closed bathrooms while hot showers were running were 2.05, 3.15, and 30 ppm in homes with 6, 3, and 20 ppm, respectively, of total hydrocarbons in their water. In the two homes with the highest total concentrations, xylene accounted for roughly 63% of the concentration in air, toluene 29-32% and benzene 5-9%; in the other home 95% was benzene, the rest toluene. The author of this study suggested that odor may be a sensitive indicator of gasoline contamination in water. In the houses with high hydrocarbon contamination, an offensive odor was noticeable, especially during sampling (2313). Even though no benzene, toluene or xylene was detected in the air of three homes with less than 0.5 ppm total hydrocarbons in their water, in two of these homes gasoline odors were present in the bathroom. However, a modelling study (2314) indicates that petroleum-based pollutants (benzene, toluene, xylene) present in water at 5 to 50 ppb--levels below detectable taste or odor threshholds--may result in peak air concentrations that cause mucous membrane irritation.

Volatilization of gasoline hydrocarbons in soil is another potential source of human exposure. This exposure pathway is likely to be more significant for gasoline than other petroleum products because of its high volatility. Exposures may be more intensive when the soil is contaminated directly from leaking underground storage tanks and pipes, rather than from surface spills. In such cases, the more volatile components do not have an opportunity to evaporate before penetrating the soil. Once in the soil, the hydrocarbons evaporate saturating the air in the soil pores, and diffusing in all directions including upward to the soil surface. The vapors may diffuse into the basement of homes or other structures in the area resulting in inhalation exposures to the building's occupants.

65.2.4 Other Sources of Human Exposure

Data on ambient concentrations of gasoline in air and water as well as food and drinking water are not readily available in the literature. Exposure information on specific components may be found in other chapters of the IRP Toxicology Guide.

The volatile nature of automotive gasoline suggests that inhalation may represent a significant exposure pathway. The average concentrations of automotive gasoline to which residents of communities near bulk terminals, bulk plants, and service stations (employing no special controls) are exposed have been estimated as 1.41, 0.073, 0.026 ppm, respectively (2311). It should be emphasized that these values are averaged over a lifetime and in all cases the concentrations are estimated from emission rates. Exposure to service station employees and individuals filling their tanks at self-service operations are much

higher. At one high-volume station, the mean concentration to which an individual filling his own tank of gas was exposed (for ten minutes) was 4.2 ppm (2283).

65.3 HUMAN HEALTH CONSIDERATIONS

65.3.1 Animal Studies

65.3.1.1 Carcinogenicity

Most of the evidence regarding carcinogenicity of gasoline has been provided by a study conducted for the American Petroleum Institute (API) (2298). Reports of this study appear in several forms throughout the literature. A chronic inhalation study of gasoline vapor was conducted in mice and rats; the gasoline employed was unleaded, with the benzene content adjusted to 2%. Groups of both sexes of B6C3F1 mice and Fischer 344 rats were exposed to vapor at concentrations of 67, 292 or 2056 ppm for 6 hours per day, 5 days per week for periods ranging from 103 to 113 weeks. After as little as three months of exposure to 2056 ppm, macroscopic lesions were evident in the kidney of male rats. Microscopic observations included an increased incidence of renal disease with tubular degeneration, regeneration or cystic dilatation among males exposed to 292 or 2056 ppm. At 24 months, an increase in the occurrence of primary renal neoplasm was seen in the male rats at all doses, with some evidence of a dose-response relationship. In addition, a compound related increase in liver nodules and masses was seen in female mice exposed to the intermediate and high levels. Histopathologic examination revealed primary hepatocellular tumors (adenomas and carcinomas) in these animals.

These unexpected findings of species and sex-specific carcinogenic effects were not evident until late in the study. To better understand the significance to human health, the American Petroleum Institute contracted with Universities Associated for Research and Education in Pathology, Inc. (UAREP) for assistance in interpretation of the findings. The UAREP reviewed the chronic inhalation study, "old rat nephropathy" syndrome, and the basic morphological and functional similarities and differences in the kidneys of the rat, mouse, and man (2299). This review concluded that the significance of the hepatocellular carcinoma in female mice was questionable. The UAREP felt that other studies on different hydrocarbons demonstrated acute toxic effects on the female liver including fatty metamorphosis whereas these effects were not reported in the API chronic inhalation study.

The finding of renal carcinoma in male rats was clearly significant. The lesions were seen as early as 90 days and were dose-related. The lesions could be clearly distinguished from the old rat nephropathy, which is composed of chronic lesions involving all components of the kidney. However, administration of unleaded gasoline appeared to accentuate all the lesions characteristic of old rat nephropathy. It was not possible to evaluate the potential role of the

superimposed old rat nephropathy on the initiation, development, and progression of renal neoplasia induced by unleaded gasoline. Thus the UAREP review concluded that:

> "The chronic inhalation study demonstrated that unleaded gasoline inhalation produced acute, subchronic and chronic toxicity in the kidneys of male rats. Simultaneously, there was the development of preneoplastic lesions and ultimately the appearance of adenomas and adenocarcinomas in these male rats. The link between acute and chronic toxicity and carcinogenicity is not clear, nor can it (be) determined from the data generated in this bioassay. Although the pattern of acute and chronic non-neoplastic toxic lesions is somewhat unique for gasoline-related hydrocarbons, the morphological appearance of the preneoplastic and neoplastic lesions is similar to that produced by a number of renal carcinogens."

65.3.1.2 Mutagenicity

In general, studies of unleaded gasoline have shown no genotoxicity. Unleaded gasoline failed to induce his^+ mutants in the Ames Salmonella plate or suspension assays performed with and without metabolic activation by rat liver microsomes (2303). In cytogenetic studies, no chromosomal abnormalities were seen in the bone marrow of rats treated intraperitoneally with unleaded gasoline (2303,2304), nor were sister chromatid exchanges increased in human lymphoblasts treated in vitro (2301). When unleaded gasoline was tested in the L5178Y mouse lymphoma assay (2303) and in a similar assay employing a human lymphoblastoid line (2301), no increase in mutation frequency was observed in either system. A dose-related increase in unscheduled DNA synthesis (UDS) was observed in rat hepatocytes treated in vitro with 0.05 to 0.10% (v/v) gasoline, whereas these doses were toxic in both mouse and human hepatocyte cultures (2302). Weak UDS activity was observed in hepatocytes isolated from male and female mice treated 12 hours previously by gastric intubation with 2 g unleaded gasoline/kg (2302).

65.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Unleaded gasoline did not induce dominant lethal mutations in sperm cells of CD-1 male mice (2300). The mice were exposed to gasoline vapors for 6 hours per day, five days per week for eight weeks prior to mating with untreated females. Doses of 400 ppm and 1600 ppm did not cause any significant reduction in the fertility of the treated males, nor was any significant increase in pre- or post-implantation loss of embryos noted. It should be noted, however, that deaths amongst the males occurred during the treatment period; the cause and significance are unknown.

Tests for teratogenicity induced by the inhalation of unleaded gasoline gave negative results. No additional details were reported (2228).

65.3.1.4 Other Toxicologic Effects

65.3.1.4.1 Short-Term Toxicity

Gasolines generally act as anesthetics. They are also mucous membrane irritants (2). An oral LD_{50} of 13.6 g/kg was reported in the rat for unleaded gasoline. A single dose of 18 g/kg produced 90% mortality. A significant degree of gastrointestinal distress was observed. Necropsy revealed hemorrhagic gastroenteritis, gastrointestinal tympani and pneumonia with abscess formation (1924).

Acute anesthetic and toxic effects of gasoline vapors were studied as early as 1921 in dogs. Central nervous system effects were observed at approximately 10,000 ppm, and death at about 25,000 ppm (2290).

Toxicity of a gasoline component mixture was evaluated in a short-term inhalation study performed by Halder et al. (2292). A blend consisting of 25% (w/w) each of n-butane, n-pentane, isobutane and isopentane was vaporized to more closely approximate ambient exposure (in contrast to complete volatilization). Rats exposed to 44, 432 or 4437 ppm of vapor for 6 hours per day, 5 days per week for 3 weeks showed no clinical signs of distress. No gross or histopathologic lesions were noted, including in the kidneys. All other parameters of body and organ weights, hematology or blood chemistry were within normal range.

Studies on the acute effects of gasoline ingestion by rats revealed nephrotoxicity in male rats. Both unleaded gasoline (2291) and shale-derived distillate fuel (2294) caused reversible hyaline droplet formation (protein resorption) in the kidneys. This effect was believed due to a hydrocarbon-induced defect in the degradation of renal $a_{2\mu}$-globulin, a protein synthesized in the liver and excreted in urine, and was obvious after a single administration of as little as 2 mL/kg gasoline (2291). Over a three day period, hepatic lesions and alterations in serum chemistry and hematology were noted. By fourteen days, lymphoid depletion in the thymus was observed, as was congestion of multiple organs (2294).

Unleaded motor gasoline was slightly irritating to the shaved skin of New Zealand rabbits after a 24 hour dermal exposure to 0.5 mL. In a subacute dermal study, doses of 2.5 to 8 mL/kg were applied daily for a total of 10 days. No mortality was seen. Severe dermal irritation and weight loss were observed. Necropsy revealed pale and congested livers and kidneys (1924).

Gasoline containing tetraethyl lead caused no more injury than gasoline alone when applied to rabbit eyes. A single drop applied without local anesthetic caused discomfort and blepharospasm lasting

several minutes. The conjunctiva became mildly hyperemic but rapidly returned to normal. Ten drops applied during a 5 minute period (after local anesthesia) caused blepharospasm lasting 15 minutes. The conjunctiva became moderately edematous and hyperemic but recovery was prompt and complete (19).

65.3.1.4.2 Chronic Toxicity

To evaluate the long-term effects of gasoline inhalation, rats and monkeys were exposed to either 284 or 1552 ppm unleaded gasoline vapors or 103 and 374 ppm leaded gasoline vapor 6 hours per day, 5 days per week, for 90 days (2290). Although vomiting was noted in certain monkeys after 2 weeks exposure, no remarkable changes in body weight, hematology, or CNS responses were noted in either species. Lead deposition in the liver, kidney, brain and blood were observed in those animals treated with leaded gasoline. Upon histopathologic examination, male rats exposed to 1552 ppm unleaded gasoline displayed regenerative epithelium and dilated tubules in the kidney.

Pulmonary changes in rats exposed to leaded gasoline vapor were reviewed by Cooper (2296). Changes in male rats ranged from minor foci of interstitial fibrosis to widespread sclerosis after 6 weeks exposure to 100 ppm. After eight weeks, tachypnea and prostration were evident. Such observations were confirmed in female rats similarly exposed. Ultrastructural changes emerged sequentially as degeneration, hypertrophy and/or hyperplasia and finally development of interstitial sclerosis and irregular alveolar collapse. A number of these changes are thought related to the fact that gasoline vapor inhalation caused a decrease in pulmonary surfactant. Surfactant, functioning to decrease surface tension and stabilize surface forces, was reduced after only 5 days exposure.

Repeated exposure of albino rabbits eyes to gasoline vapor levels of 3 mg/L air daily for 10 months has been reported to cause histologically recognizable disturbances of the corneal and conjunctival epithelium. Exposure to a vapor level of 616 ppm of a C_9-C_{10} fraction of a high octane motor fuel induced cataracts in 70% of exposed rats. Exposure was for a total of 2424 hours. The petroleum fraction was composed mainly of alkyl benzenes. It contained no naphthalene, a known inducer of cataracts in animals (19).

Other changes seen in animals after chronic gasoline inhalation include a depression in body weight in rats and mice, a reduction in the incidence of cystic or enlarged uteri of female mice, and mild multifocal, dose-related pulmonary inflammation in rats (2298).

65.3.2 Human and Epidemiologic Studies

Before reviewing the adverse effects of gasoline on humans, it is important to note that human exposure is considerably different from that used in animal studies. Due to the differential volatility of the hydrocarbon compounds present in gasoline, the vapor produced under

experimental conditions does not mimic ambient vapor composition. The animals are exposed to completely volatilized gasoline whereas human exposure is to partial volatilization. The larger hydrocarbons, which are less volatile, are present in lower proportion in ambient vapors than in completely volatilized gasoline. Thus, since certain subsets of the higher molecular weight compounds are thought to be responsible for nephrotoxicity, it is likely that the animal studies overestimate the toxic effect in humans.

65.3.2.1 Short-term Toxicologic Effects

The primary mode of exposure to gasoline is by inhalation. The most common symptoms of intoxication are headaches, blurred vision, dizziness and nausea (2). Most of the adverse physical effects in humans have been documented by cases of intentional gasoline inhalation or "sniffing." Absorption of the volatile components across the lungs is generally rapid and quite efficient. Levels as low as 500-1000 ppm for 30 to 60 minutes can result in an euphoric condition consisting of ataxia (decreased muscle coordination), drowsiness and dizziness. Increased levels (1000-3000 ppm) lead to irritation, headache, nausea, and vomiting. Levels in excess of 5000 ppm can cause dizziness or deep anesthesia within minutes, and occasionally coma and death are reported (2277,2284). In general, the euphoria, lethargy and decreased sensory perception last several hours after exposure (2280). The intoxicating feeling is believed to be due to the neurotoxic effects of n-hexane and the narcotic properties of the C4 to C8 saturated hydrocarbons (2277).

Deaths from gasoline sniffing have rarely been reported. In a study of 110 "sudden sniffing deaths" occurring during the 1960's, 3.6% were thought to be associated with gasoline inhalation. Sudden death has been reported in an adolescent who exercised after inhaling gasoline fumes while siphoning gasoline from a car. Death was presumably from a cardiac arrhythmia induced by the fumes (1570).

Symptoms in severe oral intoxication are mild excitation, loss of consciousness, occasional convulsions, cyanosis, congestion and capillary hemorrhaging of the lung and internal organs, followed by death due to circulatory failure. In mild cases, symptoms include inebriation, vomiting, vertigo, dizziness, confusion and fever (12). In adults, ingestion of 20-50 g may produce severe poisoning. One case of accidental ingestion caused immediate severe burning of the pharynx and gastric region. After immediate gastric lavage, no general symptoms were noted. Liver function tests were slightly elevated, indicating hepatic damage which was probably due to gasoline's lipid solubility. Another case of accidental ingestion of gasoline presented with nausea, abdominal cramps and red-brown urine. Upon further investigations, acute reversible toxic injury was found to the upper portions of both kidney (2278). It should be noted that ingestion can be accompanied by aspiration. This can lead to chemical injury, irritation to the lung and mucosal surfaces and generalized chemical pneumonitis. Symptoms are lethargy, moderate respiratory distress with laboratory confirmation of leukocytosis and increased serum levels of

liver enzymes. Hypoxemia (low blood oxygen levels) accompanying aspiration pneumonitis accounts for the CNS manifestation, not direct CNS toxicity of the gasoline. Most symptoms are reversible within 48 hours (2279).

Dermal exposure to gasoline vapor and liquid is also possible. Considering the physical/chemical properties of the volatile components, they should be readily absorbed through the skin (2286). Liquid gasoline is irritating to the skin. Prolonged contact causes a chemical burn (2228). Hypersensitivity may develop in certain individuals (54).

Exposure of volunteers to gasoline vapors indicated no ocular irritation at a concentration of 140 ppm. Irritation of the eyes and throat was seen at vapor levels of 270 to 900 ppm. If splashed into the eye, pain and irritation occurs, but there is only slight, transient corneal epithelial disturbance (19).

65.3.2.2 Chronic Toxicologic Effects

The possible long-term effects of chronic inhalation of gasoline have been reported as anorexia, weight loss, weakness and cramps (2284). The neurological and encephalopathic effects seen in severe cases include incoordination and tremors, however, these effects appear reversible with therapy and cessation of exposure (1570). Post-mortem findings of gasoline sniffers frequently show cerebral and pulmonary edema; if death is delayed, necrosis of the liver and kidney is evident. The minor components of gasoline such as benzene, xylene and tetraethyl lead contribute more to these chronic effects than do the aliphatic hydrocarbons (2284,2277).

Infrequent or controversial effects of chronic inhalation include decreased intelligence and fetal retardation. It is known that exposure to gasoline vapors leads to increased mean blood levels (specific components not reported) in women and fetuses. In a study conducted by Hunter et al. (2282), a community of 500 American Indians with prevalent gasoline abuse showed a high incidence of mental retardation (4% of live births). Although alcohol abuse was widespread, the infants' clinical signs were not typical of fetal alcohol syndrome. Methyl mercury poisoning would account for the symptoms, however blood and hair mercury levels within the community were low. Therefore this study suggested that the retardation was due to prenatal exposure to organic lead present in the gasoline vapors.

Although there has been a rough correlation between the temporal increase in gasoline production/consumption and elevated renal cancer mortality, geographic ecologic studies comparing counties involved in petroleum refining with control counties have shown no significant increase in kidney cancer deaths. Cohort studies comparing refinery workers with the general population showed no consistent increases in standardized mortality rates of kidney cancer. However, most of these studies were not designed or analyzed with a gasoline exposure - kidney

cancer hypothesis in mind and little data are available on duration of exposure or time since first exposure in relation to kidney cancer (2281).

A retrospective case-control study (2276) was conducted to examine increased risk of renal cell carcinoma. Only 4 of the 92 cancer cases and 122 of the 1,558 non-neoplastic control patients had any occupational exposure to gasoline. This finding suggests that there is no independent effect of occupational gasoline exposure on risk for renal cell carcinoma. Thus the epidemiologic literature provides no consistent evidence for a relationship between gasoline exposure and kidney cancer in man.

A small epidemiology study recently reported to EPA found leukemia deaths in auto mechanics and gas station attendants to be in excess of standard mortality ratios; however, more definitive studies are necessary to determine if the leukemia excess is associated with gasoline, benzene or other chemicals in their work environment (2285).

65.3.3 Toxicology of Gasoline Components

A brief overview of the toxicology of the major hydrocarbon components of automotive gasoline (see Table 65-3) are summarized below. The acute toxicity values for these components are presented in Table 65-4.

n-Hexane

Hexane may be the most highly toxic member of the alkanes. When ingested, it causes nausea, vertigo, bronchial and general intestinal irritation and CNS effects. It also presents an acute aspiration hazard. Acute exposure occurs primarily through inhalation. Non-specific symptoms such as vertigo, headache, nausea and vomiting are the first to be manifested. At high concentrations, a narcosis-like state appears as a result of CNS depression. Pre-narcotic symptoms occur at vapor concentrations ranging from 1500-2500 ppm. n-Hexane irritates the eyes and mucous membranes. These effects can be seen after an exposure of 880 ppm for 15 minutes. Skin contact primarily causes fat removal and cutaneous irritation.

Chronic exposure to n-hexane vapors causes peripheral neuropathy. The first clinical sign of neural damage is a feeling of numbness in the toes and fingers. Progression leads to further symmetrical sensory impairment in the distal portions of the extremities and to loss of muscular stretching reflexes. Ultimately, symmetrical muscular weakness develops, chiefly in the distal portion of the extremities. Paralysis develops with varying degrees of impaired grasping and walking. This may include muscular atrophy (sensorimotor poly-neuropathy). The development of electrophysiological changes parallels the severity of the clinical picture. In the most severe cases, nerve conductivity is neutralized. In some cases, cranial nerve involvement is also observed. After exposure ceases, recovery begins within 6 to

TABLE 65-4

ACUTE TOXICITY OF COMPONENTS OF AUTOMOTIVE GASOLINE

Component	Oral LD_{50}	Dermal LD_{50}	LC_{50}
n-hexane	24-49 mL/kg [rat] (1935) 28,710 mg/kg [rat] (1937)	no data	33,000 ppm •4 hr [rat] (1935)
octane	<— no data —>		
dodecane	<— no data —>		
isopentane	no data	no data	1000 mg/L [mouse] (12)
isooctane	<— no data —>		
methylcyclopentane	<— no data —>		
methycyclohexane	2250 mg/kg [rat] (47)	no data	no data
cyclohexane	29,820 mg/kg [rat] (1935)	no data	no data
benzene	3800 mg/kg [rat] (59) 4700 mg/kg [mouse] (47)	no data	10,000 ppm •7 hr [rat] (47)
toluene	5000 mg/kg [rat] (47)	12,124 mg/kg [rabbit] (47)	5320 ppm •8 hr [mouse] (47)
xylenes	4300 mg/kg [rat] (47)	no data	5000 ppm •4 hr [rat] (47)
ethyl benzene	3500 mg/kg [rat] (47)	5000 mg/kg [rabbit] (59)	no data
trimethylbenzenes	no data	no data	18 mg/m^3 •4 hr [rat] (47)
1-methylnaphthalene	1840 mg/kg [rat] (47)	no data	no data
2-methylnaphthalene	1630 mg/kg [rat] (47)	no data	no data

10 months in mild to moderate cases, but may take up to 3 years in serious cases. The threshold level at which neuropathy occurs has not been firmly established but symptoms have been observed in people exposed to concentrations ranging from 10 to 200 ppm for 9-12 months.

In animals, signs of narcosis are seen after mice are exposed to vapor levels of 16,000 ppm for 5 minutes. Death generally occurred at concentrations between 43,800 and 52,000 ppm after 9-119 minutes. The oral LD_{50} is cited as 24 mL/kg for 14-day-old rats and 49 mL/kg for young adult rats.

Long-term inhalation experiments in rats suggest that the first signs of neurotoxicity appear after they are exposed to levels of 200 ppm for 24 weeks. This higher threshold to induce neurotoxicity in animals may be due to differences in metabolism. Specifically, 2-hexanol is the chief metabolite in animals, while 2,5-hexanedione which is neurotoxic, predominates in man. Chronic topical application of a solvent containing 35.2% n-hexane caused axonal swelling and myelin degeneration in chicks. No clinical signs were seen. Dosage was 1 g/kg/day for 64 days. In rabbits, topical application of 0.5 mL/day for up to 10 days caused redness, irritation and scab formation. N-hexane is neither carcinogenic or teratogenic. One in vivo study in rats that inhaled 150 ppm for 5 days found an increased number of chromosome aberrations in the bone marrow cells. No studies on mutagenicity, reproductive toxicity or carcinogenicity in man were found (12,1930,1935).

Isopentane

Isopentane is a CNS depressant. Effects may include exhilaration, dizziness, headache, loss of appetite, nausea, confusion, inability to do fine work, a persistent taste of gasoline and in extreme cases, loss of consciousness. Inhalation of up to 500 ppm appears to have no effect on humans. "Very high" vapor concentrations are irritating to the skin and eyes. Repeated or prolonged skin contact will dry and defat skin resulting in irritation and dermatitis. The LC_{50} in the mouse is estimated to be 1000 mg/L (12).

2-Methylpentane (isohexane, 3-methylpentane)

No physiological data are available but isohexanes are expected to be mucous membrane irritants and to have a low oral toxicity. Isohexanes are predicted to have narcotic properties and are documented to be cardiac sensitizers but are not expected to have neurotoxic properties (12).

Cyclohexane

Cyclohexane is a CNS depressant of low toxicity. Symptoms of acute exposure are excitement, loss of equilibrium, stupor and coma. Rarely, death results due to respiratory failure. The anesthesia which is induced is weak and of brief duration but more potent than that

caused by hexane. The oral LDLo in rabbits ranges from 5.5 to 6.0 g/kg. Within 1.5 hours the animals exhibited severe diarrhea, widespread vascular damage and collapse. Degenerative lesions were seen in the heart, lung, liver, kidney and brain. A one-hour vapor exposure to 26,752 ppm caused rapid narcosis and tremor and was lethal to all exposed rabbits. In mice, concentrations causing narcosis vary from 14,600 to 122,000 ppm.

Cyclohexane is nominally absorbed through the skin although massive applications (>180.2 g/kg) to rabbit skin resulted in microscopic changes in the liver and kidneys and caused the death of all animals.

The danger of chronic poisoning is relatively slight because this compound is almost completely eliminated from the body. No toxic changes were seen in rabbits exposed to vapor levels of 434 ppm, 6 hours daily for 50 exposures, but some microscopic changes were seen in the liver and kidneys when the exposure was to 786 ppm for the same period.

In man, no systemic poisonings by cyclohexane have been reported. A vapor level of 300 ppm is somewhat irritating to the eyes and mucous membranes. It has been reported that cyclohexane may potentiate the toxic effects of TOCP but no additional details of this interaction are available (12,17,46,54,1937).

Benzene

The primary effects of benzene inhalation and ingestion are on the central nervous system (54). Benzene is carcinogenic in both animals and man. Several reports have established a relationship between benzene exposure and leukemia. For more information, refer to Chapter 18 of the Installation Restoration Program Toxicology Guide, Volume 1.

Toluene

Toluene is a CNS depressant with a low toxicity. For more information, refer to Chapter 19 of the Installation Restoration Program Toxicology Guide, Volume 1.

Xylenes

Acute exposure to high concentrations of xylene vapors may cause CNS depression. Both the liquid and the vapor are irritating to the eyes, mucous membranes and skin (46). The National Toxicology Program recently reported that there was no evidence of carcinogenicity of mixed xylenes in either mice or rats given daily doses ranging from 250 to 1000 mg/kg by gavage for 2 years (1939).

For more information, refer to Chapter 21 of the Installation Restoration Program Toxicology Guide, Volume 1.

Trimethylbenzenes

The trimethylbenzenes occur in 3 isomeric forms. The 1,3,5-isomer (mesitylene) and the 1,2,4-isomer (pseudocumene) are toxicologically similar. High vapor concentrations (5000-9000 ppm) cause CNS depression in animals. Loss of reflexes was seen in mice exposed to 8130-9140 ppm of the 1,2,4-isomer or 8130 ppm of the 1,3,5-isomer. Rats exposed to 1700 ppm of an isomeric mixture for 10-21 days had no adverse effects or fatalities.

