

**Army Field Manual No 3-9
Navy Publication No P-467
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Potential Military Chemical/ Biological Agents and Compounds

**Headquarters
Department of the Army
Department of the Navy
Department of the Air Force
Washington, DC, 12 December 1990 PCN 320 008457 00**

P r e f a c e

This field manual provides commanders and staffs with general information and technical data concerning chemical and biological agents and other compounds of military interest. It discusses the use; the classification; and the physical, chemical, and physiological properties of these agents and compounds. It also discusses protection and decontamination of these agents. In addition, it discusses their symptoms and the treatment of those symptoms.

The proponent of this publication is Headquarters, TRADOC. Submit recommended changes for improving this publication on DA Form 2028 (Recommended Changes to Publications and Blank Forms) and forward it to Commandant, US Army Chemical School, ATTN: ATZN-CM-NF, Fort McClellan, AL 36205-5020. Send applicable Air Force comments by letter to HQ, USAF/XOOTM, Washington, DC 20330-5054.

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United States Policy

Biological Agents – No Use

- 1. The United States will not use biological agents, including toxins and all other methods of biological warfare, under any circumstances.**
- 2. The United States will strictly limit biological research to defensive measures.**

Chemical Agents – No First Use

- 1. US armed forces will not use lethal or incapacitating chemical agents first.**
- 2. The United States will strictly reserve the right to retaliate, using lethal or incapacitating chemical agents, against an enemy force that has used them on US forces.**
- 3. The authority to order or approve the first retaliatory chemical strike rests with the president of the United States.**
- 4. The United States will avoid risk to civilian populations to the maximum extent possible.**

Chapter 1

Introduction

Nations have used toxic chemical agents in the past, and we cannot ignore the possibility that they will use them in future conflicts. An understanding of chemical and biological agents and other compounds of military interest is vital to our ability to cope with their possible use against our forces. To develop an understanding of chemical and biological agents and other compounds of military interest, you must learn about several factors. You must learn about their historical use, the US policy regarding their use, and the properties that cause a substance to be suitable for use in military operations.

Section I. Background and Policy

Background

Chemical and biological (CB) operations are not new. Historical records show previous use of chemicals, smoke, and flame in warfare. In World War I the Allies and the Germans used them extensively. Many nations developed and manufactured agents during World War II, and some have used these agents since then.

As with chemicals, crude forms of biological warfare started in ancient times. Poisoning of water supplies with rotting carcasses was common practice. In the 1300s the Tartars catapulted corpses of plague victims over the walls and into the besieged city of Kaffa. History suggests that fleeing survivors of this siege caused the “black death,” a plague epidemic that swept Europe. Pizarro in the 1500s and the British in the 1700s introduced smallpox among Indians in the Americas as a means to win a war.

Documentation exists of more recent use of chemical agents and some biological agents in Afghanistan, Southeast Asia, and Southwest Asia.

After World War I various international accords recognized the potential for chemical and biological weapons and outlawed these weapons. Most nations, including the Soviet Union, signed these treaties. However, recent evidence indicates some nations have not adhered to these bans. A growing number of nations can employ biological and chemical, as well as conventional, munitions.

In addition to CB agents, related materials, such as irritants and herbicides, lend themselves to modern warfare. The United States must prepare to defend against these weapons and materials and to retaliate when appropriate. The next paragraph further discusses US policy on the use of these weapons.

United States Policy

The United States seeks to achieve a verifiable, worldwide ban on chemical weapons. Until a verifiable ban is achieved, the US policy is to deter enemy chemical weapons use through denying the enemy a significant military advantage for such use. US chemical weapons deterrence includes a viable NBC defense capability and a credible retaliatory capability.

The United States will not use chemical weapons first against an enemy but reserves the right to retaliate should an enemy use chemical weapons against US or Allied

forces. Only the president may order chemical weapons retaliation.

Current US policy states that we will not use herbicides in war, unless our adversaries first employ them and the president directs their use in retaliation. Executive Order 11850 unilaterally renounces first use of herbicides in war, except to control vegetation within US bases and installations or around their immediate defensive perimeters.

The United States renounces the first use of riot control agents (RCAs) in war except in defensive military modes to save lives, such as in —

- Riot control situations in areas under direct and distinct US military control. This includes the control of rioting prisoners of war.
- Situations in which civilians are used to mask or screen attacks, and these agents can reduce or avoid civilian casualties.
- Rescue missions in remote or isolated areas. Examples are recovering downed aircrews and passengers and rescuing escaping prisoners of war.
- Rear-echelon areas outside the zone of immediate combat to protect convoys from civil disturbances, terrorists, and paramilitary operations.
- Security operations regarding the protection or recovery of nuclear weapons.

The president must approve the use of riot control agents in war.

The United States will not use biological agents, including toxins, regardless of source or manner of production, or other methods of biological warfare under any circumstances. The United States will strictly limit its biological and toxin research program to defensive measures, such as production of vaccines, antidotes, treatment, and protective equipment. US policy is in accordance with the 1925 Geneva Protocol and the 1972 Biological Weapons Convention, both of which the United States has signed and ratified.

Section II. Militarily Significant Aspects of Chemical Agents

Classification of Chemical Agents and Miscellaneous Compounds

We classify chemical agents and compounds according to physical state, physiological action, and use. The terms persistent and nonpersistent describe the time chemical agents remain in an area. These terms do not classify these agents technically. We define chemical agents apart from military chemical compounds. Chemical agent use kills, seriously injures, or incapacitates people. These agents include blood, nerve, choking, blister, and incapacitating agents. On the other hand, military chemical compounds are less toxic. Military chemical compounds include riot control agents, herbicides, smoke, and flame materials. The term excludes chemical agents.

Physical State

Chemical agents and military chemical compounds may exist as solids, liquids, or gases. To a certain extent the state in which an agent normally exists determines its use, duration of effectiveness, and physiological action. It also determines the type of munition used for its dissemination.

Physiological Action

We classify agents and compounds by their physiological actions as follows:

Choking Agents

Choking agents attack lung tissue, primarily causing pulmonary edema (“dryland drowning”). These chemical agents irritate and inflame tissues from the nose to the lungs, causing a choking sensation.

Nerve Agents

These chemical agents, when inhaled, ingested, or absorbed into the body through the skin, inhibit cholinesterase enzymes throughout the body. This inhibi-

tion permits acetylcholine, which transmits many nerve impulses, to collect at its various sites of action. The major effects are—

- Muscle stimulation with uncoordinated contractions, followed by fatigue and eventual paralysis.
- Pinpointed pupils; tightness in chest; nausea, vomiting, and diarrhea; and secretions from the nose, mouth, and air passages.
- Disturbances in thought, convulsions, coma, and depression of vital centers of the brain, leading to death.

Blood Agents

The body absorbs these chemical agents, including the cyanide group, primarily by breathing. They poison an enzyme called cytochrome oxidase, blocking the use of oxygen in every cell in the body. Thus, these agents prevent the normal transfer of oxygen from the blood to body tissues. The lack of oxygen rapidly affects all body tissues, especially the central nervous system.

Blister Agents (Vesicants)

Both exterior and interior parts of the body readily absorb these chemical agents. These agents cause inflammation, blisters, and general destruction of tissues. Agent vapors attack moist tissue. Vulnerable areas include the eyes, mucous membranes, and respiratory tract. Eyes are very susceptible to blister agent.

Tear Agents (Lacrimators)

These compounds cause a large flow of tears and irritation of the skin. Some of these compounds are very irritating to the respiratory tract. They sometimes cause nausea and vomiting.

Vomiting Agents (Sternutators)

These compounds cause nausea and vomiting. They also cause coughing, sneezing, pain in the nose and throat, nasal discharge, or tears. A headache may follow.

Defoliants

These compounds cause trees, shrubs, and other plants to shed their leaves prematurely.

Plant Growth Regulators

These compounds regulate (stimulate or inhibit) plant growth.

Desiccants

These compounds remove water from plant tissues, causing the plants to dry and shrivel.

Soil Sterilants

These compounds make a soil incapable of supporting higher plant life. Their effects may last one growing season or many years.

Use

Chemical agents and military chemical compounds may be grouped according to use.

Chemical Agents

Chemical agents may be either toxic or incapacitating.

Toxic agents. Toxic agents can produce incapacitation, serious injury, or death when used in field concentrations.

Duration of Effectiveness of Chemical Agents

Several factors determine the time a chemical agent remains effective. These include the method of dissemination and the physical properties of the agent. Factors also include weather, terrain, and target conditions.

Method of Dissemination

The size of the particles disseminated greatly influences the effectiveness of liquid or solid agents. Vapors or aerosols (air-contaminating agents) do not persist as long as do droplets of agents used to contaminate terrain and materiel. In explosive munitions the degree of division depends upon several factors. These factors are the amount and the type of burster charge and the fuzing of the munition (air or ground burst). Nonexploding types of munitions, such as aerosol generators and spray tanks, can vary the degree of dispersion. Thus, these types influence the duration of effectiveness of agents.

Physical Properties of the Agent

Vapor pressure and volatility influence the rate of evaporation. These properties are especially important in

This category includes lethal and damaging (blister) agents. Lethal chemical agents in field concentrations can produce death.

Incapacitating agents. Incapacitating agents produce temporary physiological or mental effects, or both. Effects may persist for hours or days after exposure. They may make individuals unable to perform their assigned duties. Victims do not usually require medical treatment, but treatment speeds recovery.

Military Chemical Compounds

These compounds include riot control agents, training agents and compounds, smokes, and herbicides.

Riot control agents (RCAs) produce transient irritating or disabling effects. These effects disappear within minutes after exposure ends. Governments widely use these compounds for domestic law enforcement purposes.

Training agents and compounds are chemicals that are authorized for use in training. These chemicals enhance proficiency for operating in an NBC environment.

Smokes are used for obscuring, screening, deceiving, and identifying and signaling.

Herbicides are chemical compounds that will kill or damage plants.

Note: This manual seeks to show only the general relationship between chemical agent properties and uses. FM 3-10-1, FM 3-11, FM 3-50, and FM 20-33 specifically cover use concepts.

determining the duration of effectiveness of an agent. Gases (vapors), aerosols, and highly volatile liquids tend to disperse rapidly after release. Thus, they present an immediate short-duration hazard. Large drops or splashes remain a hazard longer than finely divided particles. Also, viscous materials tend to adhere and not spread or flow readily. This can increase persistency. Appendix A discusses physical properties.

Weather Conditions

Many weather factors influence the duration of effectiveness. The most important are temperature, temperature gradient, wind speed, relative humidity, and precipitation. See FM 3-6 for a detailed discussion of the impact of weather on duration.

Temperature

The higher the ground or surface temperature, the quicker a liquid chemical agent will evaporate from it. Low temperatures may freeze some agents, thus reducing the immediate contact hazard, but will increase persistency.

Temperature Gradient

Often the temperature of the layer of air next to the ground is different to that of the air layers above. This gives rise to a temperature gradient. Agents in a vapor state will remain near the ground during stable (inversion) conditions. When an unstable (lapse) condition exists, air layers mix and agents disperse more quickly.

Wind Speed

High winds increase the rate of evaporation of liquid chemical agents. High winds also disperse chemical clouds more rapidly than low winds. A low wind speed allows agent to persist longer. Also, the rate of spread will be slow. Vapors and aerosols disperse rapidly in open country; dangerous concentrations may remain longer in woods, foxholes, and built-up areas.

Relative Humidity

Relative humidity has little direct effect on most chemical agents. However, the choking agent phosgene (CG) and the blister agent lewisite (L) rapidly decompose at relative humidities over 70 percent.

Precipitation

Heavy or lasting rains will wash liquid agent contamination to low areas and stream beds and present a lingering hazard. Light rainfall can cause recurrence of a contact hazard. Snow tends to wash agents from the air. Snow cover reduces the vapor concentration above the contaminated

area but, in combination with the lower temperatures, increases the duration.

Conditions of Terrain or Target

Vegetation, soil, and contours play an important part in the duration of effectiveness of an agent at the target. See FM 3-6 for a detailed discussion of terrain impact.

Vegetation

Liquid chemical agents cling to vegetation, increasing the area for contact and evaporation. Because of low wind speeds and reduced temperatures, heavily wooded and jungle areas retain vapors longer.

Soil

Toxic liquids quickly soak into porous surfaces and evaporate more slowly than from nonporous surfaces. This increases the duration of any vapor hazard, although it reduces vapor cloud concentration.

Contours

Toxic clouds follow the contour of the surface of the terrain. Chemical clouds tend to go around obstacles, such as hills. Concentrations persist in hollows, low ground, depressions, foxholes, and buildings. Rough ground, including that covered with tall grass or brush, slows chemical cloud movement. Flat country (unless covered with tall grass or brush) allows an even, steady movement. Urban areas form local "heat islands" that may alter significantly the normal temperature gradient.

Requisites and Desirable Features of Chemical Agents

The combined physical, chemical, and toxicological properties of a substance determine its suitability and effectiveness as a chemical agent. These factors influence whether the substance meets the requisite and desirable features of a chemical agent.

Requisites

A chemical agent must be—

- Toxic. Through its chemical properties in small concentrations it will produce damaging or lethal effects on man, animals, or plants.
- Stable or capable of stabilization during the period between its production and use.
- Producible from readily available raw materials in adequate quantities for effective military use.
- Capable of dissemination from a device feasible for field use in sufficient concentration to produce the desired effect on the target.

- Capable of being handled and transported without extensive precautions.

Desirable Additional Features

A chemical agent should—

- Have little or no corrosive action on the munition or container during storage.
- Have such inherent properties that complete protection from the chemical agent is difficult for enemy personnel. If possible, the agent should be capable of minimizing the effectiveness of the protective equipment of potential enemies.
- Have a known physiological mechanism of action, protective measures, and a method of medical treatment or prophylaxis.
- Be difficult to detect by ordinary methods before the onset of physiological and/or psychological effects. (Colorless, odorless, and nonirritating toxic chemical agents are desirable.)

Physical Properties

The physical properties impact on how a chemical agent or compound is used. These properties also impact upon the defensive measures against its use. Some of the more important physical properties are vapor density, vapor pressure, volatility, melting and freezing points, and liquid and solid densities. The vapor density determines whether the agent is lighter or heavier than air; it thus determines whether the agent will settle to low areas or float away and dissipate in the atmosphere. Vapor pressure determines

the volatility and the rate of evaporation of an agent. The rate of evaporation has a major effect upon the vapor concentration. It also affects the duration of an agent hazard after dissemination. The boiling and freezing points of chemical agents influence their operational use and the means of disseminating them. See Appendix A for a discussion of physical properties. Appendix B tabulates chemical agent physical properties and other data.

Chemical Properties

The chemical properties of an agent influence its stability, toxicity, and reactivity with water and other substances.

also be toxic. Examples include lewisite and other agents containing arsenic.

Hydrolysis

Hydrolysis is the reaction of a compound with the elements of water whereby decomposition of the substance occurs. The reaction produces one or more new substances.

Rapid hydrolysis aids in lowering the duration of effectiveness of toxic chemical agents. For example, in the presence of water or water vapor, lewisite (L) rapidly hydrolyzes. Therefore, it has a shorter duration of effectiveness than distilled mustard (HD).

New substances (hydrolysis products) form when an agent or compound reacts with water. In certain cases hydrolysis does not completely destroy the toxicity of an agent or compound. The resulting hydrolysis products may

Stability in Storage

Stability in storage determines the practical usefulness of an agent or chemical compound. If a candidate agent decomposes in storage, it will have little value regardless of any other properties that may recommend it. The addition of stabilizers to agents will slow decomposition and polymerization.

Action on Metals, Plastics, Fabrics, and Paint

The action between an agent or compound and certain materials limits the use of that chemical or material. Chemicals that are acids or form acids have a corrosive effect on metals, leather, fabrics, and paints, except chemical agent-resistant coatings.

Physiological Aspects

Chemical agents have various physiological effects upon the human body. Most agents are used for their toxic effects to produce a harmful physiological and/or psychological reaction when applied to the body externally, when breathed, or when taken internally. Most agents cause a disorganization of body functions, as described in Chapter 2 for individual chemical agents and in Chapter 3 for chemical compounds.

Routes of Entry

Chemical agents may enter the body by several routes. Any part of the respiratory tract, from the nose to the lungs, may absorb inhaled gases, vapors, and aerosols. Moist tissues, such as the lungs or eyes, absorb vapors most rapidly. The skin, especially areas affected by sweat, can also absorb vapors. The surface of the skin, eyes, and mucous membranes can absorb droplets of liquids and solid particles. Wounds or abrasions are probably more susceptible to absorption than the intact skin. Chemical

agents can contaminate food and drink, and therefore the body can absorb them through the gastrointestinal tract. The onset and severity of signs may vary, depending upon the route as well as the amount of exposure.

Some agents are highly toxic if absorbed through the skin or eyes; others are nontoxic by those routes. Nerve agents exert their full toxic effects through the skin, the eyes, and the lungs. The primary blood agent hazard results from inhalation, not skin or eye absorption because of agent volatility. Liquid hydrogen cyanide (AC) can be toxic by absorption through the skin or the eyes. However, liquid AC rarely exists in a military situation. Skin and eye effects, although severe, are usually local. Blister agents damage skin and any other tissues that they contact; if absorbed in sufficient quantity, these agents can cause systemic poisoning. The vomiting compounds and choking agents exert their effects only if inhaled. The tear compounds normally have little effect on the body except temporary irritation to the eyes and upper respiratory tract.

Dosage

The dose is the amount of compound the body takes in or absorbs. It is usually expressed as milligrams per kilogram (mg/kg) of body weight. Median lethal dose describes the degree of toxicity of a substance.

Median Lethal Dosage (LD₅₀) of Liquid Agent

The LD₅₀ is the amount of liquid agent expected to kill 50 percent of a group of exposed, unprotected personnel.

Median Incapacitating Dosage (ID₅₀) of Liquid Agent

The ID₅₀ is the amount of liquid agent expected to incapacitate 50 percent of a group of exposed, unprotected individuals.

For airborne chemical agents, the concentration of agent in the air and the time of exposure are the important factors that govern the dose received. The dosage may be inhaled (respiratory) or absorbed through the eyes (ocular) or through the skin (percutaneous). Dosages are based on short exposures – ten minutes or less. Toxicity is generally identified by reference to the lethal dosage,

Median Lethal Dosage (LC_{t50}) of a Vapor or Aerosol

The median lethal dosage of a chemical agent employed for inhalation as a vapor or aerosol is generally the LC_{t50}. The LC_{t50} of a chemical agent is the dosage (vapor concentration of the agent multiplied by the time of exposure) that is lethal to 50 percent of exposed, unprotected personnel at some given breathing rate. It varies with the degree of protection provided by masks and clothing worn by personnel and by the breathing rate. If individuals are breathing faster, they will inhale more agent in the same time, increasing the dose received.

Median Incapacitating Dosage (IC_{t50}) of a Vapor or Aerosol

For inhalation effect, the median incapacitating dosage is the IC_{t50}. The IC_{t50} is the amount of inhaled vapor that is sufficient to disable 50 percent of exposed, unprotected personnel. The unit used to express IC_{t50} is mg-min/m³.

Note: You may also express dosages in amounts other than the median dosage. For example, the LC_{t25} is the dosage of vapor that would kill 25 percent of a group of exposed, unprotected personnel; IC_{t90} is the vapor dosage that would incapacitate 90 percent of a group of exposed, unprotected personnel.

Modifying Factors

After exposure to a chemical agent vapor a person may show signs and symptoms that are less or more severe than expected. Severity depends upon some of the following variables:

- How long the person held his or her breath during short exposure.
- Speed with which he or she donned the mask.
- Proper fit of the mask.
- Whether the body absorbed the agent through the skin.
- Whether the agent stimulated the rate of breathing.
- Rate and depth of breathing of the person at the time of exposure.
- Amount of physical exertion of the person at the time of exposure.
- Rate of detoxification, especially if exposure was long.

For tabulation purposes we ignore such variables. The Ct values measure the amount of agent a person receives when breathing at a normal rate in a temperate climate with average humidity. Dosages are given for 70-kilogram (kg) individuals with very light activity (for example, desk work) with a breathing rate of 15 liters per minute. These values provide a basis to compare various agents.

The skin vapor dosage is equal to the time of exposure in minutes of a person's unprotected skin multiplied by the concentration of the agent cloud. The particle size, the time, and the concentration affect the physiological effectiveness of skin and respiratory vapor dosages. Retention by the lungs and absorption through the skin are functions of physical characteristics, such as particle size.

Rate of Detoxification

The human body can detoxify some toxic materials. This rate of detoxification is the rate at which the body can counteract the effects of a poisonous substance. It is an important factor in determining the hazards of repeated exposure to toxic chemical agents.

Most chemical agents are essentially cumulative in their effects. The reason is that the human body detoxifies them very slowly or not at all. For example, a one-hour exposure to HD or CG followed within a few hours by another one-hour exposure has about the same effect as a single two-hour exposure. Continued exposure to low concentrations of HD may cause sensitivity to very low concentrations of HD. Other chemical agents also have cumulative effects. For example, an initial exposure to a small (less than lethal) amount of Sarin (GB) would decrease cholinesterase levels; a second quantity less than the LD₅₀ could be enough to kill. (Although the body can detoxify it to some extent, GB is essentially cumulative.)

Some compounds have a detoxification rate that is significant. Because the body detoxifies such chemical agents as AC and cyanogen chloride (CK) at a fairly rapid rate, it takes high concentrations of these agents to produce maximum casualty effects.

Rate of Action

The rate of action of a chemical agent is the rate at which the body reacts to or is affected by that agent. The rate

varies widely, even to those of similar tactical or physiological classification. For example, blister agent HD causes no immediate sensation on the skin. Skin effects usually occur several hours later (some cases result in delays of 10 to 12 days before symptoms appear). In contrast, lewisite produces an immediate burning sensation on the skin upon contact and blistering in about 13 hours. Decontamination immediately (within four to five minutes) will prevent serious blister agent effects.

With the single exception of arsine (SA), the nerve agents and the blood agents are very fast acting. Vomiting compounds also exert their effects within a short time after inhalation. In general, agents that are inhaled or ingested will affect the body more rapidly than those that contact the skin. To avert death, first-aid measures, such as administering antidotes, generally must follow within a few minutes after the absorption of a lethal dosage of any agents.

Agent Mixtures

Mixing chemical agents with each other or with other materials can alter the characteristics and effectiveness of agents. This alteration occurs through changes in physical properties, physiological effects, or toxicity.

Mixtures may lower the freezing point, increasing agent effectiveness over a wider temperature range. Distilled mustard has a freezing point of 14.5°C (55°F); but a mixture (37:63) of it and lewisite will freeze at -25°C.

The addition of thickeners or thinners to agents will increase or decrease persistency. Soman mixed with thickeners will increase persistency. Riot control agents mixed with thinners will decrease persistency.

In addition to changing the physical properties, mixing agents together will create special problems through their physiological effects. These problems can produce difficulty in identification, immediate and delayed effects, or contact and vapor hazards occurring simultaneously. Some mixtures would make it difficult to maintain the seal of the mask.

Mixing some agents can increase the toxic effects, either by a synergistic effect or by an improved absorption through the skin. For example, dimethylsulfoxide (DMSO) can penetrate the skin and carry substances mixed with it into the body at a very rapid rate.

Section III. Militarily Significant Aspects of Biological Agents

Classification of Biological Agents

Biological agents can be classified according to their biological type, uses, operational effects, and physiological action. The terms persistent and nonpersistent describe the continuing hazard posed by the agent remaining in the environment. Do not use these terms to classify biological agents.

Types of Biological Agents

Biological agents can be classified as pathogens, toxins, or other agents of biological origin, such as bioregulators/modulators (BRMs).

Pathogens

Pathogens are disease-producing microorganisms, such as bacteria, mycoplasma, rickettsia, fungi, or viruses. Pathogens are either naturally occurring or altered by random mutation or recombinant deoxyribonucleic acid (DNA) techniques. TM 3-216, FM 8-9, and FM 8-33 detail the characteristics of naturally occurring pathogens.

Toxins

Toxins are poisons naturally produced through the metabolic activities of living organisms. They are organic chemical compounds, such as proteins, polypeptides, and

alkaloids, that come from a variety of biological sources. These sources include microorganisms and various plants and animals. Although toxins were initially isolated from living organic sources, manufacture of some by chemical synthesis or other biochemical processes is feasible. Industrial fermentation processes can obtain large amounts of highly concentrated bacterial toxins. Laboratories can synthesize toxins composed of only 10 to 12 amino acids.

Bioregulators/Modulators

Bioregulators/modulators (BRMs) are biochemical compounds, such as peptides, that occur naturally in organisms. These peptides and other small molecules can act as neurotransmitters and/or can modify neural responses. It is feasible to produce some of these compounds by chemical synthesis. It is probable that neuropeptides will become available soon as a result of research in the medical community. Although BRMs have potential as biological agents, this manual does not include them.

Uses

Biological agents can be directed against personnel, plants, animals, or materiel. Food and industrial products can be rendered unsafe or unfit for use by contamination

or by the effects resulting from contamination with biological agents.

Antipersonnel

Biological antipersonnel agents are those that are effective directly against humans. The Threat would select these agents on the basis of the agents' ability to cause death or disability. The Threat might use these agents against selected persons or groups or to produce mass casualties over large areas. This use could result in physical and psychological effects that could weaken or destroy the ability to resist aggression. Potential biological antipersonnel agents include toxins, bacteria, rickettsiae, viruses, and fungi.

Antianimal

Biological antianimal agents are those that could be employed against animals to incapacitate or destroy them through disease. The main purpose of the use of these agents is to affect humans indirectly by limiting their food supply. TM 3-216 contains information on potential antianimal agents.

Antiplant

Biological antiplant agents are live organisms that cause disease or damage to plants. An enemy may use these agents to attack food or economically valuable (cash or money) crops. The enemy could thereby reduce a nation's ability to resist aggression. TM 3-216 describes some antiplant agents.

Antimateriel

Antimateriel agents are organisms that degrade or break down some item of materiel. Most of the materiel damage done by microorganisms is a result of natural contamination that grows only under very special conditions of temperature and relative humidity. Fungi are responsible for damage to fabrics, rubber products, leather goods, and foodstuffs. Some bacteria can use petroleum products as an energy source, causing residues that might clog fuel or oil lines. Other bacteria produce highly acidic compounds that cause pitting in metals. The use of antimateriel biological agents for military purposes appears unlikely. However, with advancing technology these agents could create potential problems with stockpiled materiel.

Operational Effects

The effects produced by biological agents can influence the continued operational effectiveness of units in the field. Biological agents can produce incapacitation, serious injury, or death. Biological agents are categorized arbitrarily as lethal or incapacitating. Some microorganisms or toxins will cause diseases that are usually lethal unless the target

population is immune. Others will cause illnesses that are essentially incapacitating.

