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U.S. Department of Homeland Security
Washington, DC 20528

Homeland Security
Science and Technology

March 20, 2018

SENT BY ELECTRONIC MAIL TO: **john@greenewald.com**

John Greenewald, Jr.
27305 W. Live Oak Road
Suite 1203
Castaic, CA 91384

Re: **2018-STFO-00103**

Dear Mr. Greenewald:

This is the electronic final response to your Freedom of Information Act (FOIA) request to the Department of Homeland Security (DHS) Science and Technology Directorate (S&T), dated March 6, 2018, and received by this office on March 6, 2018. You are seeking you requested: (U) Chemical Agent Fact Book, Accession Number: ADB362101, Personal Author(s): Levine, Eric S., Grasso, Paul S., Chesler, William P., Ashman Steven N., Hawkins, Jill C., Sharp, Sharon L.; Corporate Author: DEPARTMENT OF HOMELAND SECURITY, ABERDEEN PROVING GROUND, MD CHEMICAL SECURITY ANALYSIS CENTER; Report Date: May 2010, Descriptive Note: Final rept. 5 Jan 2009-21 May 2010, Pages:220 Page(s), Report Number: DHS/CSAC-BK-09-001 (DHSCSACBK09001) , XJ - DHS/CSAC (XJDHSCSAC), Monitor Series: DHS/CSAC (DHSCSAC). [REQUESTDESCRIPTION].

A search of the DHS S&T Office of National Labs for documents responsive to your request produced a total of 220 pages. Of those pages, I have determined that 219 pages of the records are releasable in their entirety, and 1 page is partially releasable, pursuant to Title 5 U.S.C. § 552 (b)(6), FOIA Exemptions 6.

You have a right to appeal the above withholding determination. Should you wish to do so, you must send your appeal and a copy of this letter, within 90 days of the date of this letter, to: Privacy Office, Attn: FOIA Appeals, U.S. Department of Homeland Security, 245 Murray Lane, SW, Mail Stop 0655, Washington, D.C. 20528-0655, following the procedures outlined in the DHS FOIA regulations at 6 C.F.R. Part 5 § 5.8. Your envelope and letter should be marked “FOIA Appeal.” Copies of the FOIA and DHS FOIA regulations are available at www.dhs.gov/foia.

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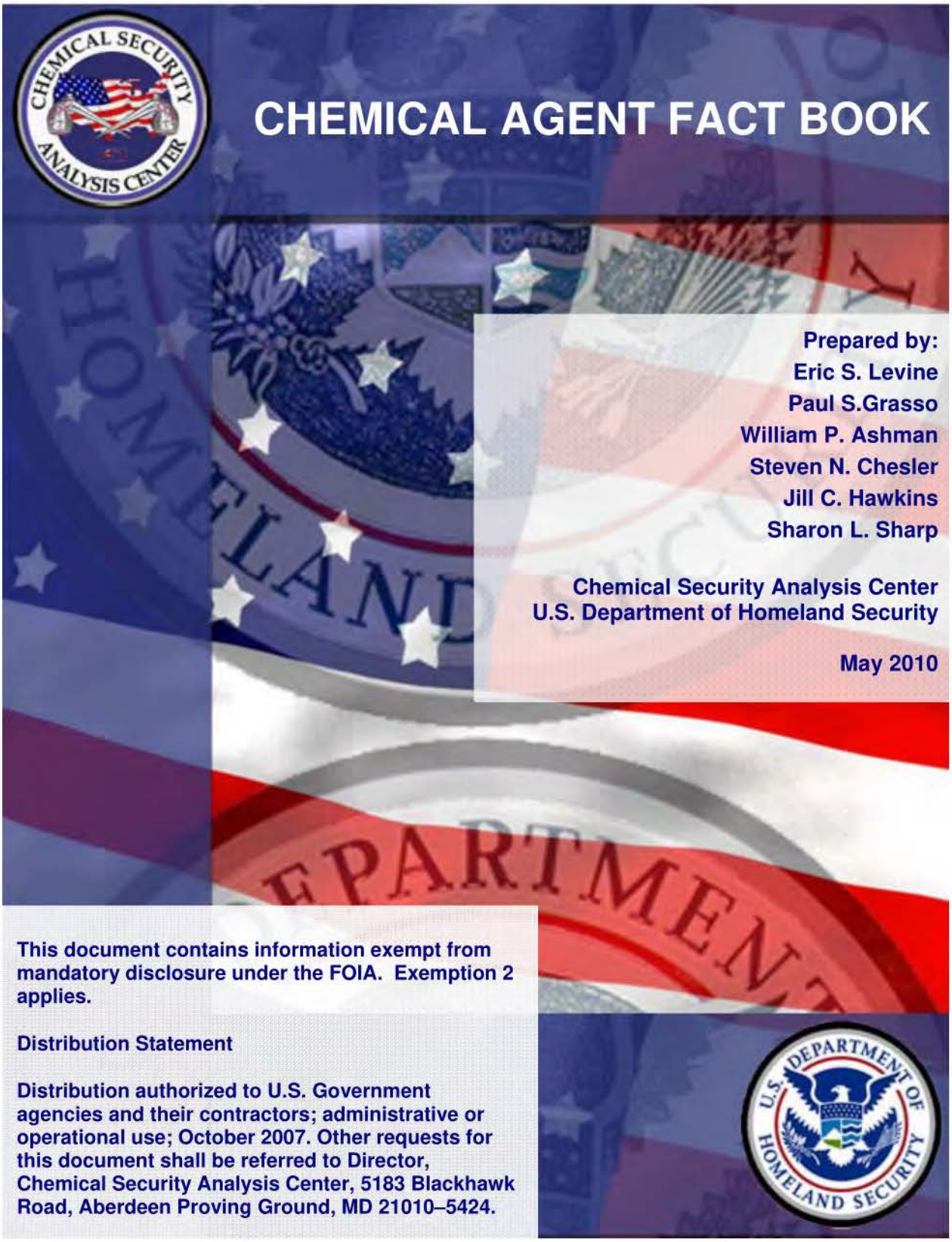
If you need any further assistance or would like to discuss any aspect of your request, please contact the analyst below who processed your request and refer to **2018-STFO-00103**. You may send an e-mail to stfoia@hq.dhs.gov, call 202-254-5700, or you may contact our FOIA Public Liaison in the same manner. Additionally, you have a right to right to seek dispute resolution services from the Office of Government Information Services (OGIS) which mediates disputes between FOIA requesters and Federal agencies as a non-exclusive alternative to litigation. If you are requesting access to your own records (which is considered a Privacy Act request), you should know that OGIS does not have the authority to handle requests made under the Privacy Act of 1974. You may contact OGIS as follows: Office of Government Information Services, National Archives and Records Administration, 8601 Adelphi Road-OGIS, College Park, Maryland 20740-6001, e-mail at ogis@nara.gov; telephone at 202-741-5770; toll free at 1-877-684-6448; or facsimile at 202-741-5769.

Sincerely,

Sally Harris

Sally Harris
FOIA Officer

Enclosure(s): Chemical Agent Fact Book, 220 pages



CHEMICAL AGENT FACT BOOK

Prepared by:
Eric S. Levine
Paul S.Grasso
William P. Ashman
Steven N. Chesler
Jill C. Hawkins
Sharon L. Sharp

Chemical Security Analysis Center
U.S. Department of Homeland Security

May 2010

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U.S. Department of Homeland Security
Science & Technology



Chemical Security Analysis Center

CHEMICAL AGENT FACT BOOK

Prepared by:

Eric S. Levine
Noblis Contractor
Falls Church, VA 22042

Paul S. Grasso
Edgewood Chemical Biological Center
Aberdeen Proving Ground, MD 21010

William P. Ashman
Steven N. Chesler
Jill C. Hawkins
Sharon L. Sharp
Battelle Memorial Institute Contractor
Columbus, OH

U.S. Department Of Homeland Security
Chemical Security Analysis Center
Aberdeen Proving Ground, MD 21010–5424

May 2010

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Paul S. Grasso
William P. Ashman
Steven N. Chesler
Jill C. Hawkins
Sharon L. Sharp

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1.0 MILITARY AGENTS

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NERVE AGENTS

Homeland Security
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Chemical Security Analysis Center

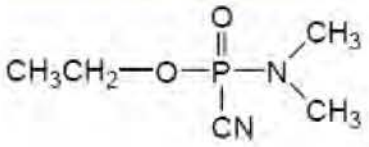
Tabun (GA)

AGENT OVERVIEW

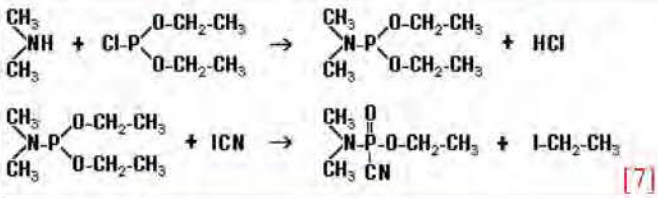
Tabun, also known by its NATO designation GA, is an extremely toxic man-made chemical warfare agent whose sole application is as a nerve agent. GA is classified as a weapon of mass destruction by the United Nations by UN Resolution 687, and production and stockpiling GA was outlawed by the Chemical Weapons Convention (CWC) of 1993. GA was discovered in 1936 by a German scientist who synthesized sarin (GB) two years later. During World War II, Germany developed chemical weapons using both GA and GB but never used them.[1][2][3]

CHEMICAL IDENTIFICATION

Common chemical name	Tabun
CAS number	77-81-6[4]
IUPAC name	Dimethylamino(ethoxy)phosphoryl]formonitrile[5]
Synonyms	Ethyl dimethylamidocyanophosphate; dimethylaminoethoxy-phosphoryl cyanide; dimethylaminocyanophosphoric acid ethyl ester; cyanodimethylaminoethoxyphosphine; dimethylamine-cyanoethoxyphosphine oxide; ethyl dimethylaminocyano-phosphonate; phosphoramidocyanidic acid, dimethyl, ethyl ester; dimethylamidoethoxyphosphoryl cyanide; dimethylamino-cyanphosphorsaeureaethylester (German); dimethylphosphor-amidocyanidic acid, ethyl ester; ethyl dimethylphosphor-amidocyanidate; ethylester-dimethylamid kyseliny kyanfosfonove (Czech)[4]
Empirical formula	$C_5H_{11}N_2O_2P$[4]

Chemical structure	 [4]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially[5] • Theft and divergence: Possible in third world nations[3] • Synthesis: Synthetic pathways well known[6]
Synthetic pathways	• Precursors: Diethyl N,N-dimethylphosphoramidate; dimethylamine; dimethylamine hydrochloride; phosphorus oxychloride; phosphorus pentachloride; phosphorus trichloride; potassium cyanide; sodium cyanide[6] • Precursor availability: Most are limited by the CWC.
Synthesis	 [7]

PHYSICAL PROPERTIES

Physical form	Colorless to brown liquid; odor faintly fruity; no odor when pure[4]
Molecular weight	162.13 Daltons[4]
Liquid/solid density	1.0756 g/mL @ 25 °C[8]
FP/MP	-50 °C (FP)[9]
Boiling point	248 °C (extrapolated)[10]
Viscosity	2.277 cP @ 25.0 °C (liquid)[8]; 6.20 x 10^{-3} cP @ 25.0 °C (vapor)[8]
Surface tension	32.5 dynes/cm @ 25 °C (liquid)[8]
Vapor pressure	0.057 mm Hg @ 25.0 °C[10]
Volatility	497 mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[10]
Vapor density (Air=1)	5.6 (calculated from the Ideal Gas Law)[10]
Solubility	Solubility in water is 7.2 g/100 g @ 20 °C[11]; very soluble in organic solvents.[12]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	$t_{1/2}$ = 8.5 hr @ 20 °C and pH 7; slow in water but fairly rapid with strong acids and alkalis with self-buffering at pH 4-5; autocatalytic below pH 4[13]
Hydrolysis products	Hydrogen cyanide and dimethylaminocyanophosphonic acid are formed, which further hydrolyze to produce methylphosphonic acid and pinacolyl alcohol.[13]
Photo	Probably reacts to strong UV[14]
Thermal	Decomposes completely @ 150 °C after about 3-3.25 hr[15]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[16] 
DOT identification number	UN 2810[16]	
DOT safety placard(s)	[16] 	[16] 
		[16] 

CLINICAL PRESENTATION

Time to effect	• Inhalation: Inhaled tabun produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 min.[16] • Skin: Liquid tabun may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hours; larger exposures may cause death within minutes to hours.[16] • Ingestion: No information is available on the time course of effects following ingestion of tabun.[16]
Exposure signs/symptoms	Localized sweating, muscular twitching, pinpoint pupils, runny nose, tightness of the chest with shortness of breath, nausea, vomiting, involuntary defecation and urination, convulsions, coma, and respiratory arrest[11]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to

	break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[16]
Morbidity and mortality	• LCt_{50}: 62.1 mg-min/m^3[17] • ECt_{50} (severe): 44.4 mg-min/m^3[17] • ECt_{50} (mild): 0.34 mg-min/m^3[17] • LD_{50} (percutaneous): 1125 mg/70 kg man[17] • ED_{50} (severe percutaneous injury): 675 mg/70 kg man[17]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hours, can be taken as a pretreatment for nerve agent exposure.[18]
Antidote/Treatment	Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity. There is generally no benefit in giving more than three injections of 2-PAM Cl, but atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[18]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[19] • Laboratory: GC/MS[20] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions[19]
Environmental persistence	8 hours in water at 20 °C and pH 7.4[21]
Formulations	Tabun is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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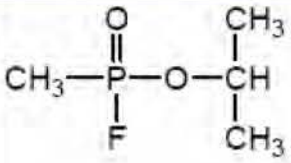
Sarin (GB)

AGENT OVERVIEW

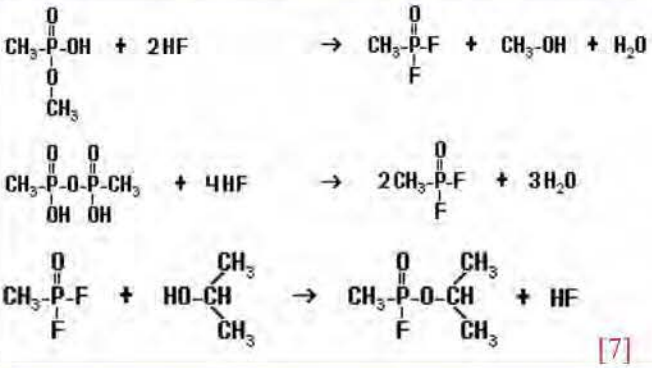
Sarin, also known by its NATO designation GB, is an extremely toxic substance whose sole application is as a nerve agent. As a chemical weapon, it is classified as a weapon of mass destruction by the United Nations by UN Resolution 687. Production and stockpiling of GB was outlawed by the Chemical Weapons Convention (CWC) of 1993. Nerve agents are the most toxic and rapidly acting of the known chemical warfare agents. Sarin originally was developed in 1938 in Germany as a pesticide. In the early 1950s NATO adopted GB as a standard chemical weapon, and both the U.S.S.R and the U.S. produced GB for military purposes. Iraq used GB against Iran during the 1980–88 war. In 1988, over the span of two days in March, the ethnic Kurd city of Halabja in northern Iraq (population 70,000) was bombarded with chemical and cluster bombs, which included GB, in the Halabja poison gas attack. An estimated 5,000 people died. In 1995, the Aum Shinrikyo sect released an impure form of GB in the Tokyo Subway resulting in twelve fatalities.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Sarin
CAS number	107-44-8[2]
IUPAC name	2-(Fluoro-methyl-phosphoryl)oxypropane[3]
Synonyms	Fluorisoproopoxymethylphosphine oxide; Isopropyl methyl-fluorophosphate; Isopropylmethanefluorophosphonate; Iso-propoxymethylphosphoryl fluoride; Propoxyl-2-methyl-phosphoryl fluoride; Phosphonofluridicacid, methyl-, isopropyl ester; Isopropylester kyseliny methylfluorfosfonove (Czech); O-Isopropyl methylphosphonofluoridate; Isopropyl-methylphosphoryl fluoride; Methylphosphonofluoridic acid isopropyl ester; Methylphosphonofluoridic acid 1-methylethyl ester; Phosphine oxide, fluoroisopropoxymethyl-; Phosphoric acid, methylfluoro-, isopropyl ester; Methylfluorphosphor-saeureisopropylester (German)[2]
Empirical formula	$C_4H_{10}FO_2P$[2]

Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially[4] • Theft and divergence: Possible in third world nations[5] • Synthesis: Synthetic pathways well known
Synthetic pathways	• Precursors: Ammonium bifluoride, dimethyl methylphosphonate, diethylphosphite, dimethylphosphite, hydrogen fluoride, methylphosphonous difluoride, methylphosphonyl dichloride, methylphosphonyl difluoride, potassium bifluoride, potassium fluoride, phosphorus trichloride, sodium bifluoride, sodium fluoride, thionyl chloride, trimethyl phosphate[6] • Precursor availability: Most are limited by the CWC
Synthesis	 [7]

PHYSICAL PROPERTIES

Physical form	Colorless liquid; no odor when pure[2]
Molecular weight	140.09 Daltons[2]
Liquid/solid density	1.887 g/mL @ 25 °C[8]
FP/MP	-56 °C (FP)[9][10][11]
Boiling point	150 °C (extrapolated)[9]
Viscosity	1.397 cP @ 25.0 °C (liquid) (extrapolated); 7.19 x 10^{-3} cP @ 25.0 °C (vapor)[12]
Surface tension	25.9 dynes/cm @ 25.0 °C[12]
Vapor pressure	2.48 mm Hg @ 25 °C (calculated from vapor pressure)[9]

Volatility	18,700 mg/m^3 @ 25 °C[9]
Vapor density (Air=1)	4.8 (calculated)[2]
Solubility	Completely miscible with water and common organic solvents[2][13]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Varies with pH and temperature; at 20 °C, $t_{1/2}$ = 27 min @ pH 1; $t_{1/2}$ = 3.5 hr @ pH2; $t_{1/2}$ = 80 hr @ pH 7; $t_{1/2}$ = 5.4 min @ pH 10; and $t_{1/2}$ = 0.6 min @ pH 11[14]
Hydrolysis products	Under acidic conditions, hydrogen fluoride (HF) and isopropyl methylphosphonic acid are formed which further hydrolyze to produce methylphosphonic acid (MPA) and isopropanol. Under alkaline conditions, methylfluorophosphonic acid and isopropyl alcohol are initially formed which further hydrolyze to produce MPA and HF.[15]
Photo	Strong UV[16]
Thermal	Complete decomposition occurs within 2.5 hr @ 150 °C[17]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[18] 
DOT identification number	UN 2810[18]	
DOT safety placard(s)	[18] 	[18] 	[18] 

CLINICAL PRESENTATION

Time to effect	• Inhalation: Inhaled sarin produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 min.[18] • Percutaneous: Liquid sarin may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hr; larger exposures may cause death within minutes to hours.[18] • Ingestion: No information is available on the time course of effects following ingestion of sarin.[18]

Exposure signs/symptoms	Localized sweating, muscular twitching, pinpoint pupils, runny nose, tightness of the chest with shortness of breath, nausea, vomiting, involuntary defecation and urination, convulsions, coma, and respiratory arrest[19]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[20]
Morbidity and mortality	• LCt_{50}: 31 mg-min/m^3[21] • ECt_{50} (severe): 22 mg-min/m^3[21] • ECt_{50} (mild): 0.35 mg-min/m^3[21] • LD_{50} (percutaneous): 1275 mg/70 kg man[21] • ED_{50} (severe percutaneous injury): 750 mg/70 kg man[21]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hr, is a pretreatment drug for nerve agent exposure.[22]
Antidote/Treatment	Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, 2-PAM Cl must be administered within minutes to a few hours following exposure to be effective. There is also generally no benefit in giving more than three injections of 2-PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[18]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical Agent, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[23] • Laboratory: GC/MS[24] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions[23]
Environmental persistence	In distilled water at 25 °C, the hydrolysis half-lives range from 75 hr at pH 7 to 0.8 hr at pH 9. The hydrolysis rate increases in seawater due to the catalytic effect of ions; the seawater hydrolysis half-life at pH 7.6 and 25 °C is about 1 hr.[25]

Formulations	Sarin is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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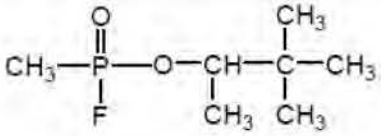
Soman (GD)

AGENT OVERVIEW

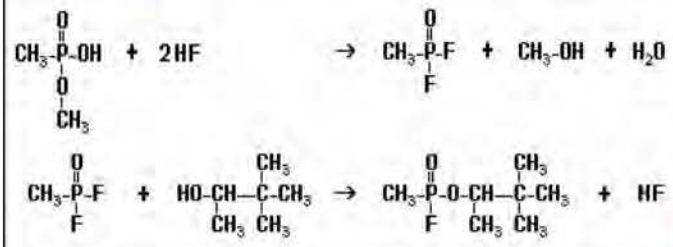
Soman, also known by its NATO designation of GD, is an extremely toxic substance whose sole application is as a nerve agent. As a chemical weapon, it is classified as a weapon of mass destruction by the United Nations by UN Resolution 687. Production and stockpiling of GD was outlawed by the Chemical Weapons Convention of 1993. Nerve agents are the most toxic and rapidly acting of the known chemical warfare agents. GD was discovered by Richard Kuhn in Germany in 1944. NATO countries decided not to produce GD because pinacolyl alcohol was commercially, however the former Soviet Union did weaponize it. Of all the G-agents, GD is the most difficult to medically mitigate due its extremely fast conversion from a reversible cholinesterase adduct to an irreversible adduct.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Soman
CAS number	96-64-0[3]
IUPAC name	3-[Fluoro(methyl)phosphoryl]oxy-2,2-dimethylbutane[4]
Synonyms	3,3-Dimethyl-n-but-2-yl methylphosphonofluoridate; 3,3-dimethyl-2-butyl methylphosphonofluridate; 2-butanol, 3,3-dimethyl-, methylphosphonofluoridate; methylphosphono-fluoridic acid, 3,3-dimethyl-2-butyl ester; 1,2,2-trimethylpropylmethyphosphonofluoridate; 1,2,2-trimethylpropylester kyseliny methylfluorfosfonove (Czech); methylphosphonofluoridic acid 1,2,2-trimethylpropyl ester; phosphonofluoridic acid, methyl-, 1,2,2-trimethylpropyl ester; phosphine oxide, fluoromethyl (1,2,2-trimethylpropoxy)-; methyl pinacolyl phosphonofluoridate; pinacolyl methylfluorophosphonate; fluoromethylpinacolyl-oxyphosphine oxide; methyl pinacolyloxyfluorophosphine oxide; pinacolyl methanefluorophosphonate; pinacoloxy-methylphosphoryl fluoride; methylfluoropinacolyl-phosphonite;

	methylfluorphosphorsaeurepinakolylester (German); methyl pinacolyloxy phosphorylfluoride; methyl pinacolylphosphonofluoridate; pinacoloxymethylphosphoryl fluoride; pinacolyl methylphosphonofluoride; pinacolyl-oxymethylphosphoryl fluoride; pynacolyl methylfluoro-phosphonate[3]
Empirical formula	$C_7H_{16}FO_2P$[3]
Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Soman is a Schedule 1 CWC chemical and therefore is not available for purchase.[5] • Theft and divergence: Possible in third world nations • Synthesis: Synthetic pathways well known
Synthetic pathways	• Precursors: Ammonium bifluoride; dimethyl methylphosphonate; diethylphosphite; dimethylphosphite; hydrogen fluoride; methylphosphonous difluoride; methylphosphonyl dichloride; methylphosphonyl difluoride; pinacolone; pinacolyl alcohol; potassium bifluoride; potassium fluoride; phosphorus trichloride; sodium bifluoride; sodium fluoride; thionyl chloride; trimethyl phosphite[6] • Precursor availability: Most are limited by the CWC
Synthesis	 [7]

PHYSICAL PROPERTIES

Physical form	Colorless liquid @ 25 °C. Odor is fruity; impurities give it the odor of camphor[3]
Molecular weight	182.17 Daltons[3]
Liquid/solid density	1.0222 g/mL @ 25 °C[8]
FP/MP	-42 °C (MP)[9][10]; generally solidifies to a noncrystalline, glasslike material[3]
Boiling point	198 °C (extrapolated) decomposes[11]

Viscosity	3.167 cP @ 25.0 °C (liquid)[3]; 5.90 x 10^{-3} cP @ 25.0 °C (vapor)[3]
Surface tension	24.5 dynes/cm @ 25.5 °C[3]
Vapor pressure	0.401 mm Hg @ 25 °C
Volatility	3930 mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[3]
Vapor density (Air=1)	6.3 (calculated from the Ideal Gas Law)[3]
Solubility	Solubility in water is 2.1 g/100 g @ 20 °C[12][13]; very soluble in organic solvents[14]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Varies with pH; using a 3 mM solution of GD @ 25 °C, $t_{1/2}$ = 3 hr @ pH 2; $t_{1/2}$ = 45 hr @ pH 6.65; $t_{1/2}$ = 60 hr @ pH 10; complete hydrolysis occurs in less than 5 min in a 5% NaOH solution[15]
Hydrolysis products	Under acidic conditions, hydrogen fluoride (HF) and pinacolyl methylphosphonic acid (PMPA) are formed, which further hydrolyze to produce methylphosphonic acid (MPA) and pinacolyl alcohol.[15]
Photo	Probably reacts to strong UV as does sarin[16]
Thermal	Above 150 °C[3]; unstabilized decomposes in 4 hr @ 130 °C[3]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[17] 
DOT identification number	UN 2810[17]	
DOT safety placard(s)	[17] 	[17]  [17] 

CLINICAL PRESENTATION

Time to effect	• Inhalation: Inhaled soman produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 min.[17] • Skin: Liquid soman may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hours; larger exposures may cause death within minutes to hours.[17]

	• Ingestion: No information is available on the time course of effects following ingestion of soman.[17]
Exposure signs/symptoms	Localized sweating, muscular twitching, pinpoint pupils, runny nose, tightness of the chest with shortness of breath, nausea, vomiting, involuntary defecation and urination, convulsions, coma, and respiratory arrest[12]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[17]
Morbidity and mortality	• LCt_{50}: 31 mg-min/m^3[18] • ECt_{50} (severe): 22.2 mg-min/m^3[18] • ECt_{50} (mild): 0.4 mg-min/m^3[18] • LD_{50} (percutaneous): 275 mg/70 kg man[18] • ED_{50} (severe percutaneous injury): 157 mg/70 kg man[18]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hr, is a prophylactic against soman intoxication.[19]
Antidote/Treatment	Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity. 2-PAM Cl is only effective for a few minutes after agent exposure, therefore its immediate administration is essential. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[19]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[20] • Laboratory: GC/MS[21] • Observation: None.
Decontamination—property & equipment	Strong bleach, caustic solutions[20]
Environmental persistence	Days to weeks[15][22]