The fatal intraperitoneal dose of the 1,2,4-isomer for the guinea pig is 1.788 g/kg, while the fatal dose of the 1,3,5-isomer by the same route is 1.5-2 g/kg for the rat. For the 1,2,3-isomer, an oral LDLo of 5000 mg/kg has been reported in the rat. Trimethylbenzene liquid is a primary skin irritant. Deposition into the lungs causes pneumonitis at the site of contact.

The only report of human exposure described symptoms of nervousness, tension, anxiety, asthmatic bronchitis, hypochromic anemia and changes in the coagulability of the blood. Vapor concentrations ranged from 10-60 ppm. Exposure was to a mixture containing 30% of the 1,3,5-isomer and 50% of the 1,2,4-isomer (2,12).

Naphthalene

Ingestion or prolonged inhalation of naphthalene produces nausea, vomiting and disorientation. It is irritating to the skin and eyes and prolonged vapor exposure has led to cataract formation in humans (17). Hemolytic anemia is the most severe effect associated with naphthalene exposure, but this effect is seen predominantly in individuals with an enzyme deficiency (54).

Gasoline Additives

Additives used in automotive gasoline are listed in Table 65-2. The toxicological information which was available is outlined below.

Tetraethyl lead (TEL)

Acute exposure to TEL causes symptoms of headache, anxiety, insomnia, fatigue and appetite loss (38). The more severe effects are seizures and acute metabolic encephalopathy which is characterized by hallucinations, disorientation, violence and paranoia (2277). The contribution of TEL to the short-term effects of gasoline inhalation is not clear. It is not known if the amount inhaled during a single episode of gasoline "sniffing" is sufficient to cause the hallucinations and behavioral changes caused by TEL alone or if TEL potentiates the short-term effects of other volatile hydrocarbons present in gasoline; however, the long-term effects are currently considered to be due to TEL (2277). The oral LD_{50} in the rat is 14 mg/kg (19). More information on TEL can be found in Chapter 54 of the Installation Restoration Program Toxicology Guide, Volume 2.

Tetramethyl lead

Tetramethyl lead affects the nervous system in animals and causes signs of increased irritability. Although not documented, it is expected to produce psychosis, mania and convulsions in humans (46). In the rat, an oral LD_{50} of 109 mg/kg was reported (47).

It is likely that intoxication by tetramethyl lead will be similar to that caused by tetraethyl lead (46). Information on tetraethyl lead can be found in Chapter 54 of the Installation Restoration Program Toxicology Guide, Volume 2.

Methylcyclopentadienyl manganese tricarbonyl (MMT)

In its concentrated form, MMT is highly toxic by all routes of exposure. The primary site of action in animals is the CNS, where the effects of MMT are similar to those caused by tetraethyl lead. The oral LD_{50} in the rat is 50 mg/kg. Human exposure data are limited. It is expected that when MMT is blended with fuels, it has a low order of toxicity.

Concentrated MMT penetrates the skin readily. When 5-15 mL was spilled on a worker's skin, nausea, headache and giddiness resulted in a 2-5 minute period; however, gasoline solutions are not as readily absorbed as the pure material (2,1937,1409).

Ethylene dibromide (EDB)

EDB is irritating to the eyes and mucous membranes. It also causes symptoms of CNS depression. Acute exposures have resulted in lung, liver and kidney damage (1745,1759,38,54). EDB is carcinogenic in rodents by oral, inhalation and dermal routes (142,1606,1743,1744). ACGIH has classified EDB as a suspected human carcinogen with a recommendation that exposure be avoided (3). The oral LD_{50} in the rat is 146 mg/kg (1759). More information on EDB can be found in Chapter 45 of the Installation Restoration Program Toxicology Guide, Volume 2.

1,2-Dichloroethane

Acute ingestion or inhalation of 1,2-dichloroethane results in symptoms of CNS depression, gastrointestinal upset and systemic injury to the liver, kidneys and lungs (12). The oral LD_{50} in the rat is 670 mg/kg (47). More information on 1,2-dichloroethane can be found in Chapter 9 of the Installation Restoration Program Toxicology Guide, Volume 1.

Methyl-t-butyl ether (MTBE)

In rats, an oral LD_{50} of 4 mL/kg was reported (1937). In recently conducted acute and subchronic tests, it was reported that MTBE caused a deepening of barbiturate sleep, a reduction of

spontaneous motor activity and reduced performance connected with disturbances of the motor coordination system; however the severity of these effects does not indicate serious toxic damage to the CNS. The study concluded that MTBE "does not even minimally increase the neurologic effects with respect to gasoline itself." The level of exposure or the species which were tested were not reported (2293).

t-Butyl alcohol

At high concentrations, t-butyl alcohol causes narcosis in animals and it is expected to cause the same effect in humans. Other than slight skin irritation, no effects have been reported from industrial exposure. The oral LD_{50} in the rat is 3500 mg/kg (46).

Ethanol

Ethanol is irritating to the eyes and mucous membranes. It is also a CNS depressant. The acute toxicity of ethanol is low for both animals and man. Overexposure causes ataxia, incoordination and drowsiness (2,46). An oral LD_{50} of 14 g/kg was reported for the rat (47).

Methanol

Methanol causes optic neuropathy and metabolic acidosis. Poisoning has occurred primarily from ingestion of adulterated alcoholic beverages. After ingestion there is a latency period of 18 to 48 hours after which exposed individuals develop symptoms of nausea, abdominal pain, headache and shortness of breath. Visual symptoms range from blurred or double vision to changes in color perception, constricted visual fields and complete blindness. Other symptoms of intoxication include dizziness, behavioral disturbances, neuritis and acidosis. The degree of acidosis has been found to parallel the severity of the poisoning. Evidence suggests that exposure to vapor concentrations of 200-375 ppm causes recurrent headaches and visual disturbances are seen at vapor levels of 1200-8300 ppm (2,46). An oral LD_{50} of 13 g/kg was reported in the rat (47).

Tri-ortho-cresyl phosphate (TOCP)

TOCP affects the spinal cord and peripheral nervous system. Symptoms of acute exposure, including nausea, vomiting, diarrhea and abdominal pain, are followed by a latent period of 3 to 30 days. At this time, there is muscle soreness, numbness of fingers,. calf muscles and toes which progresses to foot and wrist drop. These effects are manifested after ingestion, inhalation or dermal absorption (54). An oral LD_{50} of 1160 mg/kg has been reported in the rat (47). More information on TOCP can be found in Chapter 49 of the Installation Restoration Program Toxicology Guide, Volume 2.

Isopropyl alcohol

Isopropyl alcohol has moderate narcotic properties. Ingestion causes CNS depression and it is expected that sustained inhalation of high vapor concentrations will produce the same effect. It is also irritating to the eyes and mucous membranes (2,46). An oral LD_{50} of 5840 mg/kg was reported for the rat (47).

65.3.4 Levels of Concern

The ACGIH (3) recommends an occupational exposure limit of 300 ppm for automotive gasoline, with a short-term exposure limit of 500 ppm.

No other criteria or standards have been established with regard to human health and safety.

65.3.5 Hazard Assessment

A single study (2298) on the potential carcinogenic effects of gasoline is available. Mice and rats were exposed by inhalation to the vapors of unleaded gasoline (benzene content, 2%), 6 hours per day, 5 days per week for two years. Exposure levels ranged from 67 to 2056 ppm. Dose-related renal carcinomas were observed in gasoline-exposed male rats. The significance of the sex-specific and species-specific findings is unclear. Mutagenicity studies suggest no genotoxic effects for unleaded gasoline (2301,2303,2300,2304). Negative teratogenic findings were also reported (2228), although details are lacking.

Animals studies indicate kidney damage is the predominant toxic effect of acute ingestion and chronic inhalation exposure to unleaded gasoline (2290,2294). Pulmonary changes (fibrosis and sclerosis) were also evident with inhalation exposure (2296).

Humans exposed via inhalation to 500-1000 ppm gasoline for 30 to 60 minutes develop ataxia, drowsiness and dizziness; levels of 1000-3000 ppm result in irritation, headache, nausea and vomiting; exposure to greater than 5000 ppm can cause deep anesthesia within minutes, and occasionally, coma and death (2277,2284).

Ingestion of 20 to 50 g of gasoline may produce severe intoxication in adults (12). Symptoms of poisoning are similar to those noted above for inhalation exposures.

65.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of automotive gasoline in soil and water requires collection of a representative field sample and laboratory analysis for the specific major components attributed to gasoline; however, the relative concentrations of the constituents, and

even the constituents themselves, will vary with time and distance from the site of initial contamination due to weathering. The major component categories in automotive gasoline have been identified as the following:

n-alkanes
branched alkanes
cycloalkanes
benzene and alkylbenzenes
naphthalenes

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques may be used to identify the principal components in automotive gasoline. Fuel samples, and probably any samples collected in the field which are primarily organic in nature, may require the separation (prior to GC or GC/MS analysis) of the aliphatic, monoaromatic and polycyclic aromatic hydrocarbon fractions using liquid solid column chromatography; the various column eluates, with or without dilution in carbon disulfide, can then be analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (i.e., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet extraction or sonication methods (1422). An aliquot of the sample extract, with or without concentration, is then analyzed by GC or GC/MS. Sampling and analysis considerations for some specific components in gasoline, i.e., benzene, toluene, xylenes, ethyl benzene and naphthalene have been addressed in Volume 1.

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component, but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorotrifluoroethane). The "oil and grease" content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; extraction methods are those described above for aqueous and soil samples.

A detection limit for automotive gasoline was not determined; the detection limit for specific components is expected to be in the range of $\mu g/L$ for aqueous samples and $\mu g/g$ for non-aqueous samples.

COMMON SYNONYMS:	CAS REG. NO.:	AIR W/V CONVERSION FACTORS at 25°C (1967)
White spirits Mineral spirits Solvent naphtha Dry cleaning safety solvent	8052-41-3 NIOSH NO.: WJ8925000	5.77 mg/m³ ≈ 1 ppm 0.173 ppm ≈ 1 mg/m³
	APPROXIMATE COMPOSITION: linear and branched alkanes, 30-50% cycloalkanes, 30-40% aromatics, 10-20% benzene, trace olefins, trace	MOLECULAR WEIGHT: 135-145 (average)

REACTIVITY	Stoddard solvent is considered to be a miscellaneous combustible material for compatibility classification purposes. Reactions of such substances with non-oxidizing mineral acids may evolve heat and usually innocuous gases. Those with oxidizing mineral acids or organic peroxides or hydroperoxides may produce heat, fire, and toxic gases, while those with strong oxidizing agents or alkali or alkaline earth elemental metals may produce heat, fire, and innocuous gases. Nitrides evolve heat, fire, and flammable gases. Strong reducing agents evolve heat and flammable gases. Reactions with explosive materials may result in an explosion. There are also unspecified incompatibilities with bases and selected amines (38,507,511).

PHYSCIO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(2)
	• Color: colorless	(2)
	• Odor: mild petroleum	(507)
	• Odor Threshold: 0.9 ppm	(1970)
	• Liquid Density (g/ml at 20°C): 0.77	(507)
	• Freezing/Melting Point (°C): no data	( )
	• Boiling Point (°C): 154-202°	(2)
	• Flash Point (°C): 37.8-60 (variable)	(23,38, 51,507)
	• Flammable Limits in Air, % by Volume: (0.8-1.1) - 6.0	(38,51,506)
	• Autoignition Temperature (°C): 227-260 (variable)	(23,38, 51,506)
	• Vapor Pressure (mm Hg at 20°C): 3	(507)
	• Saturated Concentration in Air (mg/m³ at 20°C): 2.2 x 10^4 to 2.4 x 10^4	(ADL estim)
	• Solubility in Water (mg/L at 20°C): insoluble	(507)
	• Viscosity (cp at 20°C): 0.91-0.95	(5)
	• Surface Tension (dyne/cm at 20°C): no data	( )

PHYSICO-CHEMICAL DATA (continued)	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 3.16-7.06 (*) • Soil Adsorption Coefficient, K_{oc}: 700-5.5 x 10^6 (*) • Henry's Law Constant (atm•m^3/mol at 20°C): 4.4 x 10^{-4} - 7.4 (*) • Bioconcentration Factor: no data ( )
PERSISTENCE IN THE SOIL-WATER SYSTEM	Stoddard solvent hydrocarbons are expected to be relatively mobile and moderately persistent in most soil systems. Persistence in deep soils and ground water may be higher. Volatilization, photooxidation and biodegradation are potentially important fate processes. Surface spills are expected to be weathered by evaporation and photooxidation. Downward migration of weathered surface spills and sub-surface discharges represent a potential threat to underlying ground water. Biodegradation of C_7-C_{12} hydrocarbons is expected to be significant under environmental conditions favorable to microbial oxidation; naturally-occurring, hydrocarbon-degrading microorganisms have been isolated from polluted soils and, to a lesser extent, non-polluted soils.
PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water systems is the contamination of ground water drinking water supplies resulting from large spills of Stoddard solvent or leaking underground storage tanks. Vapors from leaked or spilled solvent may diffuse through soils and migrate into structures resulting in inhalation exposures. Inhalation exposures may also occur from the direct volatilization of surface spills. Ingestion with food is not expected to be significant.
HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (38): Overexposure to Stoddard solvent causes irritation of the eyes, nose and throat and may cause dizziness. Prolonged overexposure to the liquid may cause skin irritation. Toxicity Based on Animal Studies: LD_{50} (mg/kg): oral -- no data; skin -- no data LCLo (mg/m^3): inhalation [cat] 10,000•2.5 hr. (47) Long-Term Effects: Kidney damage Pregnancy/Neonate Data: Negative Mutation Data: Negative Carcinogenicity: No data

*Range of values for representative hydrocarbons from major component classes (see Table 67-2).

HANDLING PRECAUTIONS (38,507)	Handle only with adequate ventilation • Vapor levels of 500 to 1000 ppm: chemical cartridge respirator with a full facepiece and organic vapor cartridges • 1000 to 5000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece; gas mask with organic vapor canister • Chemical goggles if there is probability of eye contact • The use of impermeable gloves is advised to prevent skin irritation.

EMERGENCY FIRST AID TREATMENT (38,507)	Ingestion: Do not induce vomiting. Get immediate medical attention • Inhalation: Move victim to fresh air. Give artificial respiration if necessary. Get medical attention • Skin: Remove contaminated clothing. Wash skin with soap and water. If irritation persists after washing, get medical attention • Eye: Flush with large amounts of water for 15 minutes. If irritation persists, get medical attention.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): 500 ppm
- AFOSH PEL (8-hr TWA): 500 ppm

Criteria
- NIOSH IDLH (30-min): 5000 ppm
- ACGIH TLV® (8-hr TWA): 100 ppm
- ACGIH STEL (15-min): 200 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; Stoddard solvent is not a priority pollutant.
- Aquatic Life
  No criterion established; Stoddard solvent is not a priority pollutant.

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;
- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;
- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Occupational Safety and Health Act (OSHA)
  Employee exposure to Stoddard solvent shall not exceed an 8-hour time-weighted-average of 500 ppm (298).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated petroleum naphtha as a hazardous material which is subject to requirements for packaging, labeling and transportation (306).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Petroleum and coal tar distillates with flash points below 21°C are classified as flammable substances and are subject to packaging and labeling regulations. Because of the variable composition of other petroleum and coal tar distillates (excluding those used as motor

fuels), they are considered preparations and their labeling shall be done in accordance with the procedures outlined in the Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (solvents).

EEC Directives - Proposed

Proposal for a Council Directive on the Dumping of Waste at Sea (1793) EEC has proposed that the dumping of crude oil, petroleum hydrocarbons, lubricants and hydraulic fluids at sea be prohibited.

67.1 MAJOR USES AND COMPOSITION

67.1.1 Major Uses

Stoddard solvent is produced from a straight-run distillate of paraffinic or mixed base crude oil. It is used as a diluent in paints, coatings and waxes; as a dry cleaning agent; as a degreaser and cleaner and as a herbicide (2).

67.1.2 Composition

Stoddard solvent is a mixture of C_7 through C_{12} hydrocarbons, predominantly C_9 through C_{11}, with a boiling range between 160°C to 210°C. Flashpoint dry-point test and odor data are used to classify Stoddard solvent into the following four types: regular Stoddard solvent, 140 flash solvent, odorless solvent, and low end point solvent. Chemically, Stoddard solvent is a mixture of 30-50% straight and branched alkanes, 30-40% cycloalkanes, and 10-20% aromatics. Benzene and olefins are present in trace quantities only (1967,2228). The 140 flash aliphatic solvent is composed of organic compounds with carbon chain lengths ranging from C5 to C12. Its boiling range is 185-207°C and it is composed of 60.8% paraffins, 24.5% monocycloparaffins, 11.2% dicycloparaffins, 3.03% alkyl benzenes, 0.3% indans and tetralins, and 0.07% benzenes (1967). Both types will be discussed in some sections of the chapter which follows.

A characterization of the individual hydrocarbon components of Stoddard solvent was not available. Table 67-1 presents the available characterization by chemical classes.

67.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

A discussion of the environmental behavior of Stoddard solvent is limited by the lack of analytical data defining its specific components. Many of the hydrocarbons expected to be components of Stoddard solvent were addressed previously in the more extensive environmental fate section of Chapter 64. The general discussions of aliphatic and aromatic hydrocarbons and their behavior in soil/ground-water systems will not be repeated here; the reader is referred to the relevant sections of Chapter 64.

67.2.1 Equilibrium Partitioning Model

In general, soil/ground-water transport pathways for low concentrations of pollutants in soil can be assessed by using an equilibrium partitioning model. For the purposes of assessing the environmental transport of Stoddard solvent, a group of specific hydrocarbons within the C_7-C_{12} range was selected from the dominant hydrocarbon classes, i.e., alkanes, cycloalkanes, and aromatics; there are no available data to confirm the presence of the selected

TABLE 67-1

Composition Data for Stoddard Solvent
(Reference 1967)

Carbon Range	C_7-C_{12}
Straight/Branched Alkanes	48%
Cycloalkanes	38%
Aromatics	
Benzenes	0.1%
Alkylbenzenes	14%
Indans/Tetralins	< 1%

hydrocarbons in a typical Stoddard solvent sample. Table 67-2 identifies the selected hydrocarbons and presents the predicted partitioning of low soil concentrations of those hydrocarbons among soil particles, soil water, and soil air. The portions associated with the water and air phases of the soil are expected to have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil indicate that sorption is expected to be an important process for all the dominant hydrocarbon categories. Partitioning to the soil-vapor phase in this model is not very important for the C_7-C_{12} hydrocarbons. The alkyl benzenes have higher water solubilities and transport with infiltrating water may be important for these compounds; volatilization is still expected to be low. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a significant percent of the aromatic hydrocarbons is predicted to be present in the soil-water phase and available for transport with flowing ground water.

In interpreting these results, it must be remembered that this model is valid only for low soil concentrations (below aqueous solubility) of the components. Large releases of solvent (spills, leaking underground storage tanks) may exceed the sorptive capacity of the soil, thereby filling the pore spaces of the soil. In this situation, the hydrocarbon mixture would move as a bulk fluid and the equilibrium partitioning model would not be applicable.

TABLE 67-2

EQUILIBRIUM PARTIONING OF POTENTIAL
STODDARD SOLVENT HYDROCARBONS IN MODEL ENVIRONMENTS[a]

COMPOUND	Log K_{OW}	K_{oc}[b]	H[c]	UNSATURATED TOPSOIL (%)			SATURATED DEEP SOIL[d] (%)	
				Soil	Water	Air	Soil	Water
Octane	5.18 (e)	73,000	2.96	97.4	0.01	2.6	97.7	0.3
Dodecane	7.06 (f)	5.5×10^6	7.4	99.9	0.0001	0.09	99.9	0.004
Trimethylpentane	4.87 (f)	36,000	1.9-3.3	94.7	0.01	5.3	99.3	0.7
Methylcyclohexane	4.10 (f)	6,070	0.39	95.9	0.08	4.0	96.2	3.8
Trimethylcyclohexane	5.02 (h)	50,500	1.6	98.0	0.01	2.0	99.5	0.5
Xylenes	3.16 (e)	700	7×10^{-3}	98.8	0.7	0.5	74.4	25.6
Trimethylbenzenes	3.65 (h)	2,150	5×10^{-3}	99.6	0.2	0.2	90.0	10.0
Naphthalene	3.30 (e)	962	4.82×10^{-4}	99.4	0.5	0.03	80.2	19.8
Methylnaphthalenes	3.87 (e)	3,570	4.4×10^{-4}	99.8	0.1	0.01	93.7	6.3

[a] Calculations based on Mackay's equilibrium partioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

[b] Reference 652.

[c] Taken from Reference 74 unless otherwise specified. Units equal $atm \cdot m^3/mol$.

[d] Used sorption coefficient $K_p = 0.001 \times K_{oc}$.

[e] Reference 29.

[f] Arthur D. Little, Inc., estimate according to equations provided in Reference 31.

[g] Reference 10.

[h] Reference 31.

67.2.2 Transport and Transformation Processes

Transport and transformation of Stoddard solvent constituents will depend on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly in the percolating ground waters and be sorbed less strongly on the soils, thus being transported more rapidly, and may or may not be susceptible to degradation by chemical or biological action. Thus, as was shown in Figure 65-1, the relative concentrations of the constituents of the solvent will vary with time and distance from the site of contamination. This effect is called "weathering." (This term is also used to describe the changes to petroleum materials following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation are all involved.)

There are no available data specific to the transport and transformation of Stoddard solvent in soil/ground-water systems. In general, the low water solubility and moderate vapor pressure of Stoddard solvent suggest that volatilization with subsequent photooxidation in the atmosphere may be important. Even though the most volatile hydrocarbons (i.e., $< C_7$) are not expected to be major components of Stoddard solvent, volatilization from surface soils is expected to be a major fate process for the alkanes which have very low water solubility. The aromatic hydrocarbons likely to be present in Stoddard solvent are moderately soluble in water and may be available to be dissolved in and transported with infiltrating water. Sorption to organic materials may limit the actual rates of leaching and volatilization from soils.

As discussed in detail in Chapter 64, large surface spills or subsurface discharges of petroleum distillates may result in a separate organic phase on the surface of the ground water. Migration of the organic phase may be very different from that of the ground water itself and the solvent hydrocarbons dissolved in the ground water.

Biodegradation may be an important transformation process for Stoddard solvent in soil/ground-water systems; some photooxidation of surface spills may also occur. Data presented in Chapter 64 suggest that microorganisms capable of degrading C_7 to C_{12} aliphatic and aromatic hydrocarbons are not uncommon in the environment, and under conditions favorable to microbial activity, biodegradation may be rapid. It should be mentioned that Walker et al. (2257) state that even under optimum conditions, total and complete biodegradation of petroleum hydrocarbons is not expected to occur except possibly over an extremely long time period.

Overall, ground water underlying soil contaminated with Stoddard solvent hydrocarbons may be vulnerable to contamination by at least some of these components. The type of spill (surface vs. sub-surface) is of importance since volatilization from the surface may be a significant removal process particularly for the lower molecular weight

aliphatics. At this point, it should be mentioned that environmental fate/exposure/toxicology chapters for xylene and naphthalene listed in Table 67-2 were included in other chapters of the IRP Toxicology Guide.

67.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that the major components of Stoddard solvent are volatile but vary in their potential for bioaccumulation and sorption to soil. They range from moderately to strongly sorbed to soil, and their potential for bioaccumulation ranges from low to high. The variability in the properties of the components suggests they have somewhat different exposure pathways.

Spills of Stoddard solvent would result in the evaporative loss of the more highly volatile components, leaving those of lesser volatility in the soil. The fraction remaining in the soil is expected to be relatively mobile assuming the spill is large enough to exceed the sorptive capacity of the soil. Gravity will carry the bulk fluid to the saturated zone of the soil. There, the more soluble components (aromatic and lower molecular weight aliphatic compounds) will dissolve into the ground water or form emulsions with it, while the insoluble fraction will float as a separate phase on top of the water table. The movement of dissolved hydrocarbons in ground water is much greater than for the separate liquid phase, reaching distances of hundreds to thousands of meters compared to tens of meters for the separate liquid phase. In the presence of cracks and fissures, however, the flow of the separate phase is greatly enhanced.

The movement of Stoddard solvent in ground water may contaminate drinking water supplies, resulting in ingestion exposures. Groundwater discharges to surface water or the movements of contaminated soil particles to surface water drinking water supplies may also result in ingestion exposures, as well as in dermal exposures from the recreational use of these waters. The uptake of Stoddard solvent by fish and domestic animals is not expected to be a significant exposure pathway for humans because the hydrocarbons with the greatest potential for bioaccumulation, polycyclic aromatic compounds, account for such a small fraction of the mixture.

Volatilization of Stoddard solvent in soil is another potential source of human exposure. Once in the soil, the hydrocarbons evaporate, saturating the air in the soil pores, and diffusing in all directions including upward to the surface. The vapors may diffuse into the basements of homes or other structures in the area, resulting in inhalation exposures to the buildings' occupants. Exposures may be more intensive when the soil is contaminated directly from leaking underground storage tanks and pipes rather than from surface spills. In such cases the more volatile components do not have an opportunity

to evaporate before penetrating the soil. Obviously, such an exposure scenario requires a substantial release of Stoddard solvent into the soil, and is more likely to occur if the solvent is being handled in bulk rather than in drums.

67.2.4 Other Sources of Human Exposure

Data on the ambient concentrations of Stoddard solvent in air and water as well as in food and drinking water are not readily available in the literature. Exposure information on some specific components may be found in other chapters of this guide. Groups expected to receive the largest exposure to Stoddard solvent include those who use it as a solvent cleaner. Inhalation exposures are likely, as are dermal exposures if protective gloves and clothing are not worn. The same is also true for those using paints or paint thinners that contain Stoddard solvent. Dry cleaners using Stoddard solvent can also expect to experience inhalation and dermal exposures. Although traces of the solvent may remain on clothes after dry cleaning, inhalation and dermal exposures that result from wearing dry-cleaned clothes are not expected to be significant.