Lethal Agents

Lethal biological agents are those that could cause significant mortality. Lethal agents can cause death in susceptible people, but from a practical standpoint death occurs only in a certain percentage of those exposed. The mortality rates vary according to several factors. These factors include the characteristics of the agent, the route of entry, the dose received, and, in the case of pathogens, the ability of the host to resist infection.

Incapacitating Agents

Incapacitating agents usually do not kill healthy adults. However, these agents can cause death in certain groups, such as the very young, the aged, or the infirm. Incapacitating agents can cause infection or disease with militarily significant disability among susceptible, exposed people.

Transmissible Agents

Pathogens can be further classified as transmissible or nontransmissible agents. Some pathogens cause disease that is transmissible from person to person, which can lead to an epidemic. However, other microorganisms are nontransmissible. Toxins are not living organisms; their effects cannot spread from person to person.

Physiological Action

The clinical effects of toxins may closely resemble those of chemical warfare agents, such as nerve, blister, vomiting, or choking agents. Most toxins of military significance cause casualties primarily in one of two ways. These toxins can be classified as either neurotoxins or cytotoxins by the way they act.

Neurotoxins

Neurotoxins interfere with nerve impulse transmission and could be called nerve toxins. The neurotoxins exert highly specific effects upon the nervous system. Some neurotoxins cause symptoms similar to those of chemical nerve agents leading to convulsions and rigid paralysis. However, the mechanism causing the symptoms does not usually inhibit acetylcholine esterase. Many neurotoxins block the transmission of impulses along nerve and muscle fibers. These neurotoxins can cause numbness or extreme weakness, tremors, and muscular incoordination leading to severe muscle weakness and flaccid (limp or rag-doll) paralysis. Confusion, headache, blurred vision, and light sensitivity (because of dilation of pupils) may occur. Some neurotoxins affect the central nervous system. Neurotoxins tend to act rapidly.

Cytotoxins

Cytotoxins cause cellular destruction or interfere with metabolic processes, such as cell respiration or protein synthesis. Cytotoxins exert effects upon a variety of tissues or systems. These tissues or systems include the digestive, respiratory, and circulatory systems and the skin.

Symptoms of exposure may resemble those of disease or of various chemical agents. Cytotoxin effects may include irritation, blistering, and lesions of the skin; nausea or vomiting; hemorrhaging, bloody diarrhea and vomit; difficulty in breathing or sudden death.

Duration of Effectiveness of Biological Agents

The duration of effectiveness of a biological agent refers to the persistency of the agent in the environment. It varies greatly between agents. It depends on the characteristics of the agent, the influence of environmental factors, and any residual hazard generated through resuspension of settled biological particles by vehicle and troop movements or wind.

Physical, Chemical, and Biological Properties

The duration of effectiveness of a biological agent does not generally relate to its physical properties; vapor pressure or volatility are not significant factors for biological agents. Some toxins (for example, *Staphylococcus enterotoxin*, Type B) are stable in the environment and are more resistant to heat, hydrolysis, or vaporization than G- or V-series nerve agents. The chemical structure of toxins can strongly influence the stability of the agent to environmental factors. High-molecular-weight toxins, such as proteins, are usually more sensitive to ultraviolet (UV) light, heat, and oxidation than low-molecular-weight, non-protein toxins. Many toxins are water-soluble.

Because pathogens are live—exhibiting feeding, excretory, respiratory, reproductive, and defensive functions—any factors that reduce the viability will reduce the duration of effectiveness. Environmental conditions affect most pathogens significantly unless altered or protected.

Weather and Terrain Influences

Solar (ultraviolet) radiation, relative humidity, wind speed, and temperature gradient are the most important

weather factors in determining duration of effectiveness. Ultraviolet light affects most biological pathogens and some toxins (especially high-molecular-weight proteins). However, encapsulation (natural, such as bacterial spores, or man-made protective coverings), addition of dyes to the spray fluid, or possibly genetic engineering (of pathogens) may protect some agents from sunlight and other destructive natural forces. Impurities in crude toxin cultures can stabilize the toxins and/or enhance toxicity.

FM 3-3 and FM 3-6 discuss the field behavior of biological agents. These manuals also discuss the impact of weather effects and terrain features (soil, vegetation, relief) on duration. FM 8-9 discusses in detail the atmospheric influence on biological aerosols of pathogens.

Methods of Dissemination

Biological agents may be disseminated as aerosols, liquid droplets (toxins only), or dry powders. To a certain extent the state in which an agent normally exists determines its use, duration of effectiveness, and physiological action. It also determines the type of system used for its dissemination. Live microorganisms usually grow in a moist environment. Therefore, these agents may be disseminated in a liquid medium as wet aerosols. However, the technology exists to store microbiological materials as a powder (usually by a freeze-dried process), suitable for dissemination. Dissemination of spores and certain toxins as dry powders is likely. Many toxins are water-soluble, and dissemination could be as sprays or wet aerosols. In general, agents disseminated as a dry powder will survive longer than those disseminated as wet aerosols.

Characteristics of Likely Potential Biological Warfare Agents

Major militarily significant characteristics for all biological warfare (BW) agents include —

- A susceptible population.
- Highly infectious or toxic properties.
- Availability or adaptability to a large-scale production.
- Stability in storage, in handling, and after dissemination.
- Suitability for aerosol dispersion.

Advances in technology have increased the capability for production and modification of biological materials. The Threat could use a considerable number of toxic or dis-

ease-causing agents against US forces. Several factors would limit the selection. These limiting factors include biological properties, environmental factors, and methods of dissemination. FM 8-9 addresses biological operations and selection of live biological agents based on their characteristics.

Route of Entry

The type of symptoms produced by biological agents depends not only on the agent characteristics but also upon the route of entry. The places where pathogens gain entry

into the body are the portals of entry. The three important portals of entry are the skin, the respiratory tract, and the digestive tract. The respiratory system is much more susceptible to penetration than are the other portals of entry. The lungs have a large surface area, very thin air sacs, and a large blood supply. The body is more resistant to invasion by microorganisms through the digestive tract and the skin. However, penetration across the skin and mucous membranes may occur. This is particularly true of abraded (broken) surfaces. Toxins (for example, mycotoxins) may also have a direct action on the skin or mucous membranes.

Biological agents may be encountered by natural routes, such as in water and food or by vectors. However, the respiratory route appears most likely to cause mass casualties. As a result of inhalation, many pathogens will initially produce flu-like symptoms or other effects on the respiratory system. Within one to five days most pathogens will produce a unique pattern of illness. The pattern may be fever, sore throat, stiff neck, rash, neurologic or mental abnormalities, pneumonia, diarrhea, dysentery, hemorrhaging, or jaundice. Toxins absorbed through the respiratory tract might produce signs and symptoms very different from those acquired through natural occurrence.

For certain organisms causing gastrointestinal diseases, the digestive tract is the expected route of entry. Typical symptoms include nausea, vomiting, diarrhea, or dysentery. The gastrointestinal tract is often the natural route of infection or intoxication for toxins (for example, *Botulinum* toxin and *Staphylococcal* enterotoxins). The signs and symptoms would be similar to the natural infection, however, the onset may be much more rapid.

Dosage

Most BW agents are, by weight, thousands of times more lethal or effective than equivalent amounts of chemical warfare agents found in modern chemical arsenals. These BW agents also have greater downwind hazard distances associated with air-contaminating clouds than do chemical agents.

Comparison of Pathogens and Toxins

Biological agents, whether biological toxins or pathogens, can be lethal or incapacitating. However, because pathogens are live agents but toxins are nonliving biochemical compounds, there are major differences. These differences are in their toxicity, stability, lethality, and time to effects, as well as persistence in the field. FM 3-3 addresses field characteristics of biological agents. Table 1-1 summarizes important pathogen and toxin field properties.

The response to the toxic agent is a function of the total dose received, the length of exposure, and the route of

Infective Dose

The infective dose is the number of microorganisms or spores required to produce an infection. It is comparable to the effective dose for chemical agents.

Lethal Dose

Some pathogens produce toxins that can result in disease (for example, *Botulinum*, cholera, diphtheria, typhus). The median lethal dose (LD₅₀) expresses the toxicity of blotoxins. It is obtained from experimental animal investigation. The extreme toxicity of many toxins causes the lethal dose to be much smaller than that of chemical agents. Hence, units of micrograms (µg) or even nanograms (ng) may be used instead of milligrams (mg) in expressing toxicity.

Most toxicity data are based on injection (into the blood or into the body cavity) into animals. Estimates for human toxicity are made from animal data. Some human toxicity data are based on accidental contact, ingestion, or inhalation of these natural poisons.

Rate of Action

The rate of reaction to toxins varies widely. Rapid-acting toxins generally incapacitate within minutes. Delayed-acting agents may take several hours to days to incapacitate. The times given for the onset of symptoms and the descriptions apply to dosages at or about LD₅₀ unless noted. Dosages much larger than LD₅₀ may occur during toxin employment in a BW attack, especially within zone I of the potential biological hazard area. Personnel exposed to these dosages may experience a faster onset and more severe symptoms. Additional symptoms may also occur.

The time to maximum effects for pathogens is normally more than 24 hours (unless the pathogen produces a toxin). However, the incubation periods of microorganisms used in BW may be far shorter than those expected by examining the natural disease. Initial dose inhaled may be many times the infective dose. In addition, selective breeding or genetic engineering may have altered the incubation period.

exposure. Dosages given for toxicity are for 70-kilogram individuals with very light activity (for example, desk work) and a breathing rate of 15 liters per minute. Increased breathing rate (for example from increased activity) will decrease the respiratory dosages proportionally because a greater volume of agent is inhaled in the same time.

Dosages given are for less than two-minute exposure. The same total dosages received through longer exposure times at lower concentrations will reduce the symptoms somewhat.

Table 1-1. Important field properties of pathogens and toxins.

Field Characteristic	Pathogens	Toxins
Dose Requirement	Very low; agent reproduces in host	Low (1/1,000 of nerve agent dose); may have cumulative effects
Area Coverage	Very large (approximately four times predicted chemical downwind hazard distance)	Very large
Time to Effects	Hours to days (antipersonnel); weeks to years (antiplant)	Minutes to hours (most neurotoxins); hours to days (many cytotoxins)
Control	Difficult, especially if agent is contagious or forms spores	More predictable than pathogens; much like chemical agents
Persistency	Hours to days unless protected (spores or capsules) or altered	Days to weeks for some
Stability to Weather Effects	Many killed by sunlight if not protected naturally (for example, spore) or artificially (for example, encapsulated). Wind and/or low humidity can increase dehydration; cold favors survivability	Many resistant to environmental effects; resist hydrolysis (most are soluble in water)

Chapter 2

Chemical Agents and Their Properties

Chemical agents can be separated into groups according to the potential severity of their effects: lethal, blister, and incapacitating agents. This chapter contains the physical, chemical, and physiological properties of specific chemical agents that might be used or encountered in the field. It also gives brief information on their use, detection, identification decontamination, and the protective measures to be taken against them. As noted, Appendix B gives a comparison of the properties of chemical agents. Temperatures are listed in degrees Celsius (C); other data are in various metric units. Appendix C presents a table of English-metric equivalents. Appendix D presents temperature conversions.

Section I. Lethal Chemical Agents

Lethal chemical agents are those agents that primarily cause deaths among target personnel. They include the choking, nerve, and blood agents.

Choking Agents

Choking agents injure an unprotected person chiefly in the respiratory tract (the nose, the throat, and particularly the lungs). In extreme cases membranes swell, lungs become filled with liquid, and death results from lack of oxygen; thus, these agents “choke” an unprotected person. Fatalities of this type are called “dryland drownings.”

Phosgene (CG)

CG, normally a chemical agent with a short agent-cloud duration, was used extensively in World War I. In fact, more than 80 percent of World War I chemical agent fatalities resulted from CG.

CG is a colorless gas with an odor similar to that of new-mown hay, grass, or green corn, which may go unnoticed until at toxic levels. It tends to hug the ground; vapors may linger for some time in trenches and low places under calm or light winds. CG readily condenses to a colorless liquid below 46°F (7.8°C). It reacts rapidly with water, so rain, fog, and dense vegetation reduce the concentration in the air.

CG is used as a delayed-casualty agent that causes fluid buildup in the lungs that can cause dryland drowning. During and immediately after exposure, coughing and wheezing are likely however, exposure to low concentrations causes no ill effects for three hours or more. The severity of poisoning cannot be estimated from the immediate symptoms. The full effect is not usually apparent until three or four hours after exposure. Severe cases can result in dryland drowning, usually within 24 hours. With proper care a victim can recover if the amount of CG received is less than lethal. The protective mask provides protection. If a person inhales some agent, he should continue normal combat duties unless he has respiratory distress.

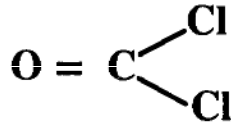
Selected data regarding the chemical properties and toxicity of this agent follow (Table 2-1). These include Chemical Abstracts Service (CAS) registry number and Registry of Toxic Effects of Chemical Substances (RTECS) reference number.

Table 2-1. Phosgene (CG).

Alternate designations: Collognite; Zusatz; Green Cross; D substance.
Chemical name: Carbonyl chloride.
Synonyms: Carbon oxychloride; chloroformyl chloride; fosgeen; fosgen; fosgene; koolstofoxychloride; phosgen.
CAS registry number: 75-44-5.
RTECS number: SY5600000.

Physical and Chemical Properties

Structural formula:

Molecular formula: COCl₂

Molecular weight: 98.92

Physical state	Colorless gas.
Odor	New-mown hay, grass, or green corn.
Boiling point	7.6°C.
Freezing point	-128°C.
Liquid density at 20°C	1.373 (pure CG); 1.381 (plant purity).
Vapor density (compared to air)	3.4.
Vapor pressure	1,173 millimeters of mercury (mm Hg) at 20°C; 555 mm Hg at 0°C; 365 mm Hg at -10°C. This is in the range of freon refrigerant.
Volatility	4,300,000 mg/m ³ at 7.6°C; 2,200,000 mg/m ³ at -10°C; 528,000 mg/m ³ at -40°C.
Latent heat of vaporization	59 calories per gram.
Flash point	None.
Decomposition temperature	800°C.
Solubility	Limited in water; decomposes immediately. Completely miscible (mixes) in most organic solvents.
Rate of hydrolysis	Rapid under usual field conditions. Rain destroys effectiveness. Heavy vegetation, jungle, and forests cause considerable loss by hydrolysis on leafy surfaces.
Hydrolysis products	Hydrochloric acid and carbon dioxide.
Stability in storage	Stable in steel containers if CG is dry.
Action on metals or other materials	None when CG is dry; acidic and corrosive when moist.

Toxicity Data

LC ₅₀	3,200 mg-min/m ³ .
IC ₅₀	1,600 mg-min/m ³ .
Rate of detoxification	Not detoxified; cumulative.
Skin and eye toxicity	Mild eye irritation.
Rate of action	Delayed. Immediate irritation in high concentrations. Exposure to low concentration may cause no ill effects for three hours or more.

Other Data

Protection required	Protective mask.
Decontamination	For confined areas, aeration; not required in the field.
Persistency	Short. Vapor may persist for some time in low places under calm or light winds and stable atmospheric conditions (approximately 30 minutes in summer; approximately 3 hours at -20°C).
Use	Delayed-action casualty agent.

Diphosgene (DP)

DP (Table 2-2) is a colorless liquid with an odor similar to that of new-mown hay, grain, or green corn. DP has a much higher boiling point than CG. Because DP has a stronger tearing effect, it has less surprise value than CG when used on troops. Furthermore, its lower volatility (vapor pressure) adds to the difficulty of setting up an effective surprise concentration.

DP can produce delayed or immediate casualties, depending upon the dosage received. Because the body converts DP to CG, the physical effects are the same for both agents. Immediate symptoms may follow exposure to a high concentration of DP; a delay of three hours or more may elapse before exposure to a low concentration causes any ill effects. The protective mask provides protection from DP.

Table 2-2. Diphosgene (DP).

Alternate designations: Difosgene; Perstoff; Surpalite; Green Cross.	
Chemical name: Trichloromethyl chloroformate.	
CAS registry number: 503-38-8.	
RTECS number: LQ7350000	
Physical and Chemical Properties	
Structural formula:	
Molecular formula: C ₂ Cl ₄ O ₂ .	
Molecular weight: 197.85.	
Physical state	Colorless oily liquid.
Odor	New-mown hay, grain, or green corn.
Boiling point	127° to 128°C.
Freezing point	-57°C.
Liquid density	1.653 at 20°C.
Vapor pressure	4.2 mm Hg at 20°C; 1 mm Hg at 0°C.
Volatility	12,000 mg/m ³ at 0° C; 45,000 mg/m ³ at 20°C; 270,000 mg/m ³ at 51.7°C. The volatility of DP is much lower than that of CG. It is approximately twice that of water and one-third that of ethyl alcohol at 25°C.
Latent heat of vaporization	57.4 calories per gram.
Flash point	None.
Decomposition temperature	300°C to 350°C. (Yields two molecules of CG, which decomposes at 800° C.)
Solubility	Limited in water; good in organic solvents.
Rate of hydrolysis	Slow at ordinary temperatures.
Hydrolysis products	Hydrogen chloride (HCl) and carbon dioxide.
Stability in storage	Unstable because of conversion to CG.
Action on metals or other materials	Metals act as catalyzers in conversion to CG.
Toxicity Data	
LCt ₅₀	3,000 mg·min/m ³ for resting troops. Because the effects of DP are cumulative, the Ct does not significantly change with variations in time of exposure (within reasonable limits).
ICt ₅₀	1,600 mg·min/m ³ for resting troops.
Rate of detoxification	Not detoxified; cumulative.
Skin and eye toxicity	No effect on skin; slight tearing effect.
Rate of action	Delayed. Although immediate symptoms may follow exposure to a high concentration of DP, a delay of three hours or more may elapse before exposure to a low concentration causes any ill effects.
	continued

Table 2-2, Diphosgene (DP) continued

Other Data	
Protection required	Protective mask.
Decontamination	Live steam, ammonia, and aeration for confined area; not required in the field.
Persistence	About 30 minutes to 3 hours in summer; 10 to 12 hours in winter.
Use	Delayed- or immediate-action casualty agent, depending upon dosage rate.

Nerve Agents.

Nerve agents are organophosphate ester derivatives of phosphoric acid. They are generally divided into the G-agents, which in the unmodified state are volatile, and the V-agents, which tend to be more persistent. Even G-agents are capable of being thickened with various substances to increase the persistence and penetration of the intact skin. The principal nerve agents are Tabun (GA), Sarin (GB), Soman (GD), and VX. (In some countries the V-agents are known as A-agents.)

The G-agents are fluorine- or cyanide-containing organophosphates. In pure form they are colorless liquids. Their solubility in water ranges from complete miscibility for GB to almost total insolubility for GD. They have a weakly fruity odor but in field concentrations are odorless. Clothing gives off G-agents for about 30 minutes after contact with vapor; consider this fact before unmasking.

The V-agents are sulfur-containing organophosphorous compounds. They are oily liquids with high boiling points, low volatility, and resultant high persistency. They are primarily contact hazards. They are exceptionally toxic; the limited amount of vapor they produce is sufficient to be an inhalation hazard. They have very limited volubility in water and are hydrolyzed only minimally. V-agents affect the body in essentially the same manner as G-agents.

The nerve agents are all viscous liquids, not nerve gas per se. However, the vapor pressures of the G-series nerve agents are sufficiently high for the vapors to be lethal rapidly. The volatility is a physical factor of most importance. GB is so volatile that small droplets sprayed from a plane or released from a shell exploding in the air may never reach the ground. This total volatilization means that GB is largely a vapor hazard. At the other extreme agent VX is of such low volatility that it is mainly a liquid contact hazard. Toxicity can occur from the spray falling on one's skin or clothes and from touching surfaces on which the spray has fallen. GD is also mainly a vapor hazard, while GA can be expected to contaminate surfaces for a sufficiently long time to provide a relevant contact hazard.

Thickeners added to GD increase persistence in the field. The thickened agents form large droplets that provide a greater concentration reaching the ground and a greater contact hazard than the unthickened forms.

The relative volubility of these compounds in water and soil is of significance because it relates to their disposition.

The ability of GB and GA to mix with water means that water could wash them off surfaces, that these agents can easily contaminate water sources, and that they will not penetrate skin as readily as the more fat-soluble agents VX and GD. G-agents spread rapidly on surfaces, such as skin; VX spreads less rapidly, and the thickened agents very slowly. The moist surfaces in the lungs absorb all the agents very well.

Both the G- and V-agents have the same physiological action on humans. They are potent inhibitors of the enzyme acetylcholinesterase (AChE), which is required for the function of many nerves and muscles in nearly every multicellular animal. Normally, AChE prevents the accumulation of acetylcholine after its release in the nervous system. Acetylcholine plays a vital role in stimulating voluntary muscles and nerve endings of the autonomic nervous system and many structures within the central nervous system. Thus, nerve agents that are cholinesterase inhibitors permit acetylcholine to accumulate at those sites, mimicking the effects of a massive release of acetylcholine. The major effects will be on skeletal muscles, parasympathetic end organs, and the central nervous system.

Individuals poisoned by nerve agents may display the following symptoms:

- Difficulty in breathing.
- Drooling and excessive sweating.
- Nausea.
- Vomiting, cramps, and loss of bladder/bowel control.
- Twitching, jerking, and staggering.
- Headache, confusion, drowsiness, coma, and convulsion.

The number and severity of symptoms depend on the quantity and route of entry of the nerve agent into the body. When the agent is inhaled, a prominent symptom is pinpointing of the pupils of the eyes and dimness of vision because of the reduced amount of light entering. However, if exposure has been through the skin or by ingestion of a nerve agent, the pupils may be normal or only slightly to moderately reduced in size. In this event, diagnosis must rely upon the symptoms of nerve agent poisoning other than its effects on the pupils.

Exposure through the eyes produces a very rapid onset of symptoms (usually less than 2 to 3 minutes). Respiratory exposure usually results in onset of symptoms in 2 to 5

minutes; lethal doses kill in less than 15 minutes. Liquid in the eye kills nearly as rapidly as respiratory exposure.

Symptoms appear much more slowly from skin absorption. Skin absorption great enough to cause death may occur in one to two hours. Respiratory lethal dosages kill in one to ten minutes, and liquid in the eye kills nearly as rapidly. Very small skin dosages sometimes cause local sweating and tremors but little other effects. Nerve agents are cumulative poisons. Repeated exposure to low concentrations, if not too far apart, will produce symptoms.

Treatment of nerve agent poisoning includes use of the nerve agent antidote (atropine and 2-PAM chloride). Atropine blocks acetylcholine; 2-PAM Cl, reactivates the enzyme AChE. As time passes without treatment the binding of nerve agents to AChE "ages" and the 2-PAM Cl can no longer remove the agent. Certain agents, such as GD,

that age rapidly may resist treatment if it is not prompt. Therefore, an antidote enhancer, pyridostigmine bromide (PB), is available to US forces in active theaters of operation. PB pretreatment increases the victim's survivability when the antidote is used after exposure to nerve agents.

Tabun (GA)

GA is a brownish to colorless liquid that gives off a colorless vapor. GA (Table 2-3) was the first of the nerve agents developed by the Germans before World War II. It is about 30 times as toxic as phosgene, which was used in WWI. It enters the body primarily through the respiratory tract, but it is also highly toxic through the skin and digestive tract. It is approximately 20 times more persistent than GB but not as stable in storage.

Table 2-3. Tabun (GA).

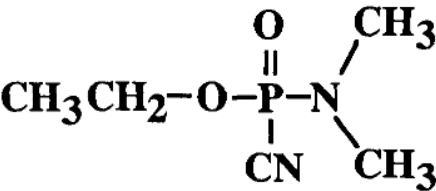
Alternate designation: Le-100.	
Chemical name: Ethyl N,N-dimethylphosphoramidocyanidate.	
Synonyms: Ethyl dimethylamidocyanophosphate; dimethylamidoethoxyphosphoryl cyanide.	
CAS registry number: 77-81-6.	
RTECS number: TB4550000.	
Physical and Chemical Properties	
Structural formula:	
	
Molecular formula: C ₅ H ₁₁ N ₂ O ₂ P.	
Molecular weight: 162.13.	
Physical state	Colorless to brown liquid.
Odor	Faintly fruity; none when pure.
Boiling point	220°C to 246°C at 760 mm Hg; 120°C at 9.75 mm Hg.
Melting point	-50°C.
Vapor density (compared with air)	5.63.
Liquid density	1.073 at 25°C.
Vapor pressure	0.037 mm Hg at 20°C; 0.006 at 0°C. This is about the same as mustard.
Volatility	858 mg/m ³ at 30°C; 328 mg/m ³ at 20°C (atmospheric pressure); 90 mg/m ³ at 0°C. This is approximately one-twentieth that of water.
Latent heat of vaporization	79.56 calories per gram (average value between 25°C and 50°C).
Flash point	78°C.
Decomposition temperature	Decomposes completely at 150°C for 3-1/4 hours. GA undergoes considerable decomposition when explosively disseminated.
Solubility	Slightly soluble in water: 9.8 percent at 25°C; 7.2 percent at 20°C. Readily soluble in organic solvents, such as alcohols, ethers, gasoline, oils, and fats.
continued	

Table 2-3, Tabun (GA) continued

Rate of hydrolysis	Slow with water but fairly rapid with strong acids or alkalies; self-buffering at pH 4.5. Autocatalytic below pH 4, because of presence of hydrogen cyanide (HCN). Half-life, 8.5 hours at pH 7 (20°C); 7 hours at pH 4 to 5. The rate of hydrolysis is increased by the presence of phosphate.
Hydrolysis products	Hydrogen cyanide and other products.
Stability in storage	GA is stable for several years when stored in steel containers at ordinary temperatures.
Action on metals or other materials	Slightly corrosive to steel.

Toxicity Data

Threshold eye effects (miosis)	2.5 mg-min/m ³ (estimated).
LC ₅₀ (respiratory)	Approximately 400 mg-min/m ³ (resting).
IC ₅₀ (respiratory)	Approximately 300 mg-min/m ³ (resting).
LC ₅₀ (percutaneous)	Unknown; probably between 20,000 and 40,000 mg-min/m ³ .
LD ₅₀ (percutaneous)	1 to 1.5 mg/person.
Rate of detoxification	Slight but definite.
Skin and eye toxicity	Eyes: Very high toxicity; much greater through eyes than through skin. Very low concentration of vapor causes pupil of eyes to constrict, resulting in difficulty in seeing in dim light. Skin: Very toxic. Decontamination of smallest drop of liquid agent is essential. Liquid penetrates skin readily.