Formulations	Soman is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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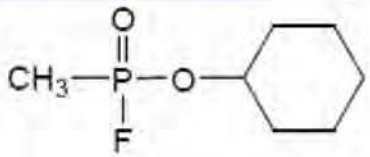
Cyclosarin (GF)

AGENT OVERVIEW

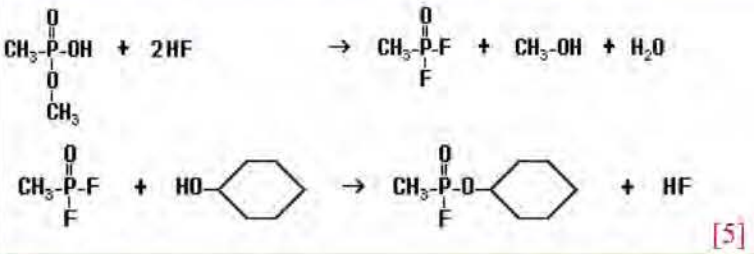
Cyclosarin, also known by its NATO designation of GF, was probably first synthesized during WW II as part of the systematic study of organophosphate nerve agents undertaken by the Germans after their potential military utility was identified. As a chemical warfare agent, GF is classified as a weapon of mass destruction by the United Nations by UN Resolution 687. Production and stockpiling of GF was outlawed by the Chemical Weapons Convention (CWC) of 1993.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Cyclosarin
CAS number	329-99-7[2]
IUPAC name	[fluoro(methyl)phosphoryl]oxycyclohexane[3]
Synonyms	Cyclohexyloxyfluoromethylphosphine oxide; cyclohexyl methylfluorophosphate; phosphonofluoridic acid, methyl-, cyclohexyl ester; methyl cyclohexylfluorophosphonate[2]
Empirical formula	$C_7H_{14}FO_2P$[2]

Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially[4] • Theft and divergence: Possible in third world nations[4] • Synthesis: Synthetic pathways well known[4]
Synthetic pathways	• Precursors: Ammonium bifluoride; cyclohexylamine; cyclohexanol; diethyl phosphite; dimethyl methylphosphonate; dimethylphosphite; hydrogen fluoride; methylphosphonous difluoride; methylphosphonyl dichloride; methylphosphonyl difluoride (DF); phosphorus trichloride; potassium bifluoride; potassium fluoride; sodium bifluoride; sodium fluoride; thionyl chloride; trimethyl phosphite[4] • Precursor availability: Most are limited by the CWC
Synthesis	 [5]

PHYSICAL PROPERTIES

Physical form	Colorless liquid @ 25 °C[6]
Molecular weight	180.16 Daltons[2]
Liquid/solid density	1.1276 g/mL @ 25 °C[7]
FP/MP	-12 °C (MP)[2]
Boiling point	228 °C (extrapolated)[8]
Viscosity	5.41 cP @ 25.0 °C (liquid)[9]; 6.15 x 10^{-3} cP @ 25.0 °C (vapor)[9]
Surface tension	32.3 dynes/cm @ 25.5 °C[9]
Vapor pressure	0.0843 mm Hg @ 25.5 °C[8]
Volatility	898 mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[8]
Vapor density (Air=1)	6.2 (calculated from the Ideal Gas Law)[8]
Solubility	Solubility in water is 3.7 g cyclosarin/100 g @ 20 °C[9]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	$t_{1/2}$ = 42 hr @ 25 °C using a 0.003 M solution of cyclosarin in distilled water[10]
Hydrolysis products	Hydrogen fluoride, cyclohexylmethylphosphonic acid[11]
Photo	Probably reacts to strong UV[12]
Thermal	Completely decomposes within 2 hr @ 150 °C[13]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	[14] 
	Flammability	
	Reactivity	
	Special Properties	
DOT identification number	UN 2810[14]	
DOT safety placard(s)	[14] 	[14] 
		[14] 

CLINICAL PRESENTATION

Time to effect	• Inhalation: Inhaled GF produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 min.[15] • Skin: Liquid GF may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hr; larger exposures may cause death within minutes to hours.[15] • Ingestion: No information is available on the time course of effects following ingestion of GF.[15]
Exposure signs/symptoms	Localized sweating, muscular twitching, pinpoint eye pupils, runny nose, tightness of the chest with shortness of breath, dimness of vision, headache, cramps, nausea, vomiting, involuntary defecation and urination, twitching, jerking, staggering, convulsions, drowsiness, coma, and respiratory arrest[16]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their

	undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[15]
Morbidity and mortality	• LCt_{50}: 31.0 mg-min/m^3[17] • ECt_{50} (severe): 22.2 mg-min/m^3[17] • ECt_{50} (mild): 0.17 mg-min/m^3[17] • LD_{50} (percutaneous): 262 mg/70 kg man[17] • ED_{50} (severe percutaneous injury): 150 mg /70 kg man[17]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hr, is a pretreatment drug for nerve agent exposure[18]
Antidote/Treatment	Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, 2-PAM Cl must be administered within minutes to a few hours (depending on the agent) following exposure to be effective. There is also generally no benefit in giving more than three injections of 2-PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[15]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[2] • Laboratory: GC/MS[19] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions[20]
Environmental persistence	Days to weeks (estimated)[11]
Formulations	GF is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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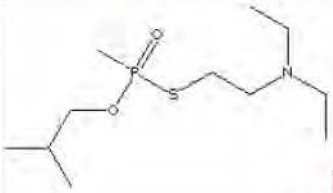
Russian VX (VR)

AGENT OVERVIEW

Two similar chemical compounds were developed by both Russia and the U.S. The Russian compound R-VX, also known as VR, has the same chemical composition and formula as VX, which was developed by the U.S. These two compounds differ only in structural conformation. R-VX occurs in both thickened (viscous) and non-thickened varieties, requiring two destruction processes to address both types effectively.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Russian VX
CAS number	159939-87-4[3]
IUPAC name	N,N-diethyl-2-(methyl-(2-methylpropoxy)phosphoryl)sulfanylethanamine[4]
Synonyms	O-isobutyl S-(2-diethylaminoethyl)methyl phosphothioate; O-isobutyl S-(2-diethylaminoethyl)methyl thiophosphonate; O-isobutyl S-(N,N-diethylaminoethyl)methylphosphonothioate; phosphonothioic acid, methyl-, S-(2-diethylamino)ethyl)O-(2-methylpropyl) ester; R 33; R-33; Russian V-gas; RVX; R-VX; S-(N,N-DEA)IMPT[3][5]
Empirical formula	$C_{11}H_{26}NO_2PS$[3]

Chemical structure	 [5]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: VR is not available commercially. • Theft and divergence: Possible from current stockpiles • Synthesis: Difficult
Synthetic pathways	• Precursors: Isobutyl methylphosphonate, phosphorus pentasulfide, phosphorus trichloride, and sulfur[5] • Precursor availability: Isobutyl methylphosphonate not available commercially
Synthesis	Contact CSAC

PHYSICAL PROPERTIES

Physical form	Oily consistency; colorless when pure[1]; odorless[6]
Molecular weight	267.38 Daltons[1][5]
Liquid/solid density	1.003 g/cm^3 @ 25 °C[1]
FP/MP	Contact CSAC
Boiling point	323 °C[1]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	0.048 Pa @ 20 °C, 0.084 Pa @ 25 °C (calculated values)[7]
Volatility	9.06 mg/m^3 @ 25 °C; 5.27 mg/m^3 @ 20 °C; 0.467 mg/m^3 @ 0 °C (calculated from vapor pressure)[7]
Vapor density (Air=1)	Heavier than air[5]
Solubility	Contact CSAC

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Rate of hydrolysis is slow.[1]
Hydrolysis products	Isobutyl methylphosphonate[5]; isobutyl methylphosphonic acid, 2-(diethylamino)ethanethiol, and P,P-diisobutyl dimethyl diphosphonate[1]
Photo	Not found

Thermal	Not found

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	Not found
	Flammability	
	Reactivity	
	Special Properties	
DOT identification number	Not found	
DOT safety placard(s)	Not found	

CLINICAL PRESENTATION

Time to effect	Signs and symptoms of overexposure may occur within minutes or hours depending upon dose.[1]
Exposure signs/symptoms	Inhalation: Headaches and pressure sensation, runny nose and nasal congestion, salivation, tightness in the chest, nausea, vomiting, giddiness, anxiety, difficulty in thinking, difficulty sleeping, nightmares, muscle twitches, tremors, weakness, abdominal cramps, diarrhea, involuntary urination and defecation. With severe exposure symptoms progress to convulsions and respiratory failure.[1] Skin: Headaches and pressure sensation, runny nose and nasal congestion, salivation, tightness in the chest, nausea, vomiting, giddiness, anxiety, difficulty in thinking, difficulty sleeping, nightmares, muscle twitches, tremors, weakness, abdominal cramps, diarrhea, involuntary urination and defecation. With severe exposure symptoms progress to convulsions and respiratory failure.[1] Eye: Miosis (constriction of pupils) and visual effects[1]
Personal decontamination	Eye: Flush eyes immediately with water for 10–15 min.[1] Skin: Remove contaminated clothing; wash contaminated skin with copious amounts of soap and water immediately using 10% sodium carbonate solution, or 5% liquid household bleach; rinse well with water to remove decontamination.[1] Ingestion: Do not induce vomiting.[1]
Morbidity and mortality	No VR toxicity estimates are available for humans, but the VR subcutaneous LD_{50} in guinea pigs is 11.3 µg/kg, similar to the VX LD_{50} of 8.9 µg/kg.[8] Lethal human VR doses by other routes might be similar to those for VX (e.g., VX LCt_{50} of 12 mg-min/m^3).[9]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hr, is a pretreatment drug for nerve agent exposure.[6]
Antidote/Treatment	Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[5][6]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Nerve agent sensitive chemical agent detectors (e.g., CAM, M18A2, M256, etc.) and papers (e.g., M8, M9) may be used for detection. However, detection limits have not been determined for this agent. Some reports suggest that this agent may be misidentified by automated detectors.[5] • Laboratory: Gas chromatography; NMR spectroscopy[7] • Observation: None
Decontamination—property & equipment	Surface decontamination may be accomplished using hypochlorite bleach slurries, dilute alkalis, or DS2 decontaminating solution. Steam and ammonia may be used for the decontamination of confined spaces.[5]
Environmental persistence	Heavily splashed liquid persists for long periods of time, especially in cold weather.[1]
Formulations	Russia weaponized a polymethyl methacrylate-thickened VR and plain VR. A binary version may also have been developed.[5]

The information cutoff date for this factsheet is 14 May 2010.

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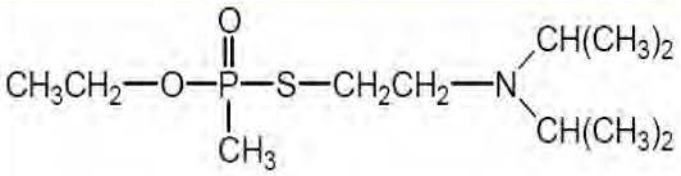
VX

AGENT OVERVIEW

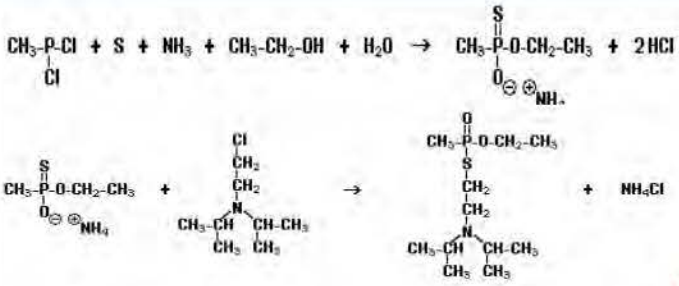
Ethyl {[2-[di(propan-2-yl) amino] ethylsulfanyl} methylphosphinate (VX) is an extremely toxic substance whose sole application is as a nerve agent. It is classified as a weapon of mass destruction by the United Nations in UN Resolution 687, and production and stockpiling of VX was outlawed by the Chemical Weapons Convention of 1993. VX is one of a family of organophosphates, known as phosphonothiolates, that were investigated in the late 1940s by the British as possible pesticides or chemical warfare agents. The British chemical warfare establishment, which initiated more systematic studies of the class at the facility at Porton Down, shared information on VX with the U.S. This eventually led to VX being selected by the United States for mass production as its second generation nerve agent in 1958. It has been estimated that Iraq synthesized over 50 tons of VX and may have used it against the Kurds in Northern Iraq. In the early 1990s VX was synthesized and used to commit assassinations by the Aum Shinrikyo organization in Japan.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	VX
CAS number	50782-69-9[3]
IUPAC name	Ethyl {[2-[di(propan- 2-yl) amino] ethylsulfanyl} methylphosphinate[4]
Synonyms	O-Ethyl-S-(2-diisopropylaminoethyl) methyl phosphonothiolate; S-(2-diisopropylaminoethyl)-O-ethyl methyl phosphonothiolate; ethyl-S-dimethylaminoethylmethylphosphonothiolate; phosphonothioic acid, methyl-, S-(2-(diisopropylamino)ethyl) O-ethyl ester; ethyl S-2-diisopropylaminoethyl methylphosphono-thiolate; ethyl-S-diisopropylaminoethyl methylthiophosphonate; methylphosphonothioic acid S-(2-(bis(methylethyl)amino)ethyl) O-ethyl ester; O-ethyl-S-2-diisopropylaminoethylester; kyseliny methylthiofosfonove (Czech)[3]
Empirical formula	$C_{11}H_{26}NO_2PS$[3]

Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: As a Schedule 1 chemical under the Chemical Weapons Convention, VX is not available commercially.[5] • Theft and divergence: Possible in third world nations • Synthesis: Synthetic pathways difficult but well known[6]
Synthetic pathways	• Precursors: Diethyl methylphosphonite; diisopropylamine; diisopropyl aminoethyl chloride; O-ethyl O-2-diisopropylaminoethyl methylphosphonite (code designation QL); O-ethylmethyl-phosphonothioic acid (also known as EMPTA); ethyl hydrogen methylphosphonite; methylphosphonous dichloride (also known as SW); methylphosphonous difluoride; methylphosphonothioic dichloride (also known as SWS); phosphorus pentasulfide; phosphorus trichloride; sulfur[2] • Precursor availability: Most are Limited by the CWC[5]
Synthesis	 [6]

PHYSICAL PROPERTIES

Physical form	Colorless or straw-colored odorless liquid[7][8][9]
Molecular weight	267.37 Daltons[3]
Liquid/solid density	1.0083 g/mL @ 25 °C[10]
FP/MP	<-51 °C (MP)[11]
Boiling point	292 °C (extrapolated)[12]
Viscosity	10.04 cP @ 25.0 °C (liquid)[3]; 5.13 x 10^{-3} cP @ 25.0 °C (vapor)[3]
Surface tension	31.3 dynes/cm @ 25.0 °C[3]
Vapor pressure	8.78 x 10^{-4} torr @ 25.0 °C[3]
Volatility	12.6 mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[3]

Vapor density (Air=1)	9.2 (calculated from the Ideal Gas Law)[3]
Solubility	Water solubility of VX is 5% @ 21.5 °C[3]; miscible with water below 9.4 °C[10]; soluble in common organic solvents[11]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Hydrolysis rate of VX varies with temperature and concentration. At 22 °C, $t_{1/2}$ = 1.8 min in 1.25 M NaOH; $t_{1/2}$ = 10.8 min in 0.25 M NaOH; $t_{1/2}$ = 31 min in 0.10 M NaOH; $t_{1/2}$ = 3.3 hr in 0.01 M NaOH; $t_{1/2}$ = 20.8 hr in 0.001 M NaOH; and $t_{1/2}$ = 60 hr in pure water.[13]
Hydrolysis products	VX hydrolyzes via three different pathways (P-S, P-O, and C-S), which vary significantly with temperature and pH. At pH <12, the P-O bond cleavage path produces ethyl methylphosphonate (EMPA) and the toxic S-[2-diisopropyl-aminoethyl] methylphosphonothiolate ion (EA 2192). At room temperature EA 2192 reacts very slowly with OH^- ($t_{1/2}$ = 7.4 days in 1.0 M NaOH), eventually producing less toxic products.[13][14] Using an equimolar ratio of VX and water at elevated temperatures appears to reduce the persistency of EA 2192.[15]
Photo	VX does not absorb UV radiation above 290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.[16]
Thermal	Decomposition half-life 34.5 hr @ 150 °C, 1.6 hr @ 200 °C, 36 sec @ 295 °C[8]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	[17] 
	Flammability	
	Reactivity	
	Special Properties	
DOT identification number	UN 2810[9][16]	
DOT safety placard(s)	[9] 	

CLINICAL PRESENTATION

Time to effect	• Inhalation: Inhaled VX produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 min.[18] • Skin: Liquid VX may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hr; larger

	exposures may cause death within minutes to hours.[18] • Ingestion: Not found
Exposure signs/symptoms	Sweating, muscular twitching, pinpoint pupils, rhinorrhea (runny nose), tightness of the chest with shortness of breath, dimness of vision, cramps, nausea, vomiting, convulsions, coma, and respiratory arrest[18]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[18]
Morbidity and mortality	• LCt_{50}: 15 mg-min/m^3 (MV 15, t=2 min)[19]; 39-70 mg-min/m^3[20]; 30 mg-min/m^3[9] • ECt_{50} (severe): 10 mg-min/m^3[19]; 25 mg-min/m^3[9] • ECt_{50} (mild): 0.1 mg-min/m^3[19]; 0.09 mg-min/m^3[9] • LD_{50} (percutaneous): 3.93 mg/70 kg man[21]; 5 mg/70 kg man[19]; 10 mg/70 kg man[9] • ED_{50} (severe percutaneous injury): 1.57 mg/70 kg man[19]; 2 mg/70 kg man[19] • ED_{50} (Severe ingestion injury): Ingestion of nerve agents is expected to be relatively rare compared to inhalation exposure or skin contact; however, they are readily absorbed from the GI tract and are highly toxic.[22]

COUNTERMEASURES

Prophylaxis	Pyridostigmine bromide, 30 mg every 8 hr, is a pretreatment drug for soman exposure and should also be effective for VX.[3]
Antidote/Treatment	**Antidote:** Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity. There is generally no benefit in giving more than three injections of 2-PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl.[18] **Treatment:** Administer antidotes and provide supportive care.[18]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[23]

	• Laboratory: GC/MS[23] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions[23]
Environmental persistence	When released on soil, the concentration of VX will decline to low levels in a few days, after which the degradation is much slower.[24][25] The half-life on vegetative surfaces is 1-2 days.[26] In seawater, the half-life of VX is approximately 230 days at 5 °C.[27]
Formulations	Produced either as VX itself, or as two ingredients that when mixed together produce VX. One component in the binary is O-ethyl O-2-diisopropylaminoethyl methylphosphonite and the other is a source of sulfur. Sulfur sources used include materials identified by the codes NE, which is sulfur to which an anticaking material has been added, and NM, a mixture of dimethylpolysulfides and sulfur.[2]

The information cutoff date for this factsheet is 14 May 2010.

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RIOT CONTROL AGENTS

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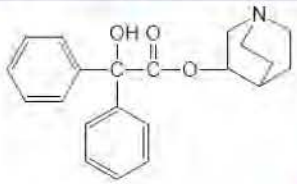
3-Quinuclidinyl Benzilate (BZ)

AGENT OVERVIEW

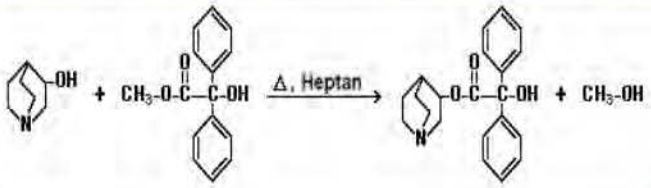
Following WWII, the U.S. military investigated a wide range of possible nonlethal, psychobehavioral, and chemical incapacitating agents. One of these, 3-quinuclidinyl benzilate, an anticholinergic compound assigned the NATO code BZ, was synthesized and studied by Abood and Biel at Edgewood Arsenal between 1959 and 1975. Claims have been made that an estimated 2800 soldiers were exposed to BZ during these experiments, and that a number of these subjects suffered from long-term effects afterwards. According to the U.S. Army, BZ was produced at the Pine Bluff Arsenal between 1962 and 1965, but production ceased due to its varied and unpredictable effects on enemy troops. This agent never saw chemical warfare agent (CWA) operational use against enemy forces. In 1988, destruction of U.S. stockpiles began and is now complete. Incapacitating agents differ from other CWAs in that the lethal dose is theoretically many times greater than the incapacitating dose. BZ acts by competitively inhibiting muscarinic receptors in the parasympathetic nervous system, which innervates numerous organ systems, including the eye, heart, respiratory system, skin, gastrointestinal tract, and bladder. Sweat glands, innervated by the sympathetic nervous system, also are modulated by muscarinic receptors. BZ effects via any route of exposure are slow in onset and long in duration. The U.S. Army Toxic and Hazardous Materials Agency demilitarized the U.S. inventory of munitions containing the incapacitating agent BZ in 1982. As employed in the munitions, BZ is blended with an energetic pyrotechnic mixture comprising of 50% BZ, 23% KC103, 9% S, and 18% $NaHCO_3$.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	3-Quinuclidinyl benzilate
CAS number	6581-06-2[3]
IUPAC name	1-Azabicyclo[2.2.2]octan-8-yl 2-hydroxy-2,2-di(phenyl)acetate[4]
Synonyms	Benzilic acid, 3-quinuclidinyl ester; 1-azabicyclo (2.2.2) octan-3-ol, benzilate; benzeneacetic acid, alphahydroxyalpha-phenyl-,1-azabi-cyclo (2.2.2)oct-3-yl ester; 3-chinuclidylbenzilate; 3-(2,2-diphenyl-2-hydroxyethanoyloxy)-quinuclidine; 3-quinuclidinol benzilate; 3-quinuclidyl benzilate; EA 2277; CS

	4030; oksilidin; QNB[3]
Empirical formula	$C_{21}H_{23}NO_3$[3]
Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: In small amounts as a pharmaceutical • Theft and divergence: Possible, but only in small amounts • Synthesis: Synthetic pathways well known
Synthetic pathways	• Precursors: benzilic acid; methyl benzilate; 3-quinuclidinol[5] • Precursor availability: Common organic chemicals
Synthesis	 [1]

PHYSICAL PROPERTIES

Physical form	White crystalline solid with no odor[2]
Molecular weight	337.42 Daltons[3]
Liquid/solid density	Bulk: 0.51 g/cm^3; crystal: 1.33 g/cm^3[3]
FP/MP	167.5 °C (MP)[5]
Boiling point	412 °C (extrapolated)[5]
Viscosity	Contact CSAC
Surface tension	Contact CSAC
Vapor pressure	1.43 x 10^{-10} mm Hg @ 25 °C (extrapolated)[5]
Volatility	2.60 x 10^{-6} mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[5]
Vapor density (Air=1)	11.6 (calculated from the Ideal Gas Law)[3]
Solubility	Solubility in water is approximately 1.18 g/L[5]; slightly soluble in water; soluble in dilute acids and common organic solvents such as alcohol and chloroform; insoluble in aqueous alkali[2]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	$t_{1/2}$ = 6.7 hr @ 25 °C and pH 9.8; $t_{1/2}$ = 1.8 min @ 25 °C and pH 13; $t_{1/2}$ = 3 to 4 wk @ 25 °C in moist air and pH 7; $t_{1/2}$ = 12 min @ 34°C and pH 12; $t_{1/2}$ = 1.4 hr @ 50 °C and pH 8.5; $t_{1/2}$ = 9.5 hr @ 100 °C and pH 0[6]
Hydrolysis products	3-Quinuclidinol and benzylic acid[7]
Photo	Not reported
Thermal	Stable up to the melting point. During prolonged heating at temperatures approximately 170 °C, BZ begins to decompose, producing carbon dioxide, benzophenone, benzhydrol, and other products. The rate of decomposition is both temperature- and purity-dependent.[8]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[9] 
DOT identification number	UN 2810[10]	
DOT safety placard(s)	[10] 	[10] 
		[10] 

CLINICAL PRESENTATION

Time to effect	The onset of action is approximately 1 hr, with peak effects occurring 8 hr post-exposure. Symptoms gradually subside over 2-4 days.[11]
Exposure signs/symptoms	Depending on the dose and time post-exposure, a number of CNS effects may manifest which include: restlessness, apprehension, abnormal speech, confusion, agitation and tremor, and picking movements. Hallucinations are prominent, and they may be benign, entertaining, or terrifying to the patient experiencing them. Exposed patients may have conversations with hallucinated figures, or they may misidentify persons they typically know well. Simple tasks typically performed well by the exposed person may become difficult. Motor coordination, perception, cognition, and new memory

	formation are altered as CNS muscarinic receptors are inhibited. Severe effects include ataxia, stupor, and coma. Other possible symptoms include impaired vision, nausea, difficulty urinating, and complaints of bodily overheating. Mydriasis resulting in photophobia is expected; tachycardia, vomiting, dry mucus membranes of the mouth and throat, slurred speech, inhibition of sweating, enlarged bladder, and hyperthermia may occur.[7]
Personal decontamination	Remove the patient/victim from the contaminated area. Remove all clothing, at least down to their undergarments, and place the clothing in a labeled durable 6-mil polyethylene bag. Prior to decontamination, cover all open wounds. Wash the patient/victim's contaminated skin thoroughly with a soap and water solution, rinse in warm water. Be careful not to break the patient/victim's skin during the decontamination process. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where additional emergency medical treatment can be provided.[10]
Morbidity and mortality	• The LD_{50} is estimated to be similar to that of atropine (~100 mg). Other factors (i.e., patient's preexisting health status) and the time from exposure to medical care, are also important.[7] • ECt_{50}: 100 mg-min/m^3[3]

COUNTERMEASURES

Prophylaxis	None identified.
Antidote/Treatment	Some military references suggest the use of physostigmine to temporarily increase synaptic acetylcholine concentrations. Physostigmine poses its own risks of side effects and interactions with other drugs and should only be used by persons familiar with its safe use. Provide appropriate supportive care.[10]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Mobile Mass Spectrometer (MM1)[3] • Laboratory: GC/MS[12] • Observation: None
Decontamination—property & equipment	Rapid hydrolysis in 0.5 N sodium hydroxide suggests alcoholic caustic as a decontamination system; also inactivated by weak acidic solutions of hypochlorite[6]
Environmental persistence	BZ in the ground might be expected, eventually, to migrate with groundwater. At high pH, the solubility would be low and the BZ would tend to be adsorbed on clays or organic matter. At low pH, BZ would achieve a high solubility relative to its toxicity, and have an extremely long half-life.[5]
Formulations	BZ is not currently manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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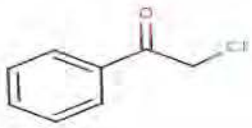
2-Chloroacetophenone (CN)