67.3 HUMAN HEALTH CONSIDERATIONS

67.3.1 Animal Studies

67.3.1.1 Carcinogenicity

There are no carcinogenicity data available for Stoddard solvent.

67.3.1.2 Mutagenicity

Stoddard solvent is not mutagenic in either *in vitro* or *in vivo* systems. The American Petroleum Institute evaluated Stoddard solvent in 3 tests (1914). In the Ames assay, there was no significant increase in the numbers of revertant colonies of *Salmonella typhimurium* strains TA98, 100, 1535, 1537 or 1538 both with and without microsomal activation. Negative results were also reported in the L5178Y mouse lymphoma assay and in a dominant lethal assay in which CD rats were administered ip doses of 0.087, 0.289 or 0.868 mL/kg/day for 5 days. Gochet *et al*. (1968) reported negative results in the micronucleus test on mouse bone marrow cells and in the *in vitro* induction of sister chromatid exchange in human lympohocytes.

67.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

In one study, there were no treatment-related effects on implantation, fetal resorption or number of viable fetuses after mated female CD rats were exposed to vapor levels of 100 or 300 ppm 6 hours daily on days 6 through 15 of gestation. In the high exposure group there was a statistically significant increase in the total incidence of fetuses with ossification variation but the types and relative

incidence were comparable to historical controls (1969). A study conducted by API also reported negative results in rats. No details were given (2308).

67.3.1.4 Other Toxicologic Effects

67.3.1.4.1 Short-term Toxicity

Stoddard solvent vapor is a mild narcotic and a mucous membrane irritant (46). A comprehensive series of studies have been conducted by Carpenter and associates (1970) to evaluate the toxicity of both Stoddard solvent and 140 flash aliphatic solvent. The Stoddard solvent used had a flash point of 109°F (43°C) and a boiling range of 307-382°F (153-194°C). Rats had no ill effects after 8 hours at 420 ppm while the no-effect level for dogs was 510 ppm in the same time period.

Eight hours at 1400 ppm was not lethal to rats but signs included eye irritation, bloody exudate around the nostrils and slight loss of coordination. Similar signs were seen after exposure to 800 ppm for 8 hours, but there was no loss of coordination (1970). A female beagle exposed to 1400 ppm had eye irritation, salivation, tremors and convulsions within a 5 hour period while a second was asymptomatic during and after the 8 hour inhalation period. Both animals survived. All cats inhaling 1700 ppm died within an 8 hour exposure period (1970). The 140 flash aliphatic solvent had a boiling range of 363-402°F (183-206°C). Exposure to vapor levels of 33 or 43 ppm for 8 hours had no effect on either dogs or rats, respectively. Cats exposed to vapor levels of 43 ppm for 6 hours also had no adverse effects (1971).

Rector et al. exposed rats, guinea pigs, rabbits, dogs and monkeys to mineral spirits which met Stoddard solvent specifications. The animals were exposed 8 hours daily, 5 days per week for a total of 30 exposures to vapor levels of 290 ppm. The only effects seen were minor congestion and emphysema of guinea pig lungs (1972).

Grant reported that Stoddard solvent caused little injury on direct contact with the rabbit eye (19).

67.3.1.4.2 Chronic Toxicity

There is evidence that long-term exposure to Stoddard solvent causes toxic effects on the kidneys of male rats. These changes are limited to the proximal portion of the tubule and are characterized by an increase in the incidence of regenerative tubular epithelia and hyalin droplet nephropathy (2309). Some rat strains appear to be more susceptible than others. The predisposition of male rats to the occurrence of hyalin droplets is thought to be related to the large amount of protein excreted by the male kidney (2309).

When Sprague-Dawley and Fischer 344 rats of both sexes were exposed to Stoddard solvent vapor at concentrations of 100 or 800 ppm, 6 hours daily, 5 days per week for 8 weeks, kidney changes were seen in males only. The Fischer 344 rats appeared to be slightly more responsive than were the Sprague-Dawley rats. The primary structural change was an increased incidence of regenerative tubular epithelia in the cortex. At the corticomedullary junction there were dilated tubules filled with proteinaceous material. Changes in urine parameters were observed after 4 to 8 weeks of exposure. In male rats, these included a reduction in urine concentrating ability, an increase in total urine protein and glucose and an increase in the excretion of epithelia cells in the urine. None of these changes were observed in female rats (2309).

Phillips and Egan (1974) exposed Sprague-Dawley rats of both sexes to dearomatized white spirit (flash point 104°F/40°C) at vapor levels of 300 or 900 ppm 6 hours daily, 5 days per week for up to 12 weeks. They observed nephrotoxicity in male rats only from both exposure groups. The effects began 4 weeks after the onset of exposure and were indicative of mild tubular toxicity. The incidence and severity increased with increasing concentrations and exposure duration. There were no other significant toxic effects.

In a similar study, Carpenter (1970) exposed male rats to 330 ppm for 65 days on the same dosing schedule and observed marked tubular regeneration which they attributed in part to the inherent murine nephrosis of the Harlan-Wistar rats employed. Phillips and Egan (1974) upon re-evaluation of Carpenter's data, found the kidney changes to be identical to those observed in their study. They concluded that the hydrocarbons eliciting the most pronounced renal tubular changes have a boiling range of 120-200°C and a carbon length of C8-C11.

In a 12-month study, male Sprague-Dawley rats exposed to vapor levels of 6500 mg/m^3 white spirit, 8 hours daily, 5 days per week had a decreased urinary concentrating ability, a decreased net acid excretion following a mild ammonium chloride load and an increased urinary lactate dehydrogenase (LDH) activity all of which indicate an alteration in the distal tubule of the kidney (1973).

No toxic effects were reported in male Harlan-Wistar rats exposed to 140 flash aliphatic solvent at vapor levels of up to 37 ppm, 6 hours daily, 5 days per week for 72 days or in dogs exposed for 73 days (1971).

In a 28-day dermal toxicity study, Stoddard solvent was classified as a moderate irritant in male and female animals (species was not reported) at a dose of 200 mg/kg. At a dose of 1000 mg/kg, it was a moderate irritant to females and a severe irritant to males. It was a severe irritant to both sexes at 2000 mg/kg (2310).

67.3.2 Human and Epidemiologic Studies

67.3.2.1 Short-term Toxicologic Effects

Stoddard solvent is an eye, nose and throat irritant in humans. Acute exposure to high vapor concentrations can cause headaches and produce narcotic effects (38). Pedersen and Cohr (1975) found that 6 hour exposures to vapor levels of 50-200 ppm white spirit produced dryness of the mucous membranes, anorexia, nausea, vomiting, diarrhea and fatigue. In another study, one of six volunteers exposed to a vapor level of 150 ppm for 15 minutes experienced eye irritation while all six reported irritation after 15 minutes at 470 ppm. Two subjects at this level also reported slight dizziness (1970).

Inhalation of 17-49 ppm 140 flash aliphatic solvent 15 minutes per day for 2 days caused slight temporary dryness of the eyes (1971).

Acute exposure to Stoddard solvent was also found to prolong reaction time and impair short-term memory for visual stimuli. The subjects were exposed to vapor levels of 4000 mg/m^3 for 35-40 minutes (1976).

Dermal exposures to the liquid have caused dermatitis and jaundice (38).

67.3.2.2 Chronic Toxicologic Effects

Industrial exposures to unknown but fairly high concentrations over long periods have resulted in headaches, eye, nose and throat irritation, fatigue, bone marrow hypoplasia, and in extreme cases, death (38).

NIOSH (1967) has reported numerous cases of long-term dermal and inhalation exposure.

Scott et al. (2332) reported 4 cases of aplastic anemia in individuals known to have been exposed to Stoddard solvent. Three of these cases were fatal. In the first fatality, Stoddard solvent and carbon tetrachloride exposures occurred 2 or 3 times a month for a 2-year period. At this time the patient experienced excessive uterine bleeding, purpura and moderate bone marrow hypoplasia. At autopsy, focal hyperplasia was found. In the second case, dermal exposure to Stoddard solvent occurred 4 or 5 times a week during a 6 month period. This individual had also been taking diphenhydramine and tripelennamine hydrochloride for several years to control seasonal allergies. Two months after exposure ended, symptoms of anemia were seen. Autopsy revealed moderate bone marrow hypoplasia (2332).

In the third fatal case, dermal exposure occurred over a 2-year period. Symptoms included purpura, pallor, fatigue and slightly hypoplastic bone marrow. Autopsy findings revealed marked hypoplasia. The patient had denied using other potentially toxic solvents (2332).

The fourth case was an individual who had used a Stoddard-type solvent in a large open tub, once a year for 20 years, usually indoors. A slight reduction in all formed blood elements was seen. The patient survived after a splenectomy was performed. The authors concluded that these cases implicated Stoddard-type solvents as possible myelotoxic agents but since no information was given on solvent composition, it is not possible to rule out other myelotoxic compounds such as benzene (2332).

Dermal exposure to undiluted Stoddard solvent for 10 weeks resulted in follicular dermatitis and jaundice. One year after exposure, tests revealed latent jaundice and possibly permanent liver damage (2333).

67.3.3 Levels of Concern

OSHA (298) currently permits exposure to 500 ppm as an 8-hour time-weighted-average. The ACGIH (3) has set a time-weighted-average of 100 ppm, with a short-term exposure limit of 200 ppm.

67.3.4 Hazard Assessment

No carcinogenicity tests have been conducted for Stoddard solvent. Mutagenicity data are negative for bacteria and mammalian cells in culture; negative results were also obtained in a rat dominant lethal study and mouse micronucleus test (1914,1968).

Exposure of pregnant rats to vapor levels of 300 ppm, 6 hours daily during gestation was without effect (1969).

Animal studies indicate mild narcotic effects and irritation of mucous membranes with acute exposure (46). Long-term exposures result in kidney damage (2309), particularly in male rats. The incidence and severity of renal toxicity appeared to increase with concentration and exposure duration (1974,2309,1970).

In humans, acute exposure produce eye, nose and throat irritation, nausea, vomiting, diarrhea and fatigue (38,1975). High vapor concentrations can produce headaches and narcotic effects (38). Prolonged industrial exposures to very high concentrations of Stoddard solvent have been linked to fatigue and bone marrow hypoplasia (38,1967); it is unclear, however, if other myelotoxic solvents were also involved.

67.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of Stoddard solvent in soil and water requires collection of a representative field sample and laboratory analysis for the specific major components attributed to Stoddard solvent; however, the relative concentrations of the constituents, and even the constituents themselves, will vary with time and distance from

the site of initial contamination due to weathering. The major component categories in Stoddard solvent have been identified as the following:

n-alkanes
branched alkanes
cycloalkanes
benzene and alkylbenzenes

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques may be used to identify the principal components in Stoddard solvent. Samples, and probably any samples collected in the field which are primarily organic in nature, may require the separation (prior to GC or GC/MS analysis) of the aliphatic and aromatic hydrocarbon fractions using liquid solid column chromatography; the various column eluates, with or without dilution in carbon disulfide, can then be analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (i.e., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet extraction or sonication methods (1422). An aliquot of the sample extract, with or without concentration, is then analyzed by GC or GC/MS. (Sampling and Analysis Considerations for some specific components in Stoddard solvent, i.e., benzene, toluene, xylenes and ethyl benzene, have been addressed in Volume 1.)

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component, but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorotrifluoroethane). The "oil and grease" content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; extraction methods are those described above for aqueous and soil samples.

A detection limit for Stoddard solvent was not determined; the detection limit for specific components is expected to be in the range of $\mu g/L$ for aqueous samples and $\mu g/g$ for non-aqueous samples.

COMMON SYNONYMS:	CAS REG. NO.:	AIR W/V CONVERSION FACTORS at 25°C (1967)
White spirits Mineral spirits Solvent naphtha Dry cleaning safety solvent	8052-41-3 NIOSH NO.: WJ8925000	5.77 mg/m³ = 1 ppm 0.173 ppm = 1 mg/m³
	APPROXIMATE COMPOSITION: linear and branched alkanes, 30-50% cycloalkanes, 30-40% aromatics, 10-20% benzene, trace olefins, trace	MOLECULAR WEIGHT: 135-145 (average)

REACTIVITY	Stoddard solvent is considered to be a miscellaneous combustible material for compatibility classification purposes. Reactions of such substances with non-oxidizing mineral acids may evolve heat and usually innocuous gases. Those with oxidizing mineral acids or organic peroxides or hydroperoxides may produce heat, fire, and toxic gases, while those with strong oxidizing agents or alkali or alkaline earth elemental metals may produce heat, fire, and innocuous gases. Nitrides evolve heat, fire, and flammable gases. Strong reducing agents evolve heat and flammable gases. Reactions with explosive materials may result in an explosion. There are also unspecified incompatibilities with bases and selected amines (38,507,511).

PHYSCIO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(2)
	• Color: colorless	(2)
	• Odor: mild petroleum	(507)
	• Odor Threshold: 0.9 ppm	(1970)
	• Liquid Density (g/ml at 20°C): 0.77	(507)
	• Freezing/Melting Point (°C): no data	( )
	• Boiling Point (°C): 154-202°	(2)
	• Flash Point (°C): 37.8-60 (variable)	(23,38, 51,507)
	• Flammable Limits in Air, % by Volume: (0.8-1.1) - 6.0	(38,51,506)
	• Autoignition Temperature (°C): 227-260 (variable)	(23,38, 51,506)
	• Vapor Pressure (mm Hg at 20°C): 3	(507)
	• Saturated Concentration in Air (mg/m³ at 20°C): 2.2 x 10^4 to 2.4 x 10^4	(ADL estim)
	• Solubility in Water (mg/L at 20°C): insoluble	(507)
	• Viscosity (cp at 20°C): 0.91-0.95	(5)
	• Surface Tension (dyne/cm at 20°C): no data	( )

PHYSICO-CHEMICAL DATA (continued)	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 3.16-7.06 (*) • Soil Adsorption Coefficient, K_{oc}: 700-5.5 x 10^6 (*) • Henry's Law Constant (atm•m^3/mol at 20°C): 4.4 x 10^{-4} - 7.4 (*) • Bioconcentration Factor: no data ( )
PERSISTENCE IN THE SOIL-WATER SYSTEM	Stoddard solvent hydrocarbons are expected to be relatively mobile and moderately persistent in most soil systems. Persistence in deep soils and ground water may be higher. Volatilization, photooxidation and biodegradation are potentially important fate processes. Surface spills are expected to be weathered by evaporation and photooxidation. Downward migration of weathered surface spills and sub-surface discharges represent a potential threat to underlying ground water. Biodegradation of C_7-C_{12} hydrocarbons is expected to be significant under environmental conditions favorable to microbial oxidation; naturally-occurring, hydrocarbon-degrading microorganisms have been isolated from polluted soils and, to a lesser extent, non-polluted soils.
PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water systems is the contamination of ground water drinking water supplies resulting from large spills of Stoddard solvent or leaking underground storage tanks. Vapors from leaked or spilled solvent may diffuse through soils and migrate into structures resulting in inhalation exposures. Inhalation exposures may also occur from the direct volatilization of surface spills. Ingestion with food is not expected to be significant.
HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (38): Overexposure to Stoddard solvent causes irritation of the eyes, nose and throat and may cause dizziness. Prolonged overexposure to the liquid may cause skin irritation.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral -- no data skin -- no data LCLo (mg/m^3) inhalation [cat] (47) 10,000•2.5 hr.
	Long-Term Effects: Kidney damage
	Pregnancy/Neonate Data: Negative
	Mutation Data: Negative
	Carcinogenicity: No data

*Range of values for representative hydrocarbons from major component classes (see Table 67-2).

HANDLING PRECAUTIONS (38,507)	Handle only with adequate ventilation • Vapor levels of 500 to 1000 ppm: chemical cartridge respirator with a full facepiece and organic vapor cartridges • 1000 to 5000 ppm: any supplied-air respirator or self-contained breathing apparatus with full facepiece; gas mask with organic vapor canister • Chemical goggles if there is probability of eye contact • The use of impermeable gloves is advised to prevent skin irritation.

EMERGENCY FIRST AID TREATMENT (38,507)	Ingestion: Do not induce vomiting. Get immediate medical attention • Inhalation: Move victim to fresh air. Give artificial respiration if necessary. Get medical attention • Skin: Remove contaminated clothing. Wash skin with soap and water. If irritation persists after washing, get medical attention • Eye: Flush with large amounts of water for 15 minutes. If irritation persists, get medical attention.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): 500 ppm
- AFOSH PEL (8-hr TWA): 500 ppm

Criteria
- NIOSH IDLH (30-min): 5000 ppm
- ACGIH TLV® (8-hr TWA): 100 ppm
- ACGIH STEL (15-min): 200 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; Stoddard solvent is not a priority pollutant.
- Aquatic Life
  No criterion established; Stoddard solvent is not a priority pollutant.

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:

- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;
- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;
- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Occupational Safety and Health Act (OSHA)
  Employee exposure to Stoddard solvent shall not exceed an 8-hour time-weighted-average of 500 ppm (298).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated petroleum naphtha as a hazardous material which is subject to requirements for packaging, labeling and transportation (306).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Petroleum and coal tar distillates with flash points below 21°C are classified as flammable substances and are subject to packaging and labeling regulations. Because of the variable composition of other petroleum and coal tar distillates (excluding those used as motor

fuels), they are considered preparations and their labeling shall be done in accordance with the procedures outlined in the Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparations (solvents).

EEC Directives - Proposed

Proposal for a Council Directive on the Dumping of Waste at Sea (1793) EEC has proposed that the dumping of crude oil, petroleum hydrocarbons, lubricants and hydraulic fluids at sea be prohibited.

67.1 MAJOR USES AND COMPOSITION

67.1.1 Major Uses

Stoddard solvent is produced from a straight-run distillate of paraffinic or mixed base crude oil. It is used as a diluent in paints, coatings and waxes; as a dry cleaning agent; as a degreaser and cleaner and as a herbicide (2).

67.1.2 Composition

Stoddard solvent is a mixture of C_7 through C_{12} hydrocarbons, predominantly C_9 through C_{11}, with a boiling range between 160°C to 210°C. Flashpoint dry-point test and odor data are used to classify Stoddard solvent into the following four types: regular Stoddard solvent, 140 flash solvent, odorless solvent, and low end point solvent. Chemically, Stoddard solvent is a mixture of 30-50% straight and branched alkanes, 30-40% cycloalkanes, and 10-20% aromatics. Benzene and olefins are present in trace quantities only (1967,2228). The 140 flash aliphatic solvent is composed of organic compounds with carbon chain lengths ranging from C5 to C12. Its boiling range is 185-207°C and it is composed of 60.8% paraffins, 24.5% monocycloparaffins, 11.2% dicycloparaffins, 3.03% alkyl benzenes, 0.3% indans and tetralins, and 0.07% benzenes (1967). Both types will be discussed in some sections of the chapter which follows.

A characterization of the individual hydrocarbon components of Stoddard solvent was not available. Table 67-1 presents the available characterization by chemical classes.

67.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

A discussion of the environmental behavior of Stoddard solvent is limited by the lack of analytical data defining its specific components. Many of the hydrocarbons expected to be components of Stoddard solvent were addressed previously in the more extensive environmental fate section of Chapter 64. The general discussions of aliphatic and aromatic hydrocarbons and their behavior in soil/ground-water systems will not be repeated here; the reader is referred to the relevant sections of Chapter 64.

67.2.1 Equilibrium Partitioning Model

In general, soil/ground-water transport pathways for low concentrations of pollutants in soil can be assessed by using an equilibrium partitioning model. For the purposes of assessing the environmental transport of Stoddard solvent, a group of specific hydrocarbons within the C_7-C_{12} range was selected from the dominant hydrocarbon classes, i.e., alkanes, cycloalkanes, and aromatics; there are no available data to confirm the presence of the selected

TABLE 67-1

Composition Data for Stoddard Solvent
(Reference 1967)

Carbon Range	C_7-C_{12}
Straight/Branched Alkanes	48%
Cycloalkanes	38%
Aromatics	
Benzenes	0.1%
Alkylbenzenes	14%
Indans/Tetralins	< 1%

hydrocarbons in a typical Stoddard solvent sample. Table 67-2 identifies the selected hydrocarbons and presents the predicted partitioning of low soil concentrations of those hydrocarbons among soil particles, soil water, and soil air. The portions associated with the water and air phases of the soil are expected to have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil indicate that sorption is expected to be an important process for all the dominant hydrocarbon categories. Partitioning to the soil-vapor phase in this model is not very important for the C_7-C_{12} hydrocarbons. The alkyl benzenes have higher water solubilities and transport with infiltrating water may be important for these compounds; volatilization is still expected to be low. In saturated, deep soils (containing no soil air and negligible soil organic carbon), a significant percent of the aromatic hydrocarbons is predicted to be present in the soil-water phase and available for transport with flowing ground water.

In interpreting these results, it must be remembered that this model is valid only for low soil concentrations (below aqueous solubility) of the components. Large releases of solvent (spills, leaking underground storage tanks) may exceed the sorptive capacity of the soil, thereby filling the pore spaces of the soil. In this situation, the hydrocarbon mixture would move as a bulk fluid and the equilibrium partitioning model would not be applicable.

TABLE 67-2

EQUILIBRIUM PARTIONING OF POTENTIAL
STODDARD SOLVENT HYDROCARBONS IN MODEL ENVIRONMENTS[a]

COMPOUND	Log K_{ow}	K_{oc}[b]	H[c]	UNSATURATED TOPSOIL (%)			SATURATED DEEP SOIL[d] (%)	
				Soil	Water	Air	Soil	Water
Octane	5.18 (e)	73,000	2.96	97.4	0.01	2.6	97.7	0.3
Dodecane	7.06 (f)	5.5×10^6	7.4	99.9	0.0001	0.09	99.9	0.004
Trimethylpentane	4.87 (f)	36,000	1.9-3.3	94.7	0.01	5.3	99.3	0.7
Methylcyclohexane	4.10 (f)	6,070	0.39	95.9	0.08	4.0	96.2	3.8
Trimethylcyclohexane	5.02 (h)	50,500	1.6	98.0	0.01	2.0	99.5	0.5
Xylenes	3.16 (e)	700	7×10^{-3}	98.8	0.7	0.5	74.4	25.6
Trimethylbenzenes	3.65 (h)	2,150	5×10^{-3}	99.6	0.2	0.2	90.0	10.0
Naphthalene	3.30 (e)	962	4.82×10^{-4}	99.4	0.5	0.03	80.2	19.8
Methylnaphthalenes	3.87 (e)	3,570	4.4×10^{-4}	99.8	0.1	0.01	93.7	6.3

[a] Calculations based on Mackay's equilibrium partioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

[b] Reference 652.

[c] Taken from Reference 74 unless otherwise specified. Units equal $atm \cdot m^3/mol$.

[d] Used sorption coefficient $K_p = 0.001 \times K_{oc}$.

[e] Reference 29.

[f] Arthur D. Little, Inc., estimate according to equations provided in Reference 31.

[g] Reference 10.

[h] Reference 31.

67.2.2 Transport and Transformation Processes

Transport and transformation of Stoddard solvent constituents will depend on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly in the percolating ground waters and be sorbed less strongly on the soils, thus being transported more rapidly, and may or may not be susceptible to degradation by chemical or biological action. Thus, as was shown in Figure 65-1, the relative concentrations of the constituents of the solvent will vary with time and distance from the site of contamination. This effect is called "weathering." (This term is also used to describe the changes to petroleum materials following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation are all involved.)

There are no available data specific to the transport and transformation of Stoddard solvent in soil/ground-water systems. In general, the low water solubility and moderate vapor pressure of Stoddard solvent suggest that volatilization with subsequent photooxidation in the atmosphere may be important. Even though the most volatile hydrocarbons (i.e., < C_7) are not expected to be major components of Stoddard solvent, volatilization from surface soils is expected to be a major fate process for the alkanes which have very low water solubility. The aromatic hydrocarbons likely to be present in Stoddard solvent are moderately soluble in water and may be available to be dissolved in and transported with infiltrating water. Sorption to organic materials may limit the actual rates of leaching and volatilization from soils.

As discussed in detail in Chapter 64, large surface spills or subsurface discharges of petroleum distillates may result in a separate organic phase on the surface of the ground water. Migration of the organic phase may be very different from that of the ground water itself and the solvent hydrocarbons dissolved in the ground water.

Biodegradation may be an important transformation process for Stoddard solvent in soil/ground-water systems; some photooxidation of surface spills may also occur. Data presented in Chapter 64 suggest that microorganisms capable of degrading C_7 to C_{12} aliphatic and aromatic hydrocarbons are not uncommon in the environment, and under conditions favorable to microbial activity, biodegradation may be rapid. It should be mentioned that Walker et al. (2257) state that even under optimum conditions, total and complete biodegradation of petroleum hydrocarbons is not expected to occur except possibly over an extremely long time period.

Overall, ground water underlying soil contaminated with Stoddard solvent hydrocarbons may be vulnerable to contamination by at least some of these components. The type of spill (surface vs. sub-surface) is of importance since volatilization from the surface may be a significant removal process particularly for the lower molecular weight

aliphatics. At this point, it should be mentioned that environmental fate/exposure/toxicology chapters for xylene and naphthalene listed in Table 67-2 were included in other chapters of the IRP Toxicology Guide.

67.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that the major components of Stoddard solvent are volatile but vary in their potential for bioaccumulation and sorption to soil. They range from moderately to strongly sorbed to soil, and their potential for bioaccumulation ranges from low to high. The variability in the properties of the components suggests they have somewhat different exposure pathways.