Other Data

Protection required	Protective mask and protective clothing. Clothing gives off G-agents for about 30 minutes after contact with vapor; consider this fact before unmasking. Immediately remove all liquid from clothing.
Decontamination	Flush eyes with water immediately. Use the M258A1, M258 or M291 skin decontaminating kit for liquid agent on the skin. Decontaminate individual equipment with the M280 individual equipment decontamination kit. Calcium hypochlorite (HTH), supertropical bleach (STB), household bleach, caustic soda, dilute alkali solutions, or decontaminating solution number 2 (DS2) are effective on equipment. Use steam and ammonia or hot, soapy water in a confined area. Note: GA may react to form CK in bleach slurry.
Persistence	Depends upon munitions used and the weather. Heavily splashed liquid persists one to two days under average weather conditions. GA evaporates about 20 times more slowly than water. GA in water can persist about one day at 20°C and about six days at 5°C. GA persists about twice as long in sea water.
Use	Quick-acting casualty agent.

Sarin (GB)

The Germans developed GB after they developed GA, hence the designation GB (Table 2-4). It is a volatile liquid at room temperature. Pure GB is odorless and colorless.

The physiological symptoms of GB are essentially the same as those of other nerve agents.

Table 2-4. Sarin (GB).

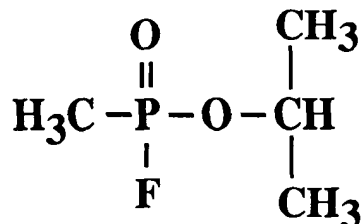
Alternate designation: Zarin.
Chemical name: Isopropyl methylphosphonofluoridate.
Synonyms: Isopropyl methylfluorophosphate; isopropoxymethylphosphoryl fluoride.
CAS registry number: 107-44-8.
RTECS number: TA8400000.

continued

Table 2-4, Sarin (GB) continued

Physical and Chemical Properties

Structural formula:

Molecular formula: C₄H₁₀FO₂P.

Molecular weight: 140.10.

Physical state	Colorless liquid.
Odor	Almost none in pure state.
Boiling point	158°C (pure); 151°C (plant grade).
Freezing point	-56°C.
Liquid density	1.102 at 20°C.
Vapor density (compared with air)	4.86.
Vapor pressure	2.10 mm Hg at 20°C.
Volatility	4,100 mg/m ³ at 0°C; 16,091 mg/m ³ at 20°C; 29,800 mg/m ³ at 30°C.
Latent heat of vaporization	80 calories per gram at 25°C.
Flash point	Nonflammable.
Decomposition temperature	Complete decomposition after 2-1/2 hours at 150°C.
Solubility	Soluble in all organic solvents, including alcohols, gasoline, oils, and fats. GB is miscible with water.
Rate of hydrolysis	Variable with pH. Half-life 7.5 hours at pH 1.8. Very rapidly hydrolyzed in alkaline solutions; half-life 5 hours at pH 9. Half-life 30 hours in unbuffered solution, 47 hours at pH 6.
Hydrolysis products	Hydrogen fluoride (HF) under acid conditions; isopropyl alcohol and polymers under alkaline conditions.
Stability in storage	Fairly stable in steel containers at 65°C. Stability improves with increasing purity.
Action on metals or other materials.	Slightly corrosive to steel.

Toxicity Data

LC ₅₀ (respiratory)	100 mg-min/m ³ (resting); 70 mg-min/m ³ (mildly active).
IC ₅₀ (respiratory)	75 mg-min/m ³ (resting); 35 mg-min/m ³ (mildly active).
LC ₅₀ (percutaneous)	12,000 mg-min/m ³ for naked person; 15,000 mg-min/m ³ for person in ordinary combat clothing.
IC ₅₀ (percutaneous)	Approximately 8,000 mg-min/m ³ with ordinary clothing.
Threshold eye effects (miosis)	1 mg-min/m ³ .
Rate of detoxification	Low; essentially cumulative.
Skin and eye toxicity	Eyes: Very high toxicity; much greater through eyes than through skin. Vapor causes pupils of the eyes to constrict; vision becomes difficult in dim light. Skin: Lethal dose (LD) is 1.7 grams per person. Liquid does not injure skin but penetrates it rapidly. Immediate decontamination of the smallest drop is essential. Vapor penetrates skin also.
Rate of action	Very rapid; death usually results within 15 minutes after absorption of fatal dose.
	continued

Table 2-4, Sarin (GB) continued

Other Data	
Protection required	Protective mask and protective clothing. Clothing gives off G-agents for about 30 minutes after contact with vapor; consider this fact before unmasking. Immediately remove all liquid from clothing.
Decontamination	Flush eyes with water immediately. Use the M258A1, M258, or M291 skin decontaminating kit for liquid agent on the skin. Decontaminate individual equipment with the M280 individual equipment decontamination kit. HTH, STB, household bleach, caustic soda, dilute alkali solutions, or DS2 are effective on equipment. Use steam and ammonia or hot, soapy water in a confined area.
Persistence	Evaporates at approximately the same rate as water or kerosene. Duration depends upon munitions used and the weather. GB is less persistent than GA.
Use	Quick-acting casualty agent.

Soman (GD)

GD is a colorless liquid that gives off a colorless vapor. Soman is the most poisonous of the G-agents, apparently because of the ease with which it can penetrate into the central nervous system. The physiological effect of GD is essentially the same as that of GA and GB. However, after

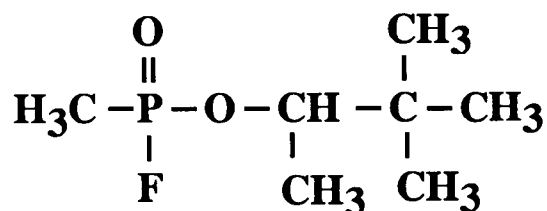
a few minutes antidotes are not as effective for GD poisoning as they are for other nerve agents. The addition of agent thickeners increases GD persistence and hazard. The usual thickened form of GD is designated TGD. VR-55 is probably another designation for thickened Soman. See Table 2-5.

Table 2-5. Soman (GD).

Alternate designation: Zoman.
Chemical name: Pinacolyl methyl phosphonofluoridate.
Synonyms: 3,3-dimethyl-n-but-2-yl methylphosphonofluoridate; methyl pinacolyl phosphonofluoridate; pinacolyl methylfluorophosphate.
CAS registry number: 96-64-0.
RTECS number: TA8750000.

Physical and Chemical Properties

Structural formula:



Molecular formula: C₇H₁₆FO₂P.

Molecular weight: 182.178.

Odor	Fruity; impurities give it the odor of camphor.
Freezing point	-42°C (generally solidifies to noncrystalline, glasslike material).
Boiling point	198°C.
Vapor density (compared with air)	6.33.
Liquid density	1.0222 at 25°C.
Vapor pressure	0.40 mm Hg at 25°C; 531 mg/m ³ at 0°C.
Volatility	3,900 mg/m ³ at 25°C; 5,570 mg/m ³ at 30°C. This is comparable to engine oil.
	continued

Table 2-5, Soman (GD) continued

Latent heat of vaporization	72.4 calories per gram at 25°C.
Flash point	High enough not to interfere with military use.
Decomposition temperature	Stabilized GD decomposes in 200 hours at 130°C. Unstabilized GD decomposes in 4 hours at 130°C.
Solubility	2.1 percent at 20°C and 3.4 percent at 0°C in water. Soluble in sulfur mustard, gasoline, alcohols, fats, and oils.
Rate of hydrolysis	Varies with pH; complete in 5 minutes in 5-percent sodium hydroxide (NaOH) solutions. Half-life at pH 6.65 and 25°C is 45 hours.
Action on metals or other materials	Slightly corrosive to metals.
Hydrolysis products	Essentially hydrogen fluoride (HF).
Stability in storage	Less stable than GA or GB.
Toxicity Data	
Threshold eye effects (miosis)	0.16 mg-min/m ³ .
LCt ₅₀ (respiratory)	70 mg-min/m ³ (mild activity for 10 minutes).
ICt ₅₀	Probably in GB and GA range.
LCt ₅₀ (percutaneous)	Estimated to be around 10,000 mg-min/m ³ .
Rate of detoxification	Low; essentially cumulative.
Skin and eye toxicity	Eyes: Very high toxicity; vapor causes pupils of eyes to contract, resulting in difficulty in seeing in dim light. Toxicity is much greater through eye than through skin. Skin: Extremely toxic by skin absorption. The estimated LD ₅₀ is 0.35 grams per person on bare skin (1.4 grams per person in ordinary clothing). Liquid does not injure skin but penetrates it rapidly. Immediate decontamination of the smallest drop is essential.
Rate of action	Very rapid. Death usually occurs within 15 minutes after absorption of fatal dose.
Other Data	
Protection required	Protective mask and protective clothing. Clothing gives off G-agents for about 30 minutes after contact with vapor; consider this fact before unmasking. Immediately remove all liquid from clothing.
Decontamination	Flush eyes with water immediately. Use the M258A1, M258, or M291 skin decontaminating kit for liquid agent on the skin. Decontaminate individual equipment with the M280 individual equipment decontamination kit. HTH, STB, household bleach, caustic soda, dilute alkali solutions, or DS2 are effective on equipment. Use steam and ammonia or hot, soapy water in a confined area.
Persistency	Depends upon munitions used and the weather. Heavily splashed liquid persists one to two days under average weather conditions. GD is calculated to evaporate about four times as slowly as water. Addition of agent thickeners can greatly increase persistency.
Use	Quick-acting casualty agent.

GF

The agent GF is a fluoride-containing organophosphate. It is a potential nerve agent. It is a slightly volatile liquid that is almost insoluble in water. It enters the body primarily through the respiratory tract but is also highly toxic

through the skin and digestive tract. It is a strong cholinesterase inhibitor. Toxicity information reports LD₅₀ values in mice from 16 µg/kg to 400 µg/kg, compared to LD₅₀ of 200 µg/kg for Sarin. It is approximately 20 times more persistent than Sarin. See Table 2-6.

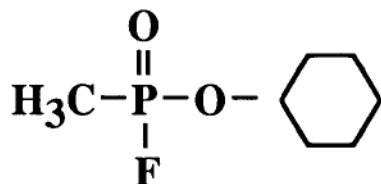
Table 2-6. GF.

Chemical name: O-Cyclohexyl-methylfluorophosphonate or cyclohexyl methylphosphonofluoridate (CMPF).

CAS registry number: 329-99-7

Chemical and Physical Properties

Structural formula:



Molecular formula: C₇H₁₄FO₂P.

Molecular weight: 180.2.

Physical state	Liquid.
Boiling point	239°C.
Freezing point	-30°C.
Liquid density	1.1327.
Vapor density	6.2.
Vapor pressure	0.044 mm Hg at 20°C.
Volatility	438 mg/m ³ at 20°C; 581 mg/m ³ at 25°C
Latent heat of vaporization	90.5 calories per gram.
Flash point	94°C.
Solubility	Almost entirely insoluble in water. 0.37% at 20°C.
Rate of hydrolysis	Very stable; hydrolyzes only when heated or in the presence of alkalies.
Stability in storage	Reasonably stable in steel at normal temperature.

Other Data

LD ₅₀ (subcutaneous)	Values are reported from 16 µg/kg to 400 µg/kg (mice).
Protection required	Protective mask and protective clothing.
Decontamination	Same as for GA or GB.
Persistence	GF is about as persistent as GA. GF evaporates about 20 times more slowly than water. Heavily splashed liquid persists one to two days under average weather conditions.
Use	Quick-acting casualty agent.

VX

The US standard V-agent is VX (Table 2-7). It is a very persistent, odorless, amber-colored liquid, similar in appearance to motor oil. Although VX is many times more persistent than the G-agents, it is very similar to GB in mechanism of action and effects. Because VX has low

volatility, liquid droplets on the skin do not evaporate quickly, thereby increasing absorption. VX by this percutaneous route is estimated to be more than 100 times as toxic as GB. VX by inhalation is estimated to be twice as toxic as GB.

Table 2-7. VX.

Chemical name: O-ethyl-S-(2-isopropylaminoethyl)methyl phosphonothiolate.

Synonyms: S-(2-diisopropylaminoethyl) o-ethyl methyl phosphonothiolate; ethyl-S-dimethylaminoethyl methylphosphonothiolate.

continued

Table 2-7, VX continued

Other Data	
Protection required	Protective mask and protective clothing.
Decontamination	HTH and STB slurries; household bleach; DS2 solution; hot, soapy water. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistence	Depends upon munitions used and the weather. Heavily splashed liquid persists for long periods under average weather conditions. In very cold weather VX can persist for months. VX is calculated to be approximately 1,500 times slower in evaporating than GB.
Use	Delayed casualty.

V_x

Another V-agent of interest is V_x, called “V sub x” (Table 2-8). Another designation for V_x is “V-gas.” The properties of V_x are similar to those of VX. It is nearly ten times more volatile than VX but is very persistent in comparison to the G-agents. The molecular weight of V_x is 211.2. Listed values are calculated, information on this agent is limited. The physiological action, protection, and decontaminants for V_x are the same as for VX.

Binary Nerve Agents (GB2 and VX2)

GB2 and VX2 are the designations for Sarin (GB) and agent VX which are formed in binary reactions. GB2 and

VX2 have been developed to decrease hazards of manufacturing, storing, and handling unitary nerve agents. In binary weapons two relatively nontoxic chemicals are mixed in flight to form the agent.

GB2 is formed by the reaction of methylphosphonic difluoride (DF) (see DF) with a mixture of isopropyl alcohol and isopropylamine (OPA) (see OPA).

VX2, binary VX, is formed by the action of O,O'-ethyl (2-diisopropylaminoethyl) methylphosphonite (see QL) with sulfur (see NE and NM).

Compounds used to produce the binary nerve agents are not chemical agents themselves; Chapter 3 discusses these compounds.

Table 2-8. V_x

Chemical and Physical Properties	
Physical state	Liquid, similar to VX.
Odor	None.
Boiling point	256°C (approximate).
Vapor density (compared with air)	7.29
Liquid density	1.062 g/cc at 25°C.
Vapor pressure	0.0042 and 0.0066 mm Hg at 20°C and 25°C, respectively.
Volatility	48 and 75 mg/m ³ at 20° and 25°C respectively.
Latent heat of vaporization	67.2 calories per gram.
Solubility	Soluble in organic solvents. Slightly soluble in water.

Blood Agents

Most blood agents are cyanide-containing compounds, absorbed into the body primarily by breathing. AC and CK are the important agents in this group. Blood agents are highly volatile and, therefore, nonpersistent even at very low temperatures. These agents can be dispersed by artillery shell, mortar, rocket, aircraft spray, or bomb. AC has an odor like bitter almonds; CK is somewhat more pungent. The odor of CK often goes unnoticed because CK is so irritating to the eyes, nose, and respiratory tract. At high

concentrations both compounds cause effects within seconds and death within minutes in unprotected personnel. The cyanides affect body functions by poisoning the cytochrome oxidase system, this poisoning prevents cell respiration and the normal transfer of oxygen from the blood to body tissues. Cyanogen chloride also acts as a choking agent. The standard protective mask gives adequate protection against field concentrations.

Hydrogen Cyanide (AC)

Although the US armed forces do not stockpile AC (Table 2-9), it is of interest because of its availability. It can be readily synthesized in large quantities and is commercially available. Potential enemies may hold large stockpiles. Some states use hydrogen cyanide for capital punishment. Pure AC is a nonpersistent, colorless liquid that is highly volatile. It is used as a quick-acting casualty agent that causes death within 15 minutes after a lethal dose has been received.

AC has a faint odor, similar to bitter almonds, that sometimes cannot be detected even in lethal concentrations. AC strongly stimulates breathing; the mask must be

put on as fast as possible. The pink color of the casualty's lips, fingernails, and skin suggests hydrogen cyanide poisoning. Exposure to high concentrations may cause instant loss of consciousness and death. A nonlethal dosage will cause moderate symptoms, but the patient can recover. Low doses have almost no effect on the body. AC is less persistent than other blood agents. The protective mask provides protection against field concentrations of AC. Liquid AC can penetrate skin. However, because of its high volatility, liquid AC is not likely to be encountered in the field, and protective clothing is required only in very unusual situations.

Table 2-9. Hydrogen cyanide (AC).

Alternate designations: Cyclone B; zyklon B.	
Chemical name: Hydrogen cyanide.	
Synonym: Hydrocyanic acid.	
CAS registry number: 74-90-8.	
RTECS number: MW6825000.	
Chemical and Physical Properties	
Structural formula:	
H-C≡N	
Molecular formula: HCN.	
Molecular weight: 27.03.	
Physical state	Colorless liquid that quickly evaporates.
Odor	Bitter almonds or peach kernels.
Freezing point	-13.3°C.
Boiling point	25.7°C.
Vapor density (compared to air)	1.007 at 25.7°C; 0.990 at 20°C; 0.978 at 0°C; 0.93 at -17.8°C.
Liquid density	0.687 g/cc at 20°C; 0.716 at 0°C.
Vapor pressure	612 mm Hg at 20°C; 265 mm Hg at 0°C.
Volatility	1,080,000 mg/m ³ at 25°C; 441,000 mg/m ³ at 0°C; 37,000 mg/m ³ at -40°C. This is approximately 50 times as volatile as water.
Latent heat of vaporization	233 calories per gram.
Flash point	-18°C. Agent ignites 50 percent of the time when disseminated from an artillery shell.
Decomposition temperature	Above 65.5°C. Forms explosive polymer on standing. Stabilized material can be stored up to 65°C.
Solubility	Highly soluble and stable in water and alcohol; soluble in ether, glycerine, chloroform, and benzene.
Rate of hydrolysis	Low under field conditions.
Hydrolysis products	Ammonia, formic acid (HCOOH), and amorphous brown solids.
Stability in storage	Unstable except when very pure. Forms explosive polymer on long standing. Will stabilize with addition of small amounts of phosphoric acid or sulfur dioxide. (See decomposition temperature.)
continued	

Table 2-9, Hydrogen cyanide (AC) continued

Action on metals or other materials	Little or none.
Toxicity Data	
LC ₅₀	Varies widely with concentration because of the rather high rate at which the body detoxifies AC. At 200 mg/m ³ concentration, the LC ₅₀ is approximately 2,000 mg-min/m ³ , whereas at 150 mg/m ³ the LC ₅₀ is approximately 4,500 mg-min/m ³ .
IC ₅₀	Varies with the concentration.
Rate of detoxification	Rapid; 0.017 mg/kg/min.
Skin and eye toxicity.	Moderate.
Rate of action	Very rapid. Incapacitation occurs within 1 or 2 minutes of exposure to an incapacitating or lethal dose. Death can occur within 15 minutes after receiving a lethal dose.
Other Data	
Protection required	Protective mask. Liquid AC can penetrate skin but, because AC has a high LC ₅₀ and because liquid AC is not likely to be encountered in the field, protective clothing is required only in unusual situations.
Decontamination	None required under field conditions.
Persistency	Short; the agent is highly volatile, and in the gaseous state it dissipates quickly in the air.
Use	Quick-acting casualty agent suitable for surprise attack.

Cyanogen Chloride (CK)

CK (Table 2-10) is a colorless, highly volatile liquid with a pungent, biting odor that will go unnoticed because of the agent's tearing and irritating properties. Although CK quickly evaporates, vapors may persist in the forest or jungle for some time under suitable weather conditions. Normally, CK is nonpersistent and is used as a quick-acting casualty agent.

CK irritates the respiratory tract similar to phosgene; fluid may accumulate in the lungs much faster than in

phosgene poisoning. Skin and eye toxicity are too low to be of military importance, but CK is highly irritating to eyes and mucous membranes. The general action of CK, interference with use of oxygen by the body tissues, is similar to that of AC. However, CK differs from AC in that it has strong irritating and choking effects and slows breathing. The protective mask protects against CK; a high concentration, however, may degrade the filter and reduce the mask's protective capability.

Table 2-10. Cyanogen chloride (CK).

Alternate designations: Mauguinite; CC; Klortsian.	
Chemical name: Cyanogen chloride.	
Synonyms: Chlorcyan; chlorine cyanide; chlorocyanogen.	
CAS registry number: 506-77-4.	
RTECS number: GT2275000.	
Physical and Chemical Properties	
Structural formula:	Cl-C≡N
Molecular formula: CNCl.	
Molecular weight: 61.48.	
Physical state	Colorless liquid that evaporates quickly.
Odor	Pungent, biting; but will go unnoticed because of its intense irritating and tearing properties.
Freezing point	-6.9°C.
Boiling point	12.8°C.
Liquid density	1.18 at 20°C.
continued	

Table 2-10, Cyanogen chloride (CK) continued

Vapor density (compared to air)	2.1.
Vapor pressure	1,000 mm Hg at 25°C.
Volatility	6,132,000 mg/m ³ at 25°C; 2,600,000 mg/m ³ at 12.8°C.
Latent heat of vaporization	103 calories per gram. This is sufficiently high to provide a satisfactory pancaking effect.
Flash point	None.
Decomposition temperature	Above 100°C.
Solubility	Slightly soluble in water; dissolves readily in alcohol, carbon disulfide, acetone, benzene, carbon tetrachloride, chloropicrin, HD, and AC.
Rate of hydrolysis	Very low.
Hydrolysis products	Hydrogen chloride and cyanic acid (CNOH).
Stability in storage	Stable at 65°C for 30 days. Will polymerize to form the solid cyanuric chloride which is corrosive. Impurities promote polymerization; may explode .
Action on metals or other materials	None if CK is dry. Attacks many common metals when stored unstabilized. See stability.

Toxicity Data

Median concentration detectable (by tearing effect)	12 mg/m ³ .
LC ₅₀	11,000 mg-min/m ³ .
IC ₅₀	7,000 mg-min/m ³ .
Rate of detoxification	0.02 to 0.1 mg/kg/min.
Skin and eye toxicity	Too low to be of military importance; highly irritating to eyes, upper respiratory tract, and lungs. CK can cause dryland drowning.
Rate of action	Immediate intense irritation. The systemic effect of CK is believed to arise from its conversion to AC in the body. In general, CK may be considered a rapid-acting chemical agent.

Other Data

Protection required	Protective mask. CK will break or penetrate a protective mask canister or filter element more readily than most other agents. A very high concentration may overpower the filter; high dosages will break down its protective ability.
Decontamination	None required under field conditions.
Persistency	Short. Vapor may persist in jungle and forest for some time under suitable weather conditions.
Use	Quick-acting casualty agent. Used for degradation of canisters or filter elements in protective mask.

Arsine (SA)

SA is a gas with a mild, garliclike odor. It is used as a delayed-action casualty agent that interferes with the functioning of the blood and damages the liver and kidneys. Slight exposure causes headache and uneasiness. In-

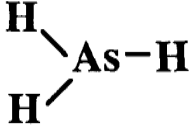
creased exposure causes chills, nausea, and vomiting. Severe exposure damages blood, causing anemia. It is a carcinogen. The protective mask provides adequate protection. See Table 2-11.

Table 2-11. Arsine (SA).

Alternate designation: Arthur.
Chemical name: Arsenic trihydride; arsine.
CAS registry number: 7784-42-1.
RTECS number: CG6475000.

continued

Table 2-22, Arsine (SA) continued

Physical and Chemical Properties	
Structural formula:	
	
Molecular formula: AsH ₃ .	
Molecular weight: 77.93.	
Physical state	Gas.
Odor	Mild, garliclike.
Boiling point	-62.5°C.
Freezing point	-116°C.
Liquid density	1.34 at 20°C.
Vapor density (compared to air)	2.69.
Vapor pressure	11,100 mm Hg at 20°C. This high vapor pressure means that SA is difficult to liquify and to store.
Volatility	30,900,000 mg/m ³ at 0°C. This is by far the highest volatility found among the compounds considered for tactical use as chemical agents. This fact, coupled with a relatively low latent heat of vaporization, qualifies SA as the most rapidly dispersing chemical agent.
Latent heat of vaporization	53.7 calories per gram at -62.5°C.
Flash point	SA ignites so easily that it cannot be used in shells. It may also explode in air.
Decomposition temperature	280°
Rate of hydrolysis	Rapid, but reaches an equilibrium condition quickly. (Under certain conditions SA forms a solid product with water that decomposes at 30°C.)
Hydrolysis products	Arsenic acids and a hydride containing fewer hydrogen atoms than SA itself.
Stability in storage	Not stable in uncoated metal containers. Metals catalyze decomposition of SA.
Action on metals or other materials	Reacts slowly with copper, brass, and nickel. Contact with other metals may also decompose it.
Toxicity Data	
LC ₅₀	5,000 mg-min/m ³ . It is estimated that 2 milligrams of SA per kilogram of body weight would be lethal to humans.
IC ₅₀	2,500 mg-min/m ³ .
Rate of detoxification	Not rapid enough to be of importance.
Skin and eye toxicity	None.
Rate of action	Effects are delayed from 2 hours to as much as 11 days.
Other Data	
Protection required	Protective mask.
Decontamination	None required.
Persistency	Short.
Use	Delayed-action casualty agent.

Section II. Blister Agents (Vesicants)

All of the blister agents are persistent, and all may be employed in the form of colorless gases and liquids. Blister agents damage any tissue that they contact. They affect the eyes and lungs and blister the skin. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when absorbed. Vesicants poison food and water and make other supplies dangerous to handle. They may produce lethalties, but skin damage is their main casualty-producing effect. The severity of a blister agent burn directly relates to the concentration of the agent and the duration of contact with the skin. In addition to casualty production, blister agents may also be used to restrict use of terrain, to slow movements, and to hamper use of materiel and installations.

During World War I mustard (H) was the only blister agent in major use. It had a recognizable, distinctive odor and a fairly long duration of effectiveness under normal weather conditions. Since then, odorless blister agents have been developed that vary in duration of effectiveness. Most blister agents are insidious in action; there is little or no pain at the time of exposure. Exceptions are lewisite and

phosgene oxime (CX), which cause immediate pain on contact. CX produces a wheal (similar to a bee sting) rather than a water blister, which the other blister agents produce.

Note: Fluid in mustard agent blisters may be quite irritating fluid in lewisite blisters is nontoxic and nonvesicant.

Blister agents can be described as mustards, arsenicals, or urticants. The mustards (H, HD, HN-1, HN-2, and HN-3) contain either sulfur or nitrogen. The next paragraphs discuss the mustards. The arsenical (ethyl-dichloroarsine [ED], methyl-dichloroarsine [MD], and phenyl-dichloroarsine [PD]) are a group of related compounds in which arsenic is the central atom. Arsenical hydrolyze rapidly and are less toxic than other agents of military interest. The discussion of arsenical chemical agents appears later in this chapter. Also later in this chapter is a discussion of urticants and, specifically, the principal urticant of military interest, CX. Mustards and arsenical are sometimes mixed to alter their properties for military effectiveness; they may also be employed with thickeners.

Mustards

This group of agents includes the sulfur mustards (H and HD) which are chlorinated thioethers, and the nitrogen mustards (HN-1, HN-2, and HN-3) which are considered derivatives of ammonia. The nitrogen mustards have nitrogen as the central atom with the hydrogen atoms replaced by various organic groups. Derivatives of the nitrogen mustards have been used in the treatment of certain types of cancer. HD and HN-3 are the principal military representatives of sulfur and nitrogen mustards.