AGENT OVERVIEW

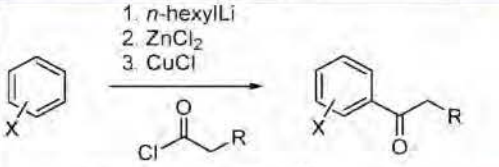
2-chloroacetophenone, also known by its codename CN, is a lacrimator developed at the end of World War I and soon afterwards was widely used by police forces. CN was used as the tear gas of choice for the 3 decades after its introduction, but its used markedly declined after the development of CS.[1] It is intensely irritating to the eyes and the mucous membranes in the nose and upper respiratory tract. For police use, it may be disseminated as a pyrotechnically generated aerosol, as a dust cloud, or in solution as a liquid spray. In spray weapons, carrier/propellant solvents include trichlorofluoroethane, 1,1,1-trichloroethane, and kerosene-type hydrocarbons.[2]

CHEMICAL IDENTIFICATION

Common chemical name	2-Chloroacetophenone[3]
CAS number	532-27-4[4]
IUPAC name	2-chloro-1-phenyl-ethanone[3]
Synonyms	Alpha-chloroacetophenone; alpha-phenacyl chloride; omega-chloroacetophenone; 1-chloroacetophenone; 2-chloro-1-phenylethanone; acetophenone, 2-chloro-; AI3-52322; BRN 0507950; CAF; CAP; CCRIS 2370; chemical Mace; chloroacetophenone; chloromethyl phenyl ketone; CN; EINECS 208-531-1; ethanone, 2-chloro-1-phenyl-; halomethyl phenyl ketone deriv. 21; HSDB 972; Mace; NCI-C55107; NSC 41666; NSC41666;; phenacyl chloride; phenacylchloride; phenyl chloromethyl ketone; phenyl-chloromethylketone; tear gas; UN1697; ZINC01673519[3]
Empirical formula	C_8H_7ClO[3]

Chemical structure	 [5]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: CN is available in bulk[6] and in original formulations of chemical Mace.[7][8] • Theft and divergence: CN is not a controlled material and is stored in bulk and transported without special security precautions. • Synthesis: Not difficult[9]
Synthetic pathways	• Precursors: N-hexyl lithium, zinc chloride, cuprous chloride[9] • Precursor availability: Common organic chemicals[9]
Synthesis	 R = H, Cl, OBn, 4-ClPhO [9]

PHYSICAL PROPERTIES

Physical form	Colorless, white, or gray crystalline solid[4] with sharp, irritating apple blossom or floral odor.[8][10][11]
Molecular weight	154.59 Daltons[12]
Liquid/solid density	1.3 g/cm^3[13]
FP/MP	58-59 °C[14] (MP)
Boiling point	247 °C @ 760 mm Hg[14]
Viscosity	Contact CSAC
Surface tension	Contact CSAC
Vapor pressure	0.005 mm Hg @ 20 °C[4]; 0.0054 mm Hg @ 20 °C[14]
Volatility	34.3 mg/m^3 @ 20 °C[15]
Vapor density (Air=1)	5.3[15]
Solubility	Relatively insoluble in water: 0.68 g/L @ 20 °C; 1.64 g/L @ 25 °C (estimated)[5][10]; soluble in alcohol, benzene, ether, and chloroform.[15]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Very slow even when alkali is added[16]
Hydrolysis products	Hydrogen chloride[15]
Photo	May be broken down by UV light[8]
Thermal	Carbon monoxide and fumes of chlorine[15]; hydrogen chloride[8]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	[15] 
	Flammability	
	Reactivity	
	Special Properties	
DOT identification number	UN 1697[15]	
DOT safety placard(s)	[15] 	[15] 

CLINICAL PRESENTATION

Time to effect	Immediate[15]
Exposure signs/symptoms	Inhalation: Exposure to CN generally causes instantaneous irritation of the eyes, respiratory tract, and skin. Initial irritation typically resolves within 15 to 30 min following decontamination.[15] Respiratory adverse health effects, such as cough, dyspnea, and accumulation of fluid in the lungs (pulmonary edema), may occur immediately after exposure or can be delayed up to 12 to 24 hr.[15] Skin: Exposure to CN generally causes instantaneous irritation of the skin. Itching, redness, and blisters can develop.[15] Adverse effects to the skin occurring within 24 hrs of exposure can be severe and may include redness and blistering.[15] Eye: Exposure to CN generally causes instantaneous irritation of the eyes with copious tear production, conjunctivitis, and blepharospasm. Initial irritation typically resolves within 15 to 30 min following decontamination. Eye irritation may persist if the eyes are rubbed. Eye redness and accumulation of fluid in the tissues surrounding the eye (periorbital edema) may take 1 to 2 days to resolve.[8][10][15]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their

	undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.[15]
Morbidity and mortality	• LCt_{50}: 7,000 mg-min/m^3 (estimate, pure aerosol); 14,000 mg-min/m^3 (estimate, commercial CN grenade)[2] • ECt_{50} (severe): 25-50 mg-min/m^3[10]; est. 50 mg-min/m^3[8]; 80 mg-min/m^3[11]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	No antidote; in the rare event of pulmonary effects following massive exposure, evacuation for hospital care is required. Severe respiratory effects, including accumulation of fluid in the lungs (pulmonary edema), may not manifest until 12 to 24 hr after exposure. Monitoring for respiratory complications has been recommended for those with marked symptoms or who have pre-existing conditions such as hyper-reactive airways (asthma). Flush eyes with copious amounts of water. For relief, eyes can be washed with a weak boric acid solution. Patient/victims with severe eye exposures should be observed closely for development of eye effects (corneal opacity and iritis). For skin exposure, wash with warm sodium bicarbonate solution (if not available, use soap and water). Skin may be treated with calamine lotion or a topical corticosteroid depending on severity. Cases with blisters should be managed as a second degree burn. Secondary infections are treated with appropriate antibiotics.[8][15][17][18]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: GC/MS[19] • Observation: Immediate incapacitation of exposed individual[4]
Decontamination—property & equipment	A solution of detergent and water (which should have a pH value of 8-10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.[15]
Environmental persistence	Short persistency in soil and on material; aquatic bioconcentration and absorption on sediment are minimal[10]
Formulations	CNC: CN in chloroform[20] CNB: CN in benzene and carbon tetrachloride[1] CNS: CN with chloropicrin (PS) in chloroform[1]

The information cutoff date for this factsheet is 14 May 2010.

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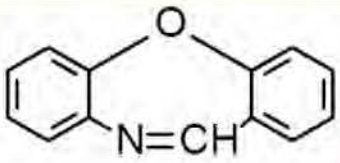
Dibenz(b,f)-1:4-oxazepine (CR)

AGENT OVERVIEW

First synthesized in 1962, dibenz(b,f)-1:4-oxazepine (CR) is more potent but less toxic than O-chlorobenzylidene malononitrile (CS). It is dispersed in a liquid solution, does not degrade in water, and therefore persists in the environment. Its effects are similar to those of CS but it is about five times more potent. Limited data are available on CR but it appears to be much safer than CS because it seems to have little effect on the lower airways or lungs. There does not appear to be persistent skin or eye effects.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Dibenz(b,f)-1:4-oxazepine[1]
CAS number	257-07-8[1]
IUPAC name	Benzo[b][1,5]benzoxazepine[3]
Synonyms	Dibenzoxazepine; CR; dibenz[b,f][1,4]oxazepine; oprea1_455217; dibenz[b,f][1,4]oxazepine; dibenz(b,f)(1,4)oxazepine; EA 3547; NSC 293779; BRN 0743986; NSC293779; ZINC00500952; MS-2513; LS-61552[4]
Empirical formula	$C_{13}H_9NO$[1]

Chemical structure	 [1]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Can be purchased in bulk[5] or prepackaged as a personal defense aide.[6] • Theft and divergence: Unregulated chemical and widely available[5] • Synthesis: Synthesis pathways well known.[7]
Synthetic pathways	• Precursors: o-Formylaminodiphenyl ether; polyphosphoric acid[7] • Precursor availability: o-Formylaminodiphenyl ether not available commercially
Synthesis	Bischler-Napieralski type ring closure of o-acylaminodiphenyl ether with polyphosphoric acid[7]

PHYSICAL PROPERTIES

Physical form	Pale yellow solid with pepper-like odor[1]
Molecular weight	195.22 Daltons[1]
Liquid/solid density	1.56 g/cm^3 @ 25 °C[8]
FP/MP	73 °C[1]
Boiling point	335 °C[8]
Viscosity	Contact CSAC
Surface tension	Contact CSAC
Vapor pressure	0.00059 mm Hg @ 20 °C[8]
Volatility	0.63 mg/m^3 @ 25 °C[8]
Vapor density (Air=1)	6.7[1]
Solubility	Slightly soluble in water[9]; soluble in polypropylene glycol, benzene, chloroform, and carbon tetrachloride.[8]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Not hydrolyzed in aqueous solutions[1]; stable in organic solutions[10]
Hydrolysis products	Unknown
Photo	Unknown

Thermal	Thermally stable

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	 [11]	
	Flammability		
	Reactivity		
	Special Properties		
DOT identification number	Not found		
DOT safety placard(s)	Not found		

CLINICAL PRESENTATION

Time to effect	Instantaneous[1]
Exposure signs/symptoms	• Inhalation: If a splash enters the nose, it causes irritation and rhinorrhea. A solution splashed in the mouth causes burning of the tongue and palate and salivation for 5 to 10 minutes.[12] Initially, some, coughing, and shortness of breath, but causes almost no effects in the lower airways and lungs.[12][13] • Skin: Skin exposure causes burning within a few minutes, which persists for 15 to 30 minutes, and an erythema lasting for 1 to 2 hours.[12][13] Erythema is well demarcated, and moderate to severe in intensity.[12][13] • Eye: Intense burning and stinging sensation, and difficulty opening the eyelids. Copious tears, blepharospasm, reddening of eyes, and conjunctival injection may occur.[12][13]
Personal decontamination	Move to fresh air. Flush eyes with copious amounts of cold water. Do not rub your eyes. Do not use any form of bleach. Soap and water can be used on skin.[1]
Morbidity and mortality	• LD_{50} (skin): >100,000 mg-min/m^3[13][14] • ECt_{50} (severe): 0.15 mg-min/m^3 (1 min exposure)[1]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Flush eyes with copious amounts of cold water. Soap and water can be used on skin.[1]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: GC/MS[15] • Observation: Immediate incapacitation of exposed individual[8]
Decontamination—property & equipment	To decontaminate equipment or surfaces, remove by using towels, rags, absorbent paper, or any other method such as scraping, shoveling, or sweeping.[1]
Environmental persistence	CR does not degrade in water, and it is quite persistent in the environment. Under suitable conditions, CR can persist on certain surfaces (especially porous) for up to 60 days.[1]
Formulations	Currently used only in solution for dissemination in liquid dispensers. The solutions in the dispensers contains 0.1% dibenzoxazepine in 80 parts propylene glycol and 20 parts water.[16]

The information cutoff date for this factsheet is 14 May 2010.

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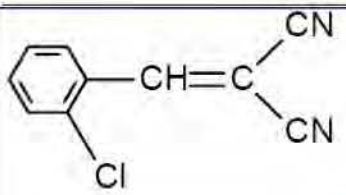
Ortho-Chlorobenzylidene Malononitrile (CS)

AGENT OVERVIEW

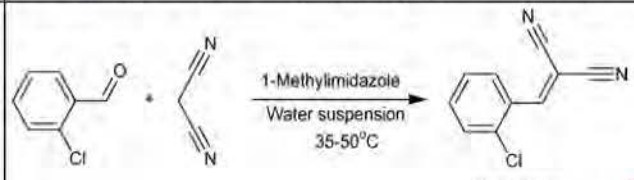
Ortho-chlorobenzylidene malononitrile (CS) is a riot control agent. It was synthesized in 1928 and has been used as the chief riot control agent since 1959. Riot control agents, also called irritants, lacrimators, and "tear gas," produce transient discomfort and eye closure to render the recipient temporarily incapable of fighting or resisting. Law enforcement agencies use them for riot control, and military forces use them for training and in combat. They have a high LCt_{50} and a low effective ECt_{50}, and therefore have a high safety ratio. Their primary objective is to cause pain, burning, or discomfort on exposed mucous membranes and skin; these effects occur within seconds of exposure but seldom persist more than a few minutes following exposure.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	O-Chlorobenzylidene malononitrile
CAS number	2698-41-1[3]
IUPAC name	2-Chlorobenzalmalononitrile[4]
Synonyms	((2-chlorophenyl)methylene)propanedinitrile; (O-chlorobenzal)malononitrile; (O-chlorobenzylidene)malononitrile; 2-chloro-BMN; 2-chlorobenzalmalonitrile; 2-chlorobenzalmalononitrile; 2-chlorobenzylidene malononitrile; 2-chlorobenzylidenemaloni-nitrile; 2-chlorobenzylidenemalononitrile; 2-chlorobmn; beta,beta -dicyano-o-chlorostyrene; CS gas; malononitrile, (O-chlorobenzylidene)-; NCI-C55118; O-chlorobenzylidenemalonic nitrile; OCBM; USAF KF-11[5]
Empirical formula	$C_{10}H_5ClN_2$[3]

Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Can be purchased in bulk[6] or prepackaged as a personal defense aid[7] • Theft and divergence: Unregulated chemical and widely available[6][7][8] • Synthesis: Well known and uncomplicated[9]
Synthetic pathways	• Precursors: 2-chlorobenzaldehyde; malononitrile[9] • Precursor availability: Widely available from chemical suppliers[9]
Synthesis	 Yield 99 % [9]

PHYSICAL PROPERTIES

Physical form	White crystalline solid[10]; pepper-like odor[3]
Molecular weight	188.6 Daltons[3]
Liquid/solid density	Bulk: 0.24-0.26 g/cm^3; crystal: 1.04 g/cm^3[11]
FP/MP	95-96 °C (MP)[3]
Boiling point	310-315 °C[3]
Viscosity	Contact CSAC
Surface tension	Contact CSAC
Vapor pressure	0.045 Pa @ 20 °C[11]
Volatility	0.71 mg/m^3 @ 25 °C[11]
Vapor density (Air=1)	6.5 (calculated)[3]
Solubility	Insoluble in water[11][3]; moderately soluble in alcohol; and freely soluble in acetone, chloroform, methylene dichloride, ethylacetate, and benzene[3]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Not found
Hydrolysis products	o-Chlorobenzaldehyde (metabolite); o-chlorobenzylmalono-nitrile (metabolite)[12]
Photo	CS is degraded by light. Seven days after mixing CS into soil samples, samples kept in the dark retained 78% of CS vs. 36% for samples exposed to light.[13]
Thermal	Can be destroyed by incineration producing hydrogen cyanide, hydrogen chloride, nitrogen oxides, phosgene, nitrogen monoxide, acetylene, and chlorobenzene[14]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [4]	
DOT identification number	UN 1693[15]		
DOT safety placard(s)	 [15]		

CLINICAL PRESENTATION

Time to effect	Immediate[3]
Exposure signs/symptoms	Inhalation: Extreme discomfort in the respiratory tract, with wheezing, coughing, and shortness of breath[16] Skin: Burning sensation, redness, and vesication resembling second degree burns[17] Eye: Intense eye pain; excessive tearing, conjunctivitis, and involuntary closing of the eyes[16][17]
Personal decontamination	Copious amounts of water[17]
Morbidity and mortality	• LCt_{50}: 52,000-61,000 mg-min/m^3 (15-90 min exposure)[16] • ECt_{50} (intolerable pain): 7 mg-min/m^3 (1 min exposure)[16]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Inhalation: Blow nose to remove any CS particles. Skin: Wash with soap and water. Eye: Rinse eyes with copious amount of pure water.[17]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: GC/MS[18] • Observation: White smoke with pepper-like odor[17]
Decontamination—property & equipment	CS causes a severe decontamination area problem. Particles disseminated by any of the standard dispersion means will adhere to the person, clothing, furnishings, or fixtures for a long time. Humid conditions will cause the odor and irritant effect to linger indefinitely. Decontamination is achieved by using an alkaline solution. A typical decontamination solution is water and 5% sodium bisulfite.[19]
Environmental persistence	CS has been found to persist in snow for as long as 30 days but its persistency in soil varied, depending on the condition of the soil.[13]
Formulations	• CS is available both unground and ground with 5% silica aerogel or treated Cab-O-Sil.[20] • CS1 has been especially formulated to prolong persistency and increase effectiveness. Unlike CS, CS1 is a free-flowing agent powder consisting of 95% crystalline CS blended with 5% silica aerogel. This formulation reduces agglomeration, increases fluidity, and achieves the desired respiratory effects when dispersed as a solid aerosol. When disturbed, CS1 reaerosolizes and can cause respiratory and eye effects.[3] • CS2 is a siliconized, microencapsulated form of CS1. This treatment improves the physical characteristics of CS by reducing agglomeration and hydrolysis. This form of CS prolongs the effectiveness for both immediate and surface contamination effects.[3] • CSX is a form of CS developed for dissemination as a liquid rather than a powder. One gram of powdered CS is dissolved in 99 grams of trioctyl phosphite. As with CS, CSX stings and irritates the eyes, skin, nose, throat, and lungs of exposed personnel.[3]

The information cutoff date for this factsheet is 14 May 2010.

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VESICANTS

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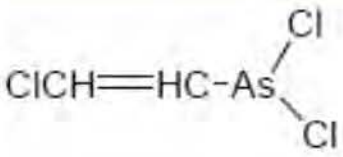
Lewisite (L)

AGENT OVERVIEW

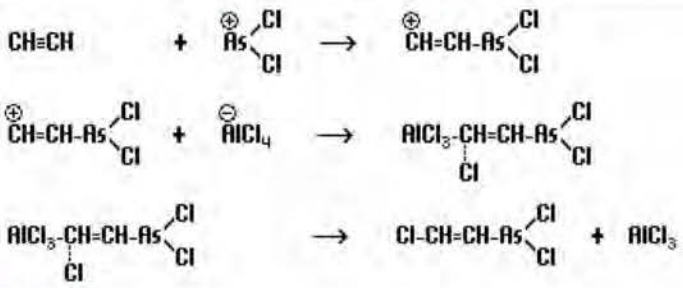
Lewisite is a strong arsenical vesicating agent with the military designation L. Lewisite is named after the American military scientist W. Lee Lewis, who produced this arsenical as a prototype chemical warfare agent (CWA) in 1918. Because it was developed so late in World War I, Lewisite was never used in that conflict. The only military use may have occurred in China during the Sino-Japanese conflict (ca. 1937-1942). Unlike mustards, which have delayed onset of clinical symptoms, the extreme irritation to eyes and skin begins almost immediately, with redness and blisters forming hours later. Significant exposure to Lewisite can cause blindness.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Lewisite
CAS number	541-25-3[2]
IUPAC name	Dichloro-[(E)-2-chloroethenyl]arsane[3]
Synonyms	L; dichloro(2-chlorovinyl)arsine; EA 1034; Lyvizit; LI; M-1; arsonous dichloride, (2-chloroethenyl)-; chlorovinylarsine dichloride; 2-chlorovinyldichloroarsine; β-chlorovinyldichloroarsine; (2-chloroethenyl) arsonous dichloride; arsine, dichloro (2-chlorovinyl)-[2]
Empirical formula	$C_2H_2AsCl_3$[2]

Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially • Theft and divergence: Possible in third world nations • Synthesis: Prepared from arsenic trichloride and acetylene with aluminum chloride as a catalyst[4][5][6]
Synthetic pathways	• Precursors: acetylene; arsenic trichloride; aluminum trichloride[4] • Precursor availability: Arsenic trichloride is limited by the CWC (Schedule 2)[7]. Acetylene and aluminum trichloride are available commercially from Sigma-Aldrich.[8]
Synthesis	 [5][6]

PHYSICAL PROPERTIES

Physical form	Brown liquid; colorless when pure[9], impurities lead to colors ranging from violet to brown.[10] Darkens to violet-black or green with time.[11] Odor is geranium-like; odorless when pure[2]
Molecular weight	207.32 Daltons[2]
Liquid/solid density	1.888 g/mL @ 20 °C[12]; 1.8793 g/mL @ 25 °C (extrap.)[2]
FP/MP	-44.7 to -1.8 °C (FP) (depending on purity and isomers present)[13], MP 0.1 °C, solidifies @ -13 °C[12]; -44.7 °C (cis isomer); -1.2 °C (trans isomer)[11]
Boiling point	190 °C (extrapolated)[2][14]; decomposes prior to boiling[15]; 190 °C @ 760 torr (decomposes)[12]; at 760 mm Hg: 169.8 °C (cis); 196.6 °C (trans)[11]
Viscosity	liquid: 4.261 cP @ 20 °C; 3.858 cP @ 25 °C; vapor: 8.36 x 10^{-3} cP @ 20.0 °C; 8.53 x 10^{-3} cP @ 25.0 °C[16]; 20.05 cP (trans); 1.69 cP (cis) @ 25 °C[11]
Surface tension	41.1 dynes/cm[2]
Vapor pressure	0.22 mm Hg @ 20 °C; 0.35 mm Hg @ 25 °C[17]

Volatility	4480 mg/m^3[18]
Vapor density (Air=1)	7.1 (calculated from the Ideal Gas Law)[2]
Solubility	In water, 500 mg/L[11]; soluble in ordinary organic solvents; insoluble in dilute mineral acids[12]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Lewisite hydrolyzes immediately when associated with water to form Lewisite oxide (solid), which then dissolves very slowly in water.[19] Readily soluble in common organic solvents, oils, and CWAs.[20]
Hydrolysis products	2-Chlorovinylarsonous acid (CVAA), 2-chlorovinylarsenious oxide (Lewisite oxide), and hydrochloric acid[21]
Photo	Vapor-phase Lewisite will be degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 1.2-1.3 days. Reaction with ozone will contribute to Lewisite's atmospheric degradation, but it will not be as rapid as reaction with hydroxyl radicals. At 10 ppm in cyclohexane solution, Lewisite does not absorb UV light above 290 nm; this suggests that direct photolysis in sunlight will not occur.[22]
Thermal	At 149 °C, 0.5% is destroyed; at 493 °C, >99.99% is destroyed.[15]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[23] 
DOT identification number	UN 1556[23]	
DOT safety placard(s)	[23] 	

CLINICAL PRESENTATION

Time to effect	• Eye: Lewisite produces pain or irritation within seconds to minutes of exposure. Redness occurs within 15 to 30 min following exposure to liquid Lewisite.[24] • Inhalation: Lewisite produces immediate burning pain; this may cause exposed patients/victims to seek protection and limit their exposure.[24]

	• Skin: Lewisite produces immediate stinging pain; redness (erythema) within 15 to 30 min, with pain and itching for 24 hrs; and blistering (vesication) within 12 hrs, with pain for 2 to 3 days. Blistering begins within hours following exposure, but the full extent of blistering does not occur for 12 to 18 hrs. The blister begins small in the center of a red area, and then expands to include the entire area of inflammation. Exposure to liquid Lewisite causes skin lesions to occur sooner than does exposure to vapor. Lewisite is absorbed by the skin within 3 to 5 min following exposure, and may result in shock.[24] • Ingestion: No information is available on the time course of effects following ingestion of Lewisite.
Exposure signs/symptoms	• Eye: Immediate stinging, burning pain, strong irritation, tear production, spasmodic blinking, eyelid edema, vesication and scarring of the cornea, perforation of the eye, and blindness[24] • Inhalation: Irritation of the nose and lower airways, immediate burning pain, violent sneezing, nosebleed, and sinus pain. Also possible are cough, difficulty breathing, pneumonitis, pulmonary edema, respiratory failure, and death.[24] Lewisite also acts systemically to produce diarrhea, restlessness, subnormal temperatures, weakness, and hypotension.[22] • Skin: Immediate stinging and burning pain, itching, irritation, erythema, vesication, and severe burns[24] • Ingestion: Nausea, vomiting, chemical burns of the gastrointestinal tract, and prostration[24]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash skin with a soap and cold or warm water solution. Be careful not to break the patient/victim's skin during the decontamination process. Cover all open wounds. Cover the patient/victim to prevent shock. Move the patient to an area where emergency medical treatment can be provided.[24]
Morbidity and mortality	• LCt_{50}: 1000 mg-min/m^3[2] • ECt_{50} (severe): 500 mg-min/m^3[2] • ECt_{50} (mild): 50 mg-min/m^3[2] • LD_{50} (skin): 1400 mg/70 kg man[2]; as low as 2.8 grams per individual (40 mg/kg)[25]; about 30 drops (2.6 g; 37.1 mg/kg), applied to the skin and not washed off or otherwise decontaminated, would be expected to be fatal to an average man[26]; 37.6 mg/kg[27] • ED_{50} (severe skin injury): 600 mg/70 kg man[2] • LD_{50} (ingestion): Ingestion of Lewisite may cause nausea and vomiting.[28] • ED_{50} (severe ingestion injury): Ingestion of Lewisite may cause nausea and vomiting.[28]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	2,3-dimercaptopropanol (British Anti-Lewisite)[29]; administer the antidote and continue to provide appropriate supportive care.[28]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[30] • Laboratory: GC/MS[31] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions; puddles of liquid must be contained by covering with vermiculite, diatomaceous earth, clay, fine sand, sponges, paper towels, or cloth towels. Remove all material and place in a container. Decontaminate area with copious amounts of household bleach. Removal of porous material, including painted surfaces, that may have absorbed liquid may be required.[25]
Environmental persistence	Short due to fast Lewisite hydrolysis[20]
Formulations	Lewisite is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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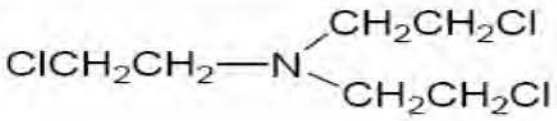
Nitrogen Mustard 3 (HN-3)