Spills of Stoddard solvent would result in the evaporative loss of the more highly volatile components, leaving those of lesser volatility in the soil. The fraction remaining in the soil is expected to be relatively mobile assuming the spill is large enough to exceed the sorptive capacity of the soil. Gravity will carry the bulk fluid to the saturated zone of the soil. There, the more soluble components (aromatic and lower molecular weight aliphatic compounds) will dissolve into the ground water or form emulsions with it, while the insoluble fraction will float as a separate phase on top of the water table. The movement of dissolved hydrocarbons in ground water is much greater than for the separate liquid phase, reaching distances of hundreds to thousands of meters compared to tens of meters for the separate liquid phase. In the presence of cracks and fissures, however, the flow of the separate phase is greatly enhanced.

The movement of Stoddard solvent in ground water may contaminate drinking water supplies, resulting in ingestion exposures. Groundwater discharges to surface water or the movements of contaminated soil particles to surface water drinking water supplies may also result in ingestion exposures, as well as in dermal exposures from the recreational use of these waters. The uptake of Stoddard solvent by fish and domestic animals is not expected to be a significant exposure pathway for humans because the hydrocarbons with the greatest potential for bioaccumulation, polycyclic aromatic compounds, account for such a small fraction of the mixture.

Volatilization of Stoddard solvent in soil is another potential source of human exposure. Once in the soil, the hydrocarbons evaporate, saturating the air in the soil pores, and diffusing in all directions including upward to the surface. The vapors may diffuse into the basements of homes or other structures in the area, resulting in inhalation exposures to the buildings' occupants. Exposures may be more intensive when the soil is contaminated directly from leaking underground storage tanks and pipes rather than from surface spills. In such cases the more volatile components do not have an opportunity

to evaporate before penetrating the soil. Obviously, such an exposure scenario requires a substantial release of Stoddard solvent into the soil, and is more likely to occur if the solvent is being handled in bulk rather than in drums.

67.2.4 Other Sources of Human Exposure

Data on the ambient concentrations of Stoddard solvent in air and water as well as in food and drinking water are not readily available in the literature. Exposure information on some specific components may be found in other chapters of this guide. Groups expected to receive the largest exposure to Stoddard solvent include those who use it as a solvent cleaner. Inhalation exposures are likely, as are dermal exposures if protective gloves and clothing are not worn. The same is also true for those using paints or paint thinners that contain Stoddard solvent. Dry cleaners using Stoddard solvent can also expect to experience inhalation and dermal exposures. Although traces of the solvent may remain on clothes after dry cleaning, inhalation and dermal exposures that result from wearing dry-cleaned clothes are not expected to be significant.

67.3 HUMAN HEALTH CONSIDERATIONS

67.3.1 Animal Studies

67.3.1.1 Carcinogenicity

There are no carcinogenicity data available for Stoddard solvent.

67.3.1.2 Mutagenicity

Stoddard solvent is not mutagenic in either *in vitro* or *in vivo* systems. The American Petroleum Institute evaluated Stoddard solvent in 3 tests (1914). In the Ames assay, there was no significant increase in the numbers of revertant colonies of *Salmonella typhimurium* strains TA98, 100, 1535, 1537 or 1538 both with and without microsomal activation. Negative results were also reported in the L5178Y mouse lymphoma assay and in a dominant lethal assay in which CD rats were administered ip doses of 0.087, 0.289 or 0.868 mL/kg/day for 5 days. Gochet *et al.* (1968) reported negative results in the micronucleus test on mouse bone marrow cells and in the *in vitro* induction of sister chromatid exchange in human lympohocytes.

67.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

In one study, there were no treatment-related effects on implantation, fetal resorption or number of viable fetuses after mated female CD rats were exposed to vapor levels of 100 or 300 ppm 6 hours daily on days 6 through 15 of gestation. In the high exposure group there was a statistically significant increase in the total incidence of fetuses with ossification variation but the types and relative

incidence were comparable to historical controls (1969). A study conducted by API also reported negative results in rats. No details were given (2308).

67.3.1.4 Other Toxicologic Effects

67.3.1.4.1 Short-term Toxicity

Stoddard solvent vapor is a mild narcotic and a mucous membrane irritant (46). A comprehensive series of studies have been conducted by Carpenter and associates (1970) to evaluate the toxicity of both Stoddard solvent and 140 flash aliphatic solvent. The Stoddard solvent used had a flash point of 109°F (43°C) and a boiling range of 307-382°F (153-194°C). Rats had no ill effects after 8 hours at 420 ppm while the no-effect level for dogs was 510 ppm in the same time period.

Eight hours at 1400 ppm was not lethal to rats but signs included eye irritation, bloody exudate around the nostrils and slight loss of coordination. Similar signs were seen after exposure to 800 ppm for 8 hours, but there was no loss of coordination (1970). A female beagle exposed to 1400 ppm had eye irritation, salivation, tremors and convulsions within a 5 hour period while a second was asymptomatic during and after the 8 hour inhalation period. Both animals survived. All cats inhaling 1700 ppm died within an 8 hour exposure period (1970). The 140 flash aliphatic solvent had a boiling range of 363-402°F (183-206°C). Exposure to vapor levels of 33 or 43 ppm for 8 hours had no effect on either dogs or rats, respectively. Cats exposed to vapor levels of 43 ppm for 6 hours also had no adverse effects (1971).

Rector et al. exposed rats, guinea pigs, rabbits, dogs and monkeys to mineral spirits which met Stoddard solvent specifications. The animals were exposed 8 hours daily, 5 days per week for a total of 30 exposures to vapor levels of 290 ppm. The only effects seen were minor congestion and emphysema of guinea pig lungs (1972).

Grant reported that Stoddard solvent caused little injury on direct contact with the rabbit eye (19).

67.3.1.4.2 Chronic Toxicity

There is evidence that long-term exposure to Stoddard solvent causes toxic effects on the kidneys of male rats. These changes are limited to the proximal portion of the tubule and are characterized by an increase in the incidence of regenerative tubular epithelia and hyalin droplet nephropathy (2309). Some rat strains appear to be more susceptible than others. The predisposition of male rats to the occurrence of hyalin droplets is thought to be related to the large amount of protein excreted by the male kidney (2309).

When Sprague-Dawley and Fischer 344 rats of both sexes were exposed to Stoddard solvent vapor at concentrations of 100 or 800 ppm, 6 hours daily, 5 days per week for 8 weeks, kidney changes were seen in males only. The Fischer 344 rats appeared to be slightly more responsive than were the Sprague-Dawley rats. The primary structural change was an increased incidence of regenerative tubular epithelia in the cortex. At the corticomedullary junction there were dilated tubules filled with proteinaceous material. Changes in urine parameters were observed after 4 to 8 weeks of exposure. In male rats, these included a reduction in urine concentrating ability, an increase in total urine protein and glucose and an increase in the excretion of epithelia cells in the urine. None of these changes were observed in female rats (2309).

Phillips and Egan (1974) exposed Sprague-Dawley rats of both sexes to dearomatized white spirit (flash point 104°F/40°C) at vapor levels of 300 or 900 ppm 6 hours daily, 5 days per week for up to 12 weeks. They observed nephrotoxicity in male rats only from both exposure groups. The effects began 4 weeks after the onset of exposure and were indicative of mild tubular toxicity. The incidence and severity increased with increasing concentrations and exposure duration. There were no other significant toxic effects.

In a similar study, Carpenter (1970) exposed male rats to 330 ppm for 65 days on the same dosing schedule and observed marked tubular regeneration which they attributed in part to the inherent murine nephrosis of the Harlan-Wistar rats employed. Phillips and Egan (1974) upon re-evaluation of Carpenter's data, found the kidney changes to be identical to those observed in their study. They concluded that the hydrocarbons eliciting the most pronounced renal tubular changes have a boiling range of 120-200°C and a carbon length of C8-C11.

In a 12-month study, male Sprague-Dawley rats exposed to vapor levels of 6500 mg/m^3 white spirit, 8 hours daily, 5 days per week had a decreased urinary concentrating ability, a decreased net acid excretion following a mild ammonium chloride load and an increased urinary lactate dehydrogenase (LDH) activity all of which indicate an alteration in the distal tubule of the kidney (1973).

No toxic effects were reported in male Harlan-Wistar rats exposed to 140 flash aliphatic solvent at vapor levels of up to 37 ppm, 6 hours daily, 5 days per week for 72 days or in dogs exposed for 73 days (1971).

In a 28-day dermal toxicity study, Stoddard solvent was classified as a moderate irritant in male and female animals (species was not reported) at a dose of 200 mg/kg. At a dose of 1000 mg/kg, it was a moderate irritant to females and a severe irritant to males. It was a severe irritant to both sexes at 2000 mg/kg (2310).

67.3.2 Human and Epidemiologic Studies

67.3.2.1 Short-term Toxicologic Effects

Stoddard solvent is an eye, nose and throat irritant in humans. Acute exposure to high vapor concentrations can cause headaches and produce narcotic effects (38). Pedersen and Cohr (1975) found that 6 hour exposures to vapor levels of 50-200 ppm white spirit produced dryness of the mucous membranes, anorexia, nausea, vomiting, diarrhea and fatigue. In another study, one of six volunteers exposed to a vapor level of 150 ppm for 15 minutes experienced eye irritation while all six reported irritation after 15 minutes at 470 ppm. Two subjects at this level also reported slight dizziness (1970).

Inhalation of 17-49 ppm 140 flash aliphatic solvent 15 minutes per day for 2 days caused slight temporary dryness of the eyes (1971).

Acute exposure to Stoddard solvent was also found to prolong reaction time and impair short-term memory for visual stimuli. The subjects were exposed to vapor levels of 4000 mg/m^3 for 35-40 minutes (1976).

Dermal exposures to the liquid have caused dermatitis and jaundice (38).

67.3.2.2 Chronic Toxicologic Effects

Industrial exposures to unknown but fairly high concentrations over long periods have resulted in headaches, eye, nose and throat irritation, fatigue, bone marrow hypoplasia, and in extreme cases, death (38).

NIOSH (1967) has reported numerous cases of long-term dermal and inhalation exposure.

Scott et al. (2332) reported 4 cases of aplastic anemia in individuals known to have been exposed to Stoddard solvent. Three of these cases were fatal. In the first fatality, Stoddard solvent and carbon tetrachloride exposures occurred 2 or 3 times a month for a 2-year period. At this time the patient experienced excessive uterine bleeding, purpura and moderate bone marrow hypoplasia. At autopsy, focal hyperplasia was found. In the second case, dermal exposure to Stoddard solvent occurred 4 or 5 times a week during a 6 month period. This individual had also been taking diphenhydramine and tripelennamine hydrochloride for several years to control seasonal allergies. Two months after exposure ended, symptoms of anemia were seen. Autopsy revealed moderate bone marrow hypoplasia (2332).

In the third fatal case, dermal exposure occurred over a 2-year period. Symptoms included purpura, pallor, fatigue and slightly hypoplastic bone marrow. Autopsy findings revealed marked hypoplasia. The patient had denied using other potentially toxic solvents (2332).

The fourth case was an individual who had used a Stoddard-type solvent in a large open tub, once a year for 20 years, usually indoors. A slight reduction in all formed blood elements was seen. The patient survived after a splenectomy was performed. The authors concluded that these cases implicated Stoddard-type solvents as possible myelotoxic agents but since no information was given on solvent composition, it is not possible to rule out other myelotoxic compounds such as benzene (2332).

Dermal exposure to undiluted Stoddard solvent for 10 weeks resulted in follicular dermatitis and jaundice. One year after exposure, tests revealed latent jaundice and possibly permanent liver damage (2333).

67.3.3 Levels of Concern

OSHA (298) currently permits exposure to 500 ppm as an 8-hour time-weighted-average. The ACGIH (3) has set a time-weighted-average of 100 ppm, with a short-term exposure limit of 200 ppm.

67.3.4 Hazard Assessment

No carcinogenicity tests have been conducted for Stoddard solvent. Mutagenicity data are negative for bacteria and mammalian cells in culture; negative results were also obtained in a rat dominant lethal study and mouse micronucleus test (1914,1968).

Exposure of pregnant rats to vapor levels of 300 ppm, 6 hours daily during gestation was without effect (1969).

Animal studies indicate mild narcotic effects and irritation of mucous membranes with acute exposure (46). Long-term exposures result in kidney damage (2309), particularly in male rats. The incidence and severity of renal toxicity appeared to increase with concentration and exposure duration (1974,2309,1970).

In humans, acute exposure produce eye, nose and throat irritation, nausea, vomiting, diarrhea and fatigue (38,1975). High vapor concentrations can produce headaches and narcotic effects (38). Prolonged industrial exposures to very high concentrations of Stoddard solvent have been linked to fatigue and bone marrow hypoplasia (38,1967); it is unclear, however, if other myelotoxic solvents were also involved.

67.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of Stoddard solvent in soil and water requires collection of a representative field sample and laboratory analysis for the specific major components attributed to Stoddard solvent; however, the relative concentrations of the constituents, and even the constituents themselves, will vary with time and distance from

the site of initial contamination due to weathering. The major component categories in Stoddard solvent have been identified as the following:

n-alkanes
branched alkanes
cycloalkanes
benzene and alkylbenzenes

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques may be used to identify the principal components in Stoddard solvent. Samples, and probably any samples collected in the field which are primarily organic in nature, may require the separation (prior to GC or GC/MS analysis) of the aliphatic and aromatic hydrocarbon fractions using liquid solid column chromatography; the various column eluates, with or without dilution in carbon disulfide, can then be analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (i.e., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet extraction or sonication methods (1422). An aliquot of the sample extract, with or without concentration, is then analyzed by GC or GC/MS. (Sampling and Analysis Considerations for some specific components in Stoddard solvent, i.e., benzene, toluene, xylenes and ethyl benzene, have been addressed in Volume 1.)

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component, but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorotrifluoroethane). The "oil and grease" content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; extraction methods are those described above for aqueous and soil samples.

A detection limit for Stoddard solvent was not determined; the detection limit for specific components is expected to be in the range of µg/L for aqueous samples and µg/g for non-aqueous samples.

COMPOSITION:

Mineral Base	Synthetic	
Linear and branched chained aliphatics	Polyglycols	Olefin oligomers
Oil in water emulsions	Phosphate esters	Alkylated aromatics
Water in oil emulsions	Silicate esters	Polybutenes
	Silicones	Cycloaliphatics
	Organic esters	Polyphenyl ethers

REACTIVITY

Many hydraulic fluids primarily consist of a blend of various hydrocarbons. Hydrocarbons are typically incompatible with strong acids, alkalies, and strong oxidizers, and may be considered miscellaneous combustible or flammable materials for compatibility classification purposes. Such substances typically evolve heat, fire, and toxic or flammable gases in reactions with oxidizing mineral acids, alkali or alkaline earth elemental metals, nitrides, organic peroxides or hydroperoxides, or strong oxidizing agents. Reactions with explosive materials may result in an explosion, while those with strong reducing agents may evolve heat and flammable gases. Non-oxidizing mineral acids generally evolve heat and innocuous gases.

Other types of hydraulic fluids may include or be comprised of various types of glycols, glycol ethers, esters, and various additives. Reactivity hazards for these must be determined on a case-by-case basis (23,505,507,511).

PHYSICO-CHEMICAL DATA

• Physical State (at 20°C): liquid	(23)
• Color: yellow brown; varies with use	(60)
• Odor: odorless to slight ammonia	(2233)
• Odor Threshold: no data	( )
• Liquid Density (g/ml at 20°C): 0.902	(60)
• Freezing/Melting Point (°C): not pertinent	(60)
• Boiling Point (°C): 190.5-287.8	(23)
• Flash Point (°C): varies with particular blend and product	( )
• Flammable Limits in Air, % by Volume: no data	( )
• Autoignition Temperature (°C): no data	( )
• Vapor Pressure (mm Hg at 20°C): no data	( )
• Saturated Concentration in Air (mg/m^3 at 20°C): not pertinent	( )
• Solubility in Water (mg/L at 20°C): no data	( )
• Viscosity (cp): 56-150 at 40°C	(21)
• Surface Tension (dyne/cm at 20°C): 36-37.5	(60)
• Log (Octanol-Water Partition Coefficient), log K_{ow}: not available	( )
• Soil Adsorption Coefficient, K_{oc}: not available	( )
• Henry's Law Constant ($atm \cdot m^3/mol$ at 20°C): not available	( )
• Bioconcentration Factor: not available	( )

PERSISTENCE IN THE SOIL-WATER SYSTEM	Hydrocarbon-based fluids are expected to be highly immobile and persistent in the soil/ground-water system. Major loss mechanisms are volatilization and aerobic biodegradation. Other ester, ether and glycol-based oils may be moderately mobile and much less persistent due to hydrolysis and biodegradation.

PATHWAYS OF EXPOSURE	The primary pathway of concern from the soil/ground-water system is the contamination of ground water drinking water supplies with hydraulic fluids, especially those based on organic and phosphate esters and polyglycols. Runoff to surface water drinking water supplies may be an important exposure pathway for mineral-oil based fluids. Inhalation exposures and ingestion with food are not expected to be significant.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (60): Minimal gastrointestinal tract irritation is expected from ingestion of hydraulic fluids. Diarrhea may occur. Pulmonary irritation may result from aspiration. Skin or eye contact may produce irritation.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral -- no data skin -- no data LC_{50} (mg/m^3) inhalation -- no data
	Long-Term Effects: No data
	Pregnancy/Neonate Data: No data
	Mutation Data: No data
	Carcinogenicity: No data

HANDLING PRECAUTIONS (60)	Wear protective gloves; goggles or faceshield.

EMERGENCY FIRST AID TREATMENT (60)	Ingestion: Do not induce vomiting. If conscious, have victim drink water or milk. Get medical attention • Inhalation: Move victim to fresh air immediately. If necessary, perform artificial respiration. Get medical attention • Skin: Remove contaminated clothing and wash affected areas with soap and water. If irritation develops, get medical attention • Eye: Flush eye with large amounts of water. Get medical attention.

Acute toxicity resulting from the ingestion of several ounces of pyridine produced severe vomiting, diarrhea, hyperpyrexia and delerium. Death occurred 43 hours post-ingestion. Autopsy revealed pulmonary edema and membranous tracheobronchitis which was thought to result from aspiration of pyridine into the lung. A small oral dose of 2 to 3 mL pyridine in man produced mild anorexia, nausea, fatigue and mental depression (17).

Hydroquinone

Hydroquinone is irritating to the skin but not corrosive. Skin lesions in man are generally described as depigmentation. Fatal human doses range from 5 to 12 grams. Systemic effects include tremors and convulsions plus occasional, severe hemolytic anemia. No effect was reported following human ingestion of 300 to 500 mg hydroquinone daily for three to five months (17).

68.3.4 Levels of Concern

No criteria or standards specific for hydraulic fluid were located. EPA (2012) does list a criterion for oil and grease which requires domestic water supplies to be virtually free from oil and grease, particularly with regard to taste and odor.

68.3.5 Hazard Assessment

Toxicological data located for hydraulic fluids are scant. No data are currently available regarding the carcinogenicity, mutagenicity or reproductive effects of these materials. Limited animal studies suggest low toxicity by oral and dermal routes in rats and rabbits (2231,1936) but also indicate the potential for increased toxicity due to additives used in various formulations (2233). In general, hydraulic fluids do not appear to be eye or skin irritants although specific formulations have produced sensitization (1936).

Long-term inhalation exposure to a mist of phosphate-based hydraulic fluid at concentrations up to 110 mg/m^3 continuously for up to 163 days produced no significant pathology in dogs, monkeys or rats; limb paralysis was noted in rabbits and chickens which were indistinguishable from effects induced by TOCP (2230). Another inhalation study resulted in the death of treated rabbits exposed to 100 mg/m^3 of a phosphate-based hydraulic fluid for up to 49 exposures (2233). Similarly exposed rats exhibited a rough coat, poor grooming and a decrease in body weight gain (2233).

68.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the presence of hydraulic fluids in soil and water requires the collection of a representative field sample and laboratory analysis for the specific major components generally attributed to hydraulic fluids; however, the relative concentrations of

the constituents, and even the constituents themselves, will vary with time and distance from the site of initial contamination due to weathering. The major component categories in hydraulic fluids have been identified as the following:

Straight and branched chain aliphatic hydrocarbons (paraffins)
Cycloparaffins
Aromatic hydrocarbons
Organic esters
Polyglycols
Phosphate esters
Silicones and silicate esters

A combination of capillary column gas chromatography (GC) and gas chromatography/mass spectrometry (GC/MS) techniques may be used to identify the principal components in hydraulic fluids. Oil samples, and any samples collected in the field which are primarily organic in nature, may require separation (prior to GC or GC/MS analysis) using liquid solid column chromatography; the various column eluates, with or without dilution in carbon disulfide, can then be analyzed by GC or GC/MS techniques. Aqueous samples need to be liquid-liquid extracted with an appropriate solvent (e.g., trichlorotrifluoroethane) prior to analysis; solid samples would be extracted with trichlorotrifluoroethane using soxhlet or sonication methods. An aliquot of the sample extract, with or without concentration, could then be analyzed by GC or GC/MS for the specific components of interest. (Sampling and analysis considerations for some specific components possibly present in hydraulic fluids, i.e., benzene, toluene, xylenes, ethyl benzene, naphthalene, TOCP and ethylene glycol, have been addressed in previous chapters.)

Alternatively, the "oil and grease" content can be measured. This determination would not be the measurement of an absolute quantity of a specific component but rather the quantitative determination of groups of components with similar physical characteristics (i.e., common solubility in trichlorofluoroethane). The oil and grease content is defined as any material recovered from extraction with trichlorotrifluoroethane and measured gravimetrically; the extraction methods are those described above for aqueous and soil samples.

A detection limit for hydraulic fluids cannot be determined; the detection limit for specific components is expected to be in the range of $\mu g/L$ for aqueous samples and $\mu g/g$ for non-aqueous samples.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): none established
- AFOSH PEL (8-hr TWA): none established

Criteria
- NIOSH IDLH (30-min): none established
- ACGIH TLV® (8-hr TWA): none established
- ACGIH STEL (15-min): none established

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories - None established

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; hydraulic fluid is not a priority pollutant.
- Aquatic Life
  No criterion established; hydraulic fluid is not a priority pollutant.

Oil and Grease (2012)

For domestic water supply: Virtually free from oil and grease, particularly from the tastes and odors that emanate from petroleum products.

For aquatic life:
- 0.01 of the longest continuous flow 96-hour LC_{50} to several important freshwater and marine species, each having a demonstrated high susceptibility to oils and petrochemicals;
- levels of oils or petrochemicals in the sediment which cause deleterious effects to the biota should not be allowed;
- surface waters shall be virtually free from floating non-petroleum oils of vegetable and animal origin as well as petroleum-derived oil.

REGULATORY STATUS (as of May 1, 1987)

Promulgated Regulations

- Federal Programs

  Toxic Substances Control Act (TSCA)
  Manufacturers and processors of the C9 aromatic hydrocarbon fraction must test it for neurotoxicity, mutagenicity, developmental toxicity, reproductive effects and oncogenicity. The C9 fraction is obtained from the reforming of crude petroleum. It consists of ethyltoluenes and trimethylbenzenes (1988). Testing will be conducted by the American Petroleum Institute. Interim reports must be submitted at 6-month intervals (1987).

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Hazardous Materials Transportation Act (HMTA)
  The Department of Transportation has designated petroleum distillates as hazardous materials which are subject to requirements for packaging, labeling and transportation (305).

- State Water Programs

  Virginia has a quality standard of 1 mg/L for petroleum hydrocarbons in ground water (981).

  Illinois has a quality standard of 0.1 mg/L for oil in the public water supply (981).

  The following states have ground water quality standards for oil and grease (981):

  Nebraska - 1 mg/L
  Virginia and Wyoming - 10 mg/L

  Other states follow EPA Ambient Water Quality Criteria for oil and grease.

Proposed Regulations

- Federal Programs

  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed listing used oil as a hazardous waste. Used oil is defined as petroleum derived or synthetic oil including, but not limited to, lubricant, hydraulic fluid, metalworking fluid, insulating fluid or coolant (1985).

  Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
  EPA has proposed a reportable quantity (RQ) of 100 kg for used oil (1985).

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Ground Water (538)
Direct discharge into ground water (i.e., without percolation through the ground or subsoil) of organophosphorous compounds, organohalogen compounds and substances which may form such compounds in the aquatic environment, substances which possess carcinogenic, mutagenic or teratogenic properties in or via the aquatic environment and mineral oils and hydrocarbons is prohibited. Appropriate measures deemed necessary to prevent indirect discharge into ground water (i.e., via percolation through ground or subsoil) of these substances shall be taken by member countries.

Directive on Fishing Water Quality (536)
Petroleum products must not be present in salmonid and cyprinid waters in such quantities that they: (1) form a visible film on the surface of the water or form coatings on the beds of water-courses and lakes, (2) impart a detectable "hydrocarbon" taste to fish and, (3) produce harmful effects in fish.

Directive on the Quality Required of Shellfish Waters (537)
The mandatory specifications for petroleum hydrocarbons specify that they may not be present in shellfish water in such quantities as to produce a visible film on the surface of the water and/or a deposit on the shellfish which has harmful effects on the shellfish.

Directive on the Discharge of Dangerous Substances (535)
Organohalogens, organophosphates, petroleum hydrocarbons, carcinogens or substances which have a deleterious effect on the taste and/or odor of human food derived from aquatic environments cannot be discharged into inland surface waters, territorial waters or internal coastal waters without prior authorization from member countries which issue emission standards. A system of zero-emission applies to discharge of these substances into ground water.