The mustards can penetrate skin and a great number of materials. These materials include wood, leather, rubber, and paints. Because of their physical properties, mustards are very persistent under cold and temperate conditions. It is possible to increase their persistency even more by dissolving them in thickeners. Mustards are less persistent in hot climates but can reach relatively high concentrations in air because of greater evaporation rate.

Levinstein Mustard (H)

Levinstein mustard is the original mustard (gas) of World War I vintage. It contains about 30-percent sulfur impurities, which give it a pronounced odor. These impurities lessen the effectiveness of H but depress its freezing point two to five degrees. Other properties of H are essentially the same as those for distilled mustard, which is discussed next.

Distilled Mustard (HD)

HD originally was produced from H by a purification process of washing and vacuum distillation. HD (Table 2-12) is a colorless to amber-colored liquid with a garliclike odor. HD has less odor and a slightly greater blistering power than H and is more stable in storage. It is used as a delayed-action casualty agent, the duration of which depends upon the munitions used and the weather. Although HD is heavier than water, small droplets will float on water surfaces and present a hazard.

The effects of HD are usually delayed for 4 to 6 hours, but latent periods have been observed for up to 24 hours. The higher the concentration, the shorter the interval of time from exposure to the first symptoms. Mustard acts first as a cell irritant and finally as a cell poison on all tissue surfaces contacted. Early symptoms include inflammation of the eyes; inflammation of the nose, throat, trachea, bronchi, and lung tissue; and redness of the skin; blistering or ulceration may follow. Effects may include a more "at-ease" attitude, vomiting, and fever, beginning about the same time as skin reddening. The eyes are very sensitive to low concentrations; skin damage requires higher concentrations. Wet skin absorbs more mustard than does dry skin. For this reason HD exerts a casualty effect at lower concentrations in hot, humid weather, because the body is moist with perspiration. The protective mask and clothing

provide adequate protection, but protection against large droplets, splashes, and smears requires impermeable clothing. HD has a very low detoxification rate; therefore, very small repeated exposures are cumulative in the body.

Individuals also can become sensitized to mustard. Amounts approaching the lethal dose make casualties

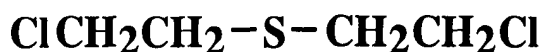
more susceptible to local and overwhelming infections than the normal individual. Injuries produced by HD heal much more slowly and are more susceptible to infection than burns of similar intensity produced by physical means or by most other chemicals.

Table 2-12. Distilled mustard (HD).

Alternate designations: HS; Kampstoff "Lost"; mustard gas; S-Lost; Schwefel-Iost; sulfur mustard; Y; Yellow Cross liquid; Yperite
Chemical name: Bis-(2-chloroethyl) sulfide.
Synonyms: 2,2'-dichloroethyl sulfide; 1,1'-thiobis(2-chloroethane).
CAS registry number: 505-60-2.
RTECS number: WQ0900000.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₄H₈Cl₂S.

Molecular weight: 159.08.

Physical state	Oily, colorless to amber liquid.
Odor	Like garlic or horseradish.
Boiling point	227.8°C (216°C calculated; decomposes).
Freezing point	14.45°C.
Solid density	1.37 g/cm ³ at 0°C.
Liquid density.	1.27 g/cm ³ at 20°C.
Vapor density (compared to air)	5.4.
Vapor pressure	0.072 mm Hg at 20°C.
Volatility	75 mg/m ³ at 0°C (solid); 610 mg/m ³ at 20°C (liquid); 2,860 mg/m ³ at 40°C (liquid).
Latent heat of vaporization	94 calories per gram. (This property is not of great importance in an agent of low volatility, as the sustained vapor concentration is essentially a function of the surrounding temperature.)
Flash point	105°C. Low enough to cause occasional ignition if explosive charges in the shell are too great.
Decomposition temperature	149°C to 177°C.
Solubility	Barely soluble in water (less than 1 percent); freely soluble in fats and oils, gasoline, kerosene, acetone, carbon tetrachloride, alcohol, PS, and FM. Miscible with DP, L, ED, PD, and the organophosphorus nerve agents.
Rate of hydrolysis	Half-life is 8.5 minutes in distilled water at 25°C and 60 minutes in salt water at 25°C. Mustard on or under water undergoes hydrolysis only if dissolved. It is only slightly soluble in water; as a result mustard may persist in water for long periods. Alkalinity and higher temperatures increase the rate of hydrolysis.
Hydrolysis products	Hydrogen chloride and thiodiglycol.
Stability in storage	Stable in steel or aluminum containers.
Action on metals or other materials	Very little when pure.
	continued

Table 2-12, Distilled mustard (HD) continued

Toxicity Data	
LD ₅₀	7 grams per person (estimated).
LC _{t50} (respiratory)	1,500 mg-min/m ³ .
IC _{t50} (respiratory)	150 mg-min/m ³ .
LC _{t50} (percutaneous)	10,000 mg-min/m ³ .
IC _{t50} (percutaneous)	2,000 mg-min/m ³ or less. Wet skin absorbs more mustard than does dry skin. For this reason, HD exerts a casualty effect at lower concentrations in hot, humid weather, because the body is then moist with perspiration. The dosage given above for skin absorption applies to temperatures of approximately 21°C to 27°C, as the body would not be perspiring excessively at these temperatures. Above 27°C perspiration causes increased skin absorption. The incapacitating dose requirement drops rapidly as perspiration increases; at 32°C 1,000 mg-min/m ³ could be incapacitating.
Eye injury (EC _{t50})	100 to 200 mg-min/m ³ .
Rate of detoxification	Very low. Even very small, repeated exposures of HD are cumulative in their effects or more than cumulative owing to sensitization. This has been shown in the postwar case histories of workers in mustard-filling plants. Exposure to vapors from spilled HD causes minor symptoms, such as "red eye." Repeated exposure to vapor causes 100-percent disability by irritating the lungs and causing a chronic cough and pain in the chest.
Skin and eye toxicity	Eyes are very susceptible to low concentrations; incapacitating effects by skin absorption require higher concentrations.
Rate of action	Delayed—usually 4 to 6 hours until first symptoms appear. Latent periods of up to 24 hours have been observed, however, and in rare cases of up to 12 days.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable clothing for protection against large droplets, splashes, and smears.
Decontamination	STB, fire, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Depends upon the amount of contamination by liquid, the munition used, the nature of the terrain and the soil, and the weather conditions. Heavily splashed liquid persists one to two days or more in concentrations that produce casualties of military significance under average weather conditions, and a week to months under very cold conditions. HD on soil remains vesicant for about two weeks. An incident in which mustard leaked and soaked into soil caused blisters after three years. HD is calculated to evaporate about five times more slowly than GB. Persistency in running water is only a few days, while persistency in stagnant water can be several months. HD is about twice as persistent in sea water.
Use	Delayed-action casualty agent.

Nitrogen Mustard (HN-1)

HN-1 (Table 2-13) is similar to mustard in its properties and effects; however, it is more volatile and less persistent than mustard but only one-fifth as damaging and not as stable. HN-1 is a colorless liquid with a faint, fishy or musty odor. It is used as a delayed-action casualty agent that has

a delay of 12 hours or more before skin-damaging symptoms are felt.

Nitrogen mustards act more quickly on the eyes than does HD. The eyes are very susceptible to low concentrations of nitrogen mustard, while a high concentration is required to significantly damage the skin or respiratory tract insofar as single exposures are concerned. Mild vapor

exposure may produce no skin lesions. Severe vapor exposure or exposure to liquid HN will result in redness of the skin, causing irritation and itching. Later blisters may appear in the red area. The skin lesions are similar to those caused by HD. The body does not detoxify HN-1; therefore, it is cumulative.

Effects on the respiratory tract are the same as those of mustard: irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. These

effects can progress to fever and labored breathing. Bronchial pneumonia may appear after 24 hours.

Following ingestion or systemic absorption, the nitrogen mustards injure the intestinal tract. Severe diarrhea, which may be bloody, occurs. Ingestion of 2 to 6 milligrams causes nausea and vomiting.

The protective mask and protective clothing provide adequate protection, but protection against large droplets, splashes, and smears requires impermeable clothing.

Table 2-13. Nitrogen mustard (HN-1).

Alternate designations: Ethyl S; NH-Lost; NOR nitrogen mustard; NSC 10873.	
Chemical name: Bis-(2-chloroethyl)ethylamine.	
Synonyms: Ethylbis(2-chloroethyl)amine.	
CAS registry number: 538-07-8.	
RTECS number: YE1225000.	
Chemical and Physical Properties	
Structural formula:	
$\text{CH}_3\text{CH}_2 - \text{N} \begin{array}{l} \diagup \text{CH}_2\text{CH}_2\text{Cl} \\ \diagdown \text{CH}_2\text{CH}_2\text{Cl} \end{array}$	
Molecular formula: C ₆ H ₁₃ Cl ₂ N.	
Molecular weight: 170.08.	
Physical state	Oily, colorless to pale yellow liquid.
Odor	Faint, fishy, or musty.
Freezing point	-34°C.
Boiling point	194°C calculated; decomposes. At atmospheric pressure HN-1 decomposes below boiling point.
Vapor density (compared to air)	5.9.
Liquid density	1.09 at 25°C.
Vapor pressure	0.24 mm Hg at 25°C.
Volatility	127 mg/m ³ at -10°C; 308 mg/m ³ at 0°C.; 1,520 mg/m ³ at 20°C; 3,100 mg/m ³ at 30°C. HN-1 closely parallels HD in the variation of volatility with temperatures and is of little value in producing a vapor hazard when weather is cold.
Latent heat of vaporization	77 calories per gram.
Flash point	High enough not to interfere with military use of the agent.
Decomposition temperature	Decomposes before boiling point is reached.
Solubility	Sparingly soluble in water; freely soluble in acetone and other organic solvents.
Rate of hydrolysis	Slow because of low solubility in water; less readily hydrolyzed than mustard.
Hydrolysis products	Hydroxyl derivatives and condensation products. (All intermediate hydrolysis products are toxic.)
Stability in storage	Adequate for use in munitions. Polymerizes slowly.
Action on metals or other materials	Slight corrosion of steel at 65°C.
	continued

Table 2-13, Nitrogen mustard (HN-1) continued

Toxicity Data	
LC ₅₀ (respiratory)	1,500 mg-min/m ³ .
LC ₅₀ (percutaneous)	20,000 mg/min/m ³ .
IC ₅₀ (percutaneous)	9,000 mg-min/m ³ .
IC ₅₀ (eye injury)	200 mg-min/m ³ .
Rate of detoxification	Not detoxified; cumulative.
Skin and eye toxicity	Eyes are very susceptible to low concentration; incapacitating effects by skin absorption require higher concentrations.
Rate of action	Delayed: 12 hours or longer.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable clothing for protection against large droplets, splashes, and smears.
Decontamination	HTH, STB, household bleach, fire, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Depends on munitions used and the weather. Somewhat shorter than duration of effectiveness for HD, the heavily splashed liquid of which persists one to two days under average weather conditions and a week or more under very cold conditions.
Use	Delayed-action casualty agent.

Nitrogen Mustard (HN-2).

HN-2 is a liquid with a fruity odor in high concentrations. It is rated as somewhat more toxic than HN-1. HN-2 affects the eyes in lower doses than do the other mustards. HN-2 has the greatest blistering power of the nitrogen mustards in vapor form but is intermediate as a liquid blistering

agent. Skin effects are delayed 12 hours or longer after exposure. The protective mask and protective clothing provide adequate protection, but protection against large droplets, splashes, and smears requires impermeable clothing. See Table 2-14. HN-2 is highly unstable and is not presently considered seriously as a chemical agent.

Table 2-14. Nitrogen mustard (HN-2).

Alternate designations: Dichloren; N-methyl-Lost; mustine; nitrogen mustard; NSC 762; S.

Chemical name: Bis-(2-chloroethyl)methylamine.

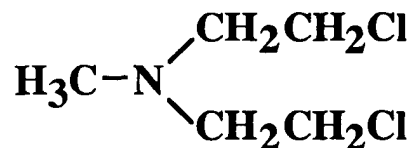
Synonyms: 2,2'-dichloro-N-methyldiethylamine; chloramine; N,N-bis(2-chloroethyl)methylamine.

CAS registry number: 51-75-2.

RTECS number: IA1750000.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₅H₁₁Cl₂N.

Molecular weight: 156.07

Physical state	Dark liquid.
Odor	Fruity in high concentrations. Like soft soap in low concentrations.
Freezing point	-65°C to -60°C.
	continued

Table 2-14. Nitrogen mustard (HN-2) continued

Boiling point	75°C at 15 mm Hg. At atmospheric pressure HN-2 decomposes below its boiling point.
Liquid density	1.15 at 20°C.
Vapor density (compared to air)	5.4.
Vapor pressure	0.29 mm Hg at 20°C; 1.25 mm Hg at 40°C.
Volatility	3,580 mg/m ³ at 25°C; 5,100 mg/m ³ at 30°C; 10,000 mg/m ³ at 40°C.
Latent heat of vaporization	78.8 calories per gram.
Flash point	High enough not to interfere with military use.
Decomposition temperature	Decomposes before boiling point is reached. Instability of HN-2 is associated with its tendency to polymerize or condense; the reactions involved could generate enough heat to cause an explosion.
Solubility	Soluble in acetone and organic solvents; sparingly soluble in water.
Rate of hydrolysis	Slow except where alkali is present. Dimerizes fairly rapidly in water.
Hydrolysis products	Complex condensates or polymers.
Stability in storage	Not stable.
Action on metals or other materials	None.

Toxicity Data

LC ₅₀ (respiratory)	3,000 mg-min/m ³ .
IC ₅₀ (eye injury)	100 mg-min/m ³ .
IC ₅₀ (percutaneous)	Somewhere between the values given for HN-1 and HN-3.
Rate of detoxification	Not detoxified.
Skin and eye toxicity	HN-2 has the greatest blistering power of the nitrogen mustards in vapor form but is intermediate as a liquid blistering agent. It produces toxic eye effects more rapidly than does HD.
Rate of action	Skin effects delayed 12 hours or longer.

Other Data

Physiological action	Same as for HD.
Protection required	See distilled mustard.
Decontamination	Same as for HD.
Persistency	Same as for HD based on evaporation; however, less than HD because of instability. See distilled mustard.
Use	Delayed-action casualty agent.

Nitrogen Mustard (HN-3).

HN-3 is the principal representative of the nitrogen mustards because its vesicant properties are almost equal to those of HD. It also is the most stable in storage of the three nitrogen mustards. Because of its low volatility, HN-3 does not constitute a grave vapor hazard to the skin in open air.

HN-3 is a liquid that has no odor in its pure form. It is used as a delayed-action casualty agent that has a persist-

ency that is considerably longer than HD. Because it is not detoxified, it is cumulative in the body. Most symptoms are delayed for four to six hours, but in some cases tearing, eye irritation, and intolerance to light develop immediately. The protective mask and protective clothing provide adequate protection, but protection against large droplets, splashes, and smears requires impermeable clothing. See Table 2-15.

Table 2-15. Nitrogen mustard (HN-3).

Alternate designations: Nitrogen mustard-3; TO.	
	continued

Table 2-15, Nitrogen mustard (HN-3) continued

Chemical name: Tris(2-chloroethyl)amine.

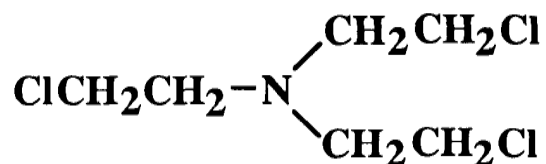
Synonyms: 2,2',2"-trichlorotriethylamine; tri(2-chloroethyl)amine.

CAS registry number: 555-77-1.

RTECS number: YE2625000.

Chemical and Physical Properties

Structural Formula:

Molecular formula: C₆H₁₂Cl₃N.

Molecular weight: 204.54.

Physical state	Oily liquid.
Odor	None when pure.
Freezing point	-3.7°C.
Boiling point	256°C calculated; decomposes. At atmospheric pressure HN-3 decomposes below boiling point.
Liquid density	1.24 at 25°C.
Vapor density (compared to air)	7.1.
Vapor pressure	0.0109 mm Hg at 25°C.
Volatility	13 mg/m ³ at 0°C; 121 mg/m ³ at 25°C; 180 mg/m ³ at 30°C; 390 mg/m ³ at 40°C. With no greater vapor toxicity than HD, HN-3 has a volatility too low to yield an effective vapor concentration even under tropical conditions.
Latent heat of vaporization	74 calories per gram.
Flash point	High enough not to interfere with military use.
Decomposition temperature	Decomposes before boiling point is reached.
Solubility	Soluble in sulfur mustards and chloropicrin. Insoluble in water, being less soluble than sulfur mustard. Soluble in ether, benzene, and acetone.
Rate of hydrolysis	Very slow because of low solubility in water.
Hydrolysis products	Hydrochloric acid and triethanolamine, N(CH ₂ CH ₂ OH) ₃ , in dilute solutions. Dimer formation in higher concentrations.
Stability in storage.	Stable enough for use as a bomb filling even under tropical conditions. However, the agent darkens and deposits a crystalline solid in storage.
Action on metals or other materials	None if HN-3 is dry.

Toxicity Data

LD ₅₀ (percutaneous)	0.7 gram per person (estimated).
LC _{t50} (respiratory)	1,500 mg-min/m ³ .
LC _{t50} (percutaneous)	10,000 mg-min/m ³ .
IC _{t50} (eye injury)	200 mg-min/m ³ .
IC _{t50} (percutaneous)	2,500 mg-min/m ³ . This information is based on estimates and indicates that HN-3 closely approaches HD in vapor toxicity and that it is the most toxic of the nitrogen mustards.
	continued

Table 2-15, Nitrogen mustard (HN-3) continued

Rate of detoxification	Not detoxified; cumulative.
Rate of action	Most symptoms are delayed four to six hours (same as for HD). In some cases eye irritation, tearing, and sensitivity to light (photophobia) develop immediately.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable clothing for protection against large droplets, splashes, and smears.
Decontamination	STB, fire, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Considerably longer than for HD (see HD).
Use	Delayed-action casualty agent.

Mustard-T Mixture (HT).

HT is a clear, yellowish, highly viscous liquid. It has a garliclike odor similar to HD. It is a mixture of 60-percent HD and 40-percent T. T is a sulfur, oxygen, and chlorine compound similar in structure to HD. HT is used as a delayed-casualty agent, the persistency of which depends on the munitions used and the weather. Properties are

essentially the same as those of HD, but HT is more stable, has a longer duration of effectiveness, and has a lower freezing point than HD. Its low volatility makes effective vapor concentrations in the field difficult to obtain. HT has a strong blistering effect. In addition to causing blisters, it irritates the eyes and is toxic when inhaled. See Table 2-16.

Table 2-16. Mustard-T mixture (HT).

Chemical name: HD Bis-(2-chloroethyl) sulfid; T Bis[2(2-chloroethylthio)ethyl] ether.

CAS registry numbers: HD 505-60-2; T 693-07-2

RTECS number: HD WQ0900000; T WQ3250000

Chemical and Physical Properties

Structural formula:



Molecular formula: HD C₄H₈Cl₂S; T C₈H₁₆Cl₂OS₂.

Molecular weight: HD 159.08; T 263.3; average 189.4 based on 60:40 weight percent.

Physical state	Clear, yellow liquid.
Odor	Like garlic.
Boiling point	Above 228°C.
Freezing point	0.0°C to 1.3°C for 60:40 mixture.
Liquid density	1.269 g/cm ³ at 25°C.
Vapor density (compared to air)	6.92 based on 60:40 mixture.
Vapor pressure	0.104 mm Hg at 25°C.
Volatility	831 mg/m ³ at 25°C.
Latent heat of vaporization	No data available.
Flash point	See HD (about 100°C).
	continued

Table 2-16, Mustard-T mixture (HT) continued

Decomposition temperature	165°C to 185°C.
Solubility	Barely soluble in water. Soluble in most organic solvents.
Rate of hydrolysis	See data on HD.
Hydrolysis products	Hydrogen chloride and thiodiglycol.
Stability in storage	Pressure develops in steel.
Action on metals or other materials	Very little when pure.
Toxicity Data	
LC ₅₀ (by any route)	None established in humans. See HD.
Rate of detoxification	See HD.
Skin and eye toxicity	Eyes are very susceptible to low concentrations. Incapacitating effects by skin absorption require higher concentrations than does eye injury. HT applied to the skin appears to be more active than HD.
Rate of action	No data available. See HD.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable protective clothing for protection against large droplets, splashes, and smears.
Decontamination	HTH, STB, household bleach, fire, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Depending on munitions used and the weather, the persistency of HT is somewhat longer than the duration of effectiveness of HD. Heavily splashed HD liquid persists one to two days under average weather conditions and a week or more under very cold conditions. It persists in water due to poor solubility.
Use	Delayed-action casualty agent.

Arsenical

The arsenical vesicants are a group of related compounds in which arsenic is the central atom. In these agents the hydrogen atoms of arsine (AsH_3) are replaced by various organic groups and chloride or cyanide. The main arsenical vesicants are lewisite (L), mustard-lewisite mixture (HL), and phenyldichloroarsine (PD).

All arsenical vesicants are colorless to brown liquids. In general, they are more volatile than mustard and have fruity to geraniumlike odors. They hydrolyze rapidly with water to lose most of their vesicant properties. They are much more dangerous as liquids than as vapors. The liquids will cause severe burns of the eyes and skin, while field concentrations of vapors are unlikely to cause permanent significant injuries. Absorption of either vapor or liquid through the skin in adequate dosage may lead to systemic intoxication or death. The rate of detoxification in sublethal amounts is rapid. Immediate decontamination is necessary to remove the liquid agents, but decontamination is not necessary for vapor unless pain is present. Inhaled vapors cause sneezing and may produce mild to moderate irritation of the upper respiratory tract. Arsenical are less toxic than other blister agents of military interest.

Lewisite (L).

Lewisite is the principal arsenical of military interest. It is a liquid with an odor like geraniums and very little odor when pure. It is used as a moderately delayed-action casualty agent with a persistency somewhat shorter than that of HD. When humidity is high, L hydrolyzes so rapidly that it is difficult to maintain a concentration sufficient to blister bare skin. It produces effects similar to mustard. One main difference is that L produces immediate pain.

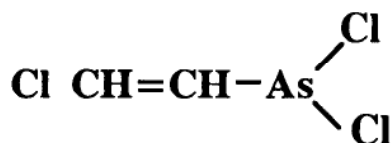
Lewisite warns of its presence by irritating the eyes and skin and has a rapid rate of action. Liquid L causes immediate burning sensation in the eyes and permanent loss of sight if not decontaminated within one minute with large amounts of water. It has about the same blistering action on the skin as does HD, even though the lethal dosage for L is much higher. Skin exposure to L produces an immediate and strong stinging sensation to the skin; reddening of the skin starts within 30 minutes. Blistering does not appear until after about 13 hours. Skin burns are much deeper than those caused by HD. When inhaled in high concentrations, lewisite may be fatal in as short a time as 10 minutes. The body does not detoxify L. See Table 2-17.

Table 2-17. Lewisite (L).

Alternate designation: Lyvizit.
Chemical name: Dichloro-(2-chlorovinyl)arsine.
Synonyms: Chlorovinylarsine dichloride; 2-chlorovinylidichloroarsine.
CAS registry number: 541-25-3.
RTECS number: CH2975000.

Chemical and Physical Properties

Structural formula:

Molecular formula: C₂H₂AsCl₃.

Molecular weight: 207.35.

Physical state	Colorless to brownish liquid.
Odor	Like geraniums. Very little odor when pure.
Freezing point	18°C to 0.1°C (depending on purity and isomers present).
Boiling point	190°C.
Vapor density (compared to air)	7.1.
Liquid density	1.89 at 20°C (much heavier than mustard).
Vapor pressure	0.087 mm Hg at 0°C; 0.394 mm Hg at 20°C. (Higher than HD at room temperature.)
Volatility	1,060 mg/m ³ at 0°C; 4,480 mg/m ³ at 20°C; 8,620 mg/m ³ at 30°C.
Latent heat of vaporization	58 calories per gram from 0°C to 190°C.
Flash point	None.
Decomposition temperature	Above 100°C.
Solubility	Soluble in organic solvents and oils; insoluble in water and dilute mineral acids. Because of its good miscibility with other chemical warfare agents, L is suitable for the preparation of tactical mixtures.
Rate of hydrolysis	Rapid for vapor and dissolved lewisite. Low solubility in water limits the hydrolysis.
Hydrolysis products	Hydrochloric acid and chlorovinyl arsenous oxide, a vesicant. The latter is a nonvolatile solid that is not readily washed away by rains. Strong alkalis destroy these blister-forming properties.
Stability in storage	Stable in steel or glass container.
Action on metals or other materials	None if L is dry.

Toxicity Data

LD ₅₀	30 mg/kg.
LC _{t50} (respiratory)	1,400 mg-min/m ³ . The intense irritation to the respiratory tract usually causes exposed personnel to mask immediately to avoid the vapor.
LC _{t50} (percutaneous)	100,000 mg-min/m ³ . When the humidity is high, L hydrolyzes so rapidly that it is difficult to maintain a vapor concentration sufficient to blister bare skin. The high vapor pressure and short duration of effectiveness of L further increase this difficulty.
IC _{t50} (percutaneous)	Over 1,500 mg-min/m ³ . Lewisite irritates the eyes and skin and gives warning of its presence.
	continued

Table 2-17, Lewisite (L) continued

Skin and eye toxicity	Even limited concentrations of L vapor (below 300 mg-min/m ³) cause extreme irritation of the eyes. Burning, pain, sensitivity to light, tearing, and swelling of the eyelids result. An exposure of 1,500 mg-min/m ³ produces severe and probably permanent corneal damage to the eyes. Liquids cause severe damage to the eye. L has about the same blistering action on the skin as HD, even though the lethal dosage for L is much higher.
Rate of detoxification	The body does not detoxify lewisite.
Rate of action	Rapid. The body absorbs L more rapidly through the skin than it absorbs the nitrogen mustards.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable protective clothing for protection against large droplets, splashes, and smears.
Decontamination	HTH, STB, household bleach, DS2, or caustic soda. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Somewhat shorter than for HD; very short duration under humid conditions.
Use	Moderately delayed-action casualty agent.

Mustard-Lewisite Mixture (HL).

HL is a variable mix of HD and L that provides a mixture of low freezing point for use in cold-weather operations or as high-altitude spray. Table 2-18 lists the properties for the mixture having the lowest possible freezing point, which is 37-percent HD and 63-percent L by weight. Other mixtures, such as 50:50, may be prepared to meet predetermined weather conditions and have advantages over the 3763 mixture because of the increased HD content. The persistency of HL depends on the munitions used and the weather.