AGENT OVERVIEW

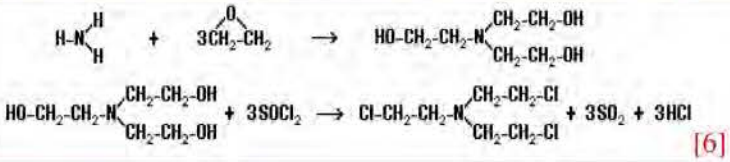
HN-3 is one of three nitrogen mustard blister agents (vesicants). Nitrogen mustards have not previously been used in warfare. The properties of nitrogen mustards are only slightly different from those of sulfur mustards, another major class of blister agents. However, exposure to nitrogen mustards may be more immediately toxic than exposure to sulfur mustard. Nitrogen mustards are alkylating agents that affect DNA and other molecules in the body. Exposure to nitrogen mustard damages the eyes, skin, and respiratory tract and suppresses the immune system. Although the nitrogen mustards cause cellular changes within minutes of contact, the onset of pain and other symptoms is delayed. Exposure to large amounts can be fatal. HN-3 is odorless when pure.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Nitrogen mustard 3
CAS number	555-77-1[2]
IUPAC name	2-Chloro-N,N-bis(2-chloroethyl)ethanamine[3]
Synonyms	2, 2', 2"-Trichlorotriethylamine; tri (2-chloroethyl)amine; tris (2-chloroethyl)amine; tris (β-chloroethyl)amine; EA 1053; HN-3; TO; TL 145; TS 160 [2]
Empirical formula	$C_6H_{12}Cl_3N$[2]

Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially in the United States.[4] Produced in limited quantities as a antineoplastic agent in Europe and as a fixing agent in textile dyes.[5] • Theft and divergence: Possible in third world nations • Synthesis: Synthetic pathways well known[6]
Synthetic pathways	• Precursors: Thionyl chloride; triethanolamine; triethanolamine hydrochloride[7][8] • Precursor availability: Most are limited by the CWC.[9]
Synthesis	 [6]

PHYSICAL PROPERTIES

Physical form	Oily dark liquid; colorless when pure[10], may be pale yellow[1]; no odor when pure[11], but may be butter almond[12], fishy, or soapy[13]
Molecular weight	204.53 Daltons[2]
Liquid/solid density	1.2352 g/mL @ 25.0 °C[14]; 1.2347 g/mL @ 25 °C[1]
FP/MP	-3.74 °C (MP)[2]
Boiling point	257 °C (extrapolated)[2], at atmospheric pressure HN-3 decomposes below the boiling point[15]
Viscosity	0.073 cP @ 25.0 °C[2]
Surface tension	40.9 dynes/cm[2]
Vapor pressure	1.4×10^{-5} atm[1][2]
Volatility	120 mg/m^3 @ 25 °C (calculated from the Ideal Gas Law)[2]
Vapor density (Air=1)	7.1 (calculated from the Ideal Gas Law)[2]
Solubility	80 mg/L @ ambient temperature[11]; 160 mg/L @ 25 °C[4] Miscible with common organic solvents.[11]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Very slow; hydrolysis is not complete even after several days unless alkali is present[16]
Hydrolysis products	Complete hydrolysis gives hydrochloric acid and triethanolamine.[10] The process involves a complex series of reactions, with formation of the hydrochloride, cyclic imonium salts, a dimer, etc.[11]
Photo	The rate constant for the vapor-phase reaction of HN-3 with photochemically produced hydroxyl radicals has been estimated as 1.1×10^{-11} cm^3/molecule-sec at 25 °C, corresponding to an atmospheric half-life of approximately 1.5 days at an atmospheric concentration of 5×10^5 hydroxyl radicals per cm^3.[5]
Thermal	Above 150 °C[15]; remains stable when explosively disseminated[11]; heated to decomposition emits hydrogen chloride and nitrogen oxide.[17]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[1] 
DOT identification number	UN 2810[12]	
DOT safety placard(s)	[1] 	[1] 

CLINICAL PRESENTATION

Time to effect	• Eye: The eyes are the organs that are most sensitive to mustard vapor; eye injury may occur within 1 to 2 hrs after severe exposure, or 3 to 12 hrs after a mild to moderate exposure.[1] • Inhalation: Airway injury may occur within 2 to 6 hrs after severe exposure and within 12 to 24 hrs after mild exposure.[1] • Skin: The symptom-free (latent) period is 6 to 12 hrs in temperate conditions; hot, humid weather strikingly increases the action of nitrogen mustards. Some skin injury may appear as late as 48 hrs after exposure.[1] • Ingestion: No information is available on the time course of effects following ingestion of nitrogen mustard.
Exposure signs/symptoms	• Eye: Eye irritation, dryness, itchy or gritty feeling, lacrimation,

	blepharospasm, miosis, edema in the eyelids, photophobia, ulceration of the cornea, and blindness[1] • Inhalation: Runny nose, sneezing, nosebleed, hoarseness progressing to "toneless" voice, barking cough, loss of taste and smell, wheezing and difficulty breathing or shortness of breath in smokers and asthmatics, and nasal and sinus pain. Also possible are acute inflammation of the upper and lower airways; necrosis of the airway lining; possible obstruction of both upper and lower airways due to formation of a false membrane or fibrous deposit; airway occlusion from inflamed and necrotic cells; and death, due to inflammatory lung disease.[1] • Skin: Skin pain, intense itching or irritation, and formation of rashes and blisters. Redness begins to appear 1 to 24 hrs after exposure (typically within 4 to 8 hours); vesication begins 2 to 18 hrs after onset of redness; systemic health effects including weakness, malaise, emesis, fever, and prostration.[1] • Ingestion: Ingestion is an uncommon route for exposure but can lead to local effects such as nausea, vomiting, pain, diarrhea, and esophageal or gastrointestinal burns.[1]
Personal decontamination	The eyes and skin must be decontaminated within 1 or 2 min after exposure to reduce tissue damage. Flush the eyes immediately with water for about 5 to 10 min by tilting the head to the side, pulling eyelids apart with fingers, and pouring water slowly into eyes. Do not cover eyes with bandages. If exposure to liquid agent is suspected, cut and remove all clothing and wash skin immediately with soap and water. If shower areas are available, showering with water alone will be adequate. However, in those cases where water is in short supply, and showers are not available, an alternative form of decontamination is to use 0.5% sodium hypochlorite solution or absorbent powders such as flour, talcum powder, or Fuller's earth. If exposure to vapor only is certain, remove outer clothing and wash exposed areas with soap and water or 0.5% solution of sodium hypochlorite. Place contaminated clothes and personal belongings in a sealed double bag. In cases of ingestion, do not induce emesis. There is no evidence that administration of activated charcoal is beneficial.[18]
Morbidity and mortality	• LCt_{50}: 1000 mg-min/m^3[2] • ECt_{50} (severe): 500 mg-min/m^3[2] • ECt_{50} (mild): 50 mg-min/m^3[2] • LD_{50} (skin): 1400 mg/70 kg man[2] • ED_{50} (severe skin injury): 600 mg/70 kg man[2]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Antidote: None Treatment: Provide appropriate supportive care[18]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical, M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[19] • Laboratory: GC/MS[20] • Observation: None
Decontamination—property & equipment	Strong bleach, caustic solutions[19]
Environmental persistence	Days to weeks[16]
Formulations	HN-3 is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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Homeland Security
Science and Technology

Chemical Security Analysis Center

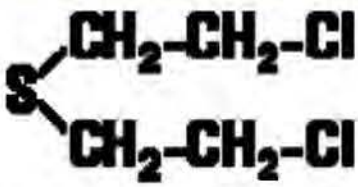
Sulfur Mustard (HD)

AGENT OVERVIEW

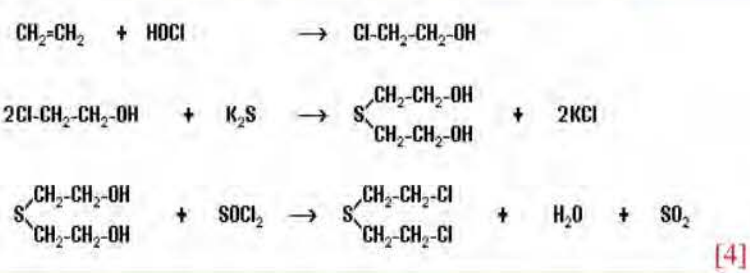
Sulfur Mustard, also known by its NATO designation HD, is an extremely toxic substance whose sole application is as a blistering agent. As a chemical weapon, it is classified as a weapon of mass destruction by the United Nations in UN Resolution 687. The synthesis of mustard gas was reported much earlier than its first use as a chemical weapon. In 1860, Frederick Guthrie observed that when ethylene reacted with chlorine a substance was produced, which in small quantities could produce toxic effects on the skin. The gas was used for the first time as an agent of chemical warfare during WWI, when it was distributed with devastating effect near Ypres in Flanders on July 12, 1917.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Sulfur mustard
CAS number	505-60-2[2]
IUPAC name	1-Chloro-2-(2-chloroethylsulfanyl)ethane[3]
Synonyms	HD; 2, 2'-dichloroethyl sulfide; 1, 1'-thiobis(2-chloroethane); β- β '-dichlorodiethyl sulphide; β ,β´-dichloroethylsulfide di-(2-chloro-ethyl) sulfide; sulfide, bis (2-chloroethyl); bis (beta-chloroethyl)sulfide; Bis (2-chloroethyl)sulfide; bis (2-chloroethyl) sulphide; 1-chloro-2-(beta-chloroethylthio)ethane; 2,2'-dichlorodiethyl sulfide; di-2-chloroethyl sulfide; beta,beta'-dichloroethyl sulfide; beta,beta-dichlor-ethyl-sulphide; 2,2'-dichloroethyl sulphide; gelbkreuz (Czech); 1,1'-thiobis(2-chloroethane)[2]
Empirical formula	$C_4H_8Cl_2S$[2]

Chemical structure	 [4]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available commercially[3] • Theft and divergence: Possible in third world nations • Synthesis: Synthetic pathways well known
Synthetic pathways	• Precursors: 2-chloroethanol; ethylene; hydrochloric acid; sodium sulfide; sulfur dichloride; sulfur monochloride; thiodiglycol[5] • Precursor availability: Most are limited by the CWC
Synthesis	 [4]

PHYSICAL PROPERTIES

Physical form	Pale yellow to dark-brown oily liquid[6], colorless when pure[7]; odor is garlic or horseradish-like[8]
Molecular weight	159.07 Daltons[2]
Liquid/solid density	1.2685 g/mL @ 25 °C (liquid)[2], 1.372 g/mL @ 0 °C (solid)[9]
FP/MP	14.45 °C (FP)[7]
Boiling point	218 °C (extrapolated); at atmospheric pressure HD starts to decompose below the boiling point[10]
Viscosity	3.951 cP @ 25 °C (liquid)[11]; 6.65 x 10^{-3} cP @ 25.0 °C (vapor)[2]
Surface tension	42.5 dynes/cm @ 25 °C[2]
Vapor pressure	9.1 Pa @ 20 °C; 14.1 Pa @ 25.0 °C; 14.7 Pa @ 25 °C[12]
Volatility	598 mg/m^3 @ 20 °C; 906 mg/m^3 @ 25 °C; both calculated from the Ideal Gas Law[10]
Vapor density (Air=1)	5.5 (calculated from the Ideal Gas Law)[2], 5.4[13]
Solubility	Solubility of HD in distilled water: 0.092 g/100 ml @ 22 °C[14]; 0.0684 g/100 ml @ 25 °C[15]. HD is freely soluble in fats and oils, gasoline,

	kerosene, most organic solvents, and CW agents.[2]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	$t_{1/2}$ = 5 min @ 25 °C via an Sn1 mechanism[16]; $t_{1/2}$ = 60 min @ 25 °C in salt water[17]; HD on or under water undergoes hydrolysis only if dissolved; the rate of HD hydrolysis is controlled by the rate of mass transfer and is very slow.[18]
Hydrolysis products	Hydrogen chloride, thiodiglycol, and sulfonium ion aggregates[18]
Photo	Vapor-phase HD will be degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals, with a half-life of about 50 hours. Direct UV photolysis, while possible, is not expected to be an important fate process.[19]
Thermal	Decomposes at 180 °C[20]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[21] 
DOT identification number	UN 2810[22]	
DOT safety placard(s)	[22] 	[22] 	[22] 

CLINICAL PRESENTATION

Time to effect	• Eye: The eyes are the organs that are most sensitive to mustard vapor; eye injury may occur within 1 to 2 hrs after severe exposure, or 3 to 12 hrs after a mild to moderate exposure.[22] • Inhalation: Airway injury may occur within 2 to 6 hrs after severe exposure, and within 12 to 24 hrs after mild exposure.[22] • Skin: The symptom-free (latent) period is 6 to 12 hrs in temperate conditions; hot, humid weather strikingly increases the action of sulfur mustard. Some skin injury may appear as late as 48 hrs after exposure.[22] • Ingestion: Nausea and vomiting is common in the first few hours after exposure, and may abate after 24 hours.[22]
Exposure signs/symptoms	• Eye: Eye irritation, dryness, itchy or gritty feeling, lacrimation,

	blepharospasm, miosis, edema in the eyelids, photophobia, ulceration of the cornea, and blindness[22] • Inhalation: Runny nose, sneezing, nosebleed, hoarseness progressing to "toneless" voice, barking cough, loss of taste and smell, wheezing and difficulty breathing or shortness of breath in smokers and asthmatics, nasal and sinus pain, acute inflammation of the upper and lower airways; necrosis of the airway lining, possible obstruction of both upper and lower airways due to formation of a false membrane or fibrous deposit, airway occlusion from inflamed and necrotic cells, and death due to inflammatory lung disease.[22] • Skin: Skin pain, intense itching or irritation, and formation of rashes and blisters. Erythema begins to appear 1 to 24 hrs after exposure (typically within 4 to 8 hrs); vesication begins 2 to 18 hrs after onset of redness; skin necrosis, systemic health effects including weakness, malaise, emesis, fever, and prostration.[22] • Ingestion: Nausea, vomiting, pain, diarrhea, possible chemical burns of the gastrointestinal tract, and prostration.[22]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds. Cover the patient/victim to prevent shock and loss of body heat. Move the patient/victim to an area where emergency medical treatment can be provided.[22]
Morbidity and mortality	• LCt_{50}: 787 mg-min/m^3[23] • ECt_{50} (severe): 46.4 mg-min/m^3[23] • LD_{50} (skin): 1140 mg/70 kg man[23]; 4480 mg/70 kg man[24]; 7000 mg/70 kg man[25] • ED_{50} (severe skin injury): 371 mg/70 kg man[23] • LD_{50} (ingestion): 0.7 mg/kg[25][26]

COUNTERMEASURES

Prophylaxis	None known
Antidote/Treatment	Provide appropriate supportive care[22]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time M9 Paper Chemical Agent; M256 and M256A1 Detector Kits, Chemical Agent; M18A2 Detector Kit; M272 Water Testing Kit, Chemical; M43A1 Detector Unit, Chemical Agent Automatic Alarm; Chemical Agent Monitor[27] • Laboratory: GC/MS[28] • Observation: None
Decontamination—	Strong bleach, caustic solutions[27]

property & equipment	
Environmental persistence	Contamination of surface soil may persist for weeks, and pockets of deeper soil may remain contaminated for years. Mustard dissolves very slowly in water; however, once dissolved it breaks down into less toxic degradation products within minutes.[29]
Formulations	Sulfur mustard is not manufactured.

The information cutoff date for this factsheet is 14 May 2010.

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2.0 TOXIC INDUSTRIAL CHEMICALS

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BLOOD AGENTS

Homeland Security
Science and Technology

Chemical Security Analysis Center

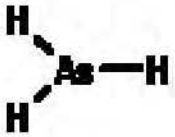
Arsine (SA)

AGENT OVERVIEW

Arsine is an extremely toxic, colorless gas used extensively in the semiconductor industry. It is known chemically as arsenic hydride, and is the arsenical analog to ammonia and a lethal gaseous form of arsenic. Arsine also is used in mining and manufacturing processes involving arsenicals and in paints and herbicides containing arsenicals. Arsine is extremely toxic and a potent hemolytic agent, ultimately causing death via renal failure. In humans arsine is absorbed via the lungs and mucosal surface of the respiratory tract. The first case of arsine poisoning was reported in 1815 after a German chemist died from an exposure to arsine in his laboratory. It is unclear whether arsine is significant as a chemical warfare agent or not. Some view that arsine is too flammable and unstable for battlefield use. However, arsine may be made from arsenides on the spot, thus circumventing the stability problem. An American document from 1926 counts arsine among the agents used by the Central Powers in WW I.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Arsine
CAS number	7784-42-1[3]
IUPAC name	Arsane[4]
Synonyms	SA; Arthur; arsenic trihydride; hydrogen arsenide, arseniuretted hydrogen; arsenic hydride; CG6475000; arsenous hydride; AsH_3; arsenic hydride; arsenowodor; arsenwasserstoff[3]
Empirical formula	AsH_3[3]

Chemical structure	 [5]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Available for purchase. • Theft and divergence: Not found • Synthesis: Synthesis pathways well known.[6][7]
Synthetic pathways	• Precursors: Sodium, zinc and aluminum arsenides; hydrogen chloride; arsenic trioxide[1] • Precursor availability: Available from commercial sources
Synthesis	Arsine may be made through hydrolysis of arsenides (sodium, zinc and aluminum): $2AsAl + 3H_20 \rightarrow 2AsH_3 + Al_2O_3$[6] Reaction of aluminum arsenide with hydrochloric acid; electrochemical reduction of arsenic compounds in acid solutions.[6] AsH_3 is generally prepared by the reaction of As^{3+} sources with H^- equivalents.[7]

PHYSICAL PROPERTIES

Physical form	Colorless gas[3]; disagreeable garlic odor[8]
Molecular weight	77.95 Daltons[3]
Liquid/solid density	1.667 g/mL (-75 °C); 1.734 g/mL (-100 °C)[3]
FP/MP	-116 °C[9]
Boiling point	-62.2 °C (extrapolated)[9]
Viscosity	0.01458 mPa.s (gas, 0 °C, 101.3 kPa)[1]
Surface tension	21.98 dynes/cm (-58.5 °C)[10]
Vapor pressure	400 mm Hg (-75 °C); 86.9 mm Hg (-100 °C)[3]
Volatility	2.55×10^6 mg/m^3 (-75 °C); 6.27×10^5 mg/m^3 (-100 °C) (both calculated from vapor pressure)[3]
Vapor density (Air=1)	2.7 (calculated)[11]
Solubility	0.028 g/100 g water (20 °C); soluble in alkalis, halogen alkanes, hydrocarbons, chloroform, and benzene[12]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Arsine hydrolysis occurs rapidly in the presence of light; slowly in the absence of light and air at 15.5 °C and pH ~7; 32% of SA is hydrolyzed within 5 hrs and about 66% within 24 hrs.[3]
Hydrolysis products	Arsenic, arsenic acids, and hydrides[7][1]
Photo	Moist arsine hydrolyzes rapidly in the presence of light.[7]
Thermal	Decomposes when heated at 300 °C, depositing arsenic, which volatilizes at 400 °C[7]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[13] 
DOT identification number	UN 2188[1]	
DOT safety placard(s)	[13] 	

CLINICAL PRESENTATION

Time to effect	Effects usually appear within 30 to 60 minutes, but may be delayed for several hours.[13]
Exposure signs/symptoms	• Inhalation: Acute arsine exposure may be fatal. Headache and a garlicky odor of the breath may be the first signs and symptoms, followed by malaise, muscle cramping, abdominal and chest pains, chills, nausea, and vomiting. Other symptoms may include decreased urine output, blood in the urine, urine colored red or green, bronze skin color, reductions in hematocrit and hemoglobin, and low blood pressure. Cardiac ischemia can occur within hours to days of exposure.[13][14] • Skin: There is little information about direct toxic effects of arsine on the skin or eyes, or about absorption through the skin. Exposure to liquid arsine (the compressed gas) can result in frostbite.[15] Abnormal pigmentation may be observed. A peculiar bronze tint been described as characteristic of arsine poisoning.[13]
Personal decontamination	Move to fresh air; no decon required under field conditions[3][15]
Morbidity and mortality	• LCt_{50}: 7500 mg-min/m^3[3]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	**Antidote:** None. British Anti-Lewisite is not effective[15][16] **Treatment:** • Inhalation: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, and pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchial spasm with beta2 agonist and corticosteroid aerosols.[13] • Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. A physician should examine the area if irritation or pain persists.[13] • Eyes: Irrigate exposed eyes with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobias persist, the patient should be seen in a health care facility.[13]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Mobile Mass Spectrometer[3] • Laboratory: Arsine in air is determined by x-ray fluorescence spectrometric analysis of the arsine gas, trapped on silver nitrate-impregnated filter paper[17]; a combination of gas chromatography with multiple ion detection mass spectrometry and hydride generation-heptane cold trap techniques can detect arsine in human urine[18]; samples collected on charcoal can be recovered by acid desorption and analyzed by atomic absorption spectrophotometry.[19] • Observation: Detection by observation is not immediate. The first signs noted may be a garlicky odor of the breath and a bronze skin pigmentation.[13]
Decontamination—property & equipment	Vapors should be allowed to dissipate.
Environmental persistence	Arsine is rapidly hydrolyzed in water to arsenic acids and hydrides.[1]
Formulations	Grades: technical; 99% pure or in mixture with other gases.[6]

The information cutoff date for this factsheet is 14 May 2010.

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Cyanogen

AGENT OVERVIEW

Cyanogen is a colorless gas with an odor of almonds.[1][2] It is used as a gas for welding and cutting heat-resistant metals, as rocket and missile propellant, and as a fumigant.[3]

CHEMICAL IDENTIFICATION

Common chemical name	Cyanogen
CAS number	460-19-5[1]
IUPAC name	Oxalonitrile[4]
Synonyms	Dicyan; dicyanogen; ethanedinitrile; oxalonitrile; oxalic acid dinitrile; carbon nitride; oxalyl cyanide; prussite; nitriloacetonitrile[2]
Empirical formula	C_2N_2
Chemical structure	 [5]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Commercially available • Theft and divergence: Shipped as a liquefied compressed gas[2] • Synthesis: Well-known
Synthetic pathways	• Precursors: Sodium cyanide; potassium cyanide; copper(II) sulfate; chloride; hydrogen cyanide; oxygen; hydrogen peroxide; copper oxide; nitrogen dioxide[1][6] • Precursor availability: Commercially available
Synthesis	Prepared by adding an aqueous solution of sodium or potassium cyanide to an aqueous solution of copper(II) sulfate or chloride. It may also be prepared from hydrocyanic acid by using copper oxide or from hydrocyanic acid and nitrogen dioxide. The continuous production of $(CN)_2$ from HCN and O_2 or H_2O_2 is utilized industrially on a small scale.[1][6]

PHYSICAL PROPERTIES

Physical form	Colorless gas; burns with purple tinged flame[1]; shipped as a liquefied compressed gas[2]; pungent, penetrating almond-like odor[1]
Molecular weight	52.03 Daltons[1]
Liquid/solid density	0.954 g/cm^3 (-21 °C)[1]
FP/MP	-27.83 °C[1]
Boiling point	-21.1 °C[1]
Viscosity	0.447 cP (liquid, -27.78 °C)[5]
Surface tension	22 dynes/cm (liquid, -21.1 °C)[5]
Vapor pressure	4300 mm Hg (25 °C)[5]
Volatility	Not found
Vapor density (Air=1)	1.8[1]
Solubility	1 g/100 g water (18 °C)[1]; soluble in ethanol, ethyl ether, diethyl ether[1]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Slowly hydrolyzed in aqueous solution[7]
Hydrolysis products	Hydrogen cyanide and cyanate[2]; oxalic acid and ammonia[7]
Photo	Cyanogen does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.[1]
Thermal	Toxic gases and vapors (such as cyanide, hydrogen cyanide, carbon monoxide, and oxides of nitrogen) may be released when cyanide decomposes.[8]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [9]
DOT identification number	UN 1026[9]	
DOT safety placard(s)	 [9]	 [9]

CLINICAL PRESENTATION

Time to effect	Exposure to 16 ppm cyanogen vapor causes immediate eye irritation, followed several minutes later by nasal irritation.[1]
Exposure signs/symptoms	Inhalation: Initially, headache, vertigo, agitation, and a burning sensation in the mouth and throat occur, followed by combative behavior, coma, seizures, and death. Tachypnea, hyperpnea, and dyspnea followed rapidly by respiratory depression are common. Pulmonary edema may occur. Skin: Papules, rashes, pruritus, and ulcerations may occur. Eye: Equally red retinal arteries and veins are common.[5]
Personal decontamination	Remove contaminated clothing and wash exposed area thoroughly with soap and water.[5]
Morbidity and mortality	• LC_{50}: 350 ppm for 1 hr[2] • EC_{50} (mild): 16 ppm[2]

COUNTERMEASURES

Prophylaxis	Wear appropriate protective gloves, clothing, and goggles -- as recommended by the manufacturer. Always wear thermal protective clothing when handling refrigerated/cryogenic liquids. Wear positive pressure self-contained breathing apparatus.[5]
Antidote/Treatment	**Antidote**: Cyanide Antidote Kit[5] **Treatment**: If needed use a Cyanide Antidote Kit.[5] Ingestion: In symptomatic patients, advanced life support including use of the cyanide antidote kit should be initiated as gastrointestinal decontamination is being prepared. Ipecac-induced vomiting is not recommended because of the potential for CNS depression and seizures. Administer activated charcoal as a slurry (240 ml water/30 g charcoal). Usual dose: 25 to 100 g in

	adults/adolescents. Consider after ingestion of a potentially life-threatening amount of poison if it can be performed soon after ingestion (generally within 1 hour). Immediately begin therapy with 100% oxygen. Inhalation: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with beta2 agonist and corticosteroid aerosols. Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. A physician should examine the area if irritation or pain persists. While cyanide can be absorbed through intact skin, most reported cases have involved whole-body immersion in cyanide solutions or large-area burns with molten cyanide solutions. Eyes: Immediately flush with running water for at least 20 minutes.[5]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Fluorometric diffusion method; spectrophotometry; spectrofluorometry; high performance liquid chromatography; gas chromatography; flame ionization detector[1] • Observation: Not found
Decontamination—property & equipment	Allow gas to dissipate
Environmental persistence	Cyanogen will volatilize from dry soil surfaces based upon its vapor pressure. Estimated volatilization half-lives for a model river and model lake are 2 hours and 3.4 days, respectively.[1]
Formulations	Grade with purity of 98.5% available[1]

The information cutoff date for this factsheet is 14 May 2010.