Directive on Toxic and Dangerous Wastes (542)
Any installation, establishment, or undertaking which produces, holds and/or disposes of certain toxic and dangerous wastes including phenols and phenol compounds; organic-halogen compounds; chrome compounds; lead compounds; cyanides; ethers and aromatic polycyclic compounds (with carcinogenic effects) shall keep a record of the quantity, nature, physical and chemical characteristics and origin of such waste, and of the methods and sites used for disposing of such waste.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Petroleum and coal tar distillates with flash points below 21°C are classified as flammable substances and are subject to packaging and labeling regulations. Because of the variable composition of other petroleum and coal tar distillates (excluding those used as motor fuels) they are considered preparations and their labeling shall be done in accordance with the procedures outlined in the Directive Relating to the Classification, Packaging and Labeling of Dangerous Preparation (solvents).

Directive on Disposal of Waste Oils (1986)
Establishments collecting and/or disposing of waste oils must carry out these operations so that there will be no avoidable risk of water, air or soil pollution.

EEC Directives - Proposed

Proposal for a Council Directive on the Dumping of Waste at Sea (1793)
EEC has proposed that the dumping of crude oil, petroleum hydrocarbons, lubricants and hydraulic fluids at sea be prohibited.

68.1 MAJOR USES AND COMPOSITION

68.1.1 Major Uses

Hydraulic fluids are used in all kinds of applications but especially in machinery that moves or lifts objects. Aircraft, automobiles, trucks, forklifts, compressors, garden tractors and many others all use hydraulic fluids in their hydraulic components to magnify a relatively small force to do useful work. Automobiles need hydraulic fluids as transmission and brake fluids, while supersonic jet and commercial aircraft use them in landing gear and other equipment (21).

68.1.2 Composition

Traditionally, most hydraulic fluids have been mineral base oils, specifically those high in paraffins. Their advantages include stability to oxidation and good resistance to foaming and wear. Another major advantage of mineral base fluids over synthetics is their lower cost (1823,1824).

The development of synthetic hydraulic fluids arose from the need for fluids with a greater range of operating temperatures. Synthetic hydraulic fluids such as the phosphate esters provide excellent fire resistance, increasing the maximum operating temperature by perhaps 150°C over mineral oils. In most aircraft, hydraulic lines pass close to high temperature parts while high altitudes and speeds can produce temperatures well below 0°C. Commonly, temperatures can range from -53°C to 260°C (1824). It is this range of operating temperatures that dictates the type of fluid and additives used. Under these conditions, synthetic fluids of high autoignition temperatures and superior temperature-viscosity characteristics are used especially if there is the possibility of fluid leakage or spray on or near hot surfaces. Table 68-1 provides a list of typical hydraulic fluids including mineral base and synthetic base fluids.

Mineral base and synthetic base hydraulic fluids are fortified with approximately 0-20 volume percent additives (1825), which in most cases are identical to those used in the crankcase oils (see Chapter 69, Table 69-2). The most common additives in hydraulic fluids are used to modify physical/chemical characteristics; they include viscosity improvers, inhibitors of rust and corrosion, and inhibitors of wear, foaming and oxidation. Generally, detergent use is minimal (21,1823,1824). Tables 68-2 and 68-3 list reported hydraulic oil/lubricating oil base stocks and their additives.

TABLE 68-1

HYDRAULIC OILS

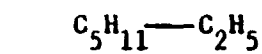
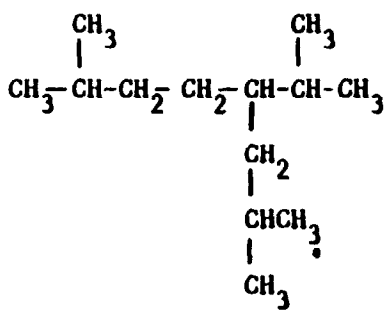
Base Stock	Structure/Composition	Properties/Characteristics
Mineral Base Oils:		
1. Straight paraffinic stock (linear and branched chained aliphatics)[a]	Examples of typical components include:  (n-paraffin)  (isoparaffin) Less typical components include: cycloparaffins, aromatic hydrocarbons, mixed aliphatic and aromatic hydrocarbons	Boiling point range approximately 300-600°C. MW approximately 150-1000. Carbon numbers approximately C_{15} - C_{50}. Densities approximately 0.8-1.0 g/mL at 15°C.

TABLE 68-1 - Continued

HYDRAULIC OILS

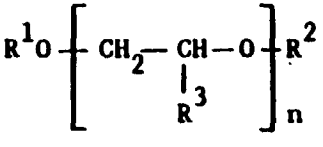
Base Stock	Structure/Composition	Properties/Characteristics
2. Oil in water emulsions. (oil is mineral base paraffinic stock)[b]	Up to 20% oil used but commonly only 1-4%. Greater than 80% water.	Used as a fire resistant hydraulic fluid. Temperature range limited to approximately 0° - 71°C.
3. Water in oil emulsions[b]	Approximately 60% oil. Approximately 40-45% water.	Used as a fire resistant hydraulic fluid.
Synthetic Base Oils:		
4. Polyoxyalkylene glycols[b] (polyglycols)		Can be formulated to be water soluble or water insoluble; the more polyethylene in character, the better the water solubility. MW typically 400-3000.
	R^1 R^3 R^2 1. H H H 2. H CH_3 H 3. C_4H_9 CH_3 H 4. C_4H_9 CH_3 C_2H_5	Densities approximately 0.95 - 1.2 g/mL.
	1. polyethylene glycol 2. polypropylene glycol 3. a monoether 4. a diether (examples of some possible R groups)	Vapor pressures of some polyglycols are reported to be less than 0.01 mm Hg at 20°C.

TABLE 68-1 - Continued

HYDRAULIC OILS

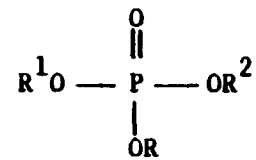
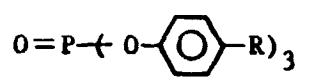
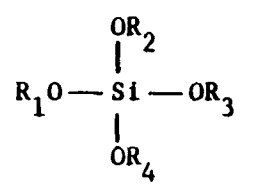
Base Stock	Structure/Composition	Properties/Characteristics
5. Phosphate esters[c]		Excellent fire resistance properties. MW typically 200-600. Densities approximately 0.9-1.5 g/mL. Boiling points for trialkylphosphates approximately 190-300°C. Used in extreme temperature applications.
	R can be H or organic groups. At least 1 R must be an organic group. Three classes: trialkylphosphates, triaryl phosphates, alkyl-aryl phosphates; i.e.,:	
		
	(a triaryl phosphate)	
	Oxygen(s) may be replaced by sulfur to give thiophosphates.	
6. Silicate esters[c]	Two classes:	MW typically 300-800. Densities approximately 0.8-1.1 g/mL at 20°C. Vapor pressures 0.1-5.0 mm Hg at 205°C. Boiling points approximately 93°-482°C. Used in extreme temperature applications.
		
	(Orthosilicates)	

TABLE 68-1 - Continued

HYDRAULIC OILS

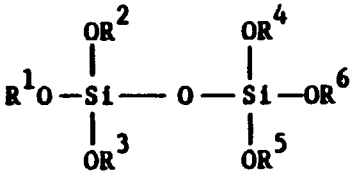
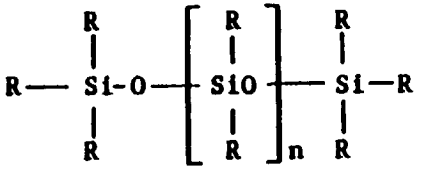
Base Stock	Structure/Composition	Properties/Characteristics
		
	(disiloxanes) (dimer silicates)	
	R's can be alkyl or aryl	
7. Silicones[c]		MW typically 1000-150,000. Densities approximately 0.75-1.1 g/mL, can be as high as 1.4 g/mL. Vapor pressures approximately 5 mm Hg at 149°C.
	R can be alkyl or aryl. Commonly R-CH_3 giving rise to the methyl and dimethyl silicone polymers	

TABLE 68-1 - Continued

HYDRAULIC OILS

Base Stock	Structure/Composition	Properties/Characteristics
8. Organic esters[c]	Includes: Monoesters (monobasic acid esters or polyolesters) diesters triester polyesters  (a diester-most common, based on a dibasic acid) (n is commonly 8-10) Diesters are derived from C_6 - C_{10} acids (i.e., adipic, azelaic, sebacic) and C_6 - C_9 alcohols (i.e., 2-ethylhexyl, 3,5,5-trimethylhexyl, isodecyl[b], and tridecyl alcohols).	MW typically 200-600; can be approximately 1000 for complex esters. Vapor pressures approximately 0.3 - 4.0 mm Hg at 205°C. Their uses include automotive engine oils (occasionally blended 50/50 with mineral oils), and jet and aircraft engines. Organic esters are the most common synthetic lubricants used. Used widely by the military in aircraft applications.

TABLE 68-1 - Continued

HYDRAULIC OILS

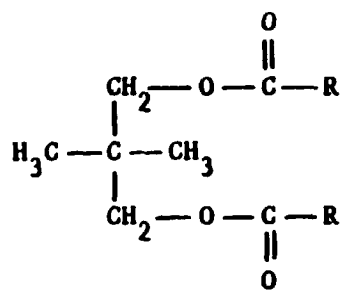
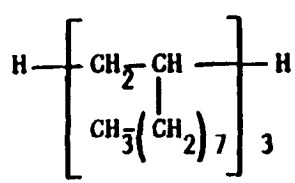
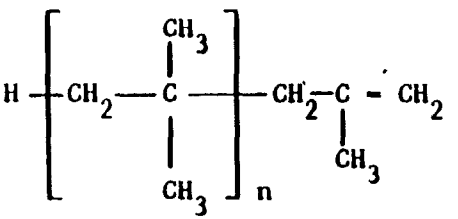
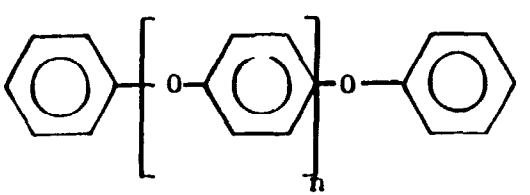
Base Stock	Structure/Composition	Properties/Characteristics
	 (a neopentyl polyol ester based on neopentyl glycol)	
9. Synthetic hydrocarbons:[a,b] Olefin oligomers (poly-alpha-olefins)	 (oligomer of 1-decene)	Resembles paraffinic mineral oils. Uses include synthetic hydrocarbon fluid in SAE 5W-20 motor oil and military aircraft fluids.
Alkylated aromatics	Typically reaction products of C_{10}-C_{14} alkyl groups and benzene/toluene/xylenes/ethylbenzenes, (i.e., dialkylated benzenes).[a]	Used in synthetic automotive engine oils.

TABLE 68-1 - Continued

HYDRAULIC OILS

Base Stock	Structure/Composition	Properties/Characteristics
Polybutenes (polyisobutylenes)	 (polyisobutylene) (typically C_{20} - C_{100})	Decomposition temperatures around 290°C. The lower MW polymers (C_{20}-C_{100}) are used as lubricants while the higher MW are used as additive viscosity index improvers. Used in many high temperature applications.
Cycloaliphatics		Used commonly as hydraulic fluids.
10. Polyphenyl ethers[c]	 Connections can be para, meta, or ortho; can be alkyl or halogen substituted (i.e., bis(p-phenoxyphenyl)ether)[b]	MW typically 150-700. Densities approximately 0.8 - 12 g/mL at 20°C.

[a] Reference 1821
[b] Reference 21
[c] Reference 1822

TABLE 68-2

SOME REPORTED MINERAL OIL AND SYNTHETIC OIL BASES FOR HYDRAULIC OIL/LUBRICATING OIL[a]

MINERAL BASE OILS[b]

straight paraffinic stock
water/oil mixtures (emulsions)
mineral oil/trialkyl thiophosphate ester blends
  e.g., $(OP(OC_2H_4SC_8H_{17})_3)$/mineral oil
mineral oil/silicate ester/polyglycol blends

SYNTHETIC BASE OILS

Organic Esters[c] (monobasic and dibasic acid esters, triesters, and polyesters)

isooctyl adipate
isodecyl adipate
2-ethylhexyl sebacate
pentaerythritol
2-ethyl-2-hydroxymethyl-1,3-propanediol
trimethylolpropane
dioctyl sebacate
di(3-methylbutyl)adipate
di(2-ethylbutyl)azelate
trimethylolethane
dibasic acid ester/silicate ester blend (~15% diester)•
dibasic acid ester/polyglycol blend
dibasic acid ester/synthetic hydrocarbon blend (~33% diester)

Polyoxyalkylene Glycols (polyglycols)[d]

polypropylene glycol
polyethylene glycol
polybutylene glycol
polyglycol/water blend
polyglycol/mineral oil/silicate ester blend
polyglycol/dibasic acid ester blend

Phosphate Esters[e]

tert-butyl-triphenylphosphate
triphenylphosphate
phenyl-m-tolyl-p-chlorophenylphosphate
tricresylphosphate
tri(2-ethylhexyl)phosphate
diorganodithiophosphate
triethylphosphate

Continued

TABLE 68-2 - Continued

SOME REPORTED MINERAL OIL AND SYNTHETIC OIL BASES FOR HYDRAULIC OIL/LUBRICATING OIL[a]

phenyl-m-trifluoromethylphenyl-1-naphthylphosphate
trixylylphosphate
trialkyl thiophosphate esters ($OP(OC_2H_4SC_8H_{17})_3$)/mineral oil blend
phosphate ester/polyglycol blends (tributoxyethyl/tributoxyethoxyethyl phosphates)
phosphate esters/dimethyl silicone polymer blend

Silicate Esters[d]

tetraethyl silicate
tetra(2-ethylhexyl) silicate
tetra(2-ethylbutyl) silicate
hexa(2-ethylbutoxy)) disiloxane
di-(2-ethylhexyl)silicate
cresyltriisopropyl silicate
silicate ester/dibasic acid ester blends
silicate ester blends with chlorofluorocarbons, mineral oils, silicones, polyglycols; e.g., bis(2-ethylhexyl)propylene glycol and butylmethyl propylene glycol/tetra alkyl orthosilicates or hexalkoxy disiloxanes

Silicones[f]

methyl, dimethyl polysiloxane
phenylmethyl polysiloxane
chlorophenyl polysiloxane
trifluoropropylmethyl polysiloxane

Synthetic Hydrocarbons[g]

alpha olefins (olefin oligomers)
2,3-dicyclohexyl-2,3-dimethyl butane
dialkylated benzene
polyisobutylene
synthetic hydrocarbon/dibasic acid ester blend (~33% diester)

Continued

TABLE 68-2 - Continued

SOME REPORTED MINERAL OIL AND SYNTHETIC OIL BASES FOR HYDRAULIC OIL/LUBRICATING OIL[a]

Others[h]

polychlorotrifluoroethylene
perfluoroheptane
trifluorotrichloroethane
bis(p-phenoxyphenyl)ether

---

[a] This table contains specific base chemicals or chemical classes used in hydraulic oils and/or lubricating oils. These chemicals may or may not be typical but all were reported in the literature as possible fluid bases.

[b] References 21,1822
[c] References 21,1826,1834
[d] References 21,1822
[e] References 1822,1829
[f] References 1822,1826
[g] References 21,1834
[h] Reference 1822

TABLE 68-3

SOME CHEMICAL ADDITIVES USED IN MINERAL AND SYNTHETIC BASE HYDRAULIC OIL/LUBRICATING OIL[a]

Chemical/Class Name	Typical Range Used
Oxidation Inhibitors	0-2.0% wt.[b] can be as much as 3.0% wt.
2,6-di-tert-butyl-p-cresol	
phenothiazine	
2,5-di-n-butylaminobenzoquinone	
2,5-di-piperidylbenzoquinone	
2,5-di-tert-butyl-p-benzoquinone	
pyridine	
quinoline	
hydroquinone	
R_3Sb or R_3SbS R=butyl or phenyl groups	
phenyl-alpha-naphthylamine	
triethanolamine	
2-naphthol	
zinc dithiophosphate	
Antiwear and Extreme Pressure Additives	0-6% wt.[c]
tricresylphosphate	
zinc diorganodithiophosphate	
zinc diisodecyldithiophosphate	
zinc di-n-butyldithiophosphate	
n-tosyltetrapropenyl succinimide	
hexadecyldiethyldithiocarbanate	
benzyl disulfide	
tungsten sulfide	
Rust and Corrosion Inhibitors	0-2.0% wt.[d] can be as high as 11.0% wt.
barium dinonylnaphthylene	
n-tosyltetrapropenyl succinimide	
zinc dithiophosphate	
dicyclohexamine	
diisobutyl ketone	
Viscosity Index (VI) Improvers	0-20% wt.[e]
polyisobutylenes	
polymethacrylates	
polyalkylstyrenes	
ethylene-propylene copolymers	
styrene-butadiene copolymers	
hydroxy cellulose ether	
silicone polymers (methyl and dimethyl polysiloxanes)	

Continued

TABLE 68-3 - Continued

SOME CHEMICAL ADDITIVES USED IN MINERAL AND SYNTHETIC BASE HYDRAULIC OIL/LUBRICATING OIL[a]

Chemical/Class Name	Typical Range Used
Detergents/Dispersants	0-20% wt.[f]
polyisobutenyl succinic anhydrides	
borated alkenyl succinimides	
oxazoline	
phosphonates and thiophosphates	
alkyl phenols and alkyl phenol sulfides	
alkyl methacrylate-dimethylaminoethyl methacrylate copolymers	
alkyl methacrylate-n-vinylpyrrolidone copolymers	
vinyl acetate-dialkyl fumarate-maleic anhydride copolymers	

[a] This table contains specific chemical additives used in hydraulic oils and/or lubricating oil. These chemicals may or may not be the typical additives but all were reported in the literature as possible chemical additives.

[b] References 21,1823,1831,1832,1834,1835,1836
[c] References 21,1821,1825,1826,1827,1833
[d] References 21,1821,1822,1823,1825
[e] References 21,1824,1825,1832,1835,1836
[f] References 21,1822,1827

68.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

68.2.1 Transport in Soil/Ground-water Systems

Most hydraulic fluids (except the more water soluble esters and glycols and oil-water emulsions) are expected to be quite immobile in the soil/ground-water environment. Bulk quantities of the oil (from a spill or improper disposal) might be carried slowly through the unsaturated zone to the top of the water table, but the high viscosity and low water solubility would mitigate this response. Most likely, at least with moderate to small spills, the oil would remain entrained in the pores of the soil near the surface. This would be more likely for low porosity and high organic carbon content soils, and less likely for sandy, porous soils.

Transport and subsequent fate of dissolved constituents of these oils will vary depending on the physicochemical (and biological) properties of the constituents. Some constituents will dissolve more quickly in the percolating ground waters, be sorbed less strongly on the soils (thus being transported more rapidly), and may be more, or less, susceptible to degradation by chemical or biological action. Thus, as was shown in Figure 65-1, the relative concentrations of the constituents of the oil will vary with time and distance from the site of initial contamination. This effect is called "weathering". (This term is also used to describe changes to oil following spills into surface waters where film spreading and breakup, and differential volatilization, dissolution and degradation all are involved.)

As noted in Tables 68-1, 68-2, and 68-3, there are a wide variety of base materials and additives that may be present in hydraulic fluids. More focused discussions of the soil/ground-water mobility and persistence of hydrocarbon-based oils are presented in Chapter 69 of this Guide. Chapter 70 (Synthetic Crankcase Oil) generally covers some of the same esters and glycols which are used in hydraulic fluids. Some data on phosphate esters are provided in Chapter 49 of this Guide.

No equilibrium partitioning model calculations (as have been given for most other chemicals in this Guide) are given for these fluids. This is due to the wide variety of materials (chemical classes) covered by the category of hydraulic fluids, to the lack of any real data on their physicochemical properties of environmental importance, and to the wide range of partitioning behaviors that could be shown from the highly immobile aliphatic and aromatic hydrocarbons to the mobile organic esters, polyglycols and phosphate ester fluids. To provide model outputs in this case would involve excessive speculation (on the needed physicochemical properties) and allow easy misuse of model results.

The aqueous phase mobility of oil constituents could be significantly enhanced if the oil was in the form of a very fine emulsion, or if the percolating ground water contained a significant amount of dissolved organic carbon (e.g., humic and fulvic acids, fatty acids, or chlorinated solvents) from other natural sources or other discharged materials. The dissolved organic carbon, much of it possibly in the form of colloidal particles, could absorb the oil constituents and assist in their transport through the soil/ground-water system.

Volatilization of constituents from the hydraulic fluids would be slow because of the low vapor pressures involved (presumably <1 mm Hg at 25°C for individual constituents, with many below 10^{-6} mm Hg). However, given that spilled oils may remain near the soil surface, making volatilization easier, that the material is resistant to leaching and degradation; and that the Henry's law constant may be moderately high, at least for the hydrocarbons, it is thus presumed that volatilization will be a major loss mechanism for spilled hydraulic fluid over time periods of weeks to years. Because the lower

molecular weight (more liquid) constituents would tend to volatilize first, the remaining material would generally have lower volatilities and lower water solubilities.

68.2.2 Transformation Processes in Soil/Ground-water Systems

An assessment of environmental persistence for hydraulic fluids is difficult given the variety of materials involved and the lack of pertinent data. Thus, most of the statements given below are both general and speculative in nature. Only the phosphate esters have been the subject of several environmental studies (see Chapter 49 of this Guide and references 1490 and 1496).

Hydraulic fluid oils are expected to be moderately persistent in the soil/ground-water environment because of their resistance to hydrolysis, oxidation and biodegradation. The general resistance to hydrolysis (for saturated and unsaturated hydrocarbons) is described by Harris (529). However, the organic esters, phosphate esters and polyglycols would be somewhat more susceptible to hydrolysis, especially under basic conditions.

The assessment of the resistance to biodegradation is more complex. Most of the molecules are so large that passage through cell walls (where metabolism or degradation is relatively easy) is hindered and much of the biodegradation must be carried out by extracellular enzymes secreted by the microbes. Such difficulties aside, many studies on petroleum hydrocarbon materials (oils as well as light distillates) have showed moderate to high eventual susceptibility to biodegradation for the bulk of the material (1842). A period of microbial adaptation may be required. The organic esters, phosphate esters and polyglycols would be expected to be more readily biodegraded.

Different constituents of the oil will differ significantly in their biodegradability for reasons related to molecular size, structure and toxicity. For example, highly branched alkanes are much less biodegradable than linear alkanes, and polycyclic aromatic hydrocarbons with three or more rings are very resistant to biodegradation (515). For all hydrocarbons, aerobic biodegradation would be expected to be much more important than anaerobic biodegradation (1841). Because of this, and because of the decrease in microbiological activity with increasing soil depth, oil constitutents reaching deep anaerobic soils could persist for very long time periods.

68.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that the components of hydraulic fluids will vary widely in their volatility, tendency to sorb to soil, and potential for bioaccumulation. However, the base stock of hydraulic fluids manufactured with mineral oils are expected to be very strongly sorbed to soil because of their high molecular weight and low water solubility. These compounds have extremely low volatility in pure form, but when present in water may have relatively

high volatility due to their low solubility. They are not expected to be readily bioaccumulated because their large size makes their passage through cell walls difficult.

Polyglycol-based hydraulic fluids and fractions composed of phosphate esters and organic esters are expected to have low volatility (because of their high water solubility and low vapor pressure) and be weakly sorbed to soil. They would also be expected to have a low potential for bioaccumulation because of their high solubility and susceptibility to biodegradation. Despite the variability in the properties of the components of hydraulic fluids, several potential exposure pathways can be inferred.

Volatilization of hydraulic fluids that are spilled or improperly disposed of is not expected to result in significant exposure of workers or residents in the area, regardless of the type of fluid. Oil-based fluids would be rapidly sorbed to the soil, and only a very small fraction of the oil would volatilize. Fluids based on polyglycols, organic esters and phosphate esters would not readily volatilize.

Ground water contamination may be a significant exposure pathway for water soluble hydraulic fluids, including oil-water emulsions, polyglycols, organic esters and phosphate esters. Exposure may occur through the direct use of ground water drinking water supplies or indirectly through ground-water discharge to surface waters. Surface waters may also be contaminated by the discharge of soil particles to which hydraulic fluids (especially mineral oil base fluids) have been sorbed. Where surface waters have been contaminated, ingestion exposures may occur from their use as drinking water supplies and dermal exposures may result from their recreational use. The uptake of hydraulic fluids by aquatic organisms or domestic animals is not expected to result in significant exposure.

68.2.4 Other Sources of Human Exposure

Data on ambient concentrations of hydraulic fluids in air and water, as well as food and drinking water, are not available in the literature. This should not be surprising since they are complex mixtures, are not distributed widely in the environment, and (except for the mineral base fluids) consist mainly of non-persistent compounds.

Aside from those involved in their manufacture, the personnel likely to receive the greatest exposure to hydraulic fluids are those employed in servicing and maintaining equipment. Although inhalation exposures are not expected to be large, these personnel may experience large dermal exposures if protective gloves and clothing are not worn during maintenance operations. Operators of hydraulic equipment would be expected to experience only small exposures because the very nature of hydraulic systems is to keep the fluid contained, and volatilization from reservoirs is likely to be minimal.

68.3 HUMAN HEALTH CONSIDERATIONS

Hydraulic fluids do not appear to be toxic to animals (2228); however, the composition and level of additives vary greatly. Major components usually include ethylene glycol, polyethylene glycol and tri-ortho cresyl phosphate (TOCP). A review of the toxicity of TOCP and ethylene glycol may be found in Chapter 49 and Chapter 43, respectively, of the IRP Toxicology Guide.

68.3.1 Animal Studies

68.3.1.1 Carcinogenicity

No specific data on the carcinogenicity of hydraulic fluids were found.

68.3.1.2 Mutagenicity

No specific studies on the mutagenicity of hydraulic fluids were found in the literature.

68.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

No specific studies were located in the literature.