HL has a garliclike odor from its HD content. It is used as a delayed-action casualty agent that is not detoxified in

the body. Contamination of the skin produces immediate stinging of the skin, which turns red within 30 minutes. Blistering, which tends to cover the entire area of the reddened skin, is delayed for about 13 hours. Liquid HL causes severe damage to eyes. The respiratory damage is similar to that produced by mustard, except in the most severe cases. In these cases fluid in the chest cavity may accompany fluid in the lungs. Liquid on the skin, as well as inhaled vapor, is absorbed and may cause poisoning throughout the body. These changes cause increased capillary permeability, which eventually causes shock and death because of the loss of fluid from the bloodstream.

Table 2-18. Mustard-lewisite (HL).

Chemical name: None (see components).	
Formula: None (see components).	
Molecular weight: 186.4 (calculated on basis of eutectic mixture of 37-percent HD and 63-percent L).	
Chemical and Physical Properties	
Physical state	Liquid.
Odor	Garliclike.
Freezing point	-42°C for plant purity HL (calculated); -25.4°C when pure.
Boiling point	Indefinite, but below 190°C.
Vapor density (compared to air)	6.5.
Liquid density	Between the densities of the components; approximately 1.66 at 20°C.
Vapor pressure	0.02 mm Hg at -10°C; 0.248 mm Hg at 20°C; 1.03 mm Hg at 40°C. (Calculated assuming mixtures obey Raoult's law. Values are somewhat higher than actual pressure.)
Volatility	240 mg/m ³ at -11°C; 2,730 mg/m ³ at 20°C; 10,270 mg/m ³ at 40°C calculated from above vapor pressure; actual volatility is somewhat lower.
Latent heat of vaporization	Intermediate between the heats of vaporization of the components.
	continued

Table 2-18, Mustard-lewisite (HL) continued

Flash point	High enough not to interfere with military use.
Decomposition temperature	Above 100°C.
Rate of hydrolysis	Rapid in the liquid or vapor state; slow at ordinary temperatures.
Hydrolysis products	Hydrogen chloride, thiodiglycol, and chlorovinylarsenious oxide. Alkaline hydrolysis destroys the blistering properties.
Stability in storage	Satisfactory in lacquered steel containers.
Action on metals or other materials	Little or none if dry.
Toxicity Data	
LC ₅₀ (respiratory)	About 1,500 mg-min/m ³ .
LC ₅₀ (percutaneous)	Above 10,000 mg-min/m ³ .
IC ₅₀ (eye injury)	About 200 mg-min/m ³ .
IC ₅₀ (percutaneous)	1,500 to 2,000 mg-min/m ³ .
Skin and eye toxicity	Very high.
Rate of detoxification	Not detoxified.
Rate of action	Produces immediate stinging of skin and redness within 30 minutes; blistering delayed about 13 hours.
Other Data	
Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable protective clothing for protection against large droplets, splashes, and smears.
Decontamination	Bleach, fire, DS2, or caustic soda. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Depends on munitions used and the weather. Somewhat shorter than that of HD, the heavily splashed liquid of which persists one to two days under average weather conditions and a week or more under very cold conditions.
Use	Delayed-action casualty agent.

Phenyldichloroarsine (PD)

PD is a colorless liquid that is used as a delayed-action casualty agent. Persistency depends on the munitions used and the weather. Although PD is classed as a blister agent, it also acts as a vomiting compound. Limited use of PD during World War I did not indicate any marked superiority over the other vomiting compounds used.

PD has an immediate effect on eyes and a delayed effect of 30 minutes to 1 hour on skin. PD blisters bare skin but wet clothing decomposes it immediately. The protective mask and protective clothing provide adequate protection, but protection against large droplets, splashes, and smears requires impermeable clothing. See Table 2-19.

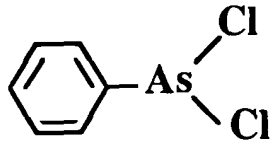
Table 2-19. Phenyldichloroarsine (PD).

Alternate designation: Pfiffikus; DJ; Sternite.
Chemical name: Phenyldichloroarsine.
Synonyms: Dichlorophenylarsine; phenylarsinedichloride.
CAS registry number: 696-28-6.
RTECS number: CH5425000.
continued

Table 2-19, Phenyldichloroarsine (PD) continued

Chemical and Physical Properties

Structural formula:

Molecular formula: $C_6H_5AsCl_2$.

Physical state	Liquid.
Odor	None.
Freezing point	-20°C.
Boiling point	252°C to 255°C.
Liquid density	1.65 at 25°C.
Vapor density (compared to air)	7.7.
Vapor pressure	0.033 mm Hg at 25°C; 0.113 mm Hg at 40°C.
Volatility	390 mg/m ³ at 25°C. If dispersed as an aerosol, it would be effective against unprotected troops although only as an agent with a short duration of effectiveness.
Latent heat of vaporization	69 calories per gram.
Flash point	High enough not to interfere with the military use.
Decomposition temperature	Stable to boiling point.
Solubility	Slightly soluble in water; miscible with alcohol, benzene, kerosene, petroleum, and olive oil.
Rate of hydrolysis	Rapid.
Hydrolysis products	Hydrochloric acid and phenylarsenious oxide.
Stability in storage	Very stable.
Action on metals or other materials	None.

Toxicity Data

Median concentration detectable (by nasal and throat irritation)	0.9 mg/m ³ .
LC ₅₀ (respiratory)	2,600 mg-min/m ³ .
IC ₅₀	16 mg-min/m ³ as a vomiting agent; 0 mg-min/m ³ as a vesicant.
Skin and eye toxicity	About 30 percent as toxic to the eyes as HD; 633 mg-min/m ³ would produce casualties by eye injury. On bare skin PD is about 90 percent as blistering as HD, but wet clothing decomposes it immediately.
Rate of detoxification	No specific information but, like related arsenicals, PD probably detoxifies rapidly in sublethal dosages.
Rate of action	Immediate effect on eyes; effects on skin are delayed 30 minutes to 1 hour.

Other Data

Protection required	Protective mask and permeable protective clothing for vapor and small droplets; impermeable protective clothing for protection against large droplets, splashes, and smears.
Decontamination	H ₂ O ₂ , STB, household bleach, caustic soda, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
	continued

Table 2-19, Phenyldichloroarsine (PD) continued

Persistency	Depends on munitions used and the weather. Somewhat shorter than that of HD under dry conditions; short duration when wet. (Heavily splashed liquid HD persists one to two days under average weather conditions and a week or more under very cold conditions.)
Use	Delayed-action casualty agent.

Ethylidichloroarsine (ED)

ED is a liquid with a fruity but biting and irritating odor. The Germans introduced ED (Table 2-20) in 1918 in an effort to produce a volatile agent with a short duration of effectiveness that would act more quickly than diphosgene or mustard and that would last longer in its effects than phenyldichloroarsine (PD).

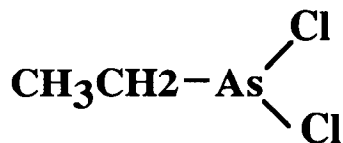
Like other chemical agents containing arsenic, ED is irritating to the respiratory tract and will produce lung injury upon sufficient exposure. The vapor is irritating to the eyes, and the liquid may produce severe eye injury. The absorption of either vapor or liquid through the skin in sufficient amounts may lead to systemic poisoning or death. Prolonged contact with either liquid or vapor blisters the skin.

Table 2-20. Ethylidichloroarsine (ED).

Alternate designation: Dick.
Chemical name: Ethylidichloroarsine.
Synonym: Dichloroethylarsine.
CAS registry number: 598-14-1.
RTECS number: CH3500000.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₂H₅AsCl₂.

Molecular weight: 174.88.

Physical state	Colorless liquid.
Odor	Fruity but biting and irritating.
Melting point	Less than -65°C.
Boiling point	156°C.
Vapor density (compared to air)	6.0.
Liquid density	1.66 at 20°C.
Vapor pressure	2.09 mm Hg at 20°C; 15.1 mm Hg at 50°C.
Volatility	6,500 mg/m ³ at 0°C; 20,000 mg/m ³ at 20°C; 27,200 mg/m ³ at 25°C.
Latent heat of vaporization	52.5 calories per gram.
Flash point	High enough not to interfere with the military use.
Decomposition temperature	Stable to boiling point.
Solubility	Soluble in ethyl chloride, alcohol, ether, benzene, acetone, and cyclohexane.
Rate of hydrolysis	Rapid.
Hydrolysis products	Hydrochloric acid and ethylarsenious oxide.
Stability in storage	Stable in steel.
	continued

Table 2-20. Ethyldichloroarsine (ED) continued

Action on metals or other materials	None on steel; attacks brass at 50°C; destructive to rubber and plastics.
Toxicity Data	
LC ₅₀ (respiratory)	3,000 to 5,000 mg-min/m ³ , depending upon the period of exposure. Because the body detoxifies ED at an appreciable rate, the product of concentration and time is not a constant; as time increases, concentration does not decrease proportionately. For example, exposure to 40 mg/m ³ for 75 minutes might have an effect similar to that produced by exposure to 30 mg/m ³ for 166 minutes.
LC ₅₀ (percutaneous)	100,000 mg-min/m ³ .
Temporary IC ₅₀ (respiratory)	5 to 10 mg-min/m ³ .
Skin and eye toxicity	Vapor is irritating but not harmful to eyes and skin except on prolonged exposure. Liquid ED has about one-twentieth the blistering action of liquid L.
Rate of detoxification	Sublethal amounts detoxify rapidly, similar to other arsenicals.
Rate of action	Irritating effect on nose and throat is intolerable after one minute at moderate concentrations; blistering effect is less delayed than with HD, which may be delayed 12 hours or longer.
Other Data	
Protection required	Protective mask and permeable protective clothing for ED vapor and small droplets; impermeable protective clothing for protection against large droplets, splashes, and smears.
Decontamination	Not usually necessary in the field. If necessary for enclosed areas, use HTH, STB, household bleach, caustic soda, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Short.
Use	Delayed-action casualty agent.

Methyldichloroarsine (MD)

MD is similar to ethyldichloroarsine. Like L and the other arsenical, MD causes immediate irritation of the eyes and nose with blistering effects delayed for hours. MD is irritating to the respiratory tract and produces lung injury upon sufficient exposure. The vapor irritates the eyes, and

the liquid may severely injure the eyes. The absorption of either vapor or liquid through the skin in sufficient amounts may lead to systemic poisoning or death. Prolonged contact with either liquid or vapor produces blistering of the skin. Vapor concentrations required for blistering effect are very difficult to attain in the field. See Table 2-21.

Table 2-21. Methyldichloroarsine (MD).

Alternate designations: Methyl-dick; Medikus.	
Chemical name: Methyldichloroarsine.	
Synonyms: Dichloromethylarsine; methylarsine dichloride.	
CAS registry number: 593-89-5.	
RTECS number: CH4375000.	
Chemical and Physical Properties	
Structural formula:	
$\text{CH}_3 - \text{As} \begin{array}{l} \diagup \text{Cl} \\ \diagdown \text{Cl} \end{array}$	
Molecular formula: CH ₃ AsCl ₂ .	
Molecular weight: 160.86	
Physical state	Liquid.
continued	

Table 2-21, Methylchloroarsine (MD) continued

Odor	None.
Freezing point	-55°C.
Boiling point	133°C.
Liquid density	1.836 at 20°C.
Vapor density (compared to air)	5.5
Vapor pressure	2.17 mm Hg at 0°C; 7.76 mm Hg at 20°C.
Volatility	74,900 mg/m ³ at 20°C.
Flash point	High enough not to interfere with military use.
Decomposition temperature	Stable to boiling point.
Latent heat of vaporization	49 calories per gram.
Solubility	Similar to ED.
Rate of hydrolysis	Very rapid.
Hydrolysis products	Hydrogen chloride and methylarsenic oxide.
Stability in storage	Stable in steel containers.
Action on metals or other materials	None on steel.

Toxicity Data

LC ₅₀	No accurate data; probably similar to ED, 3,000 to 5,000 mg-min/m ³ .
IC ₅₀ (respiratory)	25 mg-min/m ³ .
Skin and eye toxicity	Blistering action slightly less than that of HD. Has effect on eyes similar to that of L (produces corneal damage) but less severe. Vapor concentration required for blistering effect is very difficult to attain in the field.
Rate of detoxification	Detoxified at an appreciable rate.
Rate of action	Immediate irritation of eyes and nose. Blistering effect is delayed several hours.

Other Data

Protection required	Protective mask and protective clothing.
Decontamination	HTH, STB, household bleach, caustic soda, or DS2. Decontaminate liquid agent on the skin with the M258A1, M258, or M291 skin decontaminating kit. Decontaminate individual equipment with the M280 individual equipment decontamination kit.
Persistency	Relatively short.
Use	Delayed-action casualty agent.

Urticants

The urticants are compounds with a disagreeable, penetrating odor. They cause an immediate, severe, burning sensation; intense pain; and a feeling of numbness. They also cause swelling. Chemically, urticants are halogenated oximes. The most important of these is dichloroformoxime, also called phosgene oxime because of its similarity to phosgene. It may appear as a colorless, crystalline solid or as a liquid.

Phosgene oxime (CX) is one of the most violently irritating substances known. However, because of its extreme instability, pure CX is not likely to be used in military operations.

CX produces immediate pain varying from a mild pricking to almost intolerable pain resembling a severe bee sting. It has been called "nettle gas." It causes violent irritation to the mucous membranes of the eyes and nose. Even at low temperatures it has sufficient vapor pressure to produce tearing. When CX comes in contact with the skin, the area becomes pale in 30 seconds and a red ring surrounds the area. A wheal forms in about 30 minutes, the blanched area turns brown in about 24 hours, and a scab forms in about a week. The scab usually falls off in about 3 weeks. Itching may be present throughout healing, which in some cases may be delayed beyond 2 months. See Table 2-22.

Table 2-22. Phosgene oxime (CX).

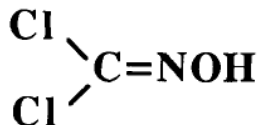
Alternate designation: Fosgen Oksim.

Chemical name: Dichloroformoxime.

Synonym: Phosgene oxime.

Chemical and Physical Properties

Structural formula:

Molecular formula: CHCl_2NO

Molecular weight: 113.9.

Physical state	Liquid above 39°C; solid below 35°C.
Odor	Intense, penetrating, disagreeable, and violently irritating.
Freezing/melting point	35°C to 40°C.
Boiling point	129°C (with decomposition).
Liquid density	Not available.
Vapor density (compared to air)	3.9.
Vapor pressure	11.2 mm Hg for solid at 25°C; 13 mm Hg for liquid at 40°C.
Volatility	1,800 mg/m ³ at 20°C; 76,000 mg/m ³ at 40°C.
Latent heat of vaporization	101 calories per gram at 40°C.
Flash point	Not available.
Decomposition temperature	Below 128°C.
Solubility	Dissolves slowly but completely in water; very soluble in organic solvents.
Rate of hydrolysis	Slow in water at pH 7 and room temperature; 5 percent in six days at ambient temperature. Not hydrolyzed by dilute acids; reacts violently in basic solutions.
Hydrolysis products	Carbon dioxide, hydrogen chloride, and hydroxylamine.
Stability in storage	Extremely unstable in presence of traces of metals or other impurities. Even traces of iron chloride may cause explosive decomposition. Pure material is stable only for one to two months. It may be stabilized by nitromethane, chloropicrin, glycine, ethyl acetate, or ether—but only in glass vessels below 20°C. Apparently, it is most stable in aromatic solvents.
Action on metals or other materials	Corrosive to most metals. See preceding paragraph.

Toxicity Data

LC ₅₀	3,200 mg-min/m ³ (estimated).
IC ₅₀	Unknown. The lowest irritant concentration after a ten-second exposure is 1 mg/m ³ . The effects of the agent become unbearable after one minute at 3 mg/m ³ .
Skin and eye toxicity	Violently irritating to eyes. Very low concentrations cause copious tearing, inflammation, and temporary blindness. Liquid on skin is corrosive.
Rate of detoxification	Not known.
Rate of Action	Rapid.

Other Data

Protection required	A properly fitting protective mask protects the respiratory system. A complete set of protective clothing will protect the remainder of the body.
	continued

Table 2-22, Phosgene oxime (CX) continued

Decontamination	Use large amounts of water or DS2 on equipment. Because of the rapid reaction of CX with the skin, decontamination will not be entirely effective after pain occurs. Nevertheless, decontaminate as rapidly as possible by flushing the area with large amounts of water to remove any agent that has not reacted with the skin.
Persistency	About two hours in soil. Relatively nonpersistent on surfaces and in water.
Use	Rapid-acting casualty agent.

Section III. Incapacitating Agents

Incapacitating agents are chemicals that cause physiological or mental effects that lead to temporary disability. Unlike riot control agents with effects lasting only a few minutes, incapacitating agents produce effects that may last for hours or days after exposure to the agent has ceased. Incapacitating agents differ from other chemical agents in that the lethal dose is many times greater than the incapacitating dose. Thus, they do not seriously endanger life except in cases exceeding many times the effective dose, and they produce no permanent injury. Medical treatment, although not required, may speed recovery.

Many compounds show potential as incapacitating agents. However, in actual use the term refers to those agents that—

- Produce their effects mainly by altering or disrupting the higher regulatory activity of the central nervous system (CNS).
- Have effects that last hours or days rather than being momentary or fleeting, as with tear agents.
- Do not seriously endanger life except at concentrations greatly exceeding the effective dose. They do not produce permanent injury.
- Allow recovery without treatment and without any permanent effects.
- Are highly potent and logistically feasible.

Incapacitating agents specifically do not include the following:

- Lethal agents that are incapacitating at sublethal doses, such as the nerve agents.
- Substances that cause permanent or long-lasting injury, such as blister agents and choking agents, and those that cause eye injury.

- Medical drugs that exert marked effects on the central nervous system, such as barbiturates, belladonna alkaloids, tranquilizers, and many of the hallucinogens. These drugs, although effective and relatively safe, are logistically infeasible for large-scale use because of the high doses required.
- Agents of temporary effectiveness that produce reflex responses that interfere with performance of duty. These include skin and eye irritants that cause pain or itching (vesicants or urticants), vomiting or cough-producing compounds (sternutators), and tear compounds (lacrimators).
- Agents that disrupt basic life-sustaining systems of the body and thus prevent the carrying out of physical activity. Examples include agents that lower blood pressure; paralyzing agents, such as curare; fever-producing agents; respiratory depressants; and blood poisons. Although theoretically effective, such agents almost invariably have a low margin of safety between the effective doses and the possible lethal doses. Thus, they affect the basic purpose of an incapacitating agent, which is to reduce military effectiveness without endangering life.

Despite restrictions imposed by the above definition, a great variety of mechanisms remain that could in theory disrupt CNS regulation and maintenance of performance. Only two general types of incapacitating agents are likely to be encountered in military use: the CNS depressants and the CNS stimulants.

Central Nervous System Depressants

CNS depressants are compounds that have the predominant effect of depressing or blocking the activity of the central nervous system, often by interfering with the transmission of information across synapses. An example of this type of agent is BZ, which appears to block the action of acetylcholine in the same way that atropine does. BZ, however, has far greater relative potency than atropine on the CNS.

Cannabinols and phenothiazine-type compounds are other potential incapacitating agents that seem to act basically as CNS depressants. The primary effects of these agents are to sedate and destroy motivation rather than disrupt the ability to think.

Other types of CNS depressants that could contain potential incapacitating agents are narcotics such as fentanyl or hypnotics.

BZ.

BZ, an odorless, white, crystalline solid, is a CNS depressant. BZ is usually disseminated as an aerosol with the primary route of entry into the body through the respiratory system; the secondary route is through the digestive tract. Skin absorption is possible with proper solvents.

BZ affects the victim's ability to remember, solve problems, pay attention, and understand instructions. Small doses of BZ cause sleepiness and decreased alertness. BZ also affects circulation of the blood, digestion,

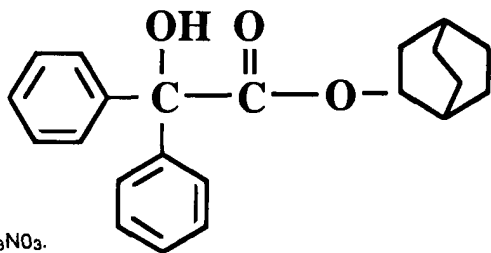
sweating, and vision. General symptoms from agent BZ are fast heartbeat, dry skin and lips, blurred near vision (increased pupil size), flushed skin, urinary retention, constipation, and sedation progressing to stupor and interference with ordinary activity. High doses produce extreme excitement, delusions, and hallucinations; high doses completely destroy the ability to perform any military task. An untreated casualty requires from three to four days to reach full recovery from the effects of BZ intoxication. See Table 2-23.

Table 2-23. BZ.

Alternate designation: Oksilidin.
Chemical name: 3-Quinuclidinyl benzilate.
Synonym: 1-azabicyclo(2.2.2)octan-3-ol, benzilate (ester).
CAS registry number: 13004-56-3 (C ₂₁ H ₂₃ NO ₃ ·HCl).
RTECS number: VD6300000 (C ₂₁ H ₂₃ NO ₃ ·HCl).

Chemical and Physical Properties

Structural formula:



Molecular formula: C₂₁H₂₃NO₃.

Molecular weight: 337.41.

Physical state	White, crystalline solid (20°C).
Odor	None.
Melting point	164°C to 167°C.
Boiling point	320°C.
Solid density	0.51 g/cm ³ (bulk); 1.33 g/cm ³ (crystal).
Vapor density	11.
Vapor pressure	Negligible.
Volatility	Negligible.
Latent heat of vaporization	62.9 calories per gram between 170°C and 194°C.
Flash point	246°C.
Decomposition temperature	Begins to decompose at about 170°C in air under prolonged heating; is almost completely decomposed after one to two hours at 200°C. Rate is both temperature- and purity-dependent.
Solubility	Slightly soluble in water; soluble in dilute acids, trichloroethylene, warm dimethylformamide, and most organic solvents, such as alcohol and chloroform; insoluble in aqueous alkali. Salts formed with inorganic and organic acids are soluble.
Rate of hydrolysis	Half-life at 25°C is 6.7 hours at pH 9.8; 1.8 minutes at pH 13 and 3 to 4 weeks in moist air. Half-life at 37°C is 95 hours at pH 7.4 and 10 hours at pH 9.
Hydrolysis products	3-Quinuclidinol and benzoic acid.
Stability in storage	Stable in most materials.

continued

Table 2-23, BZ continued

Action on metals or other materials	No effect on steel or stainless steel after three months at 71°C. Aluminum and anodized aluminum are lightly attacked after three months at 71°C.
Toxicity Data	
LC ₅₀	High; estimated to be 200,000 mg-min/m ³ .
IC ₅₀	112 mg-min/m ³ .
Inhalation threshold dose	2 mg/m ³ individual.
Rate of detoxification	From an IC ₅₀ dose severe effects last 36 hours; mild effects last 45 hours.
Rate of action	Delayed. Usual onset of symptoms occurs approximately two hours after aerosol exposure. Depending on inhaled or ingested dosage, symptoms may appear at times ranging from 30 minutes to 20 hours after exposure. Effects from skin contact may appear 36 hours later. Dimethylsulfoxide as a "carrier" increases the percutaneous effect by a factor of at least 25.
Other Data	
Protection required	The principles applied to the nerve agents apply equally as well to the incapacitating agents. It is possible that such agents will be disseminated by smoke-producing munitions or aerosols, using the respiratory tract as a route of entry. The use of the protective mask, therefore, is essential. The skin is usually a much less effective route.
Decontamination	Complete cleansing of the skin with soap and water at the earliest opportunity. If washing is impossible, use the M258A1, M258, or M291 skin decontamination kit. Symptoms may appear as late as 36 hours after contact exposure, even if the skin is washed within an hour. In fact, a delay in onset of several hours is typical. Use this time to prepare for the possibility of a widespread outbreak 6 to 24 hours after the attack. Decontaminate bulk quantities of BZ with caustic alcohol solutions. If BZ or other beiladonnoids are used as free bases, decontamination will require a solvent, such as 25-percent ethanol, 0.1N hydrochloric acid, or 5-percent acetic acid.
Persistency	Very persistent in soil and water and on surfaces.
Use	Delayed-action incapacitating agent.

Cannabinols and Phenothiazines.

Cannabinols and phenothiazine compounds are other potential incapacitating agents that seem to act basically as CNS depressants. The primary effects of these agents, however, are sedation and destruction of motivation rather than disruption of the ability to think.

Cannabinol is an active substance contained in hashish and in marijuana (*Cannabis*). Substances derived directly

from Cannabis and synthetic substances related to these parent materials have potential as incapacitants. Tetrahydrocannabinol (THC) is the principal active compound in marijuana. Table 2-24 shows the structure of the basic THC molecule.

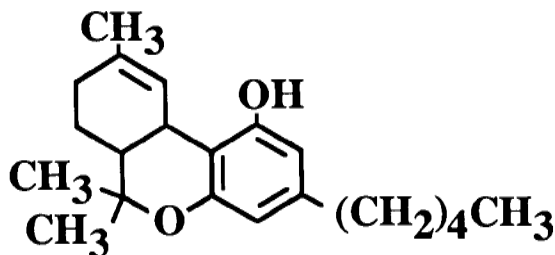
Synthetic analogues contain longer or more complex side chains and may involve the displacement of a double bond in one of the rings.

Table 2-24. THC.

CAS registry number: 33086-25-8.

RTECS number: HP8200000.

Structural formula:



Molecular formula: C₂₁H₃₀O₂.

Molecular weight: 314.51.

Inhaled natural cannabis produces effects within a few minutes. These effects peak at about one hour and subside after three to four hours. Ingested compound produces delayed, more prolonged effects. Some synthetic materials reportedly produce significant effects for up to several days. Signs and symptoms include feelings of unreality, intensification of sensations, difficulty in concentrating, lethargy, and sedation. No treatment is ordinarily required, and the effects subside spontaneously within a few hours.

Phenothiazine-like compounds have a very high safety index and would not be likely to involve any special medical care. The onset of action for phenothiazines is about five minutes, and effects last about one hour.

Fentanyl.

Fentanyls interact at the opiate receptor; that is, they act like morphine and are narcotics. Fentanyls are the most

potent painkillers therapeutically available. One analogue is 10,000 times as potent as morphine. Fentanyls depress respiration and heart rate and cause lethargy, sedation, and immobilization. Large doses produce muscle rigidity. They would probably be disseminated as aerosols. As a potential class of agents they have a rapid onset of action (10 to 90 seconds) and are extremely potent in producing incapacitation without loss of consciousness. Estimated effective intravenous doses range from 3 to 100 micrograms per kilogram ($\mu\text{g}/\text{kg}$). Effects last from minutes to several hours, depending on the structure. They can be disseminated as an aerosol. Decontamination would involve washing with water (acidified with acetic acid). Table 2-25 shows the basic structure for fentanyls.

Table 2-25. Fentanyl.