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Cyanogen Chloride (CK)

AGENT OVERVIEW

Cyanogen chloride has been called a blood agent, but it is actually a cellular poison that disrupts the oxidative processes used by the cells. It is non-persistent and is used as a quick acting casualty agent. It was used in WWI as a poison gas; currently, it is used in chemical synthesis processes.[1][2][3][4]

CHEMICAL IDENTIFICATION

Common chemical name	Cyanogen chloride
CAS number	506-77-4[1]
IUPAC name	Carbononitridic chloride[5]
Synonyms	CC; chlorcyan; chlorine cyanide; chlorocyan; chlorocyanide; chlorocyanogen; chlorure de cyanogene (French); CNCl; klortsian; mauguinite (French)[1]
Empirical formula	CNCl
Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Available from Specialty Gases of America[6] • Theft and divergence: CK is used in several manufacturing processes, and could conceivably be stolen or acquired for illicit use.[7] • Synthesis: Synthesized by the reaction of cyanide compounds with chlorine; also by reacting hydrogen cyanide, hydrogen chloride, and an oxidizing agent like hydrogen peroxide.[8]
Synthetic pathways	• Precursors: Cyanide salts; chlorine; hydrogen cyanide; hydrogen chloride; hydrogen peroxide[7] • Precursor availability: Readily available.[7]
Synthesis	$NaCN + Cl_2^- > ClCN + NaCl$[7] $HCN + HCL + H_2O_2 \rightarrow 2ClCN + 2H_2O$ in presence of Cu^{++} or Fe^{3+}[7]

PHYSICAL PROPERTIES

Physical form	Colorless gas or liquid[9]; pungent, biting odor, but odor may go unnoticed because of its intense irritating and lacrimatory (tearing) properties[10]
Molecular weight	61.47 Daltons[1]
Liquid/solid density	1.222 g/mL (0 °C)[11]; 1.202 g/mL (10 °C)[11]; 1.19 g/mL (20 °C)[10]
FP/MP	-6.9 °C[12]; -7.0 °C[10]
Boiling point	12.8 °C (calculated)[1]; 12.6 °C[10]
Viscosity	Not found
Surface tension	24.61 dynes/cm (10 °C)[11]
Vapor pressure	448 mm Hg (0 °C); 680 mm Hg (10 °C); 760 mm Hg (12.8 °C)[1]
Volatility	1.62×10^6 mg/m^3 (0 °C); 2.37×10^6 mg/m^3 (10 °C); 2.62×10^6 mg/m^3 (12.8 °C) (calculated from vapor pressure)[1]
Vapor density (Air=1)	2.1 (calculated)[1]; 2.0[10]
Solubility	71.4 g/L (20 °C) (liquefied CK in water) [1]; soluble in common organic solvents, sulfur mustard, and hydrogen cyanide[1]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	The hydrolysis half-life of CK with tap water is 180 hrs at ambient temperature and pH 7.[1]
Hydrolysis products	Hydrogen chloride and cyanic acid (CNOH)[1], ammonium chloride, NH_3, CO_2[13]
Photo	Given that CK is stable when stored in glass, even for long periods of time[10], it is likely that CK does not decompose readily upon exposure to visible light.

Thermal	Decomposes at ~149 °C[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[14] 
DOT identification number	UN 1589[14]	
DOT safety placard(s)	[14] 	

CLINICAL PRESENTATION

Time to effect	Rapid; large concentrations can potentially cause death within minutes.[1] Effects can occur immediately at about 100 mg/m^3 and can become intolerable at about 50 mg/m^3 after ~1 minute.[2]
Exposure signs/symptoms	• Inhalation: Headache, confusion, dizziness, weakness, heart palpitations, respiratory tract irritation, difficulty breathing or shortness of breath, nausea, vomiting, dilated pupils, loss of consciousness, accumulation of fluid in the lungs, coma, seizures, respiratory arrest, cardiac arrest[14] • Skin: Papules, rashes, pruritus, and ulcerations may occur.[15]
Personal decontamination	Move to fresh air.[1]
Morbidity and mortality	• LCt_{50}: 11,000 mg-min/m^3[16]; 4,000 mg-min/m^3[17] • ECt_{50} (severe): 50 mg/m^3 (intolerable irritation after 1 minute-estimate)[3] • ECt_{50} (mild): 2.5 mg/m^3 produces copious tears in a few minutes.[17]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	**Antidote:** Amyl nitrite, sodium nitrite, and sodium thiosulfate are antidotes for cyanide toxicity; however, amyl nitrite and sodium nitrite should not be administered to victims suffering from smoke inhalation. In these cases, only administer sodium thiosulfate. The described administration of nitrites is based on a patient having normal hemoglobin levels. Below normal hemoglobin levels require titration of nitrites.[14]

	Treatment: Emergency measures - in symptomatic patients, advanced life support including use of the cyanide antidote kit should be initiated as gastrointestinal decontamination is being prepared. Ipecac-induced vomiting is not recommended because of the potential for CNS depression and seizures.[15] Ingestion: If this chemical in liquid form has been swallowed, get medical attention immediately. Administer activated charcoal as a slurry (240 ml water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents. Consider after ingestion of a potentially life-threatening amount of poison if it can be performed soon after ingestion (generally within 1 hour). Immediately begin therapy with 100% oxygen.[15] Inhalation: Move patient to fresh air. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with beta2 agonist and corticosteroid aerosols.[15] Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. While cyanide can be absorbed through intact skin, most reported cases have involved whole-body immersion in cyanide solutions or large-area burns with molten cyanide solutions. Eye: In case of contact with liquefied gas, thaw frosted parts with lukewarm water. Immediately flush skin with running water for at least 20 minutes.[15]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M256A1 CADK; M272 water testing kit; M18A2 CADK; MMI[1] • Laboratory: Gas chromatography; fluorometric microdiffusion; modified Roberts-Jackson method.[7] • Observation: None, other than odor or development of symptoms.
Decontamination—property & equipment	None required under field conditions.[1]
Environmental persistence	Non-persistent[1]
Formulations	Minimum purity 97 mole %[7]

The information cutoff date for this factsheet is 14 May 2010.

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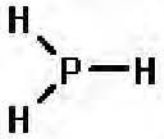
Phosphine

AGENT OVERVIEW

Phosphine is a colorless, flammable, and toxic gas with an odor of garlic or decaying fish. It can ignite spontaneously on contact with air. Phosphine is used widely in the semiconductor industry, and may be encountered in grain storage silos where it has been used as a fumigant. Certain pesticides containing zinc phosphide or aluminum phosphide can release phosphine when they come in contact with water or acid. The phosphine formed in the stomach when these solid phosphides are swallowed can result in phosphine poisoning. Exposure to even small amounts of phosphine can cause headache, dizziness, nausea, vomiting, diarrhea, drowsiness, cough, and chest tightness. More serious exposure can cause shock, convulsions, coma, irregular heartbeat, and liver and kidney damage.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Phosphine
CAS number	7803-51-2[1]
IUPAC name	Phosphane[2]
Synonyms	Hydrogen phosphide; phosphorus hydride; phosphorus trihydride; phosphoretted hydrogen[1]
Empirical formula	PH_3[1]

Chemical structure	 [3]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Common industrial gas and metal phosphides, which readily release phosphine, and are produced in very large quantities.[4][5] • Theft and divergence: Available from electronics manufacturers and agriculture enterprises. • Synthesis: Simple[5]
Synthetic pathways	• Precursors: Metal phosphides[5] • Precursor availability: Common rodenticide[5]
Synthesis	Phosphine may be prepared in a variety of ways. Industrially it can be made by the reaction of white phosphorus with sodium hydroxide, producing sodium hypophosphite as a by-product. It can be made by the hydrolysis of a metal phosphide such as aluminum phosphide or calcium phosphide. Pure samples of phosphine, free from P_2H_4, may be prepared using the action of potassium hydroxide on phosphonium iodide (PH_4I).[6]

PHYSICAL PROPERTIES

Physical form	Colorless gas, or liquefied gas under pressure[7]; odorless at high purity[7], however, impurities impart the odor of garlic or decaying fish.[1]
Molecular weight	34.0 Daltons[1]
Liquid/solid density	Liquid: 0.740 g/mL @ - 87.7 ºC[3]
FP/MP	-134 ºC[1]
Boiling point	-87.7 ºC[1]
Viscosity	0.0106 cP @ 0 ºC[3]
Surface tension	Not found
Vapor pressure	3500 kPa @ 20 ºC[3]
Volatility	Not found
Vapor density (Air=1)	1.17[1]
Solubility	0.3534 g/L @ 20 ºC[3]; soluble in alcohol, ether, cuprous chloride solutions, cyclohexanol[8]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Pure phosphine is inert.[9]
Hydrolysis products	Not found
Photo	Photodecomposes in hard UV (147 nm) to produce hydrogen and solids composed of phosphorus and mixed phosphides.[10]
Thermal	Extremely flammable and will easily ignite.[11] Products of ignition are phosphoric acid and hydrogen.[12]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[13] 
DOT identification number	UN 2199[13]	
DOT safety placard(s)	[13] 	[13]  [13] 

CLINICAL PRESENTATION

Time to effect	Adverse health effects generally occur within the first few hours following inhalation of phosphine. Cardiovascular complications may cause death within 12 to 24 h following exposure. Signs of liver damage may be delayed 48 to 72 h following exposure. Deaths that occur 24 h after exposure are usually a result of liver or kidney failure. Fluid in the lungs (pulmonary edema) may be delayed for 72 h following exposure.[13]
Exposure signs/symptoms	Earliest symptoms are usually restlessness and fatigue, with severe lung irritation, chest tightness, or burning chest pain. Additional signs may include headache; thirst; disturbances of speech, vision, and gait; nausea; abdominal pain; vomiting; and diarrhea. These may be followed by pulmonary edema, convulsions and coma. Death may occur from heart failure within four days or be delayed one to two weeks.[11] Phosphine gas produces no known adverse effects on the eyes or skin, but exposure to liquid phosphine will cause frostbite.[13]
Personal decontamination	Skin: The patient should remove all clothing and personal effects. Double-bag soiled clothing and place in a sealed container clearly labeled as a biohazard.

	Brush away any adherent solid particles and gently blot away any adherent liquid from the patient. Wash hair and all contaminated skin with copious amounts of water (preferably warm) and soap for at least 10-15 min. Decontaminate open wounds first and avoid contamination of unexposed skin. Pay special attention to skin folds, axillae, ears, fingernails, genital areas, and feet. Eye: Remove contact lenses if necessary and immediately irrigate the affected eye thoroughly with water or 0.9% saline for at least 10-15 min. Inhalation: Ensure a clear airway and adequate ventilation. Give oxygen to symptomatic patients.[14]
Morbidity and mortality	Rat: LC_{50}: 680 mg/m^3 (65-75 min exposure); 1,470 mg/m^3 (35-50 min exposure).[11] Human: LC_{LO} 1000 ppm/5 m[15]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Symptomatic patients should be admitted to hospital; asymptomatic patients should be observed for 12 h and advised to seek medical help immediately should symptoms develop. Monitor cardiac rhythm and perform a 12 lead ECG. Correct hypotension by raising the foot of the bed or expanding the intravascular volume. Apply other supportive measures as indicated by the patient's clinical condition.[14]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: GasAlert monitors (0-5 ppm)[16] • Laboratory: Gas chromatography utilizing phosphorus selective detection[17] • Observation: Not found
Decontamination—property & equipment	None.
Environmental persistence	Pure phosphine is inert, but will oxidize under influence of radiation and UV light.[9]
Formulations	As liquefied gas under its own pressure, and as metal phosphides[9]

The information cutoff date for this factsheet is 14 May 2010.

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Hydrogen cyanide (HCN)

AGENT OVERVIEW

Hydrogen cyanide is a blood agent, which is a cellular poison that disrupts the oxidative processes used by cells. It inactivates the cytochrome oxidase system. This poisoning prevents cellular respiration and the normal transfer of oxygen from the blood to body tissues. It is highly volatile and therefore non-persistent.[1] Cyanide was isolated in 1782 by the Swedish chemist Scheele from Prussian blue dye. The use of cyanide as an offensive chemical agent was proposed during the Crimean and Franco-Prussian war, but it was not until WW I that the French utilized HCN under the name Forestite. Hydrogen cyanide was used by the Germans in WW II to exterminate over 2 million soldiers and civilians.[2] Hydrogen cyanide is listed in the CWC Annex on Chemicals under Schedule 3.[3]

CHEMICAL IDENTIFICATION

Common chemical name	Hydrogen cyanide
CAS number	74-90-8[1]
IUPAC name	Formonitrile[4]
Synonyms	HCN; AC; Cyclone (Russian); Zyklon B; forestite (French); hydrocyanic acid; acide cyanhydrique (French); acido cianidrico (Italian); aero liquid HCN; blausaeure (German); blauwzuur (Dutch); carbon hydride nitride (chn); cyaanwaterstof (Dutch); cyanwasserstoff (German); Cyclon; Cyclone B; cyjanowodor (Polish); evercyn; formic anammonide; MW6825000; formonitrile; prussic acid; NA 1051; prussic acid, unstabilized; zaclondiscoids; arbon hydride nitride; zootic acid; Zyklon; nitrilomethane[1][5]
Empirical formula	HCN

Chemical structure	

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Hydrogen cyanide is sold as a gas and is also available as a technical grade liquid in concentrations of 5, 10, and 96-99.5%. Almost all grades of hydrogen cyanide contain a stabilizer such as phosphoric acid to prevent decomposition and explosion. • Theft and divergence: All passenger aircraft or railcar shipment is forbidden and the limit on cargo aircraft shipment is limited. Bulk shipments of solutions from 5-20% or greater are forbidden. • Synthesis: There are two common methods of manufacturing hydrogen cyanide. The first consists of the formation of hydrogen cyanide as a byproduct during the synthesis of acrylonitrile from the reaction of propylene and ammonia with air. The second method involves direct synthesis by the reaction of methane and ammonia with air over platinum catalysts. It can also be prepared by acidifying NaCN or potassium ferrocyanide
Synthetic pathways	• Precursors: Methane; ammonia; sodium cyanide; potassium ferrocyanide • Precursor availability: Commercially available
Synthesis	Prepared on a large scale by the catalytic oxidation of ammonia-methane mixtures; prepared in the laboratory by acidifying sodium cyanide or potassium ferrocyanide

PHYSICAL PROPERTIES

Physical form	Colorless liquid with odor of bitter almonds or peach kernels
Molecular weight	27.03 Daltons
Liquid/solid density	0.6797 g/mL (25 °C), 0.7162 g/mL (0 °C)
FP/MP	-13.3 °C (MP)
Boiling point	25.5 °C
Viscosity	0.2698 cSt (25 °C) (liquid)
Surface tension	17.78 dynes/cm (25 °C) (liquid)
Vapor pressure	760 mm Hg (25.5 °C), 746 mm Hg (25 °C), 265 mm Hg (0 °C)
Volatility	1.1×10^6 mg/m^3 (25.5 °C), 1.08×10^6 mg/m^3 (25 °C), 4.2×10^5 mg/m^3 (0 °C) (calculated from vapor pressure)

Vapor density (Air=1)	0.93 (calculated)[1]
Solubility	Miscible with common organic solvents including alcohol and ether[1][3]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Slow under acidic conditions, rapid with traces of bases or basic salts[1]
Hydrolysis products	Ammonia, formic acid, and amorphous brown solids[1]
Photo	Hydrogen cyanide is expected to be resistant to direct photolysis.[3]
Thermal	Above 65.5 °C when stabilized; forms explosive polymer on standing; stabilized material can be stored up to 65 °C.[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [8]	
DOT identification number	UN 1051[8]		
DOT safety placard(s)	 [8]		

CLINICAL PRESENTATION

Time to effect	Exposure to high concentrations causes effects within seconds and death within minutes[1]; 10 mg/L can incapacitate humans within 10-18 seconds.[2]
Exposure signs/symptoms	Inhalation: Giddiness, agitation, headache, dizziness, confusion, palpitation, chest pain, difficulty breathing, nausea, vomiting, convulsions, unconsciousness, and coma may occur.[3][9] Skin: In addition to inhalation symptoms, percutaneous exposure may result in skin irritation.[3][9] Eye: In addition to inhalation symptoms, ocular exposure may result in eye irritation and lacrimation (tearing).[3][9]
Personal decontamination	Move to fresh air; no decon normally required under field conditions[1]; for dermal exposure, wash skin thoroughly with soap and water; for eye exposure, flush eyes with lukewarm water for at least 15 minutes[9]
Morbidity and mortality	• LCt_{50}: 2,500-5,000 mg-min/m^3[1][10]; 2,000 mg-min/m^3 (0.5 min);

	20,600 mg-min/m^3 (30 min)[11] • ECt_{50} (mild): Slight effects at 20-40 mg/m^3; 50-60 mg/m^3 can be tolerated without immediate or late effects for 20-60 minutes.[12] • LD_{50} (percutaneous): 7,000-12,000 mg/m^3 (5-min exposure) for workers with self-contained respirators without effective skin protection.[12] Skin LD_{50}s for hydrogen cyanide have been estimated to be 100 mg/kg.[10][11] • LD_{50} (ingestion): 50 mg (estimate)[3]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	**Antidote:** A cyanide antidote, either hydroxocobalamin or a sodium nitrite/sodium thiosulfate kit, should be administered.[3] **Treatment:** If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. IMMEDIATELY begin administering 100% oxygen. For eye exposure, flush with lukewarm water for at least 15 minutes. For skin exposure, wash exposed skin areas twice with soap and water. For ingestion, DO NOT induce vomiting or attempt to neutralize! Activated charcoal may be administered if victims are conscious and alert. Promote excretion by administering a saline cathartic or sorbitol to conscious and alert victims.[9]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M256A1 CADK; M72 water testing kit; M18A2 CADK; M183A3, MM1[1] • Laboratory: Spectrophotometry; colorimetry; gas chromatography; electron capture detector.[2][13] • Observation: Possibly detectable by odor of bitter almonds.[1]
Decontamination—property & equipment	Neutralize with agricultural lime, crushed limestone, or sodium bicarbonate.[3]
Environmental persistence	Highly volatile and non-persistent in soil.[11] Atmospheric half-life depends on the altitude, and the rate of the hydroxyl radical reaction, approximately 2-3 years.[13][14]
Formulations	Technical (96-98%); 2, 5, and 10% solutions. All grades usually contain stabilizer, usually 0.05% phosphoric acid.[3]

The information cutoff date for this factsheet is 14 May 2010.

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Potassium Cyanide (KCN)

AGENT OVERVIEW

Potassium cyanide releases hydrogen cyanide gas, a highly toxic chemical asphyxiant that interferes with the body's ability to use oxygen. Exposure to potassium cyanide can be rapidly fatal. It has whole-body (systemic) effects, particularly affecting the brain, heart and circulatory system, and lungs. Potassium cyanide is used commercially for fumigation, electroplating, and extracting gold and silver from ores. Hydrogen cyanide gas released by potassium cyanide has a distinctive bitter almond odor, but a large proportion of people cannot detect it, and the odor does not provide adequate warning of hazardous concentrations. It is usually shipped as capsules, tablets, or pellets. Potassium cyanide absorbs water from air (is hygroscopic or deliquescent).[1]

CHEMICAL IDENTIFICATION

Common chemical name	Potassium cyanide
CAS number	151-50-8[2]
IUPAC name	Potassium cyanide[3]
Synonyms	KCN; cyanide of potassium; cyanides; cyanure de potassium (French); hydrocyanic acid, potassium salt; kalium-cyanid (German); M-44 capsules[2]
Empirical formula	KCN
Chemical structure	

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Available from manufacturers with the U.S.[2] • Theft and divergence: Possible • Synthesis pathways: Well known
Synthetic pathways	• Precursors: Potassium hydroxide; hydrogen cyanide; potassium carbonate; carbon; ammonia[4] • Precursors available commercially
Synthesis	Manufactured by the reaction of an aqueous solution of potassium hydroxide with hydrogen cyanide: $KOH + HCN \rightarrow KCN + H_2O$ Also prepared by heating a mixture of potassium carbonate and carbon with ammonia at high temperatures: $K_2CO_3 + 4C + 2NH_3 \rightarrow 2\ KCN + 3CO\uparrow + 3H_2\uparrow$[4]

PHYSICAL PROPERTIES

Physical form	White granules or crystals, faint odor of bitter almonds[2][5]
Molecular weight	65.12 Daltons[5]
Liquid/solid density	1.52-1.55 g/cm^3 @ 20 °C[2][5]
FP/MP	634 °C[5]; 634.5 °C[6]
Boiling point	1625 °C[6]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	Essentially zero[7]
Volatility	Not found
Vapor density (Air=1)	Not found
Solubility	Soluble in 2 parts cold, 1 part boiling water; 2 parts glycerol, 100 parts alcohol, 25 parts methanol[5]; 58 g/100 g water @ 20 °C[7]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Hydrolyzes rapidly in water to K^+ and CN^- ions. In water of neutral pH, not enough hydrogen cyanide gas is subsequently formed to be dangerous, except in enclosed spaces. However, if the water is acidic, significant amounts of hydrogen cyanide gas may be released. While potassium cyanide is not volatile, reaction of suspended dust particles with moisture and carbon dioxide in the air can result in release of some hydrogen cyanide.[2]
Hydrolysis products	Hydrogen cyanide[2]

Photo	Photolysis in the atmosphere is not expected to be a significant degradation pathway.[8]
Thermal	When heated to decomposition, it emits very toxic fumes of dipotassium oxide, hydrogen cyanide, and nitrogen oxides.[2][6]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[1] 
DOT identification number	UN 1680[1]	
DOT safety placard(s)	[1] 	

CLINICAL PRESENTATION

Time to effect	Effects occur rapidly following exposure to potassium cyanide. Inhalation exposure to hydrogen cyanide gas released from potassium cyanide produces symptoms within seconds to minutes; death may occur within minutes.[1]
Exposure signs/symptoms	Early symptoms of cyanide poisoning include lightheadedness, giddiness, rapid breathing, nausea, vomiting, feeling of neck constriction and suffocation, confusion, restlessness, and anxiety. Accumulation of fluid in the lungs (pulmonary edema) may complicate severe intoxications. Rapid breathing is soon followed by respiratory depression/respiratory arrest (cessation of breathing). Severe cyanide poisonings progress to stupor, coma, muscle spasms (in which head, neck, and spine are arched backwards), convulsions, fixed and dilated pupils, and death. The CNS is the most sensitive target organ of cyanide poisoning. Cardiovascular effects require higher cyanide doses than those necessary for CNS effects. In serious poisonings, the skin is cold, clammy, and diaphoretic. Blue discoloration of the skin may be a late finding. Severe signs of oxygen deprivation in the absence of blue discoloration of the skin suggest cyanide poisoning.[1]
Personal decontamination	Skin: Immediately wash or shower with soap or mild detergent and water Eyes: Wash immediately with large amounts of water Inhalation: Move to fresh air at once Ingestion: Give conscious victims large volumes of water immediately, then induce vomiting. Do not induce vomiting in unconscious victims.[7]
Morbidity and mortality	LD_{50} (ingestion): 200-300 mg/adult human (est.)[2][9]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	Amyl nitrite, sodium nitrite, and sodium thiosulfate are antidotes. For mild to moderate poisoning: Child: Administer 0.75 mL per pound of a 25% sodium thiosulfate solution intravenously over a period of 10 minutes. Adult: Administer 50 ml of a 25% sodium thiosulfate intravenously over a period of 10 minutes. For severe poisoning: Child: Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Children should receive 0.15 mL per pound of body weight of sodium nitrite (0.33 mL per kg body weight of 3% sodium nitrite) over a period of 5-20 minutes. Next, administer 0.75 mL per pound body weight of 25% sodium thiosulfate (1.65 mL per kilogram body weight of 25% sodium thiosulfate) intravenously over a period of 10 minutes. Adult: Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Administer 300 mg of a 3% solution (10 mL of a 3% solution) of sodium nitrite intravenously over a period of 5 to 20 minutes. Next, administer 12.5 g (50 mL of a 25% solution) of sodium thiosulfate intravenously over a period of 10 minutes.[2]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Titration with silver nitrate; ion exchange; continuous flow distillation; EDTA electrode method; AISI aeration method; EDTA aeration method; modified Roberts-Jackson method; EPA method for cyanides amenable to chlorination; oxidation of hemoglobin to methemoglobin and subsequent monitoring of absorption spectrum[2]
Decontamination—property & equipment	Keep water away from release. Flush spill area with hypochlorite solution. Cover in noncombustible material for proper disposal. Shovel into suitable dry container. Control runoff and isolate discharged material for proper disposal.[2]
Environmental persistence	Cyanide rapidly degrades in water and soil.[10]
Formulations	Available in various levels of purity; the article of commerce contains about 95% KCN.[2]

The information cutoff date for this factsheet is 14 May 2010.

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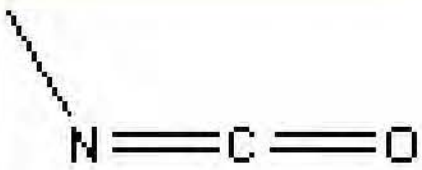
Methyl Isocyanate (MIC)

AGENT OVERVIEW

Methyl isocyanate is a very flammable colorless liquid with a pungent odor. It is irritating and corrosive to the eyes, respiratory tract, and skin. High vapor concentrations can cause severe lung injury, eye damage, and death. Methyl isocyanate is used as a chemical intermediate in the production of pesticides. It is also used to produce polyurethane foams and plastics.[1] Since the incident in Bhopal in 1984, which killed thousands of people surrounding the Union Carbide plant in India, methyl isocyanate (MIC) is now produced where it can be consumed on site.[2][3][4]

CHEMICAL IDENTIFICATION

Common chemical name	Methyl isocyanate
CAS number	624-83-9
IUPAC name	Methylimino(oxo)methane[5]
Synonyms	Methyl Isocyanate; isocyanic acid, methyl ester; CH_3NCO; iso-cyanomethane; isocyanate de methyle; isocyanatomethane; methyl carbonimide; methyl isocyanat; methylisocyanaat; metil isocianato; MIC; methylisokyanat; TL 1450; (methylimino)(oxo)methane[6]
Empirical formula	C_2H_3NO[1]

Chemical structure	

CHEMICAL AVAILABILITY

Procurement methods	• Available for purchase commercially • Theft and divergence: Possible • Synthesis pathways well known
Synthetic pathways	• Precursors: Phosgene; methylamine; monomethylformamide; N,N-diphenyl-N'-methylurea; bis(trimethylsilyl)methylamine • Precursors available for purchase commercially except for N,N-diphenyl-N'-methylurea
Synthesis	• Synthesized by reacting methylamine with phosgene, oxidizing monomethylformamide at high temperatures (> 550 °C), or heating metal methylisocyanates • Synthesized via Curtius rearrangement; by heating N,N-diphenyl-N'-methylurea; or by phosgenation of bis(trimethylsilyl)methylamine

PHYSICAL PROPERTIES

Physical form	Colorless liquid with sharp odor
Molecular weight	57.051 Daltons
Liquid/solid density	0.96 g/cm^3 @ 20 °C ; 0.9230 g/cm^3 @ 27 °C
FP/MP	-45 °C
Boiling point	39-40 °C
Viscosity	Not found
Surface tension	Not found
Vapor pressure	348 mm Hg @ 20 °C 400 mm Hg @ 20.6 °C 418.74 mm Hg @ 25 °C (calculated)
Volatility	Not found
Vapor density (Air=1)	1.42
Solubility	10 g/L @ 15 °C; 29 g/L @ 25 °C

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Reacts violently with water or steam[11]
Hydrolysis products	N-carboxymethylamine, methylamine, carbon dioxide, and N,N'-dimethylurea[2]; hydrocyanic acid[2]
Photo	Not expected to be susceptible to direct photolysis by sunlight[2]
Thermal	Toxic gases and vapors (such as hydrogen cyanide, oxides of nitrogen, and carbon monoxide) may be released in a fire involving methyl isocyanate[2]; when heated to decomposition, emits toxic fumes of nitrogen oxides and hydrogen cyanide[2]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[11] 
DOT identification number	UN 2480[11]	
DOT safety placard(s)	[11] 	[11] 

CLINICAL PRESENTATION

Time to effect	Immediate for inhalation, eye, and skin contact[2]
Exposure signs/symptoms	Inhalation: Cough, labored breathing, shortness of breath, sore throat, mucous secretions, chest pain Skin: Redness, pain, burning sensation Eyes: Tearing, pain, redness, loss of vision, permanent eye damage Ingestion: Abdominal pain, vomiting, shock or collapse[2]
Personal decontamination	Do not put yourself in danger by entering a contaminated area to rescue a victim. Provide Basic Life Support/CPR as needed. Decontaminate as follows: Inhalation: Remove the victim to fresh air and give oxygen if available. Skin: Remove and isolate contaminated clothing (including shoes) and wash skin with soap and large volumes of water for 15 minutes. Eye: Rinse eyes with large volumes of water or saline for 15 minutes. Swallowed: Do not make the victim vomit.[11]

Morbidity and mortality	• LCt_{50}: Not found • ECt_{50} (mild): 2 ppm (4.7 mg/m^3)[12]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	Inhalation: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with inhaled β2 agonist and oral or parenteral corticosteroids. Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. Eye: Irrigate exposed eyes with copious amounts of room temperature water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.[2] Antidotes for cyanide are not effective.[2]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Colorimetric paper tape detection[13] • Laboratory: HPLC[2] • Observation: None
Decontamination—property & equipment	Remove all ignition sources. Ventilate area of spill or leak. For small quantities, absorb on paper towels. Evaporate into safe place (such as fume hood). Allow sufficient time for evaporating vapors to completely clear the hood ductwork. Burn the paper in suitable location away from combustible materials. Large quantities can be reclaimed or collected and atomized in a suitable combustion chamber equipped with appropriate effluent gas cleaning device. Methyl isocyanate should not be allowed to enter a confined space, such as a sewer, because of the possibility of explosion. Alternatively, collect leaking liquid in sealable containers. Cautiously neutralize spilled liquid with caustic soda. Absorb remaining liquid in dry sand or inert absorbent and remove to safe place.[2]
Environmental persistence	"Many days"[14]

The information cutoff date for this factsheet is 14 May 2010.