68.3.1.4 Other Toxicologic Effects

68.3.1.4.1 Short-term Toxicity

MLO 82-233 is a synthetic hydrogenated polyalpha olefin with a nominal $C_{30}H_{62}$ formula while MLO 82-585 is a naphthenic type petroleum oil. Both compounds contain tricresyl phosphate with unspecified amounts of TOCP. Neither hydraulic fluid was toxic following ingestion of 5 mL/kg in Sprague-Dawley rats, dermal application of 2 mg/kg in New Zealand rabbits, or a 6-hour whole body inhalation study with 1148 mg/m^3 in Sprague-Dawley rats. No ocular irritation occurred when 0.1 mL of either fluid was instilled in the eye of albino rabbits. The synthetic hydraulic fluid also did not produce irritation when applied undiluted to the intact or abraded skin of albino rabbits; however, the petroleum hydraulic fluid produced a moderate, reversible primary skin reaction. Neither hydraulic fluid was considered a skin sensitizer or a delayed neurotoxin (2231).

Similarly, no oral or dermal toxicity was reported nor was there any indication of any eye or skin irritation or skin sensitization with a cyclotriphosphazene-based hydraulic fluid, containing 0.1% tolytriazole as a copper corrosion inhibitor, tested in Fischer 344 rats and New Zealand white rabbits under similar conditions (1936).

The Navy hydraulic fluid, Plurasafe® MC200, produced a slight skin sensitizing reaction in guinea pigs (1936). One animal responded to the challenge dose of 0.1 mL hydraulic fluid with a mild erythematous

reaction at 24 hours which increased in severity by 48 hours. One week later, animals were challenged a second time which resulted in 2 additional cases of sensitization.

Triaryl phosphate hydraulic fluids administered orally to white Vantress hens daily for 5 days resulted in signs of toxicity identical to TOCP poisoning. After a latent period of 8 to 14 days, treated birds tired easily and squatted in a characteristic pose. Leg weakness, loss of balance and clumsiness soon followed. Maximum paralysis occurred 15 to 16 days after treatment along with excessive salivation, lacrimation and severe diarrhea. Death was attributed to a combination of toxicity, starvation and dehydration (2230).

In an inhalation study involving six synthetic hydraulic fluids (2233), exposure of Sprague-Dawley rats to 6.43 mg/L (duration not stated) of one of the fluids (N501 - Supplied by Gulf R&D Company) resulted in the death of all animals within 24 hours of exposure. Signs of toxicity included rough coat, labored respiration and lethargy. The LC_{50} value was found to be 2 mg/L for a 4 hour exposure. No mortality or toxic effects were reported in rats exposed to the remaining five compounds. Toxic effects of N501 were concluded by investigators to be due to one or more of the additives.

68.3.1.4.2 Chronic Toxicity

A long-term continuous inhalation study was performed with a triaryl phosphate hydraulic fluid used by the U.S. Navy (2230). The fluid contained a mixture of tricresyl phosphates, trixylenyl phosphates and other trialkyphenyl phosphates. The TOCP content was reported to be less than 1.5%. Animals were exposed in a chamber to 1.8 to 110 mg/m^3 hydraulic fluid mist 24 hours/day for 36 to 163 days. No neurotoxic signs were reported in dogs, monkeys or rats. Rabbits exposed to high doses of hydraulic fluids (101 or 103 mg/m^3) developed lacrimation and generalized hind leg paralysis. An extensor type paralysis, lacrimation and thick, mucous salivation were reported in chickens exposed to the hydraulic fluid mist. These signs of cholinergic stimulation were indistinguishable from those induced by TOCP (see Chapter 49 of this Guide).

A 90-day aerosol exposure to a phosphate ester base hydraulic fluid, Durad MP280, resulted in toxicity 3 days after exposure to 100 mg/m^3 was initiated (2233). Rabbits became anorexic and lethargic, and cachexia and head droop were noted prior to death. All animals died by the 49th exposure day. Kyphosis (hunch back) was noted in rats exposed to 100 mg/m^3 of Durad MP280 along with a rough hair coat and unkempt appearance. A decrease in weight gain was also reported (2233).

68.3.2 Human and Epidemiologic Studies

68.3.2.1 Short-term Toxicologic Effects

No acute human data were found on hydraulic fluids.

68.3.2.2 Chronic Toxicologic Effects

No studies were found in the literature dealing with the effects of long-term exposure to hydraulic fluid oil in humans.

68.3.3 Toxicology of Hydraulic Fluid Components

The composition of hydraulic fluid varies greatly and usually depends upon the specific conditions of use. Since the exact composition of the oils is constantly changing and difficult to define, the toxicology of component classes are briefly discussed below. See Table 68-4 for the acute toxicity data of specific compounds.

Organic esters

Organic esters generally found in lubricating oils and hydraulic fluids include adipates, sebacates and dibasic acid esters. Dibasic acid esters are primarily non-toxic via ingestion or skin absorption. The only effect noted from dermal contact may be a drying of the skin (1822). Di(2-hexoxyethyl)succinate is a sebacate which is relatively non-toxic to animals. In humans it is expected to have a low toxicity. Large doses may produce CNS depression, nausea, vomiting and transient liver and kidney injury (12). Not all neopentyl esters have been tested for toxicity, but studies with trimethylopropane ester showed a toxic level comparable to that of mineral oil (1822).

Polyglycols

Ingestion of polyglycols is unlikely, but small amounts produce no toxic effect. No cases of skin irritation or skin sensitization have been reported; mild irritation to the eyelid has been reported but effects were only transitory. Usually no inhalation hazard exists but at high temperatures, where vapors are likely to form, adequate ventilation should be provided (1822).

Ucon® fluids are a mixture of polyalkylene glycols and diesters. 50-HB-260, 50-HB-5100, 25-H-2005 and 75-H-1400 are low in single-dose oral toxicity with LD_{50} values for the male rat ranging from 5.95 to >64 mL/kg bw; oral LD_{50} values for the rabbit range from 1.77 to 35.4 mL/kg bw. The lower molecular weight compounds are more toxic. A dose-related granular degeneration of the cytoplasm of the smooth muscle in the intestinal wall was noted in dogs fed 25-H-2005 for 2 years. The significance of this finding is unknown. No other adverse effects were shown. The only adverse effect observed in rats fed up to 0.5 g/kg/day of 25-H-2005 for two years was a slight growth depression in females (12).

TABLE 68-4

ACUTE TOXICITY OF SELECTED COMPONENTS OF HYDRAULIC FLUID

Compound	Oral (mg/kg)	Dermal (mg/kg)	Inhalation (ppm)
2-ethylhexylsebacate	LD_{50} [rat]: 1280	-	-
pentaerythritol	LD_{50} [mouse]: 25,500	-	-
polypropylene glycol	LD_{50} [rat]: 419	-	-
polyethylene glycol	LD_{50} [rat]: 33,750	-	-
triphenylphosphate	LDLo [rat]: 3000	-	-
tricresylphosphate	LDLo [rat]: 4680	-	-
tri-ortho-cresylphosphate	LD_{50} [rat]: 3000	-	-
	LDLo [human]: 1000	-	-
tri(2-ethylhexyl) phosphate	LD_{50} [rat]: 37,000	LDLo [rabbit]: 20,000	
triethylphosphate	LDLo [rat]: 1600	-	-
tetraethyl silicate	LDLo [rat]: 1000	-	LCLo [rat]: 1000•4 hr
tetra(2-ethylbutyl) silicate	LD_{50} [rat]: 20,000	-	-
trifluorotrichloroethane	LD_{50} [rat]: 43,000	-	TCLo [rat]: 87000•6 hr

Reference: 47

No carcinogenic effects were observed in rats orally administered Ucon® fluids in the diet or in mice dermally exposed to these compounds (13).

Polyethylene glycol applied to the open wounds of rabbits resulted in metabolic acidosis and changes in blood chemistry consistent with nephrotoxicity (2225). Effects were attributed to the metabolism of polyethylene glycol to toxic compounds (such as hydroxyglycolic and diglycolic acid homologues) which are efficient chelators of calcium. The mechanism of damage was similar to that associated with ethylene glycol-mediated renal failure. See discussion of the toxic effects of ethylene glycol in Chapter 43 of the Installation Restoration Program Toxicology Guide, Volume 2.

No adverse changes in clinical, biochemical or hematological parameters developed in rats fed 2 mL/kg/day polyethylene glycol 400 (duration not specified) (2224). Examination of monkeys administered the same treatment revealed a deposition of oxalate crystals in the cortical tubules of the kidney (2224).

Phosphate esters

Organic phosphates possess excellent thermal stability and chemical solvency properties which makes them valuable hydraulic fluid components (1822).

Organic phosphates are readily absorbed through the skin and can be inhaled. Ingestion is rare. Signs of toxicity following excessive exposure reflect stimulation of the autonomic and central nervous systems, resulting from inhibition of acetylcholinesterase and the consequent accumulation of acetylcholine. The initial effect is on smooth muscle, cardiac muscle and exocrine glands. Early signs of toxicity include intestinal cramps, tightness in the chest, blurred vision, headaches, diarrhea, decreased blood pressure, and salivation. The second stage of intoxication results from stimulation of the peripheral motor system and of all autonomic ganglia. Toxic signs include stimulation and/or paralysis of the somatic, autonomic and central nervous systems.

Chronic administration of low doses of organic phosphates produce a measurable decrease in cholinesterase activity. Toxic effects are nonexistent to slight and may result in diarrhea and tremors. Delayed paralysis in man and animals due to a degeneration of the axons in the spinal cord and peripheral nerves has also been associated with organic phosphates, particularly tri-o-cresyl phosphate (TOCP) (13). See Chapter 49 of the Installation Restoration Program Toxicology Guide, Volume 2, for a complete discussion on TOCP.

Silicate esters

The toxicity of the orthosilicates and disiloxanes vary widely and range from almost completely innocuous to rather poisonous (1822). Injection of ethyl silicate compounds into the skin of rabbits produced transient erythema, edema, and slight necrosis at the injection site. When instilled into the rabbit eye, it produced transient irritation. Inhalation of 400 ppm by rats for 7 hours/day for 30 days caused mortality and lung, liver and kidney pathological effects. Inhalation of 88 ppm caused no effects (12).

Silicones

Generally, silicones are not irritating to the skin and cause no corneal damage when splashed into the eye. Slight temporary irritation to the eye has been reported in some individuals with effects disappearing within 24 hours. Toxic materials may also be emitted during decomposition of fluorinated silicone polymers at temperatures above 570°F (1822).

In chronic feeding experiments, rats treated with hexamethyl disiloxane (HMS) showed widespread systemic irritation. Rabbits injected intradermally with HMS developed edema and necrosis at the injection sites. Siloxanes injected into the rabbit eye resulted in

transient irritation with complete clearing after 48 hours. When inhaled at 4400 ppm for 19 to 26 days, HMS caused slight depression in the rat and guinea pig, with a very slight increase in rat liver and kidney weights (12,13).

Silicone resins had no influence on health when fed for 94 days to rats, and did not result in irritation to rabbit skin or eyes. No toxic effects were reported when injected into rats intraperitoneally (12).

Rats fed a dietary level of 0.3% Antifoam A® for 2 years showed no significant toxic effect. Long-term feeding studies in mice reported similar results; however, a single subcutaneous injection of 0.2 mL antifoam showed a greater incidence of cysts at the site of injection (13).

Polydimethyl siloxane caused no evident changes when tested for reproductive and teratologic effects in rats and rabbits, or testicular effects in rabbits. Dimethylphenylmethylpolysiloxane, tris(trimethylsiloxy)phenylsilane and trifluoropropropylmethylpolysiloxane were also negative in male reproductive studies (13).

Other

Other components of hydraulic fluids include polyphenyl ethers. Studies with phenyl ether show no toxicological effects following inhalation of vapors or contact with skin. Bis(p-phenoxyphenyl)ether, bis(m-phenoxyphenyl)ether, and m-bis(m-phenoxyphenoxy)benzene cause no irritation in skin tests with rabbits and only mild transient irritation in acute eye tests. These compounds were practically non-toxic in acute oral and intraperitoneal tests with rats. Phenolic degradation products formed during use of these materials under severe conditions are expected to increase toxicity (1822).

Hydraulic Fluid Additives

Information available on additives used in hydraulic fluid is limited. Selected compounds are briefly discussed below. Refer to Table 68-5 for the acute toxicity data of specific additives.

2,6-di-tert-butyl-p-cresol

2,6-Di-tert-butyl-p-cresol, more commonly known as butylated hydroxytoluene or BHT, is used as an oxidation inhibitor in synthetic crankcase oil and hydraulic fluids.

BHT inhibits tumorigenesis when multiple doses are administered before a carcinogen while the incidence of hepatomas induced by 2-acetylaminofluorene and the number of pulmonary adenomas induced by urethane were augmented by post-treatment with BHT (17). The NCI bioassay for carcinogenic effects of BHT in rats and mice was negative (17).

TABLE 68-5

ACUTE TOXICITY OF SELECTED ADDITIVES OF HYDRAULIC FLUID

Compound	Oral (mg/kg)	Dermal (mg/kg)	Inhalation (ppm)
2,6-di-tert-butyl-p-cresol	LD_{50} [rat]: 890	-	-
phenothiazine	LD_{50} [rat]: 5000	-	-
	LDLo [child]: 425	-	-
pyridine	LD_{50} [rat]: 891	LDLo [rabbit]: 1121	LC_{50} [rat]: 4000•4 hr
quinoline	LD_{50} [rat]: 331	LD_{50} [rabbit]: 540	-
hydroquinone	LD_{50} [rat]: 320	-	-
	LDLo [human]: 29	-	-
phenyl-alpha-naphthylamine	LD_{50} [rat]: 1625	-	-
triethanolamine	LD_{50} [rat]: 8680	-	-
2-naphthol	LD_{50} [rat]: 2420	-	-
zinc dithiophosphate	LDLo [rabbit]: 2130	-	-
tricresyl phosphate	LDLo [rat]: 4680	-	-
tri-ortho-cresyl phosphate	LD_{50} [rat]: 3000	-	-
	LDLo [human]: 1000	-	-
diisobutylketone	LD_{50} [rat]: 5750	LD_{50} [rabbit]: 20,000	LCLo [rat]: 2000•4 hr LCLo [human]: 50

Reference: 47

A reported teratogenic effect of anophthalmia in rats has never been duplicated (17).

Various morphological and biochemical changes have been observed in experimental animals fed extremely high doses of BHT. Adverse effects included a dose-dependent reduction in growth rate and alveolar epithelial damage in mice which progressed to fibrosis when pure oxygen followed the BHT exposure. Dose-dependent fatalities occurred from massive hemorrhages into the pleural and peritoneal cavities while survivors suffered hemorrhages of the epididymis, testis, nasal cavity and pancreas. Liver changes in rats, mice and monkeys included enlargement, induction of microsomal enzymes and an increased synthesis of hepatic smooth endoplasmic reticulum (17).

BHT is mildly irritating to human skin and severely irritating to rabbit eyes (17).

Phenothiazine

At one time, phenothiazine was used in human medicine as an anthelmintic and urinary antiseptic. Currently, it is an important class of antipsychotic drug used to diminish motor activity and alter psychotic behavior (17,16).

Side effects of phenothiazine include toxic hepatitis and jaundice, leukocytosis, leukopenia, eosinophilia and hemolytic anemia. Dermatitis, hypersensitivity and photosensitivity have also been reported in phenothiazine treated individuals (17,16)

Tri-ortho-cresyl phosphate (TOCP)

TOCP is known to cause peripheral nervous system damage leading to neuromuscular problems (2216). For a complete discussion of the toxicological effects of TOCP, see Chapter 49 of this Guide.

Zinc dithiophosphate

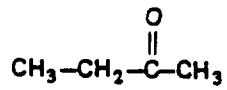
Zinc dialkyldithiophosphate (ZDDP) has a low acute systemic toxicity with an oral LD_{50} value of greater than 2 g/kg bw and a dermal LD_{50} value in excess of 3 g/kg (2317).

Undiluted ZDDP is a severe eye irritant; however, the diluted product, used as the additive in hydraulic fluids and synthetic crankcase oils, is regarded as non-irritating. Prolonged contact with undiluted ZDDP is irritating to the skin and produces moderate to severe erythema and edema. Repeated contact results in fissuring and exfoliation (2317).

In subchronic toxicity studies, ZDDP primarily affects the reproductive organs of male rabbits. Dermal application of 5 to 25% ZDDP five days a week for three consecutive weeks resulted in decreased sperm counts and some testicular atrophy (2216). Some studies suggest that the male reproductive effects may be physiological and related to body weight loss and reduced food consumption rather than to the toxic effects of ZDDP (1217).

Pyridine

Pyridine is absorbed from the respiratory and gastrointestinal tracts. Skin absorption is not significant although contact may result in dermatitis. Short-term toxic effects in animals are linked to central nervous depression. Prolonged daily administration of pyridine to rats produced hepatorenal damage (17).

COMMON SYNONYMS:	CAS REG. NO.:	FORMULA:	AIR W/V CONVERSION FACTORS at 25°C (59)
2-Butanone MEK Ethyl methyl ketone Methyl acetone	78-93-3 NIOSH NO.: EL6475000	C_4H_8O	2.94 mg/m³ ≈ 1 ppm 0.340 ppm ≈ 1 mg/m³
	STRUCTURE: 		MOLECULAR WEIGHT: 72.10

REACTIVITY	Reactions of ketones such as methyl ethyl ketone with non-oxidizing mineral acids, caustics, cyanides, mercaptans, or other organic sulfides typically produce heat, while those with alkali or alkaline earth elemental metals, nitrides or strong reducing agents evolve heat and flammable gases. Reactions with oxidizing mineral acids or other strong oxidizing agents may generate heat and fire. Those with azo or diazo compounds or hydrazines may generate heat and usually innocuous gases. Reactions with organic peroxides or hydroperoxides typically result in explosions. Various manufacturers list oxidizing agents, chlorinated hydrocarbons in the presence of alkalies, alkanolamines, amines, pyridines, ammonia, caustics, inorganic acids, isocyanates, and halogens as incompatible materials (505,507,511).

PHYSICO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(23)
	• Color: colorless	(23)
	• Odor: acetone-like	(23)
	• Odor Threshold: 5.4 ppm, 10 ppm	(384,2)
	• Liquid Density (g/ml at 20°C): 0.805	(14)
	• Freezing/Melting Point (°C): -86.4	(23)
	• Boiling Point (°C): 79.6	(14)
	• Flash Point (°C): -5.6 to -1.1 (oc); -6.7 to -3.9 (cc)	(60,507, 514)
	• Flammable Limits in Air, % by Volume: 1.7 - 2.0 to 10 - 12	(51,60, 506,507)
	• Autoignition Temperature (°C): 404 or 516	(51,60, 506,510)
	• Vapor Pressure (mm Hg at 20°C): 70.6	(59)
	• Saturated Concentration in Air (mg/m³ at 20°C): 279,071	(ADL estim)
	• Solubility in Water (mg/L at 10°C): 353,000	(67)
	• Viscosity (cp at 25°C): 0.4	(23)
	• Surface Tension (dyne/cm at 20°C): no data	( )
	• Log (Octanol-Water Partition Coefficient), log K_{ow}: 0.29	(29)
	• Soil Adsorption Coefficient, K_{oc}: 0.94	(611)
	• Henry's Law Constant (atm•m³/mol at 20°C): 4.35×10^{-5}	(1138)
	• Bioconcentration Factor: 1.86, 0.09	(1137,659)

TABLE 41-1

EQUILIBRIUM PARTITIONING CALCULATIONS FOR METHYL ETHYL KETONE IN MODEL ENVIRONMENTS[a]

Soil Environment	Estimated Percent of Total Mass of Chemical in Each Compartment		
	Soil	Soil-Water	Soil-Air
Unsaturated topsoil at 25°C[b,c]	15.2	84.3	0.5
Saturated deep soil[d]	0.4	99.6	-

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

b) Utilized estimated soil sorption coefficient: K_{oc} = 0.94 (611).

c) Henry's law constant taken as 4.35 x 10^{-5} atm•m^3/mol at 25°C (1138).

d) Used sorption coefficient K_p = 0.001 K_{oc}.

- increase with increasing soil organic matter content;
- increase slightly with decreasing temperature;
- increase moderately with increasing salinity of the soil water; and
- decrease moderately with increasing dissolved organic matter in the soil water.

No information specific to the adsorption of methyl ethyl ketone in the environment was available. Methyl ethyl ketone is highly soluble in water and its low values for log K_{ow} and K_{oc} suggest that sorption to soils/sediments does not contribute significantly to its environmental fate. Methyl ethyl ketone in the soil/ground-water system is expected to be only slightly less mobile than acetone, which was reported to migrate freely with little or no retardation.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): 200 ppm
- AFOSH PEL (8-hr TWA): 200 ppm

Criteria
- NIOSH IDLH (30-min): 3000 ppm
- ACGIH TLV® (8-hr TWA): 200 ppm
- ACGIH STEL (15-min): 300 ppm

WATER EXPOSURE LIMITS:

Drinking Water Standards - None established

EPA Health Advisories

In the absence of formal drinking water standards, the EPA (1770) has developed the following Health Advisories (formerly termed SNARLs) for noncarcinogenic risk for short and long-term exposure to methyl ethyl ketone in drinking water:

- 1 day: none established
- 10 days: none established
- long-term: 8.6 mg/L

EPA Ambient Water Quality Criteria (355)

- Human Health

  No criterion established; methyl ethyl ketone is not a priority pollutant.

- Aquatic Life

  No criterion established; methyl ethyl ketone is not a priority pollutant.

EEC Directives

Directive on Marketing and Use of Dangerous Substances (541)
Methyl ethyl ketone may not be used in ornamental objects intended to produce light or color effects by means of different phases.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Methyl ethyl ketone is classified as a flammable substance and is subject to packaging and labeling regulations.

Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
Methyl ethyl ketone is exempt from a tolerance requirement when used as a surfactant in pesticide formulations applied to growing crops (315).

Marine Protection Research and Sanctuaries Act (MPRSA)
Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

Occupational Safety and Health Act (OSHA)
Employee exposure to methyl ethyl ketone shall not exceed an 8-hour time-weighted-average (TWA) of 200 ppm (298).

Hazardous Materials Transportation Act (HMTA)
The Department of Transportation has designated methyl ethyl ketone as a hazardous material which is subject to requirements for packaging, labeling and transportation (306).

Food, Drug and Cosmetic Act (FDCA)
Methyl ethyl ketone is approved for use as an indirect food additive (362).

- State Water Programs
  There are no specific state regulations for methyl ethyl ketone.

Proposed Regulations

- Federal Programs
  Resource Conservation and Recovery Act (RCRA)
  EPA has proposed that solid wastes which contain a concentration equal to or greater than 7.2 mg/L methyl ethyl ketone be listed as hazardous in that they exhibit the characteristic defined as EP toxicity (1565).

  EPA has proposed that hazardous waste treatment, storage and disposal facilities monitor ground water for methyl ethyl ketone when EPA suspects the facilities of leaking contaminants (1754).

- State Water Programs
  No proposed regulations are pending.

REGULATORY STATUS (as of January 1, 1987)

Promulgated Regulations

- Federal Programs

Safe Drinking Water Act (SDWA)
In states with an approved Underground Injection Control program, a permit is required for the injection of methyl ethyl ketone-containing wastes designated as hazardous under RCRA (295).

Resource Conservation and Recovery Act (RCRA)
Methyl ethyl ketone is identified as a toxic, ignitable hazardous waste (U159) and listed as a hazardous waste constituent (328,329). Non-specific sources of methyl ethyl ketone-containing waste that contain at least 10% methyl ethyl ketone are solvent use (or recovery) activities (987).

Spent solvent wastes containing methyl ethyl ketone are prohibited from land disposal unless 1 or more of the following conditions apply:

- the generator is a small quantity generator;
- the waste is generated from a response action under CERCLA or a corrective action under RCRA;
- the waste is a solvent-water mixture, solvent-containing sludge or solvent-contaminated soil containing less than 1% total solvent constituents listed in 40CFR268.41.

Between November 8, 1986 and November 8, 1988, these wastes may be disposed of in a landfill or surface impoundment only if the facility is in compliance with the requirements specified in 40CFR268.5(h)(2). After November 8, 1988, all land disposal of these wastes is prohibited. These requirements do not apply if the wastes are disposed at a facility that has been granted a petition under 40CFR268.6 or an extension under 40CFR268.5 or if the waste is treated to meet specific treatment standards (1755).

Toxic Substances Control Act (TSCA)
Manufacturers, processors or distributors of methyl ethyl ketone must report production, usage and disposal information to EPA. They, as well as others who possess health and safety studies on methyl ethyl ketone, must submit them to EPA (334,335).

Comprehensive Environmental Response Compensation and Liability Act (CERCLA)
Methyl ethyl ketone is designated a hazardous substance under CERCLA. It has a reportable quantity (RQ) limit of 2270 kg. Reportable quantities have also been issued for RCRA hazardous waste streams containing methyl ethyl ketone but these depend upon the concentrations of the chemicals in the waste stream (985).

41.1 MAJOR USES

Methyl ethyl ketone is used primarily as a solvent. The coating industry uses methyl ethyl ketone extensively, accounting for 61% of its production, to manufacture gums, resins and nitrocellulose. Approximately 18% of the methyl ethyl ketone produced is used to make cements and adhesives. Other manufacturers utilize methyl ethyl ketone to produce printing ink, cleaning fluids, smokeless powders and wax (200,59,67).

41.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

41.2.1 Transport in Soil/Ground-water Systems

41.2.1.1 Overview

Methyl ethyl ketone is expected to be fairly mobile in the soil/ground-water system when present at low concentrations or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways can be assessed by using an equilibrium partitioning model, as shown in Table 41-1. These calculations predict the partitioning of low soil concentrations of methyl ethyl ketone among soil particles, soil water and soil air. Portions of methyl ethyl ketone associated with the water and air phases of the soil have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil model indicate that only 15.2% of the methyl ethyl ketone is expected to be sorbed onto soil particles. Approximately 84% is expected to partition to the soil-water phase, and is thus available to migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. For the small portion of methyl ethyl ketone in the gaseous phase of the soil (0.5%), diffusion through the soil-air pores up to the ground surface, and subsequent removal by wind, may be a significant loss pathway.