Synonyms: NCI-C56371; N-(1-phenethyl-4-piperidyl)propionanilide;

N-phenyl-N-[1-(2-phenylethyl)-4-piperidyl]propanamide;

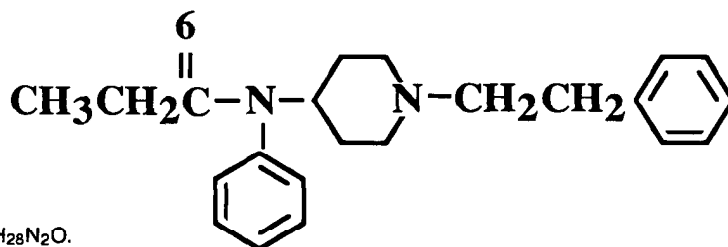
N-phenethyl-4-(n-propionylanilino)piperidine;

1-phenethyl-4-N-propionylanilinopiperidine.

CAS registry number: 437-38-7.

RTECS number: UE5550000.

Structural formula:



Molecular formula: $\text{C}_{22}\text{H}_{28}\text{N}_2\text{O}$.

Molecular weight: 336.48.

Central Nervous System Stimulants

CNS stimulants cause excessive nervous activity, often by boosting or facilitating transmission of impulses that might otherwise be insufficient to cross certain synapses. The effect is to flood the brain with too much information, making concentration difficult and causing indecisiveness

and inability to act in a sustained, purposeful manner. A well-known drug that appears to act in this manner is d-lysergic acid diethylamide (LSD). Large quantities of the amphetamines sometimes produce similar effects.

First Aid for Incapacitating Agents

Effects of small amounts of most incapacitating agents are entirely temporary. However, large doses of some, especially BZ compounds in tropical environments, can be serious and require first aid. The most important considerations are the following:

- If the casualty has a loss of sense or feeling (stupor) or is in a coma, be sure that respiration is unobstructed and turn him or her on the stomach with the head to the side to avoid strangulation should vomiting occur.

- Regard ambulatory casualties as potentially capable of resisting, and approach them with this possibility in mind. To prevent them from injuring themselves or others, confine them and isolate them, if possible, in a safe area. If no other means are available, restrain them by tying them each to a tree.
- Remove weapons and other potentially harmful materials from suspected casualties. This includes cigarettes, matches, medications, and small items they might ingest accidentally. Delirious casualties have tried to eat items bearing only a superficial resemblance to food.
- The most important single medical consideration with BZ is the possibility of heatstroke because the casualty cannot sweat. Remove excessive clothing if the temperature is more than 70°F. There is usually no danger of severe dehydration in the first 12 hours, despite dryness and coating of the lips and tongue, unless persistent vomiting occurs. Give fluids only when the casualty can drink unassisted. Check for bladder distention if voiding does not occur within 12 hours.
- Reassurance and a firm but friendly attitude by personnel providing first aid will help if the casualties appear to comprehend what is being said to them. Conversation is a waste of time, however, if a casualty is incoherent or cannot understand what is being said. In such cases, the less said the better; the casualty benefits more from prompt and vigorous restraint and evacuation to a treatment facility.

Unfamiliar agents or mixtures of agents may be encountered in future field situations. In such an instance the general principles of restraint, close observation, and supportive medical care remain valid. The judgment of the medical officer remains the only useful guide to action in these complex and unforeseeable circumstances.

See TM 8-285 for diagnosis and treatment for incapacitating agents. Symptoms and possible agent families are shown in Table 2-26.

Table 2-26. Correlation of symptoms and incapacitating agent family.

Signs and symptoms	Possible agent family
Dryness of mouth; slow pulse; elevated temperature; flushed face; blurred vision; dilated pupils; slurred or nonsensical speech; hallucinations; disrobing; mumbling and picking behavior; stupor and coma.	Anticholinergics (BZ).
Restlessness, dizziness, or giddiness; failure to obey orders, confusion; erratic behavior; stumbling or staggering; vomiting. See note.	Anticholinergics (e.g., BZ); Indoles (e.g., LSD); Cannabinols (e.g., marijuana); Other intoxications (e.g., alcohol, bromides, barbiturates, lead, etc.)
Inappropriate smiling or laughing; irrational fear; distractibility; difficulty in expressing self; perceptual distortions; labile increase in pupil size, heart rate, and blood pressure; stomach cramps and vomiting may occur.	Indoles. (Schizophrenic psychosis may mimic in some respects.)
Euphoric, relaxed, unconcerned daydreaming attitude; easy laughter; low blood pressure and dizziness on sudden standing.	Cannabinols.
Respiratory depression; slow pulse; lethargy; sedation; immobilization.	Fentanyls.
Tremor, clinging, or pleading; crying; decrease in disturbance with reassurance; history of nervousness or immaturity. See note.	

Note: Although these signs and symptoms can appear from an agent family, they may also appear from an anxiety reaction.

Chapter 3

Military Chemical Compounds and Their Properties

Military chemical compounds include vomiting and tear-producing agents (riot control agents); herbicides; and antimateriel, flame, and smoke materials. Vomiting compounds are not authorized for use by US forces in combat or in training.

This chapter contains physical and chemical properties of tear-producing and vomiting agents.

Section I. Tear-Producing and Vomiting Compounds

Tear-Producing Compounds

The tear compounds cause a flow of tears and irritation of the skin. Because tear compounds produce only transient casualties, they are widely used for training, riot control, and situations where long-term incapacitation is unacceptable. When used against poorly equipped guerrilla or revolutionary armies, these compounds have proved extremely effective. When released indoors, they can cause serious illness or death. The following paragraphs discuss the principal tear compounds of historic and current interest.

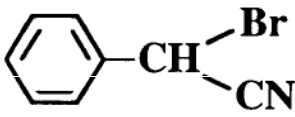
The standard tear-producing agents currently in the US Army inventory for riot control are CS, CS1, CS2, CSX, and CR. The United States considers agent CN (popularly known as mace or tear gas) and its mixtures with various

chemicals obsolete for military employment. This chapter includes these materials, however, for complete coverage of compounds with potential for use against US forces. This chapter also presents information regarding CN mixtures as an example of how agent properties can be tailored to the method of dissemination. This manual does not discuss herbicides or antimateriel, flame, or smoke materials.

Bromobenzylcyanide (CA)

CA was one of the first tear agents used. It is not as effective as CN or CS and is obsolete. CA produces a burning sensation of the mucous membranes and severe irritation and tearing of the eyes with acute pain in the forehead. See Table 3-1.

Table 3-1. Bromobenzylcyanide (CA).

Alternate designations: BBC; Iarmine; Camite.	
Chemical name: 4-Bromophenylacetylnitrile.	
Synonym: 4-Bromophenylacetoneitrile.	
CAS registry number: 16532-79-9.	
RTECS number: AL8090000.	
Chemical and Physical Properties	
Structural formula:	
Molecular formula: C ₈ H ₆ BrN.	
Molecular weight: 196.0.	

continued

Table 3-1, Bromobenzylcyanide (CA) continued

Physical state	Yellow solid or liquid, depending on temperature and purity.
Odor	Like soured or rotting fruit, but not unpleasant.
Melting point	25.5°C pure; 18.5°C plant purity.
Boiling point	242°C, but with decomposition.
Vapor density (compared to air)	6.7.
Solid density	1.52 at 20°C.
Liquid density	1.47 at 25°C.
Vapor pressure	0.011 mm Hg at 20°C.
Volatility	17 mg/m ³ at 0°C; 115 mg/m ³ at 20°C; 271 mg/m ³ at 30°C.
Latent heat of vaporization	79.5 calories per gram at 20°C; 55.7 calories per gram at boiling point.
Flash point	None; decomposes but does not burn.
Decomposition temperatures	Decomposes slowly at 60°C, more rapidly as the temperature increases. Decomposes completely at 242°C. Forms hydrobromic acid and dicyanostilbene.
Solubility	Soluble in organic liquids. Insoluble in water and cold alcohol.
Rate of hydrolysis	Very slow.
Hydrolysis products	Complex condensation products.
Stability in storage	Fairly stable in glass, lead-lined, or enamel-lined containers.
Action on metals or other materials	Vigorous corrosive action on all common metals except lead. Reaction with iron may be explosive.

Toxicity Data

LC ₅₀	Estimated 8,000 to 11,000 mg-min/m ³ . Volatility is too low to permit attaining a lethal dosage in the field. May reach lethal dosage in enclosed places.
IC ₅₀	About 30 mg-min/m ³ .
Minimal irritant concentration	0.3 mg/m ³ .
Rate of detoxification	Rapidly detoxifies at the low concentrations ordinarily encountered.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Instantaneous.

Other Data

Protection required	Protective mask.
Decontamination	Decontaminate clothing with steam or by boiling. Twenty-percent alcoholic caustic soda is effective on materiel but may damage it. Porous surfaces, such as earth, are very difficult to decontaminate.
Persistency	Depends on munitions used and the weather. Heavily splashed liquid persists one to two days under average weather conditions.
Use	Obsolete. Manual includes it to present a complete coverage of all potential military chemicals.

Chloroacetophenone (CN)

The symbol CN identifies the riot control agent popularly known as tear gas. A more effective riot control agent, CS, has replaced CN as a standard RCA. Solutions of CN, identified by the symbols CNC, CNB, and CNS, have also been employed as riot control agents. Because CN and mixtures containing CN are considered obsolete for military employment, the data that follows are primarily of academic and historical interest.

CN quickly irritates the eyes and upper respiratory passages. In higher concentrations it causes copious tearing; a tingling sensation, irritation, burning, and pain of the nose and throat; and burning and itching on tender areas of the skin, especially areas wet by perspiration. High concentrations can cause blisters. The effects are similar to those of sunburn, are entirely harmless, and disappear in a few hours. Certain individuals experience nausea following exposure to CN. See Table 3-2.

Table 3-2. Chloroacetophenone (CN).

Alternate designations: Mace; CAP; KhAf.

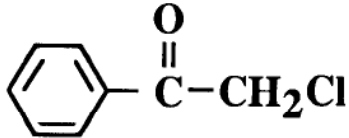
Chemical name. 2-Chloroacetophenone.

CAS registry number: 532-27-4.

RTECS number: AM6300000.

Chemical and Physical Properties

Structural formula:

Molecular formula: C₈H₇ClO.

Molecular weight: 154.59.

Physical state	Solid powder.
Odor	Fragrant; similar to apple blossoms.
Melting point	54°C pure; 46°C to 48°C plant purity.
Boiling point	248°C.
Vapor density (compared to air)	5.3.
Liquid density	1.187 at 58°C.
Solid density	1.318 at 20°C.
Vapor pressure	0.0026 mm Hg at 0°C; 0.0041 mm Hg at 20°C; 0.152 mm Hg at 51.7°C.
Volatility	2.36 mg/m ³ at 0°C; 34.3 mg/m ³ at 20°C; 1,060 mg/m ³ at 51.7°C.
Latent heat of vaporization	98 calories per gram. Like vomiting compounds, CN must be vaporized or dispersed by some means rather than depending on its own volatility.
Flash point	High enough not to interfere with military use.
Decomposition temperature	Stable to boiling point.
Solubility	Soluble in chloroform, chloropicrin, and other organic solvents; insoluble in water.
Rate of hydrolysis	Not readily hydrolyzed.
Hydrolysis products	Hydrogen chloride and a hydroxyacetophenone.
Stability in storage	Stable.
Action on metals or other materials	Tarnishes steel slightly.

Toxicity Data

LC ₅₀	7,000 mg-min/m ³ , dispersed from solvent; 14,000 mg-min/m ³ , dispersed from thermal grenade.
IC ₅₀	80 mg-min/m ³ .
Rate of detoxification	Rapid; effects disappear in minutes. High concentrations may cause skin irritation that usually disappears within a few hours.
Skin and eye toxicity	Irritating; not toxic in concentrations likely to be encountered in the field.
Rate of action	Practically instantaneous.

Other Data

Protection required	Protective mask.
Decontamination	Aeration in the field; soda ash solution or alcoholic caustic soda in enclosed areas.
Persistency	Short, because the compounds are disseminated as an aerosol.
Use	Training and control of civil disturbances.

CNC

The symbol CNC identifies a 30-percent solution of CN in chloroform. It was developed to deliver CN in liquid

form. CNC (Table 3-3) causes a flow of tears, irritates the respiratory system, and causes stinging of skin.

Table 3-3. CNC.

Chemical name: None; chloroacetophenone in chloroform.	
Chemical mixture: Chloroform – CHCl_3 (70 parts by weight). CN – $\text{C}_6\text{H}_5\text{COCH}_2\text{Cl}$ (30 parts by weight).	
Chemical and Physical Properties	
Formula weight: 128.17 (on basis of components).	
Physical state	Liquid.
Odor	Chloroform.
Freezing point	0.23°C. This is the temperature at which CN crystals separate and is not a true change of state. It is that temperature at which the solution becomes saturated with CN. If the solution cools below this point, solid matter appears and gives the appearance of freezing.
Boiling point	Variable (60°C to 247°C). Increases as chloroform boils off and mixture approaches the boiling point of pure CN.
Liquid density	1.40 at 20°C.
Vapor density (compared to air)	4.4.
Vapor pressure	61 mm Hg at 5°C; 127 mm Hg at 20°C. These are calculated values with chloroform the major contributor.
Volatility	This is an indeterminate value under field conditions because the vapor pressure of chloroform is high and the vapor pressure of CN is low. Therefore, there is not true volatility as in the case of a pure compound.
Latent heat of vaporization	Not applicable.
Flash point	None.
Decomposition temperature	Stable to the boiling point.
Rate of hydrolysis	Not readily hydrolyzed.
Hydrolysis products	Hydrogen chloride and hydroxyacetophenone. Stability in storage Adequate.
Action on metals or other materials	Slight.
Toxicity Data	
LC ₅₀	The active ingredient is CN; therefore, with allowance for the diluting action of the chloroform vapor, the LC ₅₀ would be similar to that of CN, about 11,000 mg-min/m ³ .
IC ₅₀	About 80 mg-min/m ³ .
Rate of detoxification	Rapid for sublethal exposures.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Instantaneous.
Other Data	
Protection required	Protective mask.
Decontamination	Aeration in the field; strong soda ash solution or alcoholic caustic soda in enclosed areas.
Persistency	Short, because the compound is disseminated as an aerosol.
Use	Obsolete.

CNB

The symbol CNB (Table 3-4) identifies a mixture of 10-percent CN, 45-percent carbon tetrachloride, and 45-percent benzene. It is a powerful lacrimator. US forces adopted CNB in 1920 and used it until CNS replaced it. The advantage claimed for CNB was that its lower CN content made it more satisfactory than CNC for training purposes. Actually, merely using a lower concentration would obtain the same result with CNC.

CNS

The symbol CNS (Table 3-5) identifies a mixture of 23-percent CN, 38.4-percent chloropicrin, and 38.4-percent chloroform. It is an example of multiple component mixtures developed to achieve desired dissemination characteristics. CNS was declared obsolete in 1957 and is no longer in the supply system.

In addition to having the effects described under CN, CNS also had the effects of PS, which acts as a vomiting

Table 3-4. CNB.

Chemical mixture: CN – C ₆ H ₅ COCH ₂ Cl (10 parts by weight).	
Carbon tetrachloride – CCl ₄ (45 parts by weight).	
Benzene – C ₆ H ₆ (45 parts by weight).	
Chemical and Physical Properties	
Formula weight: 119.7 (on basis of components).	
Physical state	Liquid.
Odor	Benzene.
Vapor density (compared to air)	Approximately 4.
Liquid density	1.14 at 20°C
Freezing point	-7°C to -30°C.
Boiling point	Varies from 75°C to 247°C as the two solvents vaporize.
Vapor pressure	Not pertinent, because this is almost entirely owing to the solvents.
Volatility	Undetermined, because of the effects of the solvents.
Latent heat of vaporization	Not applicable.
Flash point	Below 4.44°C (40°F).
Decomposition temperature	Above 247°C.
Rate of hydrolysis	None.
Hydrolysis products	None.
Stability in storage	Adequate.
Action on metals or other materials	Very slight.
Toxicity Data	
LC ₅₀	No specific data, but about the same as for CN (11,000 mg-min/m ³).
IC ₅₀	80 mg-min/m ³ .
Rate of detoxification	Rapid, if poisonous amounts of solvents have been inhaled.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Instantaneous.
Other Data	
Protection required	Protective masks.
Decontamination	None needed in the field. Decontaminate the CN in CNB in the presence of the solvents with a solution of 5-percent sodium hydroxide, 20-percent water, and 75-percent carbitol by weight.
Persistency	Short.
Use	Obsolete, but is included to present a complete coverage of all potential military chemicals.

compound, a choking agent, and a tear compound. CNS may cause lung effects similar to those of phosgene and also may cause nausea, vomiting, colic, and diarrhea that may persist for weeks. The lacrimatory effects of PS are much less marked than those of CN and were relatively unimportant for CNS. This is shown by the fact that tearing effects were no greater with CNS than with CNC, which contains no PS.

O-Chlorobenzylidene Malononitrile (CS)

In 1959 the US Army adopted CS for combat training and riot control purposes. By weight CS is about ten times as effective as CN. Different forms of CS have different persistence characteristics because of their formulation, dissemination, and rate of hydrolysis. CS exists as a family of four forms: CS, CS1, CS2, and CSX.

Table 3-5. CNS.

Chemical mixture: CN – C ₆ H ₅ COCH ₂ Cl (23 percent).	
Chloropicrin – CNO ₂ Cl ₃ (38.4 percent).	
Chloroform – CHCl ₃ (38.4 percent).	
Chemical and Physical Properties	
Formula weight: 141.78 (on basis of components).	
Physical state	Liquid.
Odor	Like flypaper.
Freezing point	About 2°C. This is the point at which crystals of CN separate and is not a true freezing point as in the case of a pure compound.
Boiling point	No fixed temperature; varies from 60°C to 247°C.
Liquid density	1.47 at 20°C.
Vapor density (compared to air)	About 5.0.
Vapor pressure	78 mm Hg at 20°C (calculated values that include the vapor pressure of the solvent).
Volatility	610,000 mg/m ³ at 20°C (calculated value; volatility owing to the solvent).
Latent heat of vaporization	Not applicable.
Flash point	None.
Decomposition temperature	Stable to boiling point.
Rate of hydrolysis	Does not readily hydrolyze.
Hydrolysis products	Hydrogen chloride and a hydroxyacetophenone.
Stability in storage	Stable.
Action on metals or other materials	Very little.
Toxicity Data	
LC ₅₀	11,400 mg-min/m ³ .
IC ₅₀	60 mg-min/m ³ .
Rate of detoxification	The effects of chloropicrin are long-lasting and cumulative and may prolong the effects of CNS for weeks. Such a prolonged effect may be highly undesirable in training and riot control.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Instantaneous.
Other Data	
Protection required	Protective mask.
Decontamination	Not required in the field; hot solution of soda ash and sodium sulfite for gross contamination in enclosed spaces.
Persistency	Short.
Use	Obsolete.

CS (Table 3-6) identifies the white, crystalline form. In pure form it has a characteristic pungent, pepperlike odor. CS is thermally dispersed as a solid aerosol. CS in aerosol form irritates the eyes, nose, and throat; insufficiently high concentration, it will cause militarily significant incapacitation. Incapacitation results from the individual's inability to see or preoccupation with the agent's effects.

CS produces immediate effects even in extremely low concentrations. The symptoms of exposure to training concentrations of CS include extreme burning of the eyes accompanied by copious flow of tears; coughing, difficulty

in breathing, and chest tightness; involuntary closing of the eyes; stinging or burning on moist skin; heavy mucous formation in the nose with sinus and nasal drip; and dizziness or "swimming" of the head.

Exposure to field concentrations may cause nausea and vomiting. Prolonged exposure to CS on the skin may cause severe irritation and blistering. Effects of the agent appear almost immediately and will continue as long as the individual is exposed. Affected individuals usually recover within about ten minutes in fresh air.

Table 3-6. CS.

Note: CS1, CS2, and CSX will deviate somewhat from these data, based on the percentage of CS content. See other data that follow for CS content of these mixtures.

Chemical name: O-chlorobenzylidene malononitrile.

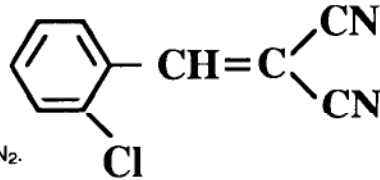
Synonym: 2-chlorbenzalmalononitrile.

CAS registry number: 2698-41-1.

RTECS number: OO3675000.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₁₀H₅ClN₂.

Molecular weight: 188.5.

Physical state	Solid, powder, or liquid.
Odor	Pungent, pepperlike.
Density	1.04 g/cc crystalline density; 0.24 to 0.26 g/cc bulk density.
Melting point	93°C to 95°C.
Boiling point	310°C to 315°C (with decomposition).
Volatility	0.71 mg/m ³ at 25°C.
Vapor pressure	0.00034 mm Hg at 20°C.
Heat of vaporization	53.6 calories per gram.
Flash point	197°C.
Decomposition temperature	Unknown.
Solubility	Soluble in hexane, benzene, methylene chloride, trioctylphosphite, acetone, dioxane, ethyl acetate, and pyridine; insoluble in water and ethanol.
Rate of hydrolysis	Rapid for dissolved CS. CS is only slightly soluble in water (about 0.008 weight percent at 25°C); thus, solid CS in water hydrolyzes relatively slowly. CS hydrolyzes more rapidly if alkalinity is increased.
Hydrolysis products	O-chlorobenzaldehyde and malononitrile.
Stability in storage	Stable.
Action on metals	Very slight action on steel.

continued

Table 3-6, CS continued

Toxicity Data	
LCt ₅₀	61,000 mg-min/m ³ .
ICt ₅₀	10 to 20 mg-min/m ³ .
Eye effects	1 to 5 mg/m ³ .
Rate of detoxification	Quite rapid. Incapacitating dosages lose their effects in 5 to 10 minutes.
Skin and eye toxicity	Highly irritating but not toxic.
Rate of action	Very rapid (maximum effects in 20 to 60 seconds).
Other Data	
Protection required	Protective mask and ordinary field clothing secured at the neck, wrist, and ankles. Personnel handling CS should wear rubber gloves for additional protection.
Decontamination	Personnel affected by CS in field concentrations should move to an uncontaminated area, face into the wind, and remain well-spaced. They should be warned not to rub their eyes or to scratch irritated skin areas. Normally, aeration is sufficient to decontaminate personnel and to dissipate ill effects of the compound in 5 to 10 minutes. Personnel contaminated with visible CS particles should flush their bodies or affected parts with cool water for 3 to 5 minutes before showering with warm or hot water. Use soap and water on equipment contaminated with CS1, CS2, or CSX. The higher the alkalinity of the soap, the better. Do not use standard decontaminants or detergents that contain chlorine bleach for CS-containing compounds; the materials can react to form compounds (epoxides, which have vesicant properties) more toxic than CS.
Munitions suitable for use	Used as filling for burning-type grenades and for capsules. CS1 is used as filling for bursting-type grenades and in bulk agent dispersers. CS2 is used as filling in bulk agent dispersers. CSX is suitable for liquid dispersers.
Persistency	Varies, depending upon amount of contamination and form of CS. CS aerosol usually has little residual hazard. CS1 can remain effective for about 7 days inside; about 14 days in open terrain under ideal conditions; rain, groundwater, and wind will appreciably reduce the duration of effectiveness of CS1. CS2 persists approximately twice as long as CS1.
Use	Training and riot control; limited tactical use in counter guerrilla operations.

CS1 has been specially formulated to prolong persistency and increase the effectiveness. Unlike CS, CS1 is a free-flowing (micropulverized) agent powder consisting of 95-percent crystalline CS blended with 5-percent silica aerogel. This formulation reduces agglomeration and achieves the desired respiratory effects when dispersed as a solid aerosol.

CS2 is CS blended with silicone-treated silica aerogel, which causes it to repel water. 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. When disturbed, CS2 reaerosolizes to cause respiratory and eye effects.

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 trioctylphosphite (TOF). As with CS, CSX stings and irritates the eyes, skin, nose, throat, and lungs of exposed personnel.

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

In 1974 the US Army approved the use of CR. CR has much greater irritating properties than CS and is about five

times more effective. In addition, CR is much less toxic than CS. CR is not used in its pure form (a yellow powder) but is dissolved in a solution of 80 parts of propylene glycol and 20 parts of water to form a 0.1-percent CR solution. It is used in solution as a riot control agent.

CR immediately and severely stings the skin, eyes, nose, and throat of exposed personnel. Eye pain, discomfort, and excessive tearing occur with sometimes painful sensitivity to strong light or temporary blindness. Nasal irritation, coughing, sneezing, and nasal drip also occur. Exposure of the skin to CR results in a stinging or burning sensation with increased irritation on moist skin. Sometimes nausea and vomiting accompany these symptoms. Symptoms can persist for 15 to 30 minutes. Severity of symptoms increases with the CR solution concentration and in any environment of high temperature and humidity. With prolonged exposure skin sensitivity (similar to that associated with a mild burn) that is renewed upon washing or rubbing may occur for hours to days.

CR does not degrade in water and is quite persistent in the environment. Under suitable conditions CR can persist on certain surfaces (especially porous) for up to 60 days. Do not use bleach to decontaminate CR. See Table 3-7.

Table 3-7. CR.

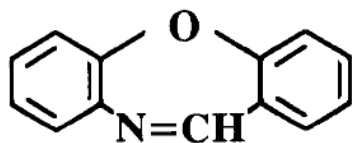
Chemical name: Dibenz-(b,f)-1,4-oxazepine.

CAS registry number: 257-07-8.

RTECS number: HQ3950000.

Chemical and Physical Properties

Structural formula:

Molecular formula: C₁₃H₉NO.

Molecular weight: 195.25.

Physical state	Yellow powder in solution.
Odor	Produces a burning sensation in nose and sinuses.
Vapor density	6.7 (calculated).
Boiling point	335°C.
Freezing point	72°C.
Volatility	0.63 mg/m ³ at 25°C.
Vapor Pressure	0.00059 mm Hg at 20°C.
Solubility	Negligible in water. Soluble in polypropylene glycol, benzene, chloroform, or carbon tetrachloride.
Rate of hydrolysis	Very slow.
Flash point	188°C.
Stability	Stable in aqueous, heated acidic, and strong alkali solutions. Thermally stable. Never mix CR with organic or inorganic bleaches or peroxide.
Decomposition products	Nitrous oxide and carbon monoxide (CO).
Storage conditions	Store in nonporous containers on nonporous surfaces. CR will leach and persist in porous materials.

Toxicity Data

IC ₅₀	0.15 mg/m ³ .
Threshold effects	0.002 mg/m ³ (respiratory tract); 0.004 mg/m ³ (eyes).
Rate of detoxification	Skin irritation persists for 15 to 30 minutes after removal of CR. Eye effects may last up to 6 hours.
Rate of action	Very rapid.