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GABA INHIBITOR AGENTS

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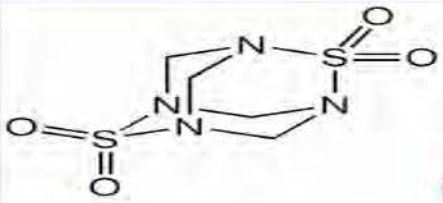
Tetramethylene Disulfotetramine (TETS)

AGENT OVERVIEW

Bayer Chemical Company disclosed the synthesis of tetramethylene disulfotetramine (TETS) in 1949. Recent years have seen a large number of mass poisonings in mainland China, particularly those caused by illicit rodenticides. This rat poison is responsible for a great percentage of death and injury in the People's Republic of China (PRC). The human oral lethal dose is estimated to be as low as 0.1 mg/kg, and TETS is widely available in open markets in mainland China—this despite being prohibited for manufacture or sale in that country. Clinical presentation of TETS ingestion is dose dependent, and poisonings may be fatal within hours. No known antidote exists, and treatment is mainly supportive.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Tetramethylene disulfotetramine[3]
CAS number	80-12-6[3]
IUPAC name	2,6-Dithia-1,3,5,7-tetraazaadamantane,2,2,6,6-tetraoxide[3]
Synonyms	Dushuqiang; four-two-four; NSC 172824; TETS; meishuming; shanbudao; tetramine; tetramethylenedisulfotetramine; 2,6-dithia-1,3,5,7-tetraazatricyclo[3.3.1.1^{3,7}]decane 2,2,6,6-tetraoxide[3][4]
Empirical formula	$C_4H_8N_4O_4S_2$[3]

Chemical structure	 [5]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: China[2] • Theft and divergence: China[2] • Synthesis: Simple[6]
Synthetic pathways	• Precursors: Sulfamide; formaldehyde[6] • Precursor availability: Common organic chemicals
Synthesis	In 1933, it was first synthesized by sulfamide and formaldehyde.[6]

PHYSICAL PROPERTIES

Physical form	White powder or cubic crystals; odorless[3][7]
Molecular weight	240.26 Daltons
Liquid/solid density	Not found
FP/MP	>270 °C[5]
Boiling point	Not found
Viscosity	Contact CSAC
Surface tension	Contact CSAC
Vapor pressure	Contact CSAC
Volatility	Contact CSAC
Vapor density (Air=1)	Contact CSAC
Solubility	Slightly soluble in water (0.25 mg/mL) and DMSO, slightly soluble in acetone, insoluble in methanol and ethanol[3]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Stable in mild acid or alkali in solutions up to 0.1 N[3]
Hydrolysis products	Not found
Photo	Contact CSAC
Thermal	Decomposes at 255-260 °C[5]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	Not found	
	Flammability		
	Reactivity		
	Special Properties		
DOT identification number	UN 2811[8]		
DOT safety placard(s)	[9] 		

CLINICAL PRESENTATION

Time to effect	The time interval between ingestion and symptom onset ranges from several minutes to half an hour (maximum 13 hours).[3]
Exposure signs/symptoms	Nausea, vomiting and abdominal pain occur in the majority of patients. Epigastric burning sensation and diarrhea have been present in some patients. Haematemesis and melaena in severe cases have been reported. Other symptoms include headache, dizziness, fatigue, anorexia, nausea, vomiting, numbness of lips, listlessness, seizures, foaming at the mouth, urinary incontinence, loss of consciousness, coma, and respiratory failure.[3]
Personal decontamination	Wash with copious amounts of water for at least 15 minutes. Remove contaminated clothing and shoes and wash before wearing.[8]
Morbidity and mortality	LD_{50} (ingestion): The human lethal dose is estimated at 7-10 mg[1] and 6-12 mg[10]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Antidote: None Treatment: Sequential hemoperfusion and continuous venovenous hemofiltration.[11] Convulsions should be controlled with appropriate anticonvulsants (e.g., sodium phenobarbital, sodium valproate, valium). Gastric lavage should be performed.[3]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: LC/MS and GC/MS[12], solid phase microextraction of TETS coupled with GC NPD[13], GC/MS[14]

	• Observation: None
Decontamination—property & equipment	Not found
Environmental persistence	Contact CSAC
Formulations	Sold at very low concentrations as rat poison.

The information cutoff date for this factsheet is 14 May 2010.

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METABOLIC AGENTS

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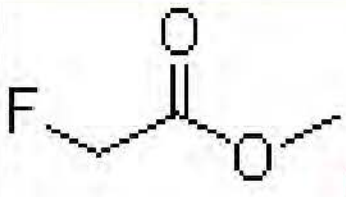
Methyl Fluoroacetate

AGENT OVERVIEW

Methyl fluoroacetate was first prepared in 1896, but was not investigated as a possible chemical warfare agent until the 1930s. During World War II, there were plans to use fluoroacetates as water contaminants because of their stability in water solution and their lack of taste or odor. The fluoroacetates are highly toxic when inhaled, injected, and to some extent when absorbed through the skin. They act as convulsant poisons with a delayed effect.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Methyl fluoroacetate
CAS number	453-18-9[2]
IUPAC name	Methyl 2-fluoroacetate[3]
Synonyms	Methyl fluoroacetate; fluoracetic acid methyl ester; fluoroacetic acid, methyl ester; EINECS 207-218-7; TL 551; BRN 1740631; methylester kyseliny fluoroctove (Czech); ZINC02040592; acetic acid, fluoro-, methyl ester; LS-12171[3]
Empirical formula	$C_3H_5FO_2$[4]

Chemical structure	 [4]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Commercially available from Canada and China[3] • Theft and divergence: Not found • Synthesis: Straightforward[5]
Synthetic pathways	• Precursors: Potassium fluoride; methyl chloroacetate[5] • Precursor availability: Common organic chemicals
Synthesis	Methyl fluoroacetate was first prepared in 1896 by Swarts in small yield by the action of silver or mercurous fluoride on methyl iodoacetate. Methyl fluoroacetate can be made in larger yield by mixing methyl chloroacetate with potassium fluoride in a rotating autoclave at 220 °C for 4 hours.[1]

PHYSICAL PROPERTIES

Physical form	Liquid; faint fruit-like odor[5]
Molecular weight	92.07 Daltons[6]
Liquid/solid density	1.1613 g/mL @ 15 °C[7]
FP/MP	-40 °C[6]
Boiling point	104.5 °C[7]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	4100 Pa @ 25 °C[8]
Volatility	Not found
Vapor density (Air=1)	Not found
Solubility	15 g/100 ml water @ 0 °C[9]; soluble in alcohol, ether, acetone, light petroleum, carbon tetrachloride, benzene, glacial acetic acid, and 2:2'-dichlorodiethyl sulfide, and partly soluble in carbon disulfide[5]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Very slow (2.5% in 60 hours @ 22-24 °C)[9]
Hydrolysis products	fluoroacetic acid[9]

Photo	Not found
Thermal	Not found

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	 [10]	
	Flammability		
	Reactivity		
	Special Properties		
DOT identification number	UN 2927[10]		
DOT safety placard(s)	 [10]		

CLINICAL PRESENTATION

Time to effect	Toxic action is delayed.[11][5]
Exposure signs/symptoms	Skin color change (red or white), pain, irritation, watery eyes, blurred vision, nausea, vomiting, chest tightness, shortness of breath, drowsiness, confusion, convulsions[10]
Personal decontamination	Remove all contaminated clothes and footwear immediately unless stuck to skin. Drench the affected skin with running water for at least 10 minutes if substance is still on skin. Rinse eyes with water for 15 minutes.[10]
Morbidity and mortality	LD_{50} (ingestion): 0.22 mg/kg (rat), 0.5-2.0 mg/kg est. (human)[3]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	**Antidote** For sodium fluoroacetate, a combination of calcium gluconate (130 mg/kg) with sodium succinate (240 mg/kg) was found to be an effective antidote (for mice) if the two solutions were either injected at separate sites or mixed in the same syringe just prior to injection.[12] Intravenous glyceryl monoacetate (monoacetin) and ethanol administration have been advocated to prevent or reverse the toxic effects of fluoroacetate. However, their safety and efficacy have not been demonstrated in humans.[13] **Treatment** Ingestion: Wash out mouth with water. Do not induce vomiting. If conscious, give two cups of water to drink immediately. If unconscious, check for

	breathing and apply artificial respiration if necessary. If unconscious and breathing is okay, place in the recovery position. Inhalation: Remove casualty from exposure. If conscious, ensure the casualty sits or lies down. If unconscious and breathing is okay, place in the recovery position. If unconscious, check for breathing and apply artificial respiration if necessary. If breathing becomes bubbly, have the casualty sit and provide oxygen if available.[10]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: High performance liquid chromatography has been used to detect sodium fluoroacetate.[14] • Observation: Not found
Decontamination—property & equipment	Not found
Environmental persistence	Not found
Formulations	Not found

The information cutoff date for this factsheet is 14 May 2010.

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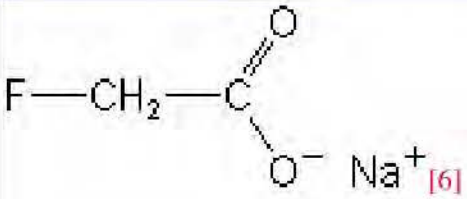
Sodium Fluoroacetate

AGENT OVERVIEW

Sodium fluoroacetate was discovered by German military chemists in WW II. The chemical was shown to be highly potent, but it was difficult to deliver, and required ingestion or injection for optimal effect.[1] Sodium fluoroacetate was introduced as a rodenticide in the U.S. in 1946. However, its considerable efficacy against target species is offset by comparable toxicity to other mammals and, to a lesser extent, birds and its use as a general rodenticide was therefore severely curtailed by 1990. Sodium fluoroacetate has been licensed in the U.S. for use against coyotes, which prey on sheep and goats, and in Australia and New Zealand to kill unwanted introduced species.[2]

CHEMICAL IDENTIFICATION

Common chemical name	Sodium fluoroacetate
CAS number	62-74-8[3]
IUPAC name	Sodium 2-fluoroacetate[4][5]
Synonyms	2-Fluoroacetic acid; acide-monofluoracetique; acido monofluoroacetico; compound 1080; cymonic acid; FAA; fluoroacetate; fluoracetato de sodium; fluoroacetic acid sodium salt; fluoroethanoic acid; gifblaar poison; MFA; monofluorazijnzuur; monofluoressigsaure; monofluoroacetate; monofluoroacetic acid; sodium monofluoroacetate; sodium perfluoroacetate[3]
Empirical formula	$C_2H_2FO_2.Na$[3]

Chemical structure	 [6]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Restricted pesticide[7] • Theft and divergence: Possible but unlikely due to the small amount used in the U.S. • Synthesis: Simple
Synthetic pathways	• Precursors: Methyl fluoroacetate; methyl chloroacetate; potassium fluoride; cobalt hydrogen fluoride; formaldehyde; ethyl chloroacetate; potassium fluoride[8][9][10] • Precursor availability: Common chemicals (except for methyl fluoroacetate)[11]
Synthesis	• Add NaOH to methyl fluoroacetate.[8] • Condense methyl chloroacetate with potassium fluoride and then convert the fluoroester to the sodium salt with sodium hydroxide.[9] • React cobalt hydrogen fluoride and formaldehyde at high pressure to give fluoroacetic acid and then use sodium hydroxide to convert to sodium fluoroacetate.[9] • React ethyl chloroacetate and potassium fluoride to form ethyl fluoroacetate, and then treat with a methanol solution of sodium hydroxide.[10]

PHYSICAL PROPERTIES

Physical form	White solid powder; faint, vinegar-like odor[12]
Molecular weight	100.02 Daltons[3]
Liquid/solid density	Not found
FP/MP	200 °C (FP)[12]
Boiling point	Decomposes before boiling[12]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	6.54×10^{-7} mm Hg @ 25 °C (est.)[13]
Volatility	Very low[3]
Vapor density (Air=1)	Not found
Solubility	111 g/100 g water @ 25 °C 5 g/100 g methanol @ 25 °C

	1.4 g/100 g ethanol @ 25 °C 0.04 g/100 g acetone @ 25 °C 0.004 g/100 g carbon tetrachloride @ 25 °C [14]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Expected to be stable in aqueous solution at any pH[2][3]
Hydrolysis products	Not found
Photo	Not found
Thermal	When heated to decomposition it emits highly toxic fumes of sodium oxide and hydrogen fluoride.[15]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[16] 
DOT identification number	UN 2629[16]	
DOT safety placard(s)	[16] 	

CLINICAL PRESENTATION

Time to effect	1 hour (ingestion)[17]
Exposure signs/symptoms	Nausea, excessive salivation, vomiting, diarrhea, blurred vision, tingling sensations; muscular twitching to convulsions alternating with coma and depression; and heart failure. Other symptoms include numbness, low blood pressure, hyperactivity, respiratory depression or arrest, cyanosis (blue tint to the skin and mucous membranes), and ventricular fibrillation[12]
Personal decontamination	Skin - Wash three times with soap and water; flush eyes with lukewarm water for at least 15 minutes.[12]
Morbidity and mortality	LD_{50} (ingestion): 0.5-2.0 mg/kg est. (human)[2]; 2-10 mg/kg[17]

COUNTERMEASURES

Prophylaxis	None

Antidote/Treatment	**Antidote:** A combination of calcium gluconate (130 mg/kg) with sodium succinate (240 mg/kg) was found to be an effective antidote (for mice) if the two solutions were either injected at separate sites or mixed in the same syringe just prior to injection.[18] Intravenous glyceryl monoacetate (monoacetin) and ethanol administration have been advocated to prevent or reverse the toxic effects of fluoroacetate. However, their safety and efficacy have not been demonstrated in humans.[17] **Treatment:** Inhalation: Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support.[12] Dermal/Eye: Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support. Remove contaminated clothing as soon as possible. If eye exposure has occurred, eyes must be flushed with lukewarm water for at least 15 minutes. Wash exposed skin areas three times with soap and water.[12] Ingestion: Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support. Vomiting may be induced with syrup of Ipecac. If elapsed time since ingestion of sodium fluoroacetate is unknown or suspected to be greater than 30 minutes, do not induce vomiting: Ambulate (walk) the victims and give large quantities of water. If vomiting has not occurred after 15 minutes, Ipecac may be re-administered. Continue to ambulate and give water to the victims. If vomiting has not occurred within 15 minutes after second administration of Ipecac, administer activated charcoal. Promote excretion by administering a saline cathartic or sorbitol to conscious and alert victims.[12]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: High performance liquid chromatography[19]; gas chromatography[15] • Observation: None
Decontamination—property & equipment	Not found
Environmental persistence	Fluoroacetate is rapidly broken down by biological action into harmless compounds in natural soil and water systems.[20]
Formulations	Usually marketed as an aqueous solution containing 0.5% nigrosine as a black warning color, which is used to prepare baits. In the United Kingdom it is formulated as a 5% solid concentrate and a 0.375% bait for dilution with water to 0.25%. Sale and use are under strict control.[2]

The information cutoff date for this factsheet is 14 May 2010.

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NON-LETHAL CHEMICALS

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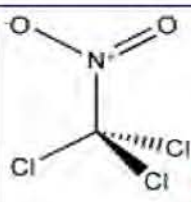
Chloropicrin (PS)

AGENT OVERVIEW

Chloropicrin (PS) is a soil fumigant used for its broad biocidal and fungicidal properties, primarily in high-value crops such as strawberries, peppers, onions, tobacco, flowers, tomatoes, and nursery crops. John Stenhouse, a Scottish chemist and inventor, synthesized chloropicrin in 1848. It was first tested as a pre-plant soil fumigant in 1920. Because PS is toxic by all routes of entry, it has the potential for widespread destruction as a chemical warfare agent. Chloropicrin was used in WW I as a chemical warfare agent because of its potent activity as a lacrimator.[1][2][3]

CHEMICAL IDENTIFICATION

Common chemical name	Chloropicrin
CAS number	76-06-2[4]
IUPAC name	Trichloronitromethane[5]
Synonyms	PS; chloropicrine; chlorpicrin-1; dolochlor; ENT-27; G 25; Larvacide; methane, trichloronitro-; microlysin; NCI-C00533; nitrochloroform; nitrotrichloromethane; pic-clor; picfume; picride; ProFume A; trichloronitromethane[4]
Empirical formula	CCl_3NO_2[4]

Chemical structure	 [4]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Widely available, common organic chemical • Theft and divergence: Possible from large-scale agricultural operations • Synthesis: Simple[6]
Synthetic pathways	• Precursors: Aqua regia; acetone[6] • Precursor availability: Common chemicals
Synthesis	Chlorination of acetone by aqua regia[6]

PHYSICAL PROPERTIES

Physical form	Slightly oily, colorless liquid[7]; irritating and pungent odor[7][8]
Molecular weight	164.376 Daltons[4]
Liquid/solid density	1.64 g/mL @ 25 °C[8]
FP/MP	-64 °C[9]
Boiling point	112 °C[8]
Viscosity	1.078 cP @ 21.1 °C[8]
Surface tension	32 dynes/cm @ 20 °C[8]
Vapor pressure	2,440 Pa @ 20 °C; 3,200 Pa @ 25 °C[9]
Volatility	164,500 mg/m^3 @ 20 °C; 210,700 mg/m^3 @ 25 °C[10]
Vapor density (Air=1)	5.7[10]
Solubility	1.6 g/L @ 25 °C in water, miscible in most organic solvents[9]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	The half-life of chloropicrin in water exposed to light was 31.1 hours.[9]
Hydrolysis products	Carbon dioxide, bicarbonate, chloride, nitrate, and nitrite[9]
Photo	The half-life of chloropicrin in air exposed to simulated sunlight was 20 days.[9]
Thermal	Chloropicrin decomposes explosively when heated above its boiling point.[10]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[11] 
DOT identification number	UN 1580[12]	
DOT safety placard(s)	[12] 	

CLINICAL PRESENTATION

Time to effect	Irritation of the eyes, upper and lower airways, and skin occur rapidly following exposure to chloropicrin. Initial irritation typically resolves within 15 to 30 minutes following decontamination. Gastrointestinal symptoms following ingestion of chloropicrin may persist for weeks. Adverse neurological and musculoskeletal effects may persist from weeks to months.[10]
Exposure signs/symptoms	• Inhalation: Severe irritation leading to coughing, choking, difficulty breathing or shortness of breath; pulmonary edema; chest wall pain; nausea, vomiting, and diarrhea; headache; dizziness; anxiety; fatigue; and bluish discoloration of the skin[10] • Skin: Severe skin irritation and redness of the skin, possibly resulting in blisters[10] • Eye7: Intense painful irritation and tear production[10] • Ingestion: Abdominal pain, nausea, vomiting, sore throat, burns in the mouth, difficulty breathing, dizziness, and bluish discoloration of the skin[10]
Personal decontamination	Use neutral or slightly basic solutions with sulfides, such as sodium sulfide. Do not use acidic solutions for decontamination; acids reduce PS to CX, a blister agent.[13]
Morbidity and mortality	LD_{50} (inhalation): 20,000 mg-min/m^3 (estimate)[14]; LC_{Lo}: 2400 mg-min/m^3[15]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	• **Antidote:** None[10]

	• **Treatment:** Inhalation: Evaluate respiratory function and pulse. Ensure that the patient/victim has an unobstructed airway. If shortness of breath occurs or breathing is difficult, administer 100% humidified oxygen as needed to maintain oxygenation and comfort. Assist ventilation as required. Always use a barrier or bag-valve-mask device. If breathing has ceased, provide artificial respiration. Monitor for spasmodic narrowing of the large airways (bronchospasm), and treat bronchospasms with β2 agonists and corticosteroids. Monitor for spasmodic contraction of the voice box (laryngospasm), and treat it if it occurs. Examine the moist lining of the respiratory tract (mucous membranes) for corrosive effects.[10] Skin: Wash with neutral or slightly basic solutions with sulfides, such as sodium sulfide. Do not use acidic solutions for decontamination; acids reduce PS to CX, a blister agent.[10] Eyes: Immediately wash eyes with large amounts of tepid water for at least 15 minutes.[10] Ingestion: Ensure that the patient/victim has an unobstructed airway. Do not induce vomiting or administer charcoal. If the patient/victim is alert and able to swallow, immediately administer 4 to 8 ounces of milk or water (not to exceed 4 ounces/120 mL in a child).[10]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Colorimetric tube devices that have a sensitivity of at least 0.15 ppm for chloropicrin[12] • Laboratory: Negative chemical ionization mass spectrometry[16]; air sampling tubes followed by gas chromatography utilizing electron capture detection[17] • Observation: None
Decontamination—property & equipment	Use neutral or slightly basic solutions with sulfides, such as sodium sulfide. Do not use acidic solutions for decontamination; acids reduce PS to CX, a blister agent.[10]
Environmental persistence	Short due to high volatility and photodecomposition
Formulations	Produced in numerous different pesticides and fumigants, with varying concentrations of chloropicrin.[18]

The information cutoff date for this factsheet is 14 May 2010.