In saturated, deep soils (containing no soil air and negligible soil organic carbon), almost all of the methyl ethyl ketone (99.6%) is predicted to be present in the soil-water phase (Table 41-1) and available for transport with flowing ground water. Sorption onto deep soils (0.4%) is not expected to be significant. Overall, ground water underlying methyl ethyl ketone-contaminated soils with low organic content is expected to be vulnerable to contamination.

41.2.1.2 Sorption on Soils

The mobility of methyl ethyl ketone in the soil/ground-water system (and its eventual migration into aquifers) is governed by the extent of its sorption on soil particles. In general, sorption on soils is expected to:

PERSISTENCE IN THE SOIL-WATER SYSTEM	Methyl ethyl ketone is expected to migrate in the soil/ground-water system with very little retardation. Volatilization from near-surface soils may occur; however, vapor concentrations in soil are expected to be very low whenever water is present. Biodegradation of methyl ethyl ketone has been demonstrated and persistence in environments with active microbial populations is not expected.

PATHWAYS OF EXPOSURE	The primary pathway of concern from a soil/ground-water system is the migration of methyl ethyl ketone to ground-water drinking water supplies. Inhalation may be important in some situations. Bioaccumulation of methyl ethyl ketone is not likely to be an important exposure pathway.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (13): Eye, nose and throat irritation are usually the first symptoms to appear in methyl ethyl ketone exposure. At high concentrations, CNS depression and narcosis along with congestion of the lungs, liver and kidneys are observed.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral 2737 [rat] (47) skin 13,000 [rabbit] (47) LC_{50} (mg/m^3) inhalation [mouse] 40,000•2 hr (47)
	Long-Term Effects: Dermatitis
	Pregnancy/Neonate Data: Fetotoxic at 3000 ppm
	Mutation Data: Conflicting data
	Carcinogenicity: No data

HANDLING PRECAUTIONS (54)	Handle chemical only with adequate ventilation • Vapor concentration of 200 - 1000 ppm: chemical cartridge respirator with organic vapor cartridge and full facepiece • 1000 - 30,000 ppm: gas mask with organic vapor canister, any supplied air respirator or self-contained breathing apparatus with full facepiece • Butyl, natural rubber, neoprene, nitrile, PE, PVA or PVC gloves, apron and boots to prevent repeated or prolonged skin contact with the liquid • Chemical goggles if there is probability of eye contact.

EMERGENCY FIRST AID TREATMENT (54)	Ingestion: Give large quantities of salt water and induce vomiting if victim is conscious. Get medical attention • Inhalation: Move victim to fresh air immediately and perform artifical respiration if necessary • Skin: Remove contaminated clothing. Wash skin with soap and water • Eye: Irrigate with large amounts of water immediately.

41.2.1.3 Volatilization from Soils

Transport of methyl ethyl ketone vapors through the air-filled pores of unsaturated soils may occur in near-surface soils. However, modeling results suggest that a very small fraction of the methyl ethyl ketone loadings will be present in the soil-air phase.

In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to a lesser extent, the vapor phase diffusion coefficient (31).

The Henry's law constant (H), which provides an indication of a chemical's tendency to volatilize from solution, is expected to increase significantly with increasing temperature. Moderate increases in H have also been observed with increasing salinity and the presence of other organic compounds (18). These results suggest that the presence of other materials may significantly affect the volatilization of acetone, particularly from surface soils. No information was available for the two other physicochemical properties influencing volatilization, i.e., the vapor-soil sorption coefficient and the vapor phase diffusion coefficient.

Experimental mass transfer coefficient (25°C) were determined for methyl ethyl ketone at several depths and mixing rates (1121). Volatilization half-lives calculated from the mass transfer coefficients ranged from 1.05 days for high mixing (2020 rpm) of a 15 cm aqueous solution to 2.25 days for low mixing (557 rpm) of a 20 cm aqueous solution. Lande et al. (1137) calculated an approximate half-life of 138 hours (5.75 days) for the evaporation (20°C) of methyl ethyl ketone from aqueous solution. It can be expected that the rate of volatilization may vary for aqueous environmental systems.

The significance of methyl ethyl ketone volatilization in the environment is not documented; data on volatilization from soils, in particular, are not available. Since methyl ethyl ketone is not strongly adsorbed to soil, some volatilization at the surface may occur; however, the ability of methyl ethyl ketone to be transported with soil water is significant. Furthermore, methyl ethyl ketone has been reported in rainwater samples (1136) suggesting that, due to its high water solubility, any methyl ethyl ketone lost due to volatilization may be washed out of the atmosphere and returned to the soil/water system.

41.2.2 Transformation Processes in Soil/Ground-water Systems

No information on the hydrolysis of methyl ethyl ketone in the soil/ground-water system was available; under normal environmental conditions, hydrolysis is not expected to occur at a rate competitive with volatilization or biodegradation. The portion of methyl ethyl

ketone that has been released from the soil into the air will return to the soil via atmospheric washout or eventually u photochemical oxidation.

Methyl ethyl ketone is expected to be susceptible to exten microbial biodegradation in pure cultures, mixed cultures, and ac vated sludge systems (1137). Several authors (1132,1133) have repor the biodegradation of methyl ethyl ketone by microbes grown on propa or by soil bacteria grown on C1-C8 aliphatic hydrocarbons; oxidati was observed even where methyl ethyl ketone was unable to suppo growth of the organism. Methyl ethyl ketone degradation by one of fou tested yeast cultures was also reported (1131).

After five days of incubation, degradation of methyl ethyl ketone, as determined by BOD_5 tests with acclimated sewage seed or microbes from polluted waters, ranged from 48% to 88%; degradation after 20 days was observed to be 69% to 89% (880,881,882,1127). Dojlido (1135) reported 100% degradation in 8 days for 200 mg/L methyl ethyl ketone and in 9 days for 400 mg/L. Chou et al. (1126) report 77% utilization of methyl ethyl ketone in an anaerobic reactor; the same authors report 100% anaerobic degradation by enriched methane cultures after an 8-day lag.

In actual soil/ground-water systems, the concentration of microorganisms capable of biodegrading methyl ethyl ketone may be low, and is expected to drop off sharply with increasing depth; prediction of biodegradation rates in the environment is not possible. However, in environments with sufficient microbial populations, methyl ethyl ketone is not expected to persist.

41.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that methyl ethyl ketone has a moderate volatility, is very weakly adsorbed to soil, and has no significant potential for bioaccumulation. This compound may volatilize from the soil surface, but that portion not removed by volatilization is likely to be mobile in ground water. These fate characteristics suggest several potential exposure pathways.

Volatilization of methyl ethyl ketone from a disposal site could result in inhalation exposure to workers or residents in the area. In addition, the potential for ground water contamination is high, particularly in sandy soils. It has been detected in ground water associated with hazardous waste sites. Mitre (83) reported that methyl ethyl ketone has been found at 10 of the 546 National Priority List (NPL) sites. It was detected at 4 sites in ground water, 4 in surface water, and 4 in air. However, it may not be commonly analyzed for at NPL sites as it is not a priority pollutant. These data, as well as the properties of methyl ethyl ketone, suggest that drinking water exposure from ground water contamination is likely to be its primary route of exposure from soil/ground-water systems.

The movement of methyl ethyl ketone in ground water may result in discharge to surface water. As a result, ingestion exposures may occur resulting from the use of surface waters as drinking water supplies, and dermal exposures may result from the recreational use of surface waters. Such exposures are likely to be lower than those from drinking contaminated ground water due to biodegradation and/or volatilization of methyl ethyl ketone in surface water. Any pathways related to the uptake by aquatic organisms or domestic animals from surface waters are likely to be less significant than other sources of exposure due to the low BCF for methyl ethyl ketone.

41.2.4 Other Sources of Human Exposure

Methyl ethyl ketone is widely used as a industrial solvent, coating, and adhesive. As such, there are a number of sources of human exposure. Data, however, are somewhat lacking. For example, it is not commonly measured in drinking water.

The production and use of methyl ethyl ketone has led to its presence in the atmosphere. Brodzinksy and Singh (84) summarized air monitoring data for a number of pollutants. For methyl ethyl ketone, they reported 181 data points for urban/suburban areas. All results for these samples were less than the detection limit. In source-dominated areas, the median concentration reported was 0.19 $\mu g/m^3$ for 33 data points.

Dermal exposure is expected to be common due to the prevalence of methyl ethyl ketone as a solvent in various products. For example, two surveys were conducted in Japan on the solvent content of a variety of products. They found methyl ethyl ketone in 26% of the paints, 21% of the inks, 23% of the adhesives, 11% of the thinners, and 8% of the degreasers that were sampled. While most of these products were used in occupational settings, some may be used by consumers (1140,1141).

The ketones are naturally occurring components of food. Lande et al. (1137) reviewed the literature and found methyl ethyl ketone in a wide variety of foods including cheese (0.3 ppm), milk (0.08 ppm), cream (0.2 ppm), bread, oranges and rum. This compound appears to be a common component of the diet although a total exposure from this source can not be evaluated without additional data (1137).

41.3 HUMAN HEALTH CONSIDERATIONS

41.3.1 Animal Studies

41.3.1.1 Carcinogenicity

No data were available with regard to the carcinogenic potential of methyl ethyl ketone.

41.3.1.2 Mutagenicity

Methyl ethyl ketone showed no evidence of mutagenicity when tested in TA102 and TA104 strains of Salmonella typhimurium (1001). It was shown to be marginally positive, at best, in a Chinese V-79 hamster cell assay which indicates the ability of compounds to inhibit gap junction-mediated intercellular communication (1013). Zimmerman (1011), using the diploid yeast strain D61.M of Saccharomyces cerevisiae, found methyl ethyl ketone strongly induced mitotic aneuploidy (having more or less than the normal number of chromosomes), but no other types of detectable genetic effects.

41.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Schwetz et al. (1003) exposed pregnant Sprague-Dawley rats to 1000 or 3000 ppm of methyl ethyl ketone vapor in a chamber for 7 hours a day on days 6 through 15 of gestation. No evidence of maternal toxicity was observed. A retardation of fetal development and a significant increase in the number of anomalous skeletons (19% vs. 0 in the control group) were observed at both the 1000 and 3000 ppm treatment levels. Examination of the litters of the dams exposed to 3000 ppm methyl ethyl ketone revealed 2 fetuses with acaudia (no tails) and imperforated anus, and 2 fetuses with brachygnathia (shortened lower jaw). Though these unusual anomalies were not statistically significant, Schwetz did note that they had never before been observed in control animals in the Sprague-Dawley strain in his laboratory.

Due to a lack of dose-related response in the previous experiment, Deacon et al. (1005) duplicated the study in order to determine the repeatability of the unusual anomalies observed by Schwetz. Experimental design was identical. Exposure to 3000 ppm methyl ethyl ketone produced a slight maternal toxicity as shown by a decreased maternal weight gain. Minor anomalies in fetal development included an extra lumbar rib and delayed ossification of the cervical centra which were noted by Deacon to occur at low incidence historically among control rats in his laboratory. The findings presented in this follow-up study indicate that methyl ethyl ketone produced a slight fetotoxic effect at the 3000 ppm exposure level, but no embryotoxic or teratogenic response in rats.

41.3.1.4 Other Toxicologic Effects

41.3.1.4.1 Short-term Toxicity

Acute exposure to methyl ethyl ketone generally results in irritation to the eyes, nose and throat, CNS depression, emphysema and congestion of the liver and kidneys. The oral LD_{50} range for rats is reported to be 2.7-3.6 g/kg (47,13) while the reported LC_{50} value in the mouse is a two-hour exposure to 40,000 mg/m^3 methyl ethyl ketone (47).

Patty et al. (1028) describe the effects of airborn methyl ethyl ketone on guinea pigs at concentrations of 0.33, 1.0, 3.3 or 10.0% for up to 810 minutes. Signs of toxicity included irritation of the nose and eyes, incoordination, narcosis, gasping and death. Necropsy revealed emphysema, slight congestion of the brain and marked congestion of the systemic organs, particularly the lungs.

Exposure of guinea pigs to 10% methyl ethyl ketone vapor for 30 minutes resulted in opaque corneas. Examination eight days later showed that the eyes had returned to normal, indicating a reversibility of the damage (1028).

It has been well established that ketones as a class can cause narcosis at high concentrations (13,1029). However, it is believed (1012) that ketones may also be capable of producing a modification of behavior or impairment of judgement at lower concentrations. Geller et al. (1012) evaluated the effect of low-level methyl ethyl ketone exposure on delayed match-to-sample tasks in the baboon. Four juvenile baboons were exposed to 100 ppm methyl ethyl ketone, 24 hours a day for 7 days. Accuracy of performance was minimally affected. However, extra responses during the delay period were recorded and the response time was significantly increased. These effects were considered to be an early manifestation of the incoordination and narcosis which are observed at much higher concentrations.

Tham et al. (1006) demonstrated that a component of the equilibrium system, the vestibulo-ocular reflex (VOR), was depressed in rats when methyl ethyl ketone was infused into the circulatory system at a rate of 70 μM/kg/minute for 60 minutes, resulting in blood levels of 100 ppm. Nystagmus (a rapid, involuntary movement of the eye ball) was induced by accelerated rotation to study vestibular function. Depression of the VOR is considered an early sign of intoxication prior to the onset of a general depression of the central nervous system.

41.3.1.4.2 Chronic Toxicity

A subchronic inhalation study performed by Cavender et al. (1008) exposed male and female Fischer 344 rats to 1250, 2500 or 5000 ppm methyl ethyl ketone in a chamber for 6 hours/day, 5 days/week for 13 weeks. The only clinical observation was a decrease in mean body weight in the group receiving 5000 ppm methyl ethyl ketone. Increases in liver weight and liver to body weight ratios were noted in both males and females in the 5000 ppm group and a depression of brain weight in females in the 5000 ppm group at necropsy. No lesions were found that could be attributed to methyl ethyl ketone.

Methyl ethyl ketone has been shown to shorten the latency period for the onset of neurotoxic effects of methyl butyl ketone and n-hexane in a number of species. Altenkirch (1002) studied the response of the nervous system to chronic repeated exposures of 10,000 ppm pure hexane, 10,000 ppm methyl ethyl ketone/n-hexane (ratio of 1:9) or 6000 ppm pure methyl ethyl ketone in rats. Motor neuropathy with giant swelling of

axons in the peripheral and central nervous system, as well as severe potentiation of hexane neurotoxicity and shortened onset of morphological and clinical signs developed in animals exposed to the methyl ethyl ketone/hexane mixture. Motor impairment of the methyl ethyl ketone/hexane treated rats varied from a waddling gait and eversion of hind limbs to quadriplegia. Methyl ethyl ketone alone did not produce neuropathy. Rats exposed to extremely high concentrations of pure methyl ethyl ketone (6000-10,000 ppm), however, developed severe bronchopneumonia and died.

The potentiation effect of methyl ethyl ketone on hexane-induced neuropathy has also been observed with methyl butyl ketone (1029,1030). Rats intoxicated by continuous exposure to air containing methyl ethyl ketone and methyl butyl ketone vapor in a ratio of 1125:225 ppm developed clinical evidence of neuropathy after 25 days; rats inhaling 225 ppm methyl butyl ketone alone exhibited neuropathy after 66 days.

Methyl ethyl ketone potentiates the neurotoxic effects of methyl butyl ketone and hexane presumably by stimulating their metabolism to neurotoxic metabolites (1015). Both hexane and methyl butyl ketone share common products in their metabolic pathways, i.e., 2,5-hexanediol and 2,5-hexanedione. Hexanedione is believed to be the neurotoxic agent (1030,1014). Administration of hexane and methyl ethyl ketone together results in a significant increase in the activity of mixed-function oxygenase enzymes in rats (1004) and the urinary excretion of 2,5-hexanedione is increased after administration of the methyl ethyl ketone/methyl butyl ketone mixture to rats (1002). Furthermore, administration of 2,5-hexanedione produced effects indistinguishable from hexane or methyl butyl ketone neurotoxicity (1014).

41.3.2 Human and Epidemiologic Studies

41.3.2.1 Short-term Toxicologic Effects

Berg et al. (1016) reported a case of retrobular neuritis in an 18-year-old male exposed to methyl ethyl ketone while removing paint in an enclosed area. Symptoms included a dull headache, mild vertigo and diminished vision in both eyes. Ophthalmic examination and testing 2 hours later revealed marked enlargment of the blind spot and superior arcuate-type defects in both eyes. Blood analysis showed the presence of methanol and formaldehyde. Thirty-six hours after exposure, vision returned to normal. Berg postulated that the patient had suffered optic nerve toxicity induced by methanol formed from the metabolism of methyl ethyl ketone.

Munies and Wurster (1031) demonstrated that methyl ethyl ketone in contact with the skin resulted in a partial dehydration of the stratum corneum. Wahlberg (1009) showed that the spontaneous transient whitening of the skin caused by excessive exposure to methyl ethyl ketone is due to a change in structure and the removal of the skin lipids rather than by vasoconstriction.

41.3.2.2 Chronic Toxicologic Effects

Smith and Mayer (1032) investigated the effects of methyl ethyl ketone on a group of factory workers using methyl ethyl ketone as a solvent. Routes of exposure included both immersion of bare hands in the solvent and inhalation of 300-600 ppm. No duration of exposure was given. A number of workers developed severe dermatitis. Several workers also experienced numbness in the fingers, arms and legs. Symptoms disappeared when exposure to methyl ethyl ketone was discontinued.

The potentiation of various hexacarbon neuropathies by methyl ethyl ketone is of particular interest in cases of solvent abuse. Altenkirch (1033) reported 25 cases of clinically severe toxic polyneuropathy in people addicted to inhaling methyl ethyl ketone-containing solvents. The peripheral motor defects took 2.5-3 years to become apparent. The effects were considered to be due to an axonal transmission disorder which destroyed peripheral and central axons (0049).

Altenkirch (1004) also described what was known as the Berlin Poisoning Affair. In 1974, a solvent manufacturer changed its formulation to help stop inhalation abuse. The hexane content was reduced from 31 to 16% and 11% methyl ethyl ketone was added as a denaturant. An epidemic-like outbreak of 19 severe neuropathy syndromes occurred soon after the new formulation was available to the public. Neurological effects included considerable weight loss, muscular weakness affecting all four extremities or paralysis of all four extremities, extreme muscular atrophy, and respiratory disorders. In some cases, visual disturbances occurred and facial nerves were affected. Individuals examined up to 4 years after the incident still exhibited muscular atrophy, muscular weakness and sensory defects. This incident further supports findings that the neurotoxic properties of hexane can be potentiated by methyl ethyl ketone.

A female shoe-factory worker developed sensorimotor neuropathy after years of working with a glue containing 20% methyl ethyl ketone and 8% hexane (1007). The woman developed sensory and motor neuropathy in the lower limbs, and the absence of deep reflexes. This condition slowly regressed once exposure to the solvent mixture ceased.

41.3.3 Levels of Concern

The EPA (1770) has established a Health Advisory for noncarcinogenic risk for long-term exposure to methyl ethyl ketone of 8.6 mg/L in drinking water.

The OSHA (298) standard is 200 ppm (590 mg/m^3) averaged over an 8-hour work-shift. The ACGIH (3) recommends a threshold limit value of 200 ppm, with a short-term exposure limit of 300 ppm (885 mg/m^3). The threshold limit value was set to prevent injurious effects and minimize complaints about odor and irritation (46).

41.3.4 Hazard Assessment

Methyl ethyl ketone exhibits a low toxicity subsequent to acute and chronic exposures. The oral LD_{50} value for rats is in the 2.7 to 3.6 g/kg range (47,13); the inhalation LC_{50} for mice for a two hour exposure is 40,000 mg/m^3 (47). At high concentrations (e.g., 10% in air), methyl ethyl ketone can induce narcosis (1028) but low level chronic exposure (2500 ppm for 90 days) produced no adverse effects in rats (1008).

Exposure to methyl ethyl ketone can produce local irritation of the eyes, upper respiratory tract and skin (1016,1031,1032). If splashed into the eyes, painful irritation and corneal injury may result (1028). Direct skin contact may produce dermatitis and defat the skin (1032,1009). Short-term human exposure to methyl ethyl ketone can produce headache, eye and throat irritation.

Studies in both humans and animals indicate that methyl ethyl ketone potentiates (i.e., shortens the time of onset) of peripheral neuropathy caused by either n-hexane or methyl n-butyl ketone. Methyl ethyl ketone itself does not induce peripheral neuropathy (1002,1029,1030).

The reproductive, mutagenic and carcinogenic activity of methyl ethyl ketone have not been thoroughly investigated and require further research. Female rats exposed via inhalation to over 1000 ppm methyl ethyl ketone resulted in fetotoxic effects. A low incidence of malformations was observed in one study (1003) but could not be duplicated using an identical experimental design (1005), suggesting that methyl ethyl ketone produces minor fetotoxic effects but is not a teratogen in rats.

Conflicting data are available regarding the mutagenicity of methyl ethyl ketone. Negative results were obtained in an Ames assay (1001), a marginally positive response at best in a Chinese V-79 hamster cell test (1013) and a strongly positive induction of aneuploidy in a yeast test (1011). There are no data available on the carcinogenic activity of methyl ethyl ketone.

41.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the concentration of methyl ethyl ketone in soil and water requires collection of a representative field sample and laboratory analysis. Due to the volatility of methyl ethyl ketone, care is required to prevent losses during sample collection and storage. Soil and water samples are collected in airtight containers with no headspace; analysis should be completed within 14 days of sampling. In addition to the targeted samples, quality assurance samples such as field blanks, duplicates, and spiked matrices should be included in the analytical program.

Methyl ethyl ketone is not included among the EPA-designated priority pollutants. However, EPA Methods 624, 1624 (65) 8015 and 8240 (63) would be appropriate methods of choice for the analysis of methyl ethyl ketone in aqueous samples. An inert gas is bubbled through the aqueous sample in a purging chamber at ambient temperature, transferring the methyl ethyl ketone from the aqueous phase to the vapor phase and onto a sorbent trap. The trap is then heated and backflushed to desorb the methyl ethyl ketone and transfer it onto a gas chromatographic (GC) column. The GC column is programmed to separate the volatile organics; methyl ethyl ketone is then detected with a flame ionization detector (Method 8015) or a mass spectrometer (Methods 624, 1624, and 8240).

The EPA procedures recommended for methyl ethyl ketone analysis in soil and waste samples, Methods 8015 and 8240 (63), differ from the aqueous procedures primarily in the method by which the analyte is introduced into the GC. The recommended method involves dispersing the soil or waste sample in methanol or polyethylene glycol to dissolve the methyl ethyl ketone. A portion of the solution is then combined with water and purged as described above. Other sample introduction techniques include direct injection and a headspace method.

Methyl ethyl ketone detection limits for the various methods were not determined but would be in the range of 1-50 μg/L for aqueous samples and 1 μg/g for non-aqueous samples.

COMMON SYNONYMS:	CAS REG. NO.:	FORMULA:	AIR W/V CONVERSION FACTORS at 25°C (12)
1,2-Ethanediol Glycol alcohol Permanent anti-freeze EG	107-21-1 NIOSH NO.: KW2975000	$C_2H_6O_2$	2.54 mg/m³ = 1 ppm 0.365 ppm = 1 mg/m³
	STRUCTURE:		MOLECULAR WEIGHT:
	$HO-CH_2-CH_2-OH$		62.07

REACTIVITY	The National Fire Protection Association reports that mixture of ethylene glycol with chlorosulfonic acid, oleum, or sulfuric acid causes the pressure and temperature to increase in a closed container. Reactions of glycols with non-oxidizing mineral acids typically generate heat, while those with oxidizing mineral acids, organic peroxides or hydroperoxides, or other strong oxidizing agents may evolve heat and fire. Compatibility charts further indicate that reactions of glycols with organic acids, isocyanates, or epoxides may initiate violent polymerization, while those with alkali or alkaline earth elemental metals or strong reducing agents may evolve heat; flammable gases and fire. Reactions with nitrides may produce heat, flammable gases, and an explosion, while those with azo or diazo compounds or hydrazines may evolve heat and generally innocuous gases (505,511).

PHYSICO-CHEMICAL DATA	• Physical State (at 20°C): liquid	(54)
	• Color: colorless	(54)
	• Odor: none	(12)
	• Odor Threshold: no data	( )
	• Liquid Density (g/ml at 20°C): 1.1135	(69)
	• Freezing/Melting Point (°C): -13	(21)
	• Boiling Point (°C): 197.6	(21)
	• Flash Point (°C): 111 (cc), 116 (oc)	(60,506, 507)
	• Flammable Limits in Air, % by Volume: 3.2-?	(51,60, 506)
	• Autoignition Temperature (°C): 398-400 or 413	(51,60, 506)
	• Vapor Pressure (mm Hg at 20°C): 0.05	(67)
	• Saturated Concentration in Air (mg/m³ at 20°C): 340	(67)
	• Solubility in Water (mg/L at 20°C): complete	(507)
	• Viscosity (cp at 20°C): 19.83	(21)
	• Surface Tension (dyne/cm at 20°C): 48.4	(69)
	• Log (Octanol-Water Partition Coefficient), log K_{ow}: -1.36	(29)
	• Soil Adsorption Coefficient, K_{oc}: 0.02	(611)
	• Henry's Law Constant (atm•m³/mol at 25°C): 6.0×10^{-8}	(966)
	• Bioconcentration Factor: 0.002	(659)

PERSISTENCE IN THE SOIL-WATER SYSTEM	Ethylene glycol is expected to be highly mobile in the soil/ground-water system. Sorption onto soil is weak and volatilization is expected to be minimal. Although data on biodegradation in soil are limited, ethylene glycol is not expected to be highly persistent.