Other Data

Protection required	Protective mask and ordinary field clothing secured at neck, wrist, and ankles.
	continued

Table 3-7, CR continued

Decontamination	Personnel affected by CR in field concentrations should move to an uncontaminated area, face into the wind, and remain well-spaced. They should be warned not to rub their eyes or to scratch irritated skin areas. Normally, aeration is sufficient to decontaminate personnel and to dissipate ill effects of the compound in 5 to 10 minutes. If CR entered the eyes, flush with large amounts of cold water. Wash exposed skin gently with soap and cold water. Individuals who have ingested CR should be given lots of water or milk to drink. Do not induce vomiting. Do not use bleach, detergents, or peroxides for decontamination; this combination releases toxic fumes. Remove CR from equipment or surfaces by wiping, scraping, shoveling, or sweeping. Wipe the area with rags soaked in propylene glycol or an automotive antifreeze solution, if available; wipe with rubbing alcohol; and then scrub with nonbleach detergent and hot water before rinsing with large amounts of cold water. Place all contaminated materials used for decontamination in a storage container where they cannot affect personnel.
Persistence	As persistent as, or more persistent than, CS2. Under suitable conditions CR can persist on certain surfaces (especially porous material) for up to 60 days.
Use	Riot control agent dispersed as a spray.

Chloropicrin (PS)

PS is a pungent, colorless, oily liquid. It is very volatile and is usable during any season to produce incapacitating or lethal concentrations.

PS (Table 3-8) is a powerful irritant whose vapors cause nose and throat irritation, coughing, and vomiting. As an eye irritant, it produces immediate burning, pain, and tearing. Even in very limited concentrations PS causes the eyelids to close. In high concentrations PS damages the lungs, causing pulmonary edema. It is very soluble in fats and oils, and different organs absorb it. In the liquid form

it causes severe burns on the skin that generally result in blisters and lesions.

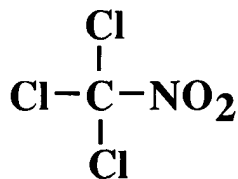
Chloropicrin was used in large quantities by "all the warring countries" during World War I. Chloropicrin was used alone; more often it was mixed with chlorine, phosgene, diphosgene and tin chloride. It was stockpiled during World War II, generally in concentrations or mixtures to produce tearing. It is more toxic than chlorine but less toxic than phosgene (CG). Chloropicrin decomposes into chlorine gas and nitrogen oxide near open fires, producing toxic fumes. The protective mask protects against vapors; protective clothing protects against the liquid agent.

Table 3-8. Chloropicrin (PS).

Alternate designations: Klorpikrin; NC; vomiting gas; G8; Aquinite; Klop.
Chemical name: Trichloronitromethane.
Synonyms: Nitrochloroform; Chloropicrin; Nitrotrichloromethane.
CAS registry number: 76-06-2.
RTECS number: PB6300000.

Chemical and Physical Properties

Structural formula:



Molecular formula: CCl₃NO₂.

Physical state	Colorless, oily liquid.
Odor	Stinging, pungent odor.
Boiling point	112°C.
	continued

Table 3-8, Chloropicrin (PS) continued

Freezing point	-69°C.
Vapor density	5.6.
Liquid density	1.66.
Vapor pressure	18.3 mm Hg at 20°C.
Volatility	165,000 mg/m ³ at 20°C.
Flash point	Not flammable.
Decomposition temperatures	Above 400°C.
Solubility	Insoluble in water; soluble in organic solvents, lipids, organophosphorus compounds, mustards, phosgene, diphosgene, and Cl ₂ .
Rate of hydrolysis	Does not hydrolyze in water.
Stability in storage	Unstable liquid; decomposes under the influence of light.
Action on metals	If dry, has little or no effect on metals.
Toxicity Data	
LCt ₅₀	2,000 mg-min/m ³ .
Lowest irritant concentration	9 mg-min/m ³ for 10 minutes.
Skin and eye toxicity	Irritates nose and throat; causes tearing; irritates lungs at higher concentrations; causes nausea and vomiting; can cause skin lesions.
Other Data	
Protection required	Protective mask for vapors.
Decontamination	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.
Persistency	Approximately 6 hours in vegetated fields.
Use	Not authorized for US military use. However, this manual discusses PS to present a complete coverage of all chemicals of potential use in military operations.
Mixtures	PG (United Kingdom, with phosgene), Klop (Germany, with Cl ₂), Green Cross (Germany, with

Vomiting Compounds (Sternutators)

Vomiting agents produce strong, pepperlike irritation in the upper respiratory tract, with irritation of the eyes and tearing. They cause violent, uncontrollable sneezing; cough; nausea; vomiting and a general feeling of bodily discomfort. The vomiting compounds listed are normally solids that vaporize when heated and then condense to form aerosols. They produce their effects by inhalation or by direct action on the eyes. Under field conditions vomiting compounds cause great discomfort to victims; when released indoors, they can cause serious illness or death. The principal vomiting agents are diphenylchloroarsine (DA), diphenylaminochloroarsine (DM; Adamsite), and diphenylcyanoarsine (DC). Chloropicrin also is a vomiting agent.

Symptoms, in progressive order, are irritation of the eyes and mucous membranes, viscous discharge from the nose similar to that caused by a cold, sneezing and coughing, severe headache, acute pain and tightness in the chest, nausea, and vomiting. The effects of DM develop more slowly than those of DA, and for moderate concentrations the effects last about 30 minutes after the person leaves the contaminated atmosphere. At higher concentrations the effects may last up to several hours.

Diphenylchloroarsine (DA)

Agent DA contains arsenic and chlorine. The Threat could use it to cause troops to remove their masks and vomit, thereby exposing them to other agents. See Table 3-9.

Table 3-9. Diphenylchloroarsine (DA).

Alternate designations: Clark I; Blue Cross agent; DIK; Klark-1; sneezing gas.

Chemical name: Diphenylchloroarsine.

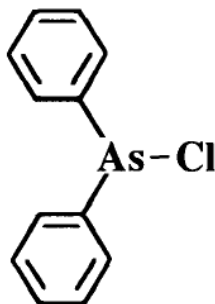
Synonym: Chlorodiphenylarsine.

CAS registry number: 712-48-1.

RTECS number: CG9900000.

Chemical and Physical Properties

Structural formula:



Molecular formula: $C_{12}H_{10}AsCl$.

Molecular weight: 264.5.

Physical state	Colorless crystals when pure.
Odor	No pronounced odor.
Vapor density (compared to air)	Forms no appreciable vapor.
Liquid density	1.387 at 50°C.
Melting point	41°C to 44.5°C.
Boiling point	333°C with decomposition.
Vapor pressure	0.0036 mm Hg at 45°C, calculated.
Volatility	48 mg/m ³ at 45°C.
Flash point	350°C.
Decomposition temperature	300°C.
Latent heat of vaporization	56.6 calories per gram. Latent heat of vaporization is of little importance in a chemical compound that is dispersed by an external heat source.
Solubility	Soluble in acetone, ethanol, carbon tetrachloride; insoluble in water.
Rate of hydrolysis	Slow in mass but rapid when finely divided.
Hydrolysis products	Diphenylarsenious oxide and hydrogen chloride. The oxide is very poisonous if taken internally.
Stability in storage	Stable when pure.
Action on metals or other material	None when dry.

Toxicity Data

LC ₅₀	15,000 mg-min/m ³ (estimated).
IC ₅₀	12 mg-min/m ³ if received over ten-minute periods; probably higher for shorter time.
Rate of detoxification	Any merely incapacitating amount is detoxified completely within one to two hours.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Very rapid, within two or three minutes after a one-minute exposure.

continued

Table 3-9, Diphenylchloroarsine (DA) continued

Other Data	
Protection required	Protective mask.
Decontamination	None required in the field. Caustic soda or chlorine used for gross contamination in enclosed spaces.
Persistency	Short, because compound is disseminated as an aerosol.
Use	Not authorized for US military use. However, this manual discusses vomiting compounds to present a complete coverage of all chemicals of potential use in military operations.

Diphenylcyanoarsine (DC)

The properties of DC are much like those of DA and DM, and the Threat would use it in the same manner. DC is more toxic than DA. The effects from a moderate con-

centration last about 30 minutes after a person leaves the contaminated atmosphere. The effects from a higher concentration may last up to several hours. See Table 3-10.

Table 3-10. Diphenylcyanoarsine (DC).

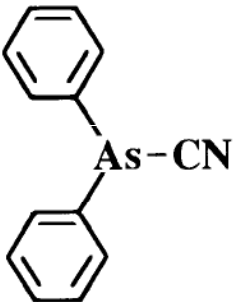
Alternate designations: Clark II.	
Chemical name: Diphenylcyanoarsine.	
Chemical and Physical Properties	
Structural formula:	
	
Molecular formula: C ₁₃ H ₁₀ AsN.	
Molecular weight: 255.0.	
Physical state	Solid.
Odor	Similar to garlic and bitter almonds.
Vapor density (compared to air)	Does not form appreciable vapor.
Liquid density	1.3338 at 35°C.
Melting point	31.5°C to 35°C.
Boiling point	350°C with decomposition.
Vapor pressure	0.0002 mm Hg at 20°C.
Volatility	2.8 mg/m ³ at 20°C.
Flash point	Low.
Decomposition temperature	About 25 percent decomposed at 300°C. Largely decomposed as a result of dispersing blast.
Latent heat of vaporization	71.1 calories per gram. Because DC has a high heat of vaporization, a source of heat must be applied to it to obtain a vapor concentration that is militarily significant.
Solubility	Soluble in chloroform and other organic solvents; insoluble in water.
Rate of hydrolysis	Very slow.
Hydrolysis products	Hydrogen cyanide and diphenylarsenious oxide.
continued	

Table 3-10 Diphenylcyanoarsine (DC) continued

Stability in storage	Stable at all ordinary temperatures.
Action on metals or other materials	None.
Toxicity Data	
LC ₅₀	10,000 mg-min/m ³ . It would be nearly impossible to build up a vapor concentration of DC that would be lethal within a practicable time.
IC ₅₀	30 mg-min/m ³ for 30-second exposure; 20 mg-min/m ³ for 5-minute exposure.
Rate of detoxification	Rapid. Incapacitating amounts lose their effect after about 1 hour.
Skin and eye toxicity	Irritating; not toxic.
Rate of action	Very rapid. Higher concentrations are intolerable in about 30 seconds.
Other Data	
Protection required	Protective mask.
Decontamination	None required in the field. Use alkali solution or DS2 for decontamination in enclosed places.
Persistence	Short, because the compound is disseminated as an aerosol.
Use	Not authorized for US military use. However, this manual discusses vomiting compounds to present a complete coverage of all chemicals of potential use in military operations.

Adamsite (DM)

Adamsite was first produced during World War I. It is disseminated as an aerosol and is effective only through the

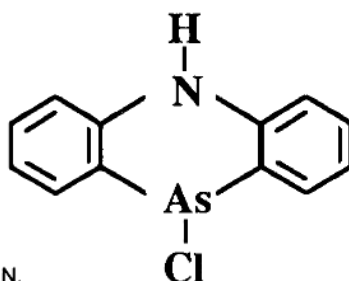
respiratory tract; it has no irritant effect on the skin. See Table 3-11.

Table 3-11. Adamsite (DM).

Alternate designations: Adamsit; R15.
Chemical name: 10-chloro-5,10-dihydrophenarsazine.
Synonyms: Diphenylaminochloroarsine; phenarsazine chloride.
CAS registry number: 578-94-9.
RTECS number: SG0680000.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₁₂H₉AsClN.

Molecular weight: 277.57.

Physical state	Light yellow to green crystals.
Odor	No pronounced odor, but irritating.
Vapor density (compared to air)	Forms no appreciable vapor.
Solid density	1.65 g/cm ³ at 20°C.
Freezing point	195°C.
	continued

Table 3-11, Adamsite (DM) continued

Boiling point	410°C, calculated.
Vapor pressure	Negligible (2×10^{-13} mm Hg at 20°C).
Volatility	Negligible.
Flash point	None under usual conditions.
Decomposition temperature	Above melting point.
Latent heat of vaporization	80 calories per gram from 200°C to 250°C; heat of sublimation 134 calories per gram.
Solubility	Soluble in furfural and acetone; slightly soluble in common organic solvents; insoluble in water. Not readily soluble in any of the liquid chemical warfare agents.
Rate of hydrolysis	Quite rapid when in aerosol form. When solid DM is covered with water, a protective oxide coating forms that hinders further hydrolysis.
Hydrolysis products	Diphenylarsenious oxide and hydrochloric acid. The oxide is very poisonous if taken internally.
Stability in storage	Stable when pure.
Action on metals or other materials	Slight when dry.
Toxicity Data	
LC ₅₀	Variable (average = 11,000 mg-min/m ³).
IC ₅₀	22 to 150 mg-min/m ³ .
Rate of detoxification	Quite rapid in small amounts. Incapacitating amounts lose their effects after about 30 minutes.
Skin and eye toxicity	Irritating; relatively nontoxic.
Rate of action	Very high. Requires only about 1 minute to temporarily incapacitate at a concentration of 22 mg/m ³ .
Other Data	
Protection required	Protective mask.
Decontamination	None needed in the field; use bleaching powder or DS2 for gross contamination in enclosed places.
Persistency	Short, because compounds are disseminated as an aerosol.
Use	Not authorized for US military use. However, this manual discusses vomiting compounds to present a complete coverage of all chemicals of potential use in military operations.

Section II. Binary Components

The binary nerve agents are GB2 and VX2. The components of GB2 are DF and a mixture of isopropyl alcohol and isopropyl amine (OPA). The components of VX2 are QL, which is designated chemically as O-(2-diisopropylaminoethyl)-O'ethyl methylphosphonite, and

NE, which is powdered sulfur with a small amount of added silica aerogel to prevent caking.

Troop exposure to these materials could occur from leaking containers, spills at production or storage facilities, or accidents during transport.

DF and DC (MPOD)

Methylphosphonic difluoride (DF) and its precursor, methylphosphonic dichloride (DC), are organophosphonic acids. They will react with alcohols to form crude lethal nerve agents, such as crude GB. High overexposure may cause inhibition of cholinesterase activity. Although much less toxic than GB, DF and DC are toxic and corrosive materials.

Troop exposure could result from leaking DF containers or accidents that disrupt packaging. Because DF and DC

are relatively volatile compounds, the primary route of exposure is expected to be the respiratory system. However, ingestion also results from inhalation exposures in animals and could occur in humans. DF and DC vapors have a pungent odor and may cause severe and painful irritation of the eyes, nose, throat, and lungs. Data provided are for DF only (Table 3-12). DC has similar properties.

Table 3-12. DF.

Alternate designation: Difluoro.

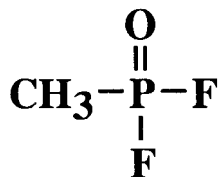
Chemical name: Methylphosphonic difluoride.

CAS registry number: 676-99-3.

RTECS number: T01840700

Chemical and Physical Properties

Structural formula:



Molecular formula: CH₃F₂PO.

Molecular weight: 100.1.

Physical state	Liquid.
Odor	Pungent, acid like.
Vapor density	3.45.
Liquid density	1.359 at 25°C.
Freezing point	-37.1°C.
Boiling point	100°C.
Vapor pressure	36 mm Hg at 25°C.
Volatility	147,926 mg/m ³ at 19.5°C.
Flash point	Not flammable.
Heat of vaporization	18.7 calories per gram.
Rate of hydrolysis	Virtually instantaneous to products (MF and HF) that are also toxic. Further hydrolysis is a slow reaction; half-times for hydrolysis of MF are 162 days at pH 7, 90 days at pH 4, and 47 days at pH 3.
Hydrolysis products	Hydrolyzes to give toxic products, MF and HF. Further hydrolysis of MF results in methylphosphonic acid.
Stability in storage	Does not spontaneously decompose but is reactive. Avoid contact with water mist or sprays, metals, alkaline materials, and some organics. Store DF in lead and wax-lined carboys, high-density polyethylene bottles, or nickel-lined containers in well-ventilated areas. Never store DF with alcohols; DF will react with alcohols to form a lethal chemical, such as crude GB.
Action on metals or other materials	Incompatible with water, glass, concrete, most metals, natural rubber, leather, and organic materials like glycols. The acidic corrosive hydrolysis products may react with some metals, such as Al, Pb, and Fe, to give off hydrogen gas, a potential fire and explosive hazard.

Toxicity Data

LC ₅₀ and IC ₅₀	Not established. The inhalation toxicity in rats, mice, and guinea pigs is on the same order as for inhaled acid gases, such as HF and HCl. Long-term inhalation exposure may cause chronic upper and lower respiratory effects. High overexposure may inhibit cholinesterase.
Skin and eye toxicity	May cause severe and painful irritation of the eyes, nose, throat, and lungs. DF hydrolyzes to HF which may cause second- or third-degree burns upon contact.
	continued

Table 3-12, DF continued

Ingestion	May cause severe tissue destruction in the gastrointestinal tract. The hydrolysis products of DF appear to be of the same order of toxicity as shown by fluoride salts. The oral LD ₅₀ for rats and mice is about 100 mg/kg.
Other Data	
Protection required	Protective mask, gloves, and protective clothing.
Decontamination	Water. Flush eyes for 15 minutes. Wash skin and seek medical help.
Use	Binary GB component.

Isopropylamine and Isopropyl Alcohol (OPA)

The mixture known as OPA (Table 3-13) is relatively nontoxic compared to nerve agents. However, it is not without hazard. It is a highly volatile and flammable liquid composed of 72-percent isopropyl alcohol and 28-percent isopropylamine. It forms toxic oxides of nitrogen as well as explosive mixtures in air.

In contact with skin and eyes OPA may cause severe irritation. Ingestion causes nausea, salivation, and severe irritation of the mouth and stomach. Inhalation may cause irritation of the lower respiratory tract, coughing, difficult breathing, or loss of consciousness.

Table 3-13. Isopropylamine and isopropyl alcohol (OPA).

Chemical name: None; mixture of 2-propanol (isopropyl alcohol) and isopropylamine .	
Chemical mixture: 2-propanol – (CH ₃) ₂ CHOH (72 percent).	
Isopropylamine – (CH ₃) ₂ CHNH ₂ (28 percent).	
Chemical and Physical Properties	
Molecular formulae: C ₃ H ₉ N and C ₃ H ₈ .	
Physical state	Clear liquid.
Odor	Alcohol and ammonia.
Vapor density	2.1.
Liquid density	0.7443 at 25°C.
Freezing point	Less than -88°C.
Boiling point	60°C.
Flash point	-9.4°C (based on amine).
Vapor pressure	197 mm Hg at 25°C.
Stability in storage	Stable but reactive, volatile, and flammable. Store OPA in a cool, well-ventilated area away from heat, open flame, and the materials in the next listed property.
Action on metals or other materials	Reacts readily with oxidizing materials and organophosphorus halides, such as DF or DC. Contact with DF or DC can produce extremely toxic compounds (GB or a chlorine compound similar to GB).
Toxicity Data	
LC ₅₀ and IC ₅₀	Not available.
Skin and eye toxicity	Irritating and toxic through the skin and eyes, mainly because of the isopropylamine in the mixture. Studies with rabbits indicate a skin LD ₅₀ of 550 mg/kg.
Ingestion	The probable lethal dose is between one teaspoon and one ounce. The oral LD ₅₀ for rats is 820 mg/kg.
Other Data	
Protection required	Protective mask and clothing.
	continued

Table 3-13, Isopropylamine and isopropyl alcohol (OPA) continued

Decontamination	Large volumes of water to flush OPA from the skin. Wash clothing with water. Absorb spilled OPA with vermiculite, earth, or sand; hold for disposal.
Use	Component of binary GB.

QL (EDMP)

One of the binary components for VX is QL, an organophosphorous ester. An additional designation for QL is EDMP, an abbreviation for O,O'-ethyl (2-diisopropylaminoethyl) methylphosphonite. The pure material is many times less toxic than VX but is by no means harmless. It reacts with moisture and other substances to produce highly toxic materials as well as flammable materials. It will ignite without application of spark or flame at 129°C (265°F). A hydrolysis product of QL ignites at a much lower temperature.

QL (Table 3-14) is a slight cholinesterase inhibitor, but the body tissue and fluids do not store it for extended

periods. Prolonged breathing of QL vapors may produce headaches and nausea.

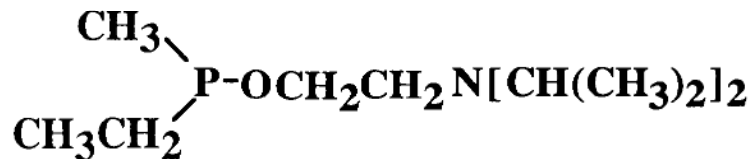
Like DF and OPA, QL is not likely to be encountered as a result of Threat action except in cases in which Threat ordnance damages containers. Other possibilities of troop exposure are leaking containers and accidents. In these cases treat QL as a flammable liquid, because it reacts with moisture to produce highly flammable diethyl methylphosphonite (TR). Never store or ship QL with sulfur or sulfur compounds, such as NE or NM.

Table 3-14. QL.

Chemical name: O-ethyl-O'(2-diisopropylaminoethyl) methylphosphonite; EDMP.
Synonym: O-(2-diisopropylaminoethyl) O'-ethyl methylphosphonite.
CAS registry number: 57856-11-8.

Chemical and Physical Properties

Structural formula:



Molecular formula: C₁₁H₂₆N₂O₂P.

Molecular weight: 235.

Physical state	Viscous liquid.
Odor	Strong, fishy.
Vapor density	8.1.
Liquid density	0.908 at 25°C.
Boiling point	232°C.
Vapor pressure	0.01 mm Hg at 25°C.
Flash point	89°C. In addition, QL has an autoignition temperature of 129°C. A hydrolysis product, O,O'-diethylmethylphosphonite (TR), has a flash point of 28°C and an autoignition temperature of 40°C. Do not use a carbon tetrachloride fire extinguisher. Use water fog, foam, CO ₂ , or bicarbonate.
Rate of hydrolysis	Rapid. QL can be hydrolyzed and oxidized.
Hydrolysis products	With an excess of water by weight, QL forms 2-diisopropylaminoethanol, ethanol, and methyl phosphorous acid. With traces of water or other proton donors QL will produce TR and O,O'-bis-(2-diisopropylaminoethyl) methylphosphonite. TR has a boiling point of 120°C and a vapor pressure of 11 mm Hg at 20°C and is highly flammable.

continued

Table 3-14 QL continued

Stability in storage	Unstable in air. Protect from water or moisture. Store away from heat or ignition sources and sulfur compounds.
Action on metals or other materials	Reacts with sulfur and sulfur compounds, producing highly toxic VX or VX-like compounds. It completely dissolves polymethylmethacrylate. It is incompatible with calcium hypochlorite (HTH), many chlorinated hydrocarbons, selenium, selenium compounds, moisture, oxidants, and carbon tetrachloride.
Toxicity Data	
LC ₅₀ and IC ₅₀	Not available. Animal studies indicate that toxicity of QL from short-term exposure is low.
LD ₅₀ (intravenous)	204 mg/kg in mice; 164 to 208 mg/kg in rabbits.
Skin and eye toxicity	Skin irritant but not a skin sensitizer or eye irritant. Animal studies have shown QL to be of relatively low toxicity, although the hydrolysis products are highly toxic.
Other Data	
Protection required	Protective mask, clothing, and gloves if danger of severe exposure exists.
Decontamination	Soda ash, slaked lime, limestone, or sodium bicarbonate. Cover spills with vermiculite, diatomaceous earth, clay, or fine sand followed by one of the decontaminants.
Use	Binary VX component.

NE and NM

NE is the designation for a powdered elemental sulfur mixture. The addition of an anticaking silica aerogel (Cab-O-Sil) prevents lumping and enhances the free-flowing characteristics of the sulfur powder. NE (Table 3-15) is one of the components for the production of binary VX.

NM is a binary chemical intermediate that can substitute for sulfur in the production of binary VX. NM is a mixture

of dimethylpolysulfides, containing some dissolved elemental sulfur. NM is a very persistent liquid material with a very obnoxious odor. There are indications of considerable risk from short-term inhalation exposures to high airborne concentrations of NM. The molecular formula for NM is (CH₃)₂S_n, where n equals 2 to 6 + (average 5); molecular weight average is 190.39.

Table 3-15. NE.

Chemical name: Sulfur (with small amount of silica aerogel).	
Chemical and Physical Properties	
Molecular formula: S ₈ .	
Molecular weight: 256.48.	
Physical state	Fine powder or yellow, crystalline solid.
Odor	Odorless when pure, but many sulfur compounds tend to be vile-smelling.
Vapor density (compared to air)	8.8, calculated. (This property is of little significance for solids at ambient temperatures.)
Solid density (specific gravity)	2.07 g/cc at 20°C.
Melting point	112.8°C.
Boiling point	444.6°C.

Section III. Miscellaneous

The Germans used chlorine in World War I. It has seen no use as a chemical agent since then; more lasting and toxic agents have replaced it. Chlorine (Table 3-16) is a powerful irritant, first on the upper and then on the lower respiratory tract.

A multitude of industrial processes use chlorine. It is also an indispensable reagent in the manufacture of chlorinated organic materials and inorganic chlorides and chlorates. It is of particular value in water purification.

Table 3-16. Chlorine.

Alternate designation: Bertholite.	
Chemical name: Chlorine.	
CAS registry number: 7782-50-5.	
RTECS number: FO2100000.	
Chemical and Physical Properties	
Molecular formula: Cl ₂ .	
Molecular weight: 70.91.	
Physical state	Greenish-yellow gas.
Odor	Pungent, like bleaching powder.
Vapor density (compared to air)	2.4.
Liquid density	1.41 at 20°C.
Melting point	-101°C.
Boiling point	-34.5°C.
Vapor pressure	4,992 mm Hg at 20°C.
Volatility	19,369,000 mg/m ³ at 20°C.
Flash point	None.
Decomposition temperature	Greater than 1,000°C.
Latent heat of vaporization	68.8 calories per gram.
Solubility	Soluble in alcohol and chlorides.
Rate of hydrolysis	Slow.
Hydrolysis products	HCl and HOCl.
Stability in storage	Stable when dry.
Action on metals or other materials	None in field concentrations if chlorine is dry. Vigorous action with metals when chlorine is moist.
Toxicity Data	
LC ₅₀	19,000 mg-min/m ³ .
IC ₅₀	1,800 mg-min/m ³ .
Rate of detoxification	Rapid.
Other Data	
Protection required	Protective mask.
Decontamination	None required.
Persistency	Short.
Use	No longer authorized. However, this manual includes chlorine for historical interest and the potential danger of a commercial accident or incident.

Chapter 4

T o x i n s

Toxins are poisonous chemicals produced by many different types of living organisms. Toxins have been implicated as the means by which certain pathogenic microorganisms produce their effects. Toxins that are highly toxic to humans and that are stable, available, and manageable are important in the threat they present in biological warfare. This chapter contains information on some of the possible Threat toxins. These toxins represent the range of toxin agents that may be available to the sophisticated user of biological weapons. However, weapon systems could incorporate many other toxins.