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OPIATES

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Homeland Security

Science and Technology

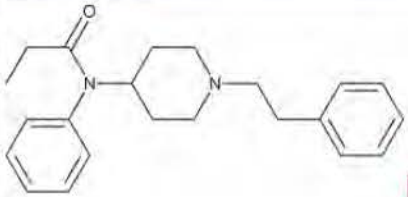
Chemical Security Analysis Center

Fentanyl

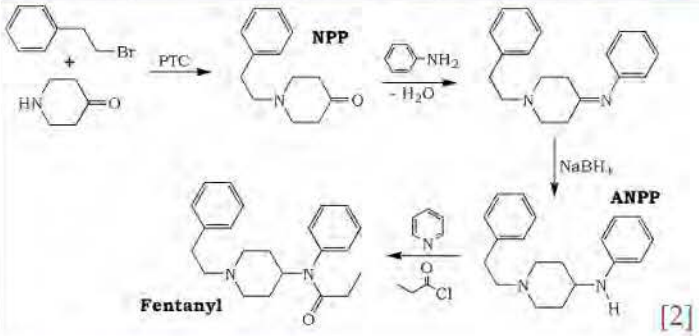
AGENT OVERVIEW

Fentanyl is an opioid analgesic, first synthesized in Belgium in the late 1950s, with an analgesic potency of about 80 times that of morphine. It was introduced into medical practice in the 1960s as an intravenous anesthetic.[1] Fentanyl is a very interesting component for underground chemistry because 1 gram of pure fentanyl is equivalent of 100 grams of very good street heroin.[2] Fentanyl is a Drug Enforcement Agency (DEA) controlled substance on Schedule II and illegal to possess without a valid DEA license or valid prescription.[3] Carfentanil is an analogue of fentanyl with an analgesic potency 10,000 times that of morphine and is used in veterinary practice to immobilize certain large animals.[4]

CHEMICAL IDENTIFICATION

Common chemical name	Fentanyl
CAS number	437-38-7[5]
IUPAC name	N-(1-(2-phenylethyl)-4-piperidinyl)-N-phenyl-propanamide[6]
Synonyms	China white; Duragesic; Fentora; Matrifen; Onsolis; Sentonil; Sublimaze[1][5]
Empirical formula	$C_{22}H_{28}N_2O$[6]
Chemical structure	 [6]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Illegal to purchase without DEA license or valid prescription[3] • Theft and divergence: Highly controlled substance[3] • Synthesis: Difficult but widely known; derivatives synthesized for the illicit drug market and called "China White"[3]
Synthetic pathways	• Precursors: Piperidone; phenethyl-tosylate[2] • Precursor availability: Common organic chemicals[7][8]
Synthesis	

PHYSICAL PROPERTIES

Physical form	Odorless white granular or crystalline powder[9][10]
Molecular weight	336.47 Daltons[11]
Liquid/solid density	1.11 g/mL (crystalline density)[12]
FP/MP	83-85 °C[12][13][14]
Boiling point	391 °C[14]
Viscosity	Not Found
Surface tension	Not Found
Vapor pressure	5.9 x 10^{-7} Pa @ 25 °C[14]
Volatility	Not Found
Vapor density (Air=1)	Not Found
Solubility	200 mg/L in water @ 25 °C; soluble in hexane[12]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Fentanyl is not expected to hydrolyze in water due to the lack of functional groups that hydrolyze under environmental conditions.[15]

Hydrolysis products	Not Found
Photo	Fentanyl does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.[15]
Thermal	Not Found

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[10] 
DOT identification number	UN 2811[10]	
DOT safety placard(s)	[10] 	[10] 

CLINICAL PRESENTATION

Time to effect	Almost immediate by injection, minutes by transdermal or oral route[16]
Exposure signs/symptoms	Symptoms of fentanyl overdose are characteristic of central nervous system depression and include lethargy and respiratory depression. Fentanyl is not detected by standard urine toxicology tests; therefore, its presence should not be excluded based on negative results. In fact, a characteristic response to the antidote naloxone (Narcan) with a negative toxicology screen is highly suggestive of fentanyl overdose.[17] Other signs of an overdose include cold, clammy skin; low blood pressure; pinpoint pupils of eyes; and slow heartbeat.[18]
Personal decontamination	Remove the patient/victim from the contaminated area and into the decontamination corridor. Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag. Thoroughly wash and rinse contaminated using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.[10]
Morbidity and mortality	LD_{50} (ingestion): The minimum lethal dose in humans has been estimated at 250 µg.[15]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Naloxone (Narcan)[17]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Analysis of biological samples by HPLC, GC, MS, and other methods[15][19] • Observation: Not found
Decontamination—property & equipment	Not found
Environmental persistence	Not found
Formulations	Skin: Transdermal patch[1][20] Oral: Dissolving tablets and lozenges[1][9] Intravenous: Fentanyl citrate sterile solution[16]

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PULMONARY AGENTS

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Ammonia, Anhydrous

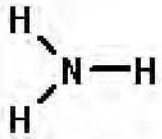
AGENT OVERVIEW

Ammonia is a high-volume chemical with production exceeding 1 million pounds annually in the U.S. Ammonia is used in at least 10 industries, and is used for building materials, furnishings, pesticide products, and others. It is widely used as a fertilizer and in chemical manufacturing. It is formed in the body during decomposition of organic materials during a large number of metabolically important reactions. Both ammonia liquid and vapors are extremely irritating, especially to the eyes. It is explosive when mixed with halogenated materials such as hypochlorites and can form nitrogen trichloride, which explodes spontaneously in air.[1][2]

During 2004, ammonia was produced by 16 companies at 32 plants in 19 states in the U.S. Fifty-four percent of total U.S. ammonia production capacity is centered in Louisiana, Oklahoma, and Texas because of their large reserves of natural gas, the dominant domestic feedstock.[3]

CHEMICAL IDENTIFICATION

Common chemical name	Ammonia
CAS number	7664-41-7[1]
IUPAC name	Azane[4]
Synonyms	AM-FOL; ammonia; ammonia (anhydrous); ammonia (anhydrous) (liquefied); ammonia gas; ammonia solution, with more than 50% ammonia; ammonia, [anhydrous]; ammonia, anhydrous, liquefied; ammonia-14N; ammoniac, anhydre (DOT French); ammoniac, anhydre, liquéfié (DOT French); ammoniac, solution, contenant plus de 50% d'ammoniac (DOT French); amoniaco, anhidro (DOT Spanish); amoniaco, anhidro, licuado (DOT Spanish); amoniaco, solución de, con más del 50% de amoniaco (DOT Spanish); anhydrous ammonia; anhydrous ammonia, liquefied; aqua ammonia; aqueous ammonia; liquid ammonia; Nitro-sil; R 717; refrigerant R717; spirit of hartshorn[5]

Empirical formula	NH_3[1]
Chemical structure	 [6]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Ammonia can be purchased in bulk quantities.[7] • Theft and divergence: Ammonia theft is common.[8] • Synthesis: Synthetic pathways well known.
Synthetic pathways	• Precursors: Atmospheric nitrogen; hydrogen source (e.g., methane, ethylene, or naphtha); carbon monoxide; hydrogen; carbon dioxide; natural gas; hot coke[9] • Precursor availability: Common chemicals
Synthesis	Ammonia is made by manufacture by a modified Haber reduction process using atmospheric nitrogen and a hydrogen source, for example, methane, ethylene or naphtha, at high temperature (400 to 600 ºC) and pressure (100 to 900 atm) in presence of an iron catalyst.[9][10] Alternatively, ammonia can be manufactured from water gas (obtained by blowing steam through incandescent coke) as a source of hydrogen, and from producer gas (obtained from steam and air through incandescent coke), as a source of nitrogen by the Haber-Bosch process.[9]

PHYSICAL PROPERTIES

Physical form	Colorless gas, or compressed liquefied gas; pungent odor[1]
Molecular weight	17.03 Daltons[1]
Liquid/solid density	Liquid: 682 g/L @ -33.5 °C; gas: 0.73 g/L @ 15 °C[6]
FP/MP	-78 °C (MP)[1]
Boiling point	-33 °C[1]
Viscosity	Gas: 9.8 x 10^{-3} cP[6]
Surface tension	Not found
Vapor pressure	888,000 Pa @ 21 °C[6]
Volatility	Not found
Vapor density (Air=1)	0.59[11]

Solubility	Very soluble in water: 33-34% @ 20 °C; 31-34% @ 25 °C; also soluble in absolute ethanol, methanol, chloroform, and ether[12]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Ammonia is a weak base, very soluble in water, and once dissolved it undergoes an acid-base equilibrium reaction with water: $NH_3 + H_2O. \Leftrightarrow NH_4^+ + OH^-$[13]
Hydrolysis products	$NH_4^+ + OH^-$[13]
Photo	$2HN_3 + h\nu \rightarrow H_2N_4 + H_2$[14]
Thermal	Thermal decomposition yields oxides of nitrogen.[15]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[16] 
DOT identification number	UN 1005 (anhydrous)[16]	
DOT safety placard(s)	[16] 	[16] 
		[16] 

CLINICAL PRESENTATION

Time to effect	Immediate for upper respiratory discomfort at concentrations above 21 mg/m^3; development of serious physiological changes from 5 minutes to 24 hours[12]
Exposure signs/symptoms	Inhalation: Respiratory tract irritation and burns, chest pain, sore throat, nausea, laryngitis, shortness of breath, pulmonary edema, and pneumonia. A pink frothy sputum, convulsions, and coma are often seen following exposure to high concentrations.[5] Skin: Severe skin burns and pain[5] Eye: Eye irritation, conjunctivitis (red, inflamed eyes), lacrimation (tearing), and corneal erosion may occur. Loss of vision is possible.[5] Ingestion: Vomiting, and oral, esophageal, and stomach burns[5]
Personal decontamination	Remove contaminated clothing and wash exposed area thoroughly with soap and water. In case of contact with substance, immediately flush skin or eyes with running water for at least 20 minutes. For minor skin contact, avoid

	spreading material on unaffected skin. Keep victim warm and quiet. Effects of exposure (inhalation, ingestion, or skin contact) to substance may be delayed. Ensure that medical personnel are aware of the material(s) involved, and take precautions to protect themselves.[9]
Morbidity and mortality	LCt_{50}: 336,000 mg-min/m^3[15]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Inhalation: Move victims to fresh air. Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support. Provide supportive care under medical supervision.[5] Skin/Eye: Remove contaminated clothing and contact lenses. Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory Do not attempt to neutralize with an acid wash; excessive liberation of heat may result. If eye exposure has occurred, eyes must IMMEDIATELY be flushed with lukewarm water for at least 15 minutes. Wash exposed skin areas THOROUGHLY with soap and water. Provide supportive care under medical supervision.[5] Ingestion: Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support. DO NOT induce vomiting or attempt to neutralize! Give the victims water or milk: children up to 1-year-old, 125 mL (4 oz or ½ cup); children 1 to 12 years old, 200 mL (6 oz or ¾ cup); adults, 250 mL (8 oz or 1 cup). Water or milk should be given only if victims are conscious and alert. Provide supportive care under medical supervision.[5]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: In general, standard air monitoring methods and technologies for ammonia involve pump-and-tube sampling approaches using short-term detector tubes with a variety of subsequent analytical approaches. Sampling is conducted by drawing a known volume of air through a solid sorbent tube using a personal sampling pump. The working concentration ranges for these methods are 0.14 to 280 mg/m^3 for a 10-L sample and 17 to 68 mg/m^3 for a 30-L sample, respectively. The current methodology used by the OSHA to determine ammonia in air (OSHA Method 188) consists of using a personal sampling pump to draw a known volume of air through a glass tube containing carbon beads impregnated with sulfuric acid. The reliable quantitative detection limit of the overall procedure is 1 mg/m^3 for a 24-L sample and 3.3 mg/m^3 for a 7.5-L sample. The time-integrated monitoring methods and technologies include filter packs and non-automatic annular denuder systems, and passive samplers. Continuous monitoring methods and technologies include:

	continuous-flow automated annular denuders; scrubber and sniffer methods; chemiluminescence NO_x monitors with an NH_3 converter automated thermodenuders; and advanced optical methods like DOAS, FTIR, PTD; and PF/LIF. Most of these instruments have been studied and validated in field experiments.[13] • Laboratory: Membrane mass spectrometry for aqueous samples[17]; in soils by ion chromatography[18]; in air by colorimetry[19] • Observation: Distinct ammonia odor is present. Additionally a white cloud of anhydrous ammonia may be visible.
Decontamination—property & equipment	Gaseous ammonia will disperse; any aqueous ammonia can be neutralized using commercial spill kits.[20]
Environmental persistence	Ammonia at natural concentrations in soil is not believed to have a very long half-life; the best estimate of the half-life of atmospheric ammonia is a few days. In low concentrations in water and soil, ammonia acts as a fertilizer to promote plant growth. Under aerobic conditions ammonia will oxidize to nitrate and does not accumulate in the environment.[12][21]
Formulations	Available as a gas or a compressed liquefied gas in various purities.[9]

The information cutoff date for this factsheet is 14 May 2010.

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Homeland Security
Science and Technology

Chemical Security Analysis Center

Chlorine

AGENT OVERVIEW

Chlorine is a greenish yellow diatomic gas. It is currently used as an industrial chemical in a variety of different industries, and it is readily available. It has been classified as a pulmonary agent, a lung injurer, a choking agent, and as a toxic industrial chemical. In April 1915, the Germans released 150 tons of chlorine in one of the first gas attacks of World War I. It is considered a potential hazard to both military and civilian populations.[1][2][3]

CHEMICAL IDENTIFICATION

Common chemical name	Chlorine[1]
CAS number	7782-50-5[1]
IUPAC name	Molecular chlorine[4]
Synonyms	Bertholite; chloor (Dutch); chlor (German); chlore (French); chlorine mol.; cloro (Italian); molecular chlorine; FO2100000[1]
Empirical formula	Cl_2[1]
Chemical structure	 [1]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Available in bulk quantities • Theft and divergence: Chlorine is transported in bulk quantities • Synthesis: Synthetic methods are readily available
Synthetic pathways	• Precursors: Sodium chloride; hydrogen chloride; sulfuric acid[2] • Precursor availability: Readily available
Synthesis	1. In the chlor-alkali electrolysis process, an aqueous solution of sodium chloride is decomposed electrolytically by direct current, producing chlorine, hydrogen, and sodium hydroxide solution.[2] 2. Chlorine (and co-product caustic soda) is made by the electrolysis of brines using mercury, membrane, or diaphragm cells.[2] 3. Chlorine is produced by the electrolysis of hydrochloric acid; oxidation of hydrogen chloride with nitrogen oxide as catalyst and absorption of steam with sulfuric acid ("KeloChlor" process).[2]

PHYSICAL PROPERTIES

Physical form	Greenish-yellow diatomic gas; disagreeable and suffocating odor[1]
Molecular weight	70.91 Daltons[1]
Liquid/solid density	1.393 g/mL @ 25 °C; 1.468 g/mL @ 0 °C (liquefied chlorine)[1]
FP/MP	-101.6 °C (FP)[1]
Boiling point	-34.7 °C[1]
Viscosity	14.0 Pa.sec @ 20 °C (gas); 340 Pa.sec @ 20 °C (liquid)[2]
Surface tension	18.4 dynes/cm @ 20 °C in contact with vapor[2]
Vapor pressure	5,750 mm Hg @ 25 °C; 2,730 mm Hg @ 0 °C[1]
Volatility	2.19 x 10^7 mg/m^3 @ 25 °C; 1.14 x10^7 mg/m^3 @ 0 °C[1]
Vapor density (Air=1)	2.4 (calculated)[1]
Solubility	0.63 g/100 g water @ 25 °C; solubility in carbon tetrachloride is 3.5% at ambient temperature[1]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	When chlorine gas (Cl_2) is dissolved in water, it rapidly undergoes an oxidation-reduction reaction (disproportionation) to form hypochlorous acid (HOCl) and chloride ion (Cl^-). This reaction is complete within seconds.[2]
Hydrolysis products	HCl and HOCl[1]
Photo	Chlorine is removed from air primarily by direct photolysis. At tropospheric

	wavelengths the chlorine molecule undergoes photodissociation, forming two chlorine radicals, which abstract a hydrogen atom from any available organic molecule to form hydrochloric acid. A lifetime of 7.3 hours was reported for the photolysis of chlorine, based on a measured rate constant of 2.3 x 10^3 s^{-1}.[2]
Thermal	Above 600 °C[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[5] 
DOT identification number	UN 1017[5]	
DOT safety placard(s)	[5] 	[5] 

CLINICAL PRESENTATION

Time to effect	The time to effect depends on the concentration and duration of exposure as well as the water content of the tissue involved and the presence of underlying cardiopulmonary disease (e.g., 430 ppm: lethal over 30 min; 1000 ppm: fatal within a few min).[2]
Exposure signs/symptoms	Inhalation: Burning of eyes, nose and mouth; lacrimation; rhinorrhea; loss of sense of smell; coughing, choking, and substernal pain; nausea; vomiting; headache; dizziness; syncope; pneumonia; hypoxemia. Pulmonary edema is common after severe exposure.[2][6] Ingestion: Vomiting may occur following initial exposure.[2][6] Skin: Dermal exposure may cause redness, pain, irritation, and cutaneous burns.[2][6] Eye: Inflammation of the eye; lacrimation; eye burns.[2][6]
Personal decontamination	Move to fresh air; rinse eyes and skin with plenty of water.[1][2][6]
Morbidity and mortality	• LCt_{50}: 9500 mg-min/m^3 (2-minute exposure)[7]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	Inhalation: Move patient to fresh air. Monitor for respiratory distress. If cough or difficulty breathing develops, evaluate for respiratory tract

	irritation, bronchitis, or pneumonitis. Administer oxygen and assist ventilation as required. Treat bronchospasm with beta2 agonist and corticosteroid aerosols.[6] Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. A physician should examine the area if irritation or pain persists.[6] Eye: Irrigate exposed eyes with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.[6] Ingestion: Seek medical assistance.[6]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Colorimetric; capillary column gas chromatography; gases/vapors by detector tubes[2] • Laboratory: Oxygen flask combustion method[2] • Observation: Not found
Decontamination—property & equipment	Property and equipment may not need decontamination, since chlorine is a gas at ambient temperatures. For runoff, keep material out of water sources and sewers. Attempt to stop leak if possible without undue personnel hazard. Do not apply water to point of leak in tank car or container. Apply water spray or mist to knock down vapors. Vapor knockdown water is corrosive or toxic and should be diked for containment.[6]
Environmental persistence	Chlorine is highly toxic to all forms of aquatic life; there is no potential for bioaccumulation or bioconcentration. Chlorine, as chlorine gas, chlorite ion, and hypochlorite, is a strong oxidant that readily reacts with organic molecules to produce a variety of chlorinated compounds. This reactivity in biological systems makes it difficult to study the pharmacokinetics of chlorine and to separate the effects of chlorine from those of the chlorine compounds and metabolites. These chlorine compounds have been shown to cause aquatic species environmental damage up to 35 days after exposure.[2]
Formulations	Semiconductor, high-purity (99.5%, liquid phase) grades; liquefied gas grade; research (min purity 99.99%), high purity (99.5%) grades.[2]

The information cutoff date for this factsheet is 14 May 2010.

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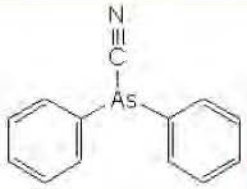
Diphenylcyanoarsine (DC)

AGENT OVERVIEW

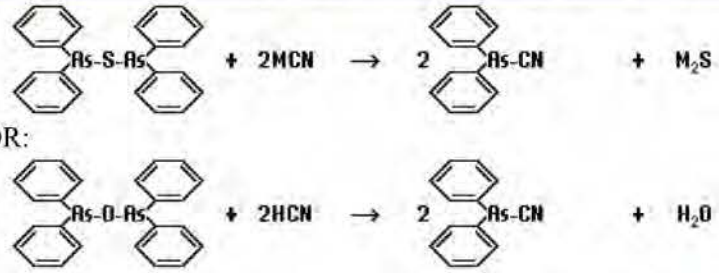
Diphenylcyanoarsine (DC) is a chemical warfare agent used as a "mask breaker" during WW I. Although previously called a vomiting agent, its primary action is irritation of the respiratory tract. Because its effect does not appear immediately but several minutes after exposure, personnel would not mask immediately, and by the time they masked a significant amount of compound would have been absorbed. Diphenylcyanoarsine's effects, such as chills, nausea, and vomiting, last for several hours after exposure. This can cause an individual to unmask, risking serious illness or death.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Diphenylcyanoarsine
CAS number	23525-22-6[1]
IUPAC name	Diphenylarsanylformonitrile[2]
Synonyms	DC; CLARK II; Clark 2; diphenylarsenous cyanide; diphenylarsinecarbonitrile; diphenylarsinous cyanide; arsinous cyanide, diphenyl; arsinecarbonitrie, diphenyl-; diphenylarsinous cyanide; blue cross; sternit[1]
Empirical formula	$C_{13}H_{10}AsN$[1]

Chemical structure	 [1]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Not available from major suppliers • Theft and divergence: Low risk since not widely used • Synthesis: Not difficult
Synthetic pathways	• Precursors: Tetraphenyl diarsine sulfide; heavy metal cyanide; tetraphenyl diarsine oxide; hydrogen cyanide[3] • Precursor availability: Not available from major suppliers
Synthesis	 OR: [3]

PHYSICAL PROPERTIES

Physical form	Colorless crystals if pure; gray to dark-brown liquid if impure[4]; odor similar to garlic or bitter almonds[1]
Molecular weight	255.15 Daltons[1]
Liquid/solid density	1.3338 g/mL @ 35 °C[1]
FP/MP	31.2 °C[1]
Boiling point	341 °C (extrapolated) (decomposes)[1]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	0.00072 mm Hg @ 35 °C (extrapolated)[1]
Volatility	9.56 mg/m^3 @ 35 °C (calculated from vapor pressure)[1]
Vapor density (Air=1)	8.8 (calculated)[1]
Solubility	0.021 g/L (37 °C) in water[5]; soluble in chloroform and other organic solvents[4]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Very slow[1]
Hydrolysis products	Hydrogen cyanide and diphenylarsenious oxide[1]
Photo	Not found
Thermal	Above 240 °C[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health	Not found	
	Flammability		
	Reactivity		
	Special Properties		
DOT identification number	UN 2810[6]		
DOT safety placard(s)	[6] 		

CLINICAL PRESENTATION

Time to effect	Rapid after high doses; several minutes after lower doses[1]
Exposure signs/symptoms	Inhalation: Causes irritation of nose and throat, salivation, and profuse secretion from the eyes and nose (symptoms last for 30 minutes to 1 hour); a feeling of suffocation and headache may last for several hours.[1] Skin: Contact with molten substance may cause severe burns.[6] Eye: Causes profuse secretion from the eyes (symptoms last for 30 min to 1 hr)[1]
Personal decontamination	If symptoms persist, the eyes, mouth and skin may be washed with water. Do not swallow the water.[1]
Morbidity and mortality	LCt_{50}: 10,000 mg-min/m^3[7] ECt_{50} (incapacitating): 30 mg-min/m^3[7] ECt_{50} (mild): 0.01 µg/L (time not specified)[4]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Ingestion: Seek medical assistance.[6]

	Inhalation: Move victim to fresh air. Apply artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; induce artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Administer oxygen if breathing is difficult.[6] Skin: Remove and isolate contaminated clothing and shoes. Immediately flush skin with running water for at least 20 min. For minor skin contact, avoid spreading material on unaffected skin.[6] Eye: Immediately flush with running water for at least 20 min.[6]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: Thin-layer chromatography[8] • Observation: None
Decontamination—property & equipment	None required outdoors. Use alkali solution or DS2 for decontamination in enclosed places.[7]
Environmental persistence	Not found
Formulations	Not found

The information cutoff date for this factsheet is 14 May 2010.

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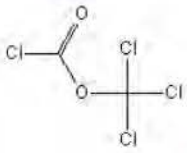
Diphosgene (DP)

AGENT OVERVIEW

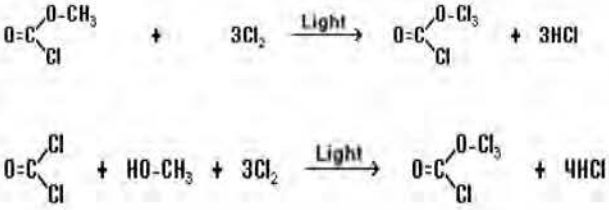
Diphosgene was produced after WW I and was intended as an improved form of phosgene. The gas mask filters that had been developed during the war to protect against phosgene were found to be ineffective against diphosgene. This was so because when diphosgene decomposed to phosgene and chloroform, the chloroform destroyed the filters, allowing the phosgene to pass through. In peacetime, diphosgene is used as a safer substitute for phosgene in synthesis.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Diphosgene
CAS number	503-38-8[2]
IUPAC name	Trichloromethyl carbonochloridate[3]
Synonyms	DP; difosgene; superpalite (British); perstoff (German); surpalite(French); green cross (German); trichloromethyl chloroformate; trichloromethyl chlorocarbonic acid ester; chloroformic acid trichloromethyl ester; trimchloromethyl chlorocarbonate; formic acid, chloro-, trichloromethyl ester; LQ7350000; carbonochloridic acid, trichloromethyl ester; difosgen; diphosgen; methanol, trichloro-, chloroformate; trichlormethylester kyseliny chlormravenci[2][4]
Empirical formula	$C_2Cl_4O_2$[2]

Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Sold by Alfa Chem[4] • Theft and divergence: Not found • Synthesis: Prepared by photochlorination of methyl formate[5]
Synthetic pathways	• Precursors: Methyl formate; methyl chloroformate[5] • Precursor availability: Commercially available
Synthesis	 [1]

PHYSICAL PROPERTIES

Physical form	Colorless oily liquid, with odor of musty hay[2]
Molecular weight	197.83 Daltons[2]
Liquid/solid density	1.656 g/mL (20 °C); 1.687 g/mL (0 °C)[2]
FP/MP	-57 °C[2]
Boiling point	127 °C[2]
Viscosity	Not found
Surface tension	Not found
Vapor pressure	4.41 mm Hg (20 °C); 0.0914 mm Hg (0 °C)[2]
Volatility	47,700 mg/m^3 (20 °C); 10,600 mg/m^3 (0 °C) (calculated from vapor pressure)[2]
Vapor density (Air=1)	6.8 (calculated)[2]
Solubility	Solubility in water is 44.6 g/L at 20°C[2]; soluble in ethanol, ether, benzene, alcohol.[5]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Slow at ambient temperature and fairly rapid at 100 °C[2]
Hydrolysis products	Hydrogen chloride and carbon dioxide[2]
Photo	Not found
Thermal	300-350 °C (yields two molecules of phosgene)[2]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [6]	
DOT identification number	UN 1076[6]		
DOT safety placard(s)	 [6]		

CLINICAL PRESENTATION

Time to effect	Although immediate symptoms may follow exposure to a high concentration of DP, a delay of 3 hours or more may elapse before exposure to a low concentration causes any ill effects.[2][7]
Exposure signs/symptoms	Inhalation: Causes chemical burns to the respiratory tract. Inhalation may produce coughing, nausea, and pulmonary edema. In rare instances, exposure may cause sensitization, resulting in inflammation of the mucous membranes and in eczematous eruptions.[6] Ingestion: May cause severe and permanent damage to the digestive tract.[6] Skin: Causes skin burns; may cause skin sensitization[6] Eye: Causes tearing and eye burns[6]
Personal decontamination	Immediately flush skin with plenty of soap and water for at least 15 minutes while removing contaminated clothing and shoes.[6]
Morbidity and mortality	LCt_{50}: 1500 mg-min/m^3 (Provisional)[2]

COUNTERMEASURES

Prophylaxis	None
Antidote/Treatment	Ingestion: Do not induce vomiting. If victim is conscious and alert, give 2-4

	cupfuls of milk or water.[6] Inhalation: Move to fresh air immediately. If not breathing, give artificial respiration. If breathing is difficult, give oxygen. Do not use mouth-to-mouth respiration.[6] Skin: Immediately flush skin with plenty of soap and water for at least 15 minutes while removing contaminated clothing and shoes.[6] Eye: Do not allow victim to rub or keep eyes closed. Extensive irrigation is required (at least 30 minutes).[6]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: MM1[2] • Laboratory: The quantitative analysis of chloroformic esters can be carried out by titration, gas chromatography, or high-performance liquid chromatography (HPLC).[5] • Observation: None
Decontamination—property & equipment	Live steam, ammonia, and aeration for confined spaces[5]
Environmental persistence	Decontamination not required in field except in very cold climates[2]
Formulations	Technical: 99% purity; commercial: 97-99 wt%[5]

The information cutoff date for this factsheet is 14 May 2010.