PATHWAYS OF EXPOSURE	The primary pathway of concern from a soil/ground-water system is the migration of ethylene glycol to ground-water drinking water supplies, although no data confirm this. Inhalation or bioaccumulation of ethylene glycol are not likely to be important exposure pathways.

HEALTH HAZARD DATA	Signs and Symptoms of Short-term Human Exposure (69): Clinical symptoms include CNS dysfunction with severe metabolic acidosis, cardiopulmonary failure and acute renal failure.
	Toxicity Based on Animal Studies: LD_{50} (mg/kg) oral 7500 [mouse] (47) skin 19530 [rabbit] (47) LC_{50} (mg/m^3) inhalation -- no data
	Long-Term Effects: CNS depression, severe renal damage
	Pregnancy/Neonate Data: Teratogenic
	Mutation Data: Negative
	Carcinogenicity Classification: IARC - none assigned; NTP - under study via dietary exposure

HANDLING PRECAUTIONS (507)	NIOSH approved breathing air equipment or NIOSH approved face mask with organic vapor cartridge and dust or mist prefilter (not for use in fire fighting or in atmospheres with reduced oxygen content).

EMERGENCY FIRST AID TREATMENT (507)	Ingestion: Give large quantities of salt water and induce vomiting immediately if individual is conscious. Get medical attention • Inhalation: Move victim to fresh air immediately and perform artificial respiration if necessary • Skin: Remove contaminated clothing. Wash skin with soap and water • Eye: Irrigate with large amounts of water immediately.

ENVIRONMENTAL AND OCCUPATIONAL STANDARDS AND CRITERIA

AIR EXPOSURE LIMITS:

Standards
- OSHA PEL (8-hr TWA): none established
- AFOSH PEL (8-hr TWA): none established

Criteria
- NIOSH IDLH (30-min): none established
- ACGIH TLV® (8-hr TWA): CL: 50 ppm
- ACGIH STEL (15-min): none established

WATER EXPOSURE LIMITS:

Drinking Water Standards
None established

EPA Health Advisories
In the absence of formal drinking water standards the EPA (383) has developed the following Health Advisories (formerly termed SNARLs) for noncarcinogenic risk for short- and long-term exposure to ethylene glycol in drinking water:

- 1 day: 19 mg/L
- 10 days: none established
- long-term: 5.5 mg/L

EPA Ambient Water Quality Criteria (355)
- Human Health
  No criterion established; ethylene glycol is not a priority pollutant.
- Aquatic Life
  No criterion established; ethylene glycol is not a priority pollutant.

REGULATORY STATUS (as of January 1, 1987)

Promulgated Regulations

- Federal Programs

  Federal Insecticide, Fungicide and Rodenticide Act (FIFRA)
  Ethylene glycol is exempt from a tolerance requirement when used as an antifreeze or deactivator for all pesticides used before a crop emerges from the soil and for herbicides used before or after a crop emerges (315).

  Marine Protection Research and Sanctuaries Act (MPRSA)
  Ocean dumping of organohalogen compounds as well as the dumping of known or suspected carcinogens, mutagens or teratogens is prohibited except when they are present as trace contaminants. Permit applicants are exempt from these regulations if they can demonstrate that such chemical constituents are non-toxic and non-bioaccumulative in the marine environment or are rapidly rendered harmless by physical, chemical or biological processes in the sea (309).

  Food, Drug and Cosmetic Act (FDCA)
  Ethylene glycol is approved for use as an indirect food additive (362).

  Consumer Product Safety Act (CPSA)
  Ethylene glycol-based radiator antifreeze distributed in containers intended or suitable for household use may be misbranded if they fail to bear a warning statement adequate for protection of the public health and safety (1236).

- State Water Programs

  There are no specific state regulations for ethylene glycol.

Proposed Regulations

- Federal Programs

  No proposed regulations are pending.

- State Water Programs

  No proposed regulations are pending.

EEC Directives

Directive on Marketing and Use of Dangerous Substances (541)
Ethylene glycol may not be used in ornamental objects intended to produce light or color effects by means of different phases.

Directive on the Classification, Packaging and Labeling of Dangerous Substances (787)
Ethylene glycol is classified as a harmful substance and is subject to packaging and labeling regulations.

43.1 MAJOR USES

Ethylene glycol is a colorless, odorless, hydroscopic liquid infinitely soluble in water and many organic liquids. Due to its ability to markedly reduce the freezing point of water, about 40% of all ethylene glycol production goes to the manufacturing of nonvolatile antifreeze and liquid coolant for motor vehicles. Approximately 35% is used to manufacture polyester fiber and film. Ethylene glycol is also used in hydraulic fluids, as a solvent and as a heat transfer agent, especially in solar powered hot-water heaters (59,507).

43.2 ENVIRONMENTAL FATE AND EXPOSURE PATHWAYS

43.2.1 Transport in Soil/Ground-water Systems

43.2.1.1 Overview

Ethylene glycol is expected to be highly mobile in the soil/ground-water system when present at relatively low concentrations or as a separate organic phase (resulting from a spill of significant quantities of the chemical). In general, transport pathways can be assessed by using an equilibrium partitioning model as shown in Table 43-1. These calculations predict the partitioning of low soil concentrations of ethylene glycol among soil particles, soil water and soil air. Portions of ethylene glycol associated with the water and air phases of the soil have higher mobility than the adsorbed portion.

Estimates for the unsaturated topsoil model indicate that only 0.4% of the ethylene glycol is expected to be sorbed onto soil particles. Approximately 99.6% is expected to partition to the mobile soil-water phase, and is thus available to migrate by bulk transport (e.g., the downward movement of infiltrating water), dispersion and diffusion. Since a very small portion of ethylene glycol is expected to be in the gaseous phase of the soil (less than 0.001%), diffusion through the soil-air pores up to the ground surface and subsequent removal by wind would appear to be a minor loss pathway. In saturated, deep soils (containing no soil air and negligible soil organic carbon), almost all of the ethylene glycol (99.99%) is predicted to be present in the soil-water phase (Table 43-1) and available for transport with flowing ground water. Sorption onto deep soils (less than 0.01%) is not expected to be significant. Overall, ground water underlying ethylene glycol-contaminated soils with low organic content is expected to be vulnerable to contamination.

43.2.1.2 Sorption on Soils

The mobility of ethylene glycol in the soil/ground-water system (and its eventual migration into aquifers) is governed by the extent of its sorption on soil particles. Ethylene glycol is miscible in water and, as evidenced by its negative log K_{ow} and low K_{oc}, adsorption to soil/sediments is not expected to significantly influence its environmental fate.

TABLE 43-1

EQUILIBRIUM PARTITIONING CALCULATIONS FOR ETHYLENE GLYCOL IN MODEL ENVIRONMENTS[a]

Soil Environment	Estimated Percent of Total Mass of Chemical in Each Compartment		
	Soil	Soil-Water	Soil-Air
Unsaturated topsoil at 25°C[b,c]	0.4	99.6	7×10^{-4}
Saturated deep soil[d]	8×10^{-3}	99.99	-

a) Calculations based on Mackay's equilibrium partitioning model (34,35,36); see Introduction in Volume 1 for description of model and environmental conditions chosen to represent an unsaturated topsoil and saturated deep soil. Calculated percentages should be considered as rough estimates and used only for general guidance.

b) Utilized estimated soil sorption coefficient: $K_{oc} = 0.02$.

c) Henry's law constant taken as 6×10^{-8} atm·m³/mol at 25°C (966).

d) Used sorption coefficient $K_p = 0.001\ K_{oc}$.

Lokke (862) studied the adsorption and leaching of ethylene glycol in subsoils. No adsorption was observed for ethylene glycol (0.1-90 mg/L) onto two sandy soils and one clay subsoil ranging from 0.1-0.2% organic carbon. Leaching studies performed with soil cores of sandy till showed that ethylene glycol (150-220 g/L) closely followed the movement of water with little or no retardation.

43.2.1.3 Volatilization from Soils

Transport of ethylene glycol vapors through the air-filled pores of unsaturated soils may occur in near-surface soils. However, modeling results suggest that an insignificant fraction of the ethylene glycol loading will be present in the soil-air phase.

In general, important soil and environmental properties influencing the rate of volatilization include soil porosity, temperature, convection currents and barometric pressure changes; important physicochemical properties include the Henry's law constant, the vapor-soil sorption coefficient, and, to the lesser extent, the vapor phase diffusion coefficient (31).

Data on ethylene glycol volatilization from soils, in particular, are not available. Ethylene glycol is not strongly adsorbed to soil and is highly soluble in water. Although some volatilization may occur at the surface, the low value of the Henry's law constant (6×10^{-8} atm•m^3/mole) suggests that vapor concentrations in soil will be low whenever water is present and volatilization will be minimal.

43.2.2 Transformation Processes in Soil/Ground-water Systems

No information was available on the non-biological degradation of ethylene glycol in the environment. Thermo-oxidative degradation to organic acids has been reported for ethylene glycol used as an anti-freeze mixture (866).

A variety of studies have reported that ethylene glycol can be readily biodegraded under both aerobic and anaerobic conditions (867, 865,868,864,869,879). Data on degradation by microorganisms isolated from soil are contradictory. Harada and Nagashima (871) reported growth and nongrowth with ethylene glycol as sole carbon source. Jensen (872) reported no degradation using microbes isolated from soil. Gaston and Stadtman (868) reported rapid degradation under anaerobic conditions using microbes isolated from mud.

Degradation using activated sludge microorganisms or sewage seed was rapid; complete degradation within a few days was reported in several studies (873,874,875,876,877,878). Concentrations up to 2000 ppm were shown to support microbial growth, with an optimum concentration of 200 ppm reported. However, some concentrations above 1000 ppm were inhibitory (879); concentrations above 10,000 ppm inhibited growth of activated sludge (863).

In actual soil/ground-water systems, the concentrations of microorganisms capable of degrading ethylene glycol may be low, and may drop off with increasing depth; prediction of biodegradation rates in the environment is not possible. However, since both aerobic and anaerobic degradation have been demonstrated, persistence of ethylene glycol in environments with sufficient active microbial populations is not expected.

43.2.3 Primary Routes of Exposure from Soil/Ground-water Systems

The above discussion of fate pathways suggests that ethylene glycol is essentially nonvolatile, is very weakly adsorbed to soil, and has no significant potential for bioaccumulation. These fate characteristics suggest several potential exposure pathways.

The potential for ground water contamination with ethylene glycol is high, particularly in sandy soils. It has been detected in ground water associated with hazardous waste sites. Mitre (83) reported that ethylene glycol has been found in 1 of the 546 National Priority List (NPL) sites. At this particular site it was detected in surface water. However, it may not be commonly analyzed for at NPL sites as it is not a priority pollutant and is not commonly thought to be of concern to

public health. The properties of ethylene glycol suggest that drinking water exposure from ground water contamination is likely to be its primary route of exposure from soil/ground-water systems.

The movement of ethylene glycol in ground water may result in discharge to surface water. As a result, ingestion exposures may occur resulting from the use of surface waters as drinking water supplies, and dermal exposures may result from the recreational use of surface waters. Such exposures are likely to be lower than those from drinking contaminated ground water due to the degradation of ethylene glycol in surface water. Any pathways related to the uptake by aquatic organisms or domestic animals from surface waters are likely to be less significant than other sources of exposure due to the low BCF for ethylene glycol.

41.2.4 Other Sources of Human Exposure

Ethylene glycol is used in antifreeze, hydraulic fluids, electrolytic condensors, heat exchangers and as an industrial solvent. As such, there are likely to be a number of sources of human exposure, however, data documenting these exposures are lacking.

43.3 HUMAN HEALTH CONSIDERATIONS

43.3.1 Animal Studies

43.3.1.1 Carcinogenicity

No tumors were reported when ethylene glycol was injected subcutaneously into rats and mice for 2-15 months; the levels used were not cited (1034,1035,1036). Blood (1037) found no increased incidence of tumors in rats after being fed a diet containing 1% ethylene glycol for 2 years. Ethylene glycol is currently under investigation by the NTP to determine possible carcinogenic effects via dietary exposure (0047).

43.3.1.2 Mutagenicity

Ethylene glycol was found to be inactive in the TA-98, TA-100, TA-1535 and TA-1537 strains of Salmonella typhimurium (1017,1038).

43.3.1.3 Teratogenicity, Embryotoxicity and Reproductive Effects

Maronpot et al. (1018) fed 80 pregnant Fischer 344 rats a diet containing 0, 0.04, 0.2 or 1 g/kg/day of ethylene glycol on days 6-15 of gestation. No maternal toxicity or increased incidence of teratogenic effects were observed at any of the dosage levels used. There was a statistically significant increase in delayed ossification (24% vs. 3% in control animals) and unossified (44% vs. 19% in control

animals) vertebrae centra observed in fetuses of the dams that received 1 g/kg/day of ethylene glycol. These effects were attributed to delayed maturation and were considered evidence of minimal embryotoxicity.

In a recent study, Lamb et al. (1019) continuously administered 0, 0.25, 0.5 or 1% ethylene glycol in the drinking water of male and female CD-1 mice. Mice from each treatment group were paired and allowed to mate. No treatment-related effects were observed on body weight or water consumption and no clinical signs of toxicity were evident in the parental generation. During a 14-week co-habitation period, exposure to 1% ethylene glycol in the drinking water was associated with a statistically significant decrease in the number of litters per fertile pair (4.5 vs. 4.9), the mean number of live pups per litter (10.2 vs. 10.8) and the mean live pup weight (1.53 vs. 1.63). The neonatal pups exhibited various malformations such as fused ribs, twisted spine, abnormally shaped or missing sternebrae, abnormally shaped vertebrae and cleft lip following continuous exposure of the parents to 1% (1.64 g/kg/day) ethylene glycol in the drinking water. No such defects were observed among control mice. The progeny of the F_1 generation, also continuously exposed to the 1% ethylene glycol drinking water, displayed modified craniofacial characteristics as adults which were not apparent in the neonatal period of growth. These facial abnormalities included a shortened snout and wide set eyes. Fertility in the F_1 generation was also decreased (61% vs. 80% in the control group).

Price et al. (1020) confirmed and expanded the results reported in the Lamb study (1019). Pregnant CD rats were dosed by gavage with 0, 1250, 2500 or 5000 mg/kg/day and CD-1 mice with 0, 750, 1500 or 3000 mg/kg/day of ethylene glycol on days 6-15 of gestation. No maternal deaths or distinctive clinical signs were noted, however, a significant dose-related decrease in maternal weight gain was observed at all levels in rats and at the 1500 and 3000 mg/kg/day levels in mice. Fetal body weight per litter was significantly reduced at the mid- and high-dosage levels in the rats and at all levels in the mice. The percentage of litters with malformed fetuses was significantly increased in all treatment groups and followed a dose-related trend. The most commonly observed malformations were cleft lip and palate, fused ribs, neural tube closure defects and abnormally shaped vertebrae and sternebrae. In mice, two fetuses in one litter in the 3000 mg/kg/day treatment group each exhibited a mid-facial cleft, which is an unusual defect for the CD-1 species. The shortened frontal, nasal and parietal bones observed in the F_1 mice following continuous pre- and postnatal exposure to ethylene glycol (1019) were not observed in fetal rats or mice in this study.

Union Carbide (1021) reported results of a recent inhalation study on the teratogenic effects of ethylene glycol in CD rats and CD-1 mice. The duration of the study was not provided. A reduced ossification in the humerous, zygomatic arch and hind limb metatarsals and phalanges indicated slight fetotoxicity in rats exposed to 1000 or 2500 mg/m^3

ethylene glycol. CD-1 mice also exposed to 1000 or 2500 mg/m^3 ethylene glycol experienced reduced body weight and reduced ossification at numerous skeletal locations. Teratogenicity was demonstrated at both of these concentrations in mice as shown by a statistically significant increase of external, visceral and skeletal malformations. Predominant terata included exencephaly, cleft palate, abnormal faces and facial bones, fused vertebrae and abnormal ribs.

A possible mechanism of action for the teratogenic effects of ethylene glycol has been proposed by Lamb (1019). A metabolite of ethylene glycol, oxalic acid, is known to chelate calcium. Lamb suggests that this calcium chelation may lead to hypocalcemia and may act upon fetal development by altering the biological supply of the calcium cation.

43.3.1.4 Other Toxicologic Effects

43.3.1.4.1 Short-term Toxicity

Ingestion of ethylene glycol generally results in depression followed by respiratory and cardiac failure, renal damage and possibly brain damage (54). The oral LD_{50} for mice is listed as 7500 mg/kg (47). Inhalation of ethylene glycol primarily results in depression of the CNS and hematopoietic dysfunction but rarely results in death (54). No LC_{50} value was found in the literature for ethylene glycol. The dermal LD_{50} is listed as 19,530 mg/kg in rabbits (47).

Typical signs of ethylene glycol poisoning are best exemplified in the dog. Dogs were orally given 6 ml ethylene glycol per kilogram of body weight. Signs included incoordination, increased depth and rate of respiration and increased heart rate. As progression of the poisoning continued, collapse and labored breathing ensued. Coma and death occurred within 37 hours. Necropsy revealed pulmonary and gastric hyperemia, severe toxic tubular nephrosis and renal oxalosis (1022). One to three hours after feeding dogs 9.5 ml ethylene glycol per kilogram of body weight, Grauer *et al.* (1023) observed depression, incoordination and increased fluid intake and urine output. Severe metabolic acidosis developed as the osmolal and anion gap increased. Within 6 hours, calcium oxalate crystalluria was observed, but it was not until 48 hours post-ingestion that a diminished renal excretory function was seen.

McDonald *et al.* (1046) injected 0.5 ml of 0, 0.004, 0.04, 0.4, 4 or 40% ethylene glycol solution into the corneal shelf of albino New Zealand rabbits. One treatment per day was given for 5 days. The 0.4% solution was found to be the highest concentration that was nontoxic and non-irritating. Irritation resulting from the 4 and 40% solutions consisted of swelling, discharge and conjunctival redness. All eyes returned to normal within 7 days of the last treatment. No evidence of systemic toxicity was observed.

The effect of ethylene glycol on brain function was tested in the male albino rat by Rajagopal (1024). Rats were given 10 mg/kg of a 50% aqueous solution of ethylene glycol by an intragastric tube. Urinary pH, blood pH and plasma bicarbonate levels all fell indicating a condition of metabolic acidosis. In response to the acidotic state, the renal distal tubular cells synthesized 332% more ammonia. The calcium oxalate deposition in the kidney and the oliguria caused a back diffusion of ammonia into the blood stream, resulting in a 497% increase in blood ammonia. The levels of brain amino acids (glutamate, GABA and glutamine) were altered in an attempt to detoxify the large amounts of ammonia entering the brain via the blood stream. The glutamate levels dropped 15.2% in order to utilize the ammonia to synthesize glutamine (which increased by 29.7%). The GABA level was reduced by 20.5%. This change in amino acid balance affected neurotransmission, and may be a possible explanation for the brain damage and even death seen in several cases of ethylene glycol toxicity.

43.3.1.4.2 Chronic Toxicity

The primary effect of repeated oral doses of ethylene glycol is kidney damage. Injury may occur even though oxalate crystals are not deposited in the kidney.

The effect of ethylene glycol on the kidney was studied by Roberts and Seibold (1047). Ethylene glycol was administered in levels ranging from 0.25 - 10% in the drinking water of several macaque species of monkeys. The left kidney was removed from all animals between days 6 and 13 of the experiment. Animals were sacrificed when they were uremic (the build-up of protein by-products in the blood due to inadequate kidney function) or dying. Seven out of ten animals received 15 ml/kg or more ethylene glycol. Five of these animals had deposition of calcium oxalate crystals in the proximal tubules. Tubular epithelium adjacent to the crystals was necrotic. Six animals were continued on the experiment for longer than 12 days. Three of these animals (ethylene glycol dose ranging from 33 to 137 ml/kg) had renal changes proportional to the dose given. Well marked to extreme deposition of calcium oxalate crystals in the proximal tubules along with necrosis of epithelial cells were present. Monkeys given total doses of less than 15 ml/kg ethylene glycol developed mild glomerular damage, but no calcium oxalate crystals were present. This led Roberts and Seibold to speculate that ethylene glycol or its metabolic products other than oxalic acid are capable of causing renal damage. Deposition of calcium oxalate crystals were also found in tissues other than kidney. Three animals that were found in a dying state by day 31 of the experiment had oxalate crystals present in the walls of the cerebral vessels and adjacent tissues. This study concluded that high doses of ethylene glycol causes nephrotoxic necrosis in the proximal tubules while low doses of ethylene glycol cause abnormal glomerular permeability.

Rats maintained on a diet containing 1 or 2% ethylene glycol, developed calcium oxalate bladderstones and severe renal injury and degeneration (1039).

43.3.2 Human and Epidemiologic Studies

43.3.2.1 Short-term Toxicologic Effects

The primary route of exposure to ethylene glycol in humans is by accidental or deliberate ingestion. Ingestion of about 100 mL can be fatal (12). The effects of ethylene glycol poisoning usually appear in three distinct phases. The onset of the first stage begins approximately 30 minutes to 12 hours following ingestion and predominately affects the CNS. With small doses, the victim appears drunk, but without the odor of alcohol on the breath; with large doses, stupor, coma, convulsions and possible death occur within the first 24 hours. If the individual survives beyond the initial 12-24 hours, cardiopulmonary signs become prominent. This phase is characterized by tachypnea, cyanosis, pulmonary edema and possible death within the next 24 hours. The final stage primarily affects the renal system and includes such signs and symptoms as flank pain, metabolic acidosis and anuria. Death may occur as late as 17 days post-ingestion (12).

Acute levels of ethylene glycol in the human body may lead to various metabolic problems. A 24-year-old man deliberately ingested an unknown quantity of ethylene glycol. The victim developed pulmonary edema and a decreased pulmonary compliance that fit the criteria for the Adult Respiratory Distress Syndrome (ARDS). Although many deaths from ethylene glycol have been attributed to cardiopulmonary dysfunction, this case is unusual because it represents a respiratory dysfunction in the presence of normal cardiac function (1025).

Cieciura (1026) examined renal biopsies of five patients with acute ethylene glycol poisoning on days 5, 10, 16 and 22 of hospitalization. Extensive calcium oxalate and carbonate crystals were present in the glomerular interloop spaces of the kidney which exerted mechanical as well as toxic effect on surrounding tissue. The crystals were shown to persist until 22 days post-ingestion.

Edelhauser et al. (1027) studied the effects of high concentrations of ethylene glycol on human corneas in culture. No damage to the corneal endothelium was reported when up to 5000 ppm ethylene glycol was exposed directly on the cornea for 2 hours.

43.3.2.2 Chronic Toxicologic Effects

Chronic exposure to ethylene glycol is rare in humans. Symptoms are generally listed as anorexia, oliguria, nystagmus, lymphocytosis and loss of consciousness (54).

An unusual case of chronic ethylene glycol toxicity due to inhalation was reported by Troisi (1040,1041). Thirty-eight women were exposed to a mixture containing 40% ethylene glycol, 55% boric acid and 5% ammonia at 105°C while working in an electrolytic condenser factory. Nine women suffered frequent attacks of unconsciousness 2-3 times a week. Fourteen women developed nystagmus (an involuntary rapid movement of the eye ball) and five showed an absolute lymphocytosis. The attacks ceased immediately once exposure to ethylene glycol vapor ceased.

43.3.3 Levels of Concern

The ACGIH (3) has established a ceiling limit of 50 ppm for ethylene glycol. OSHA (298) has no established standard.

The EPA (383) has developed health advisories of 19 mg/L for one-day exposure and 5.5 mg/L for long-term exposure to ethylene glycol in drinking water.

43.3.4 Hazard Assessment

Ethylene glycol is not considered to be either a carcinogenic or mutagenic hazard. A chronic feeding study using rats fed 1% ethylene glycol in the diet produced no evidence of carcinogenic activity (1037). There are no data to indicate any mutagenic activity either.

Ethylene glycol has been shown to produce dose-related teratogenic effects in rats and mice when administered by gavage or via the drinking water (1019,1020) as well as by inhalation (1037).

The principal hazard to humans appears to be associated with ingestion of large quantities of ethylene glycol. Depression of the central nervous system, serious renal injury and death may result from ingestion of about 100 mL (12). Early symptoms following ingestion are similar to alcoholic inebriation, but if untreated, can result in respiratory failure, convulsions, cardiovascular collapse and severe metabolic acidosis (12).

43.4 SAMPLING AND ANALYSIS CONSIDERATIONS

Determination of the concentration of ethylene glycol in soil and water requires collection of a representative field sample and laboratory analysis. Care is required to prevent losses during sample collection and storage. Soil and water samples should be collected in airtight containers with little or no headspace; analysis should be completed within 14 days of sampling. In addition to the targeted samples, quality assurance samples such as field blanks, duplicates, and spiked matrices should be included in the analytical program.

Ethylene glycol is not included among the EPA-designated priority pollutants, and an EPA-approved procedure for the analysis of ethylene glycol is not available. However, the recommended analytical method

for glycols (1142) is gas chromatography with flame ionization detection (GC/FID). Samples may either be directly injected onto the gas chromatographic (GC) column (aqueous and organic liquid samples) or they may first be extracted with an organic solvent (e.g., methylene chloride) and the concentrated extract injected onto the GC column (aqueous and solid samples). Detection of ethylene glycol is then accomplished by a flame ionization detector. A mass spectrometer using either electron impact (EI) or chemical ionization (CI) techniques may also be used to detect ethylene glycol.

A detection limit for ethylene glycol using these methods was not determined but would be in the range of µg/L for aqueous samples and µg/g for non-aqueous samples which have been extracted and parts-per-million (ppm) range for samples which have been directly injected.