Section I. Background

Many toxins were developed for medical use. This is especially true of toxins from microorganisms and fungi. Examples are atropine, morphine, streptomycin, and penicillin. As a result commercial processes in many countries prepare microbial and fungal products. The technology exists for bulk production of some toxins. As a general rule toxins are not chemically synthesized; they are extracted from their natural sources.

The chemical nature of toxins is diverse (see Appendix E). Some toxins are large proteins; some are smaller proteinlike (proteinaceous) compounds; others are non-proteins. The proteinaceous toxins are solids when pure but dissolve in water-based solutions. Protein-based toxins are generally less stable than nonprotein toxins. Some toxins are extremely stable and may retain their potency for years in storage. Toxins from plants and fungi tend to be more stable than those from animals.

Biological agents (including toxins) may be expected to be disseminated either as an aerosol, a liquid, or a powder (see Chapter 1). Based on the portals of entry, the characteristics of agents used, and the results desired, different methods of dissemination are feasible for biological attack. Toxins are tactical or strategic weapons. Some can effectively cover hundreds of square kilometers, and most could cover at least several square kilometers.

The Threat may use ground-bursting or airbursting munitions, aircraft spray tanks, or ground-level aerosol generators to produce aerosol clouds of toxins. Inhalation of these aerosols will produce casualties in a manner similar to that of chemical aerosols. The greatest threat occurs with exposure of individuals to the cloud. There is still a risk of respiratory, eye or oral exposure while the

aerosols dissipate and also through secondary aerosolization.

The body absorbs particulate or liquid aerosols of toxins through the respiratory tract, the skin, or mucous membranes. Because mechanical or heat stress inactivate some toxins, use of these toxins may require dissemination of large concentrations. Aerosol toxin attacks usually are not visible. However, because of the amount required to produce casualties and the color of the toxin or dissemination medium, aerosolized solids may be visible as a dust cloud or as powders on equipment and clothing.

Bursting munitions and spray tanks may produce large liquid drops to cause ground contamination, like ground contamination by chemical agents. Forces in southeast Asia and Afghanistan used this method of dissemination to employ "yellow rain" mycotoxins. The Threat could use ground-contaminating toxins to produce casualties or to deny terrain, equipment, or supply.

Droplets of a solution or a suspension of a toxin would cause surface contamination, including contamination of food or water, and the toxin could enter the body through the digestive tract. Some toxins (for example, mycotoxin T-2) are skin damaging and could penetrate the skin.

In addition to tactical or strategic employment, toxins pose a threat as weapons for covert, guerrilla, or terrorist operations. With the vast number of toxins and delivery options, the imagination of the user is the only limitation to covert dissemination of toxins. Saboteurs can contaminate closed ventilation systems, drinking water, lakes and rivers, and food supplies. Assassins can also use some agents.

Physical and environmental factors determine the effectiveness of these methods. Mechanical or heat stress inac-

tivate some toxins. (This does not apply to powdered toxin dissemination; it applies only to liquids.) Some toxins (for example, Staphylococcus enterotoxin, Type B) are stable in the environment and are more resistant than G- or V-agents to heat, hydrolysis, or vaporization. Others, such as botulinum toxin, have only a brief predictable persistence unless rendered resistant to environmental conditions. Appendix F, Table F-1, summarizes the physical and chemical properties of toxins.

The use of certain specialized techniques that are common to the production of pharmacological could influence the effectiveness of the toxin. Examples of these techniques are micronizing (air-milling) and microencapsulation. Micronizing is a technique used to reduce the particle size to increase absorption. This process is particularly important when exposure occurs primarily by inhalation. Microencapsulation can make aerosol distribution of biological agents technically more feasible. Encapsulation of agents in certain organic compounds could enhance agent survivability in the environment during dissemination. It could potentially allow more specific targeting of the agent within the body or enhance absorption and retention.

The actions and effects of toxins may closely resemble those of chemical warfare agents, such as nerve, blister, vomiting, or choking agents. Most toxins of military significance cause casualties in two general ways and can be classified by the way they act:

Neurotoxins (“nerve toxins”) interfere with nerve impulse transmission. They have highly specific effects on the nervous system. All neurotoxins do not produce the same symptoms or have the same mechanism of action. For example, they may stimulate or inhibit the release of acetylcholine, block receptors, or interfere with the activity of ion channels. Neurotoxins may cause symptoms similar to

chemical nerve agents such as pinpointing of pupils, convulsions, and rigid paralysis; or they may cause other symptoms such as blurred vision and light sensitivity due to dilation of pupils, tremors, seizures, confused behavior, extreme muscle weakness, or rigid or limp (flaccid) paralysis.

Cytotoxins (“cell poisons”) produce a variety of effects because of their distinct mechanisms. Some destroy cells. Others disrupt cell activities, such as protein synthesis, cell regulation, or other biochemical processes. Symptoms may resemble those of chemical blister, vomiting, or choking agents; or they may resemble food poisonings or diseases. Cytotoxins may cause nausea, vomiting, or diarrhea; rashes, inflammation, or blistering, jaundice; or bleeding or deterioration of tissue (necrosis). Appendix E discusses the chemical nature and mechanisms of action of toxins.

Toxins may produce lethal or nonlethal effects (Table 4-1). By weight, most toxins are thousands of times more toxic than standard chemical agents. These effects depend on the toxin, the dose received, and the route of entry. The time lapse between contamination and symptoms may vary from a few minutes to several hours. Many, if not most, of the toxins are principally a threat by aerosol. Most toxicity data for toxins, however, is not in air concentration times the time (LCt50). Essentially all toxins are at least as toxic by aerosol as by injected dose (LD50). Therefore, this manual expresses aerosol data as the dose of toxin actually received (LD50). Nearly all toxins of concern would require considerably higher oral doses than aerosol doses. Most of the large protein toxins are not a significant threat by dermal or oral exposure unless there is an open wound. Toxins, even though of biological origin, are nonliving chemical compounds; as such, they are not infectious or contagious after dissemination. A summary of selected toxin effects is in Appendix F, Table F-2.

Table 4-1. Lethality and rate of action of selected toxins.

Toxin and Time to Toxic Effects	LD₅₀* (μg/kg)	Type and Effects
Very rapid: 5 minutes		
Anatoxin A (Very Fast Death Factor [VFDF])	170 to 250	Lethal paralytic neurotoxin; chemical nerve agent symptoms.
Conotoxin	3 to 6	Lethal snail neurotoxin; bleeding at injection site; muscle weakness.
Palytoxin	0.08	Lethal neurotoxin; muscle paralysis; collapse.
Rapid: 5 minutes to 1 hour		
Diphtheria toxin	0.03	Lethal; sore throat; swollen glands.
Batrachotoxin	0.1 to 2	Lethal; frog paralytic neurotoxin; neuromuscular block.
Ricin (injected)	0.1 to 3.7	Lethal cytotoxin.
Taipoxin	2	Lethal; snake paralytic neurotoxin.
	continued	

Table 4-1, Lethality and rate of action of selected toxins continued

Saxitoxin	5 to 12 (oral) 1 (aerosol)	Lethal; numbness; muscle weakness; incoordination; respiratory distress.
Tetrodotoxin	8 (injected) 30 (oral)	Lethal neuromuscular block; numbness; loss of muscle control; loss of voice.
Alpha-latrotoxin	10	Lethal spider neurotoxin; paralytic chemical agent symptoms.
Notexin	20	Lethal snake neurotoxin; paralytic.
Beta-bungarotoxin	20	Lethal snake neurotoxin; paralytic.
Cobrotoxin	75	Lethal snake neurotoxin; paralytic.
Microcystin (Fast Death Factor [FDF])	50 to 100	Lethal cytotoxin; shivering; stupor.
Delayed: 1 to 12 hours		
Ricin (aerosol, skin, oral)	3.0 (oral)	Lethal cytotoxin; nausea; vomiting; cramps.
Staphylococcus enterotoxin B	20 (injected)* 200 (aerosol)*	Incapacitant; acute food poisoning symptoms.
Botulinum (oral)	0.0003 to .01	Lethal neurotoxin; drooping eyelids; double vision; dilated pupils; fever; paralysis.
T-2 (skin, aerosol, oral)	50 to 240 (aerosol)	Incapacitant/lethal cytotoxin; skin reddening, rash, blisters; nausea; bloody vomit, diarrhea.
Very delayed: 12 hours		
Tetanus toxin (injected)	0.0025 (human)	Lethal neurotoxin; painful muscle contractions; "lockjaw."

*In mice if no other information is given.

We know little about the persistency of toxins. Persistency depends on the physical and chemical properties of the toxin in question. Protein-based toxins are usually more sensitive to UV light, heat, and oxidation than nonprotein toxins, and would be less persistent in the environment.

Individual defensive measures normally associated with a persistent chemical agent attack will protect personnel against toxins. Upon recognition of an air- or ground-contaminating attack or onset of symptoms, personnel should immediately mask and put on all protective equipment (MOPP4). Apply standard MOPP analysis procedures to determine the MOPP level required to continue operations.

Normal field equipment and procedures cannot decontaminate water taken from sources exposed to toxins (such as rivers, ponds, or wells). Therefore, do not drink water from exposed sources. Do not consume food suspected of contamination. Water and food in approved closed containers are safe for consumption after exterior decontamination of the containers and inspection by qualified medical personnel.

Section II. Sources of Toxins

Toxin sources (Table 4-2) include bacteria, dinoflagellates, algae, molds and fungi, plants, and animals. Section III presents descriptions of specific toxins.

Use soap and water or standard decontaminants (DS2, STB, or HTH) to decontaminate equipment or supplies. Washing the skin with soap and water (or flushing the skin with copious amounts of water) will reduce the effectiveness of the toxins. Evacuate contaminated casualties in accordance with unit SOP governing the evacuation of chemical casualties.

The discussion on specific toxin characteristics outlines the sensitivity of each toxin to decontaminants. Some toxins are sensitive to alkalies, some to acids, and others to heat. However, because the sensitivities are agent-dependent, the recommended method of decontamination is removal by scrubbing with soap and water.

Medical care for victims of toxin poisoning consists primarily of supportive care. Treat or prevent shock. Monitor and support cardiac and respiratory functions as necessary. Definitive medical care requires precise identification of the toxin, a capability not available in the field for all potential toxins. Antitoxin therapy is available for some toxins after identification of the agent.

Table 4-2. Classification of selected toxins by source.

Source	Toxin
Bacteria	
<i>Bacillus anthracis</i>	Anthrax toxin
<i>Clostridium botulinum</i>	Botulinum A, B, C, D, E
<i>Clostridium tetani</i>	Tetanus toxin
<i>Cornyebacterium diphtheria</i>	Diphtheria toxin
<i>Escherichia coli</i>	Heat-labile enterotoxin LT Heat-stable enterotoxin ST
<i>Shigella dysenteriae</i>	<i>Shigella dysenteriae</i> toxin
<i>Staphylococcus aureus</i>	<i>Staphylococcus</i> enterotoxin A, B, C, D, E
<i>Vibrio cholerae</i>	Cholera toxin
Dinoflagellates	
<i>Gambierdiscus toxicus</i>	Ciguatoxin; maitotoxin
<i>Gonyaulax tamarensis</i> , <i>Gonyaulax catanella</i> and other species	Saxitoxin (shellfish poison)
<i>Ptychodiscus brevis</i> (formerly <i>Gymnodinium breve</i>)	Brevetoxin (red tide toxin)
<i>Takifugu poecilonotuss</i>	Tetrodotoxin
Algae	
<i>Anacystis</i> species, <i>Anabaena flos-aquae</i>	Anatoxin A (VFDF)
<i>Microcystis aeruginosa</i> , <i>Microcystis cyanea</i>	Microcystin (FDF)
<i>Lyngbya gracillis</i> (seaweed)	Debromoaplysiatoxin
Fungi	
<i>Aspergillus flavus</i>	Aflatoxins
<i>Fusarium</i> species	Trichothecene toxins
Plants	
<i>Abrus precatorius</i> (tropical legume)	Abrin
<i>Aconitum napellus</i>	Aconitine
<i>Ricinus communis</i> (castor bean)	Ricin
<i>Rhododendron ericaceae</i> and other Ericaceae	Grayanotoxin
<i>Veratrum album</i> (lily)	Veratridine
Animals	
<i>Palythoa</i> (soft corals)	Palytoxin
<i>Aplysia</i> (sea hare)	Debromoaplysiatoxin; aplysiatoxin
	continued

Table 4-2, Classification of selected toxins by source continued

<i>Conus geographus</i> ; <i>Conus magnus</i> (fish-hunting cone snails)	Conotoxins
<i>Mytilis</i> , <i>Saxidomus</i> , other mussels	Saxitoxin (shellfish poison)
<i>Arothron</i> species (puffer fish)	Tetrodotoxin
<i>Phyllobates aurotaenia</i> and <i>Phyllobates terribilis</i> (Colombian frog)	Batrachotoxin
<i>Bungarus multicinctus</i> (banded krait)	Alpha-bungarotoxin Beta-bungarotoxin
<i>Crotalus</i> species (rattlesnakes)	Crotoxin
<i>Naja naja atra</i> (Formosan cobra)	Cobrotoxin
<i>Laticauda semifasciata</i> (sea snake)	Erabutoxin

Bacterial Toxins

Toxins produced by microorganisms cause a number of bacterial diseases. In the past these toxins have been classed into two types – exotoxins and endotoxins. Classification of these toxins depends upon their chemical composition, resistance to heat, and method of release from the pathogen. The toxins produced by microorganisms may be excreted into the surrounding medium (exotoxins) or retained with the cell (endotoxins).

Exotoxins

Exotoxins are poisonous compounds that can diffuse and that the cells that produce them can eliminate into the surrounding medium. Bacterial exotoxins are proteins of varied molecular weights. They are a normal part of the metabolic activities of the pathogen; some are enzymes. Various *Clostridium* species produce exotoxins associated with disease. *Clostridium botulinum* toxins are responsible for botulism; *Clostridium tetani* toxins cause tetanus; *Clostridium perfringens* (causing gas gangrene) can produce ten different exotoxins. Some of these attack and destroy red blood cells; others cause death (necrosis) of

tissue. *Escherichia coli* and *Staphylococcus aureus* are two bacterial species that produce heat-stable exotoxins that have their primary action upon the digestive tract (enterotoxins). These toxins produce severe nausea, vomiting, and diarrhea, but the possibility of death is remote. Humans normally acquire these enterotoxins following ingestion of contaminated food or water, but these enterotoxins may be aerosolized for warfare. Heat, acids, or alkalis can detoxify many exotoxins because they are proteins.

Endotoxins

Many organisms (particularly certain classes of bacteria) do not elaborate a soluble toxin from the living intact cells. Instead, their toxins are associated with their cell wall and are not released until the cell disintegrates. *Rickettsia prowazekii*, which causes typhus fever, produces an endotoxin. This endotoxin causes the rapid destruction of the red blood cells and increases the permeability of blood vessels, resulting in hemorrhage.

Algal Toxins

Algal toxins are by-products of algae. Most algae grow either in fresh water or in salt water. An algal bloom may produce enough toxin to kill fish or any animals that drink the water. The types of molecules involved are diverse, ranging from simple ammonia to complicated polypeptides and polysaccharides. Production of some is rather easy; some are quite potent. Little testing has been done on the ability to weaponize them. No specific means of detection is available. The greatest potential for the algal toxins as agents lies in a subversive role. These agents could upset

the normal ecology of an area, contaminate potable water supplies, and contaminate fishing areas of indigenous populations. The physiological effects vary. These effects range from the acute toxicity of paralytic shellfish poison, which produces death in a short period, to those that induce tissue changes after long exposure. Several of the toxins have undergone extensive study because of their dramatic effect on sodium-ion channels. These sodium-ion channels help to control differences between levels of sodium and potassium ions inside and outside normal cells.

The blue-green algae and dinoflagellates represent the two groups with the greatest potential as biological agents.

Blue-Green Algae

Algae in this group are very similar to bacteria. The group contains most of the toxic freshwater algae along with some of the toxic marine species. At least eight genera have exhibited toxic characteristics. The toxins of the blue-green algae *Microcystis*, *Anabaena*, and *Aphanizomenon* affect the nervous system and represent potential sources of agents. Examples of toxins from blue-green algae include Anatoxin A, microcystin, and debromoaplysiatoxin.

Dinoflagellates

Most of the toxic dinoflagellates are marine organisms within the range of 40 to 60 microns in diameter. *Gym-*

nodinium breve (*Ptychodiscus brevis*) and *Gonyaulax* are primary sources of toxins. Their toxins are best known in the United States as the causes of red tide and paralytic shellfish poisoning (saxitoxin). A dinoflagellate (*Takifigu poecilonotus*) may produce tetrodotoxin, associated with puffer fish.

Several basic differences exist between the red tide toxin and saxitoxin. The red tide toxin is an endotoxin; it is insoluble in water and very unstable. Saxitoxin is an exotoxin; it is very water soluble and stable to heat and acids. Saxitoxin had been believed to be the most potent algal toxin known. However, maitotoxin from the dinoflagellate *Gambierdiscus toxicus* is now believed to be the most potent marine toxin.

Mycotoxins

Mycotoxins include a wide variety of chemical substances produced by molds or fungi. The toxins are exotoxins. Many molds produce more than one toxin, and in numerous cases, combinations of mycotoxins enhance toxicity. Many of these toxins and/or their producing species are threats as anticrop or antianimal agents. Some, however, are threats as antipersonnel agents. Trichothecene mycotoxins, aflatoxins, and tremorgens may be of greatest concern.

Trichothecenes

The trichothecenes came to the attention of the military primarily because of reports in the mid-1970s of yellow (and other color) powder, dust, and "rain" incidents in Southeast Asia. Historically, the main interest in trichothecenes resulted from health problems in humans and animals after they ate food contaminated with molds. *Fusarium*, *Stachybotrys*, and related fungi that infect food and grains, such as corn, rye, barley, oats, millet, straw, and hay, produce the toxins. These toxins are easily produced and moderately potent. They cause damage by ingestion, by eye or skin contact, or by inhalation. They are highly persistent and difficult to decontaminate. T-2 toxin is a highly toxic member of the very large family of trichothecene mycotoxins. This manual describes it chiefly because of it has been identified in the areas of attack; it also is a specifically defined chemical that chemical or biological means can produce. However, employment is

more likely of mixtures of agents than of a single agent; these mixtures come from crude biological extracts.

Aflatoxins

Aflatoxins are toxic nonproteinaceous compounds produced by strains of *Aspergillus flavus*. Natural grain contamination by the fungus and its toxin represents a serious problem in the USSR and other countries of the world. Aflatoxins are not only toxic; they also induce cancers, malformations, and mutations. Because their effects, although severe, are relatively slow to appear, aflatoxins may not be viable as agents. The aflatoxins have enhanced effects in combination with other mycotoxins, notably with T-2 toxin.

Tremorgens

Tremorgenic mycotoxins affect the nervous system; they produce severe trembling and loss of coordination and consciousness. Some *Aspergillus* and *Penicillium* molds produce tremorgenic mycotoxins. Tremorgens probably cause naturally occurring disorders of cattle and sheep known as "staggers." Symptoms appear in laboratory animals in about 30 minutes. Tremors and hypersensitivity to stimuli, such as noise or touch, usually last from 4 to 24 hours and then subside. At lethal dosages animals have intermittent seizures leading to death. Some tremorgens cause immobility that may last for hours; recovery follows, and the victim appears to be normal.

Plant Toxins

Many plants have parts that are poisonous if they enter the body. Potential agents include the proteinaceous toxins ricin and abrin (from castor beans and *Abrus* seeds) and

certain lipid-soluble toxins from members of the lily family. See descriptions under Ricin.

Animal Toxins

A number of animals produce toxins that are described in this manual. These toxins include batrachotoxin from the Colombian frog, palytoxin from soft corals, saxitoxin from various shellfish, conotoxins from marine snails, and

tetrodotoxin from puffer fish. Snake venoms also contain a large variety of toxic substances that affect nerves or damage muscles and membranes.

Section III. Specific Toxin Characteristics

This section describes specific toxins. Descriptions include the use, source, physical and chemical properties, route of entry, symptoms, and treatment. The descriptions

also include the rate of action, mode of action, toxicity, stability, decontamination, and comments where applicable. Figures show the structures of some of the toxins.

Anatoxin A (Very Fast Death Factor; VFDF)

Use

Anatoxin A is a lethal, very rapid, paralytic neurotoxin.

Source

A freshwater, blue-green algae, *Anabaena flos-aquae* produces Anatoxin A. Culturing the algae can produce significant quantities of Anatoxin A. Chemical synthesis could probably produce large quantities.

Physical and Chemical Properties

Anatoxin A is a bicyclic alkaloid with a molecular weight of 165. It is water-soluble, but heat, light, and alkalis will destroy it.

Route of Entry

This toxin usually enters the body by ingestion. The toxic algal blooms have caused the deaths of fish, livestock, and birds.

Rate of Action

Symptoms begin in less than five minutes.

Symptoms

When ingested, Anatoxin A causes symptoms typical of chemical nerve agents. These symptoms are twitching, incoordination, tremors, paralysis, and respiratory arrest. Death results from paralysis; it may occur within minutes or up to three hours, depending upon the dose. Death in mice occurs in two to five minutes.

Treatment

There is no specific treatment.

Mode of Action

This toxin binds to the same receptor as acetylcholine; it stimulates the nerves and muscles in a similar manner. Acetylcholinesterase does not hydrolyze Anatoxin A, so stimulation continues until the neuron becomes depolarized. Evidence shows that this toxin also inhibits acetylcholinesterase.

Toxicity

The LD₅₀ in mice ranges from 170 µg/kg to 250 µg/kg, when injected intraperitoneally (ip). Dermal LD₅₀ is greater than 2,100 µg/kg oral LD₅₀ is 5,000 µg/kg. The CAS registry number is 64285-06-9, and the RTECS number is KM5527000.

Decontamination

Use hot, soapy water.

Comments

The magnitude of the threat from Anatoxin A depends on its toxicity. If the toxicity for humans is equal to the value for mice, this toxin could be a serious threat. If the human toxicity is closer to that for ducks (LD₅₀ of 50 mg/kg ip), the threat is considerably less. Figure 4-1 shows the structure.

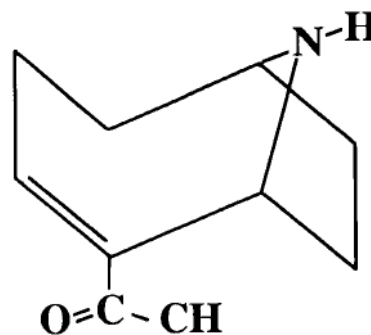


Figure 4-1. Anatoxin A (VFDF).

Batrachotoxin

Use

Batrachotoxin is a rapid-acting, lethal, paralytic neurotoxin.

Source

Batrachotoxin comes from the skin of the Colombian arrow frog (*Phylllobates aureotaenia* and related species).

South American Indians cover the points of hunting darts with a mixture of toxins secreted by these frogs. Dried natural toxin remains active for at least a year. Chemical synthesis can produce the toxin.

Physical and Chemical Properties

Batrachotoxin is a nonprotein, three-ring compound. It has a low molecular weight of 538. Batrachotoxin is not soluble in water; however, it can dissolve in nonpolar organic reagents, such as fuels, fats, and oils. Because batrachotoxin is lipid-soluble, it is probably cumulative in the body. Figure 4-2 shows the structure.

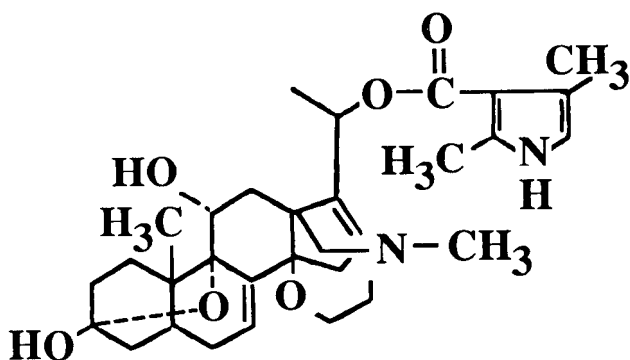


Figure 4-2. Batrachotoxin.

Route of Entry

In experiments, this toxin is usually injected. Inhalation effects should be similar.

Rate of Action

Batrachotoxin is rapid acting.

Symptoms

When given to animals, batrachotoxin causes loss of balance and coordination, profound weakness, irregular

heart rhythms, convulsions, and cyanosis (bluish skin) in rapid succession. A lethal dose in mice causes death in five to ten minutes by respiratory failure.

Treatment

Victims should receive general supportive care. They may require artificial respiration and/or cardiac resuscitation and support. No antidote or antitoxin is available. In laboratory cultures tetrodotoxin blocks the effects of this toxin.

Mode of Action

Batrachotoxin binds to sodium channels of nerve and muscle cells. It inhibits closure of the channels so the neuron becomes completely depolarized and unable to transmit a signal.

Toxicity

Batrachotoxin is about 10,000 times more lethal than Sarin. Its LD₅₀ is 0.1 to 2 µg/kg intravenously (in mice). Combining batrachotoxin with scorpion venom makes it twenty times more toxic. The CAS registry number is 23509-16-2, and the RTECS number is CR3990000.

Stability

Batrachotoxin is stable under both acidic and moderately alkaline conditions and is more active under alkaline conditions. It is somewhat nonpersistent.

Decontamination

Use soap and water to remove contamination from personnel. Because the toxin is nonpersistent, equipment would likely not require decontamination. Should decontamination prove necessary, use organic solvents if soap and water are not available.

Other Similar Toxins

Veratridine, *Aconitine*, and *Grayanotoxin* are lipid-soluble, channel-activating toxins similar to batrachotoxin; they probably have a common site and similar mechanism. These toxins are much less toxic than batrachotoxin.

- *Veratridine* is the most potent of the lipid-soluble toxins.

It comes from the lily family, genus *Veratrum*; its molecular weight is 673.

- *Aconitine* comes from the plant *Aconitum napellus*; its molecular weight is 633.
- *Grayanotoxin* comes from the leaves of rhododendron and other Ericaceae; its molecular weight is 398.

Botulinum Toxin

Use

Botulinum toxin is a lethal, delayed-action, paralytic neurotoxin. It is considerably more poisonous than nerve

gases. It causes botulism, a specific and often fatal food poisoning. Dispersion could be by aerosol.

Source

Botulinum comes from the bacteria *Clostridium botulinum* and *Clostridium parbotulinum*. These bacteria are rod-shaped, slightly motile, spore-forming, gram-positive, anaerobic bacilli. The principal reservoir of these bacteria is soil. Because these bacteria cannot grow in the presence of oxygen, natural encounter with the toxin is in improperly preserved