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Hydrogen Chloride (HCl)

AGENT OVERVIEW

Hydrogen chloride (HCl) is a colorless, corrosive gas with a pungent, suffocating odor. It is highly soluble in water, forming hydrochloric acid. Hydrogen chloride exists in gaseous and aerosol forms in the atmosphere, and the partitioning between the two forms is a function of the temperature and relative humidity. Lower temperature and higher relative humidity favor the formation of aerosol; higher temperature and lower relative humidity promote HCl in gaseous form. Hydrogen chloride dissociates in water forming hydronium ions (H_3O^+) that damage tissue. The predominant effects of inhalational HCl are due to contact with the local tissue, particularly in the upper respiratory tract. As a result of its high reactivity with the upper respiratory tract, it is unlikely that HCl would cause systemic toxicity.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Hydrogen chloride
CAS number	7647-01-0[2]
IUPAC name	Hydrogen chloride[3]
Synonyms	Acide chlorhydrique; acido cloridrico; anhydrous hydrochloric acid; anhydrous hydrogen chloride; basilin; chloorwaterstof; chlorohydric acid; chlorowodor; chlorwasserstoff; dilute hydrochloric acid; HCL; hydrochloric acid gas; hydrochloric acid mixture; hydrochloric acid, anhydrous; hydrochloride; hydrogen chloride (acid); hydrogen-chloride-anhydrous-; marine acid; muriatic acid; NA 1789; salzsaeure; soldering acid; spirit of salts; spirits of salt[2][4]
Empirical formula	HCl

Chemical structure	

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Hydrogen chloride is available for purchase. • Theft and divergence: Possible • Synthesis: Not difficult
Synthetic pathways	• Precursors: Sodium chloride; potassium chloride; sulfuric acid; sodium bisulfite; sulfur dioxide; oxygen; chlorine; hydrogen gas[2] • Precursor availability: Common chemicals
Synthesis	Reaction of salt and sulfuric acid; reaction of sodium bisulfite with sodium chloride; reaction of salt, sulfur dioxide, oxygen, and water (Hargraves process); or by burning of chlorine with hydrogen gas.[2]

PHYSICAL PROPERTIES

Physical form	Colorless gas with characteristic pungent odor[5]
Molecular weight	36.46 Daltons[6]
Liquid/solid density	1.639 g/L[5]
FP/MP	-114.2 °C[5]
Boiling point	108.58 °C in 78% water[2]; -85 °C[5][6]
Viscosity	0.405 cP (liquid, -115 °C); 0.0131 cP (vapor, 0 °C); 0.0253 cP (vapor, 250 °C)[2]
Surface tension	23 mN/cm @ -155 °C[2]
Vapor pressure	35,400 mm Hg @ 25 °C[2]
Volatility	Not found
Vapor density (Air=1)	1.268[5]
Solubility	Highly soluble in water: 823 g/L @ 0 °C; 673 g/L @ 30 °C[5]; soluble in ethanol, methanol, and ether[5]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Forms hydrochloric acid in water[2]
Hydrolysis products	Hydronium ion and chloride ion[2]

Photo	Indirect photolysis through reaction with hydroxyl radical with half-life of 11 days[3]
Thermal	Hydrochloric acid decomposes at 1782 °C[2]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[3][6] 
DOT identification number	UN 2186[6]	
DOT safety placard(s)	[6] 	[6] 

CLINICAL PRESENTATION

Time to effect	Immediately irritating when inhaled at 5 ppm or more[2]; effects may be delayed with lower doses[6]
Exposure signs/symptoms	Shock, rapid breathing and pulse, circulatory collapse, and other changes to pulse, blood pressure, and respirations[6] Inhalation: Changes in breathing patterns, irritation, changes in pulmonary function, corrosion and edema of the respiratory tract, chronic bronchitis, and non-cardiogenic pulmonary edema[6] Ingestion: Gastritis, burns, gastric hemorrhage, dilation, edema, necrosis, and strictures[6] Skin: Burns, ulceration, scarring, blanching, and irritation[6] Eye: corneal necrosis, inflammation, irritation[6]
Personal decontamination	Remove contaminated clothing and shoes immediately. Wash affected area with soap and large amounts of water for at least 15 to 20 minutes. In case of chemical burns, cover area with sterile, dry dressing. Keep affected areas cool.[2][6]
Morbidity and mortality	• LCt_{50}: 280,000 mg-min/m^3[7] • ECt_{50} (severe): 10,600 mg-min/m^3[7]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	Inhalation: Move victim to fresh air; keep warm and quiet; start artificial

	respiration if breathing stops. Ingestion: Have person drink water or milk; do NOT induce vomiting. Skin: Remove and isolate contaminated clothing and shoes. Immediately flush with running water for at least 20 minutes. For minor skin contact, avoid spreading material on unaffected skin. Eyes: Irrigate with copious amounts of tepid water for at least 15 minutes. If irritation, pain, swelling, lacrimation, or photophobia persist, the patient should be seen in a health care facility.[6]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Ion chromatography[2] • Observation: Not found
Decontamination—property & equipment	Absorb bulk liquid with fly ash or cement powder. Neutralize with agricultural lime, crushed limestone, or sodium bicarbonate.[6]
Environmental persistence	In air, hydrogen chloride will be removed by rainfall, with an atmospheric lifetime of 1-5 days. In water, hydrogen chloride dissociates readily to chloride and hydronium ions, decreasing the pH of the water. In soil, hydrogen chloride will evaporate from dry soil surfaces and dissociate into chloride and hydronium ions in moist soil.[2]
Formulations	United States Pharmacopeia (35-38%); national formulary diluted (10%); technical usually 28, 31, 35, 37 % HCl; Technical: 97.5-999% purity.[2]

The information cutoff date for this factsheet is 14 May 2010.

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Hydrogen Fluoride, Anhydrous (HF)

AGENT OVERVIEW

Anhydrous hydrogen fluoride (HF) is a colorless fuming liquid (below about 20 °C) or a colorless gas with a pungent, irritating odor. It is used as a chemical intermediate and catalyst in the production of most fluorine-containing chemicals. It is also used in pickling, etching and mineral purification [1]. HF is one of the most acidic substances known [2]. Very short contact with fumes or small quantities of the liquid can cause severe, painful burns.[3][4][5]

CHEMICAL IDENTIFICATION

Common chemical name	Hydrogen fluoride, anhydrous
CAS number	7664-39-3[4]
IUPAC name	Hydrogen fluoride[6]
Synonyms	Alsurf 45; anhydrous hydrofluoric acid; anhydrous hydrogen fluoride; antisal 2B; fluorhydric acid; fluoric acid; fluorine hydride; fluorine monohydride; HF-A; hydrofluoric acid; hydrofluoric acid gas; hydrofluoric acid, anhydrous; hydrogen fluoride; hydrogen fluoride, anhydrous; hydrogen monofluoride[4]
Empirical formula	HF[4]

Chemical structure	

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: HF can be purchased. Available commercially as a 70% solution in steel containers or as a 50% solution in plastic lined containers.[2] • Theft and divergence: Railcar and aircraft shipping is forbidden.[2] • Synthesis: well-known
Synthetic pathways	• Precursors: Sulfuric acid; calcium fluoride • Precursor availability: Readily available materials
Synthesis	Hydrogen fluoride is generally derived from the reaction of concentrated sulphuric acid on fluospar (CaF_2)[5]; the industrial processes for the manufacture of anhydrous hydrofluoric acid are all based on the action of sulfuric acid on fluorite.[2]

PHYSICAL PROPERTIES

Physical form	Colorless gas or fuming liquid, depending on temperature, with strong, irritating odor.[2][5]
Molecular weight	20.01 Daltons[7]
Liquid/solid density	0.987 g/mL @ 20 °C[7]
FP/MP	-83.1 °C[7]
Boiling point	19.51 °C[7]
Viscosity	0.256 cP @ 0 °C[2]
Surface tension	10.2 mN/m @ 0 °C[2]
Vapor pressure	400 mm Hg @ 2.5 °C[7]
Volatility	Not found
Vapor density (Air=1)	0.92[7]
Solubility	Very soluble in water, in alcohol, and in most organic solvents; slightly soluble in ether[7]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Dissociates to H^+ and F^- in water

Hydrolysis products	Fluorine and hydrogen ions
Photo	Not found
Thermal	When heated to decomposition it emits highly corrosive fumes of hydrogen fluoride.[2]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[8] 
DOT identification number	UN 1052[8]	
DOT safety placard(s)	[8] 	[8] 

CLINICAL PRESENTATION

Time to effect	Immediate to delayed effects depending on exposure time and dosage. Effects may be delayed up to 24 hours.[4]
Exposure signs/symptoms	Inhalation: Dyspnea, bronchospasm, chemical pneumonitis, pulmonary edema, chemical burns of the respiratory tract; nausea, vomiting, diarrhea, circulatory collapse[4] Skin: Contact with gas or liquefied gas may cause burns, severe injury, or frostbite.[4] It may be absorbed after dermal contact and can result in life-threatening conditions Absorption of the fluoride ion is a significant hazard mainly due to hypocalcaemia.[5] Eye: Can cause permanent blindness or eye injury. Symptoms include stinging, tearing, redness, and swelling of eyes.[4]
Personal decontamination	Flush with copious amounts of water immediately. This should be done even in an asymptomatic patient, and prior to transport to a medical facility if possible. Quickly remove and double-bag contaminated clothing while flushing exposed skin and hair with plain water or saline for at least 20-30 minutes. Cover exposed skin with a calcium-containing slurry or gel (2.5 g calcium gluconate K-Y Jelly gel).[2][5]
Morbidity and mortality	• LCt_{50}: 12,800 mg-min^3 at 2 minutes (military; calculated)[9] • ECt_{50} (severe): 2,000 mg-min^3 at 2 minutes (military; calculated)[9]

COUNTERMEASURES

Prophylaxis	
Antidote/Treatment	**Antidote** Hydrogen fluoride Burn Gel (2.5% calcium gluconate paste; not approved in the U.S.)[5] **Treatment** Ingestion: Evaluate vital signs including pulse and respiratory rate, and note any trauma. If no pulse is detected, provide CPR. If not breathing, provide artificial respiration. If breathing is labored, administer oxygen or other respiratory support. Humidified oxygen is preferred. Immediately give the victims milk or water to dilute the hydrofluoric acid.[4] Inhalation: Move victim to fresh air. Apply artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; induce artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Administer oxygen if breathing is difficult. Effects may be delayed.[4] Skin: Remove all exposed clothing and jewelry taking necessary precautions to prevent secondary exposure to health care providers. Irrigate exposed areas promptly with copious amounts of water for at least 30 minutes. Wash the skin, including hair and nails, vigorously; do repeated soap washings. Discard contaminated clothing.[4] Eye: In case of contact with liquefied gas, thaw frosted parts with lukewarm water. Immediately flush skin with running water for at least 20 minutes.[4]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Fluoride exposure can be confirmed by the determination of fluoride in the urine using a random spot urine collection.[5] • Observation: Not found
Decontamination—property & equipment	Absorb bulk liquid with fly ash or cement powder. Neutralize with agricultural lime, crushed limestone or sodium bicarbonate.[2]
Environmental persistence	Hydrogen fluoride is removed from air by wet deposition as fluoride salts with an atmospheric lifetime of 1-5 days.[2]
Formulations	Available as a gas at 99.9% purity.[2]

The information cutoff date for this factsheet is 14 May 2010.

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Hydrogen Sulfide (H_2S)

AGENT OVERVIEW

Hydrogen sulfide (H_2S) is a colorless gas having a strong odor of rotten eggs. It is shipped as a liquid confined under its own vapor pressure. Contact with the unconfined liquid can cause frostbite by evaporative cooling. Gas is very toxic by inhalation. H_2S fatigues the sense of smell, which cannot be counted on to warn of the continued presence of the gas.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Hydrogen sulfide
CAS number	7783-06-4[1]
IUPAC name	hydrogen sulfide[2]
Synonyms	Acide sulfhydrique; dihydrogen monosulfide; dihydrogen sulfide; hepatic acid; hepatic gas; hydrogen monosulfide; hydrogene sulfure; hydrogen-sulphide-; hydrosulfuric acid; idrogeno solforato; Schwefelwasserstoff; sewer gas; siarkowodor; sour gas; stink damp; sulfur hydride; sulfur hydroxide; sulfureted hydrogen; sulfuretted hydrogen; zwavelwaterstof[3]
Empirical formula	H_2S
Chemical structure	SH_2

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Commercially available • Theft and divergence: Shipped as a liquefied compressed gas[4] • Synthesis: (1) By the action of dilute sulfuric acid on a sulfide, usually iron sulfide; (2) by direct union of hydrogen and sulfur vapor at a finite temperature and pressure; or (3) as a by-product of petroleum refining.[4]
Synthetic pathways	• Precursors: Hydrogen chloride; calcium sulfide; iron sulfide; zinc sulfide; sulfuric acid[4][5] • Precursor availability: can be purchased commercially
Synthesis	Pathway 1: $2HCl(aq) + [Metal]S(s) = H_2S(g) + [Metal]Cl_2(aq)$ Pathway 2: $H_2SO_4(aq) + CaS(s) = H_2S(g) + CaSO_4(aq)$[4][5]

PHYSICAL PROPERTIES

Physical form	Colorless gas (Shipped as a liquefied compressed gas)[1] Odor: rotten egg[1]; sweet taste[4]
Molecular weight	34.08 Daltons[1]
Liquid/solid density	1.539 g/L @ 0°C[4]
FP/MP	-85.49 °C (MP)[4]
Boiling point	-60.33 °C[4]
Viscosity	0.012 cP @ 0 °C[4][6]
Surface tension	Not found
Vapor pressure	13,651 mm Hg @ 21 °C[6]; 15,200 mm Hg @ 25 °C[7]
Volatility	Not found
Vapor density (Air=1)	1.19[1]
Solubility	Water: 5.3 g/L @ 10 °C; 4.1 g/L @ 20 °C; 3.2 g/L @ 30 °C[8]; soluble in glycerol, gasoline, kerosene, carbon disulfide, crude oil[8]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	Water solutions of hydrogen sulfide are not stable; absorbed oxygen causes the formation of elemental sulfur and the solutions become turbid rapidly.[4]
Hydrolysis products	Sulfur[4]
Photo	H_2S does not undergo photolysis or react photochemically with oxygen.[4]
Thermal	When heated to decomposition it emits highly toxic fumes of sulfur oxides.[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [9]
DOT identification number	UN 1053[9]	
DOT safety placard(s)	 [9]	 [9]

CLINICAL PRESENTATION

Time to effect	Depends on concentration and exposure time. Exposure to greater than 500 ppm results in severe toxicity and death. Respiratory paralysis and death may be noted within 30 to 60 minutes. At 800 to 1000 ppm, death may be nearly immediate after 1 or more breaths.[4]
Exposure signs/symptoms	Inhalation: Respiratory depression, cyanosis, pulmonary edema, bronchitis, and dyspnea may be noted following exposure to non-fatal concentrations. Exposure to high concentrations will result in rapid respiratory paralysis leading to sudden collapse.[1] Skin: Skin exposure may result in severe pain, itching, and erythema, especially in moist areas. Cyanosis may be noted following severe exposure.[1][4] Eye: Irritation, lacrimation, painful conjunctivitis; injection of the mucous membrane that lines the inner surface of the eyelid and the exposed surface of the eyeball, corneal bulla, blurred vision and blepharospasm may be noted following exposure to 150 to 300 ppm.[1][4]
Personal decontamination	Remove contaminated clothing and wash exposed area thoroughly with soap and water. Seek medical assistance if irritation or pain persists.[1]
Morbidity and mortality	• LC_{50}: 700- 7000 mg/m^3[10] • EC_{50} (severe): 350 mg/m^3 (for pulmonary edema)[10] • EC_{50} (mild): 70 mg/m^3 (for irritation of eyes and respiratory tract)[10]

COUNTERMEASURES

Prophylaxis	Wear appropriate personal protective clothing, and approved respiratory and eye protection to prevent skin from becoming frozen from contact with the liquid or from contact with vessels containing the liquid.[1]

Antidote/Treatment	**Antidote** Nitrites may be used but are effective only if given within the first few minutes after exposure. This antidote treatment's effectiveness is open to controversy.[4] **Treatment** Inhalation: Immediately move victim to fresh air and administer 100% oxygen. Skin: Remove contaminated clothing and wash exposed area thoroughly with soap and water. Seek medical assistance if irritation or pain persists.[1]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: Not found • Laboratory: Electrolytic conductivity detector; gas chromatography flame photometric; spectrophotometry; colorimetric[4] • Observation: Not found
Decontamination—property & equipment	None usually needed, since H_2S is a gas and will dissipate by itself.
Environmental persistence	Atmosphere: Once released into the atmosphere, hydrogen sulfide will behave like many other gaseous pollutants and be dispersed and eventually removed. Residence times in the atmosphere range from about one day to more than 40 days, depending upon season, latitude, and atmospheric conditions. Soil: Anhydrous hydrogen sulfide has a boiling point of 60.3 °C at 1 atm. Consequently, when it is spilled onto soil, much will evaporate. However, since it is very soluble in water, the presence of water in soil or falling as precipitation at the time of the spill may contribute to movement in the soil. If the soil surface is saturated with moisture at the time of the spill as might be the case after a rainfall, the spill chemical will runoff and/or evaporate away.[4]
Formulations	Grades: Technical, 98.5%; purified, 99.5% min[4]

The information cutoff date for this factsheet is 14 May 2010.

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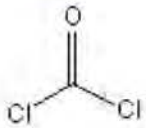
Phosgene (CG)

AGENT OVERVIEW

Phosgene was first used in the dye industry in the late 19th century by John Davy to process colorfast materials. In 1915, the Germans introduced phosgene in WW I. It is classified as a pulmonary agent that attacks lung tissue, primarily causing pulmonary edema. Vapors can linger for some time in low-lying areas under calm or light winds. Any activity or stress after exposure is likely to exacerbate the effects and turn a sublethal exposure into a lethal exposure.[1][2]

CHEMICAL IDENTIFICATION

Common chemical name	Phosgene
CAS number	75-44-5[1]
IUPAC name	Carbonyl chloride[3]
Synonyms	Carbon dichloride oxide; carbon oxychloride; carbone (oxychlorure de) (French); carbonic chloride; carbonic dichloride; carbonio (Italian); carbonio (ossiclorurdi) (Italian); carbonyl dichloride; carbonylchlorid (German); chloroformy chloride; chloroformyl chloride; collongite (French); D-gas (German); fosgeen (Dutch); fosgen (Polish); fosgene (Italian); green cross (German); koolstofoxychloride (Dutch); NCI-C60219; phosgen (German); SY 5600000; zusatz (German)[1][3]
Empirical formula	$COCl_2$[1]

Chemical structure	 [1]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: One company sells phosgene on the U.S. merchant market.[4] • Theft and divergence: Railcar and cargo aircraft shipping is forbidden.[4] • Synthesis: First prepared in 1812 by the photochemical reaction of carbon monoxide and chlorine; now commercially prepared by passing chlorine and excess carbon monoxide over activated carbon.[4]
Synthetic pathways	• Precursors: Carbon monoxide; chlorine[4] • Precursor availability: Readily available
Synthesis	$CO + Cl_2 \rightarrow COCl_2$[4]

PHYSICAL PROPERTIES

Physical form	Colorless gas that is readily liquefied; odor of musty hay or rotting fruit[1]
Molecular weight	98.92 Daltons[1]
Liquid/solid density	(Liquefied phosgene) 1.360 g/mL (25 °C), 1.402 g/mL (7.8 °C), 1.420 g/mL (0 °C)[1]
FP/MP	-128 °C[1]
Boiling point	7.8 °C[1]
Viscosity	6.84×10^{-4} Pa @ -10 °C[5]
Surface tension	34.6 mN/m at 0 °C; 20.1 mN/m at 16.7 °C; 17.6 mN/m at 34.5 °C; 15.9 mN/m at 46.1 °C[4]
Vapor pressure	1400 mm Hg (25 °C), 760 mm Hg (7.8 °C), 560 mm Hg (0 °C)[1]
Volatility	7.46×10^6 mg/m^3 (25 °C); 4.29×10^6 mg/m^3 (7.8 °C); 3.53×10^6 mg/m^3 (0 °C) (calculated from vapor pressure)[1]
Vapor density (Air=1)	3.4 (calculated)[1]
Solubility	Limited in water. Miscible with common organic solvents, petroleum, and lubricating oil.[1] Soluble in benzene, carbon tetrachloride, chloroform, toluene, and acetic acid.[4]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	$t_{½}$ = 0.25 sec at 13 °C; Does not react quickly with water in the vapor phase but it immediately reacts with liquid water.[1]

Hydrolysis products	Hydrochloric acid and carbon dioxide.[1]
Photo	Gas-phase phosgene will be slowly degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 44 years. Phosgene does not absorb at wavelengths > 290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.[4]
Thermal	800 °C (complete)[1]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	[6] 
DOT identification number	UN 1076[6]	
DOT safety placard(s)	[7] 	[7] 

CLINICAL PRESENTATION

Time to effect	Immediate to three hours. The severity of poisoning cannot be estimated from the immediate symptoms, and the full extent can be delayed up to 72 hours.[1]
Exposure signs/symptoms	Inhalation: Irritation to bronchi, trachea, pharynx, larynx, and nose. Causes pulmonary edema. Also causes tearing, dry throat, coughing, choking, dyspnea, tightness of chest, nausea, vomiting and headache.[1][2][3][4] Skin: Severe dermal burns or frostbite may develop following skin exposure to the liquefied material.[8] Eye: Severe burns or frostbite may develop following exposure to the liquefied material.[8]
Personal decontamination	Not required in field except in very cold climates[1]
Morbidity and mortality	• LCt_{50}: 1500 mg-min/m^3 (2-60 min exposure duration)[1], 2050 mg-min/m^3[4] • ECt_{50} (severe): 615 mg-min/m^3[4] • ECt_{50} (mild): 12.3 mg-min/m^3[4]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	Inhalation: Move victim to fresh air. Apply artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; induce artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Administer oxygen if breathing is difficult. Effects may be delayed.[8] Skin: Remove and isolate contaminated clothing and shoes. In case of contact with liquefied gas, thaw frosted parts with lukewarm water. Immediately flush skin with running water for at least 20 minutes. Effects may be delayed.[8] Eyes: In case of contact with liquefied gas, flush with running water for at least 20 minutes.[8]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: M182A2 CADK; MM1[1] • Laboratory: Gas chromatography, infrared, colorimetric techniques.[4] • Observation: None
Decontamination—property & equipment	Dike surface flow using soil, sand bags, foamed polyurethane, or foamed concrete. Neutralize with agricultural lime, crushed limestone or sodium bicarbonate. Dilute slowly with water. Water spill: Neutralize with agricultural lime, crushed limestone, or sodium bicarbonate. A solution of detergent and water (pH 8-10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.[8]
Environmental persistence	Gas-phase phosgene will be very slowly degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 44 years. Phosgene has a very short half-life (0.026 seconds) in aqueous solutions.[4]
Formulations	99% min. purity, liquefied gas[4]

The information cutoff date for this factsheet is 14 May 2010.

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Sulfur Dioxide (SO_2)

AGENT OVERVIEW

Sulfur dioxide (SO_2) is a colorless gas at ambient temperature and pressure. It is a water-soluble irritant gas, and is absorbed predominantly in the upper airways. As a primary component in reducing-type air pollution and occupational exposures it has been extensively studied. There are many well conducted human exposure trials, which include the targeted sensitive population of asthmatics. It is a by-product of ore smelting, coal and fuel-oil combustion, paper manufacturing, and petroleum refining.[1]

CHEMICAL IDENTIFICATION

Common chemical name	Sulfur dioxide
CAS number	7446-09-5[2]
IUPAC name	Sulfur dioxide[3]
Synonyms	SO_2; Fermenicide powder; Fermenicide liquid; sulfur oxide; sulfurous acid anhydride; sulfurous anhydride; sulfurous oxide; sulphur dioxide; schwefeldioxyd; siarki dwutlenek; oxosulfane oxide[2]
Empirical formula	SO_2
Chemical structure	 [2]

CHEMICAL AVAILABILITY

Procurement methods	• Purchase: Available commercially • Theft and divergence: Possible • Synthesis: Uncomplicated
Synthetic pathways	• Precursors: Pyrites; sulfur; sulfur trioxide; oleum[4] • Precursor availability: Common chemicals
Synthesis	Roasting pyrites in a furnace; purifying from smelting operations; burning sulfur; reaction of sulfur trioxide from oleum with sulfur[4]

PHYSICAL PROPERTIES

Physical form	Colorless gas; strong, suffocating, pungent odor[4]
Molecular weight	64.065 Daltons[2]
Liquid/solid density	2.811 g/L[4]
FP/MP	-75.5 °C[4]
Boiling point	-10.05 °C[4]
Viscosity	0.0124 mPa.s @ 18 °C (gas); 0.368 mPa.s @ 0 °C (liquid)[4]
Surface tension	28.59 mN/m (liquid, 10 °C)[4]
Vapor pressure	3.2 atm @ 20 °C[4]
Volatility	Not found
Vapor density (Air=1)	2.263 @ 0 °C[4]
Solubility	11.9% in water @ 15°C; 8.5% in water @ 25°C[4]; 25% in alcohol, 32% in methanol[4]; soluble in chloroform, ether, acetic acid, sulfuric acid[4]

DECOMPOSITION PROCESSES

Aqueous hydrolysis	With water, forms sulfurous acid (H_2SO_3)[5]
Hydrolysis products	Sulfurous acid[5]
Photo	Direct photooxidation of sulfur dioxide is minimal.[4]
Thermal	Mixed with oxygen and passed over red-hot platinum, it is converted into SO_3.[5]

TRANSPORTATION SAFETY WARNINGS

NFPA 704 diamond	Health Flammability Reactivity Special Properties	 [6]
DOT identification number	UN 1079[6]	
DOT safety placard(s)	 [6]	 [6]

CLINICAL PRESENTATION

Time to effect	• Inhalation: Immediate[4] • Skin: Immediate[4] • Ingestion: Not found
Exposure signs/symptoms	Severe irritation of skin, eyes, and respiratory tract; choking, coughing, sneezing, rhinorrhea, wheezing, dyspnea, cyanosis, chest pain, tracheitis, bronchitis, nausea, vomiting, fatigue, bronchoconstriction, pneumonitis, laryngeal/glottal edema, upper airway edema or obstruction, and increased airflow resistance; corneal damage; death may result from pulmonary edema, systemic acidosis or respiratory arrest. Contact with liquid may result in frostbite injury.[4]
Personal decontamination	Eyes: Flush with room temperature water for at least 15 minutes.[4] Skin: Flush with large amounts of water. Remove contaminated clothing.[4]
Morbidity and mortality	LCt_{50}: 402,192 mg-min/m^3 (rat)[7] LC_{Lo}: 400 ppm for 1 minute[4] EC_{50} (mild): 8-12 ppm[4] EC_{50} (severe): 50 ppm[4]

COUNTERMEASURES

Prophylaxis	None.
Antidote/Treatment	**Antidote:** None **Treatment:** Establish a patent airway. Suction if necessary. Watch for signs of respiratory insufficiency and assist ventilations if necessary. Administer oxygen by non-rebreather mask at 10 to 15 L/min. Monitor for pulmonary edema and treat if necessary. Anticipate seizures and treat if necessary. Monitor for shock and treat if necessary. For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with normal saline during transport. Do not use

	emetics. For ingestion, rinse mouth and administer 5 ml/kg up to 200 ml of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. Administer activated charcoal. Cover skin burns with sterile dressings after decontamination.[4]

ENVIRONMENTAL DETECTION AND MANAGEMENT

Agent detection	• Real time: None • Laboratory: Mass spectrometry, ion chromatography, Fourier transform infrared spectrometry[4] • Observation: None
Decontamination—property & equipment	Neutralize spilled material with limestone, soda ash, or lime.[4]
Environmental persistence	The average residence time of pollution sulfur is usually between one and five days, depending on the climate of a region.[4]
Formulations	Grades: commercial; USP; technical; refrigeration; anhydrous 99.98% min.[4]

The information cutoff date for this factsheet is 14 May 2010.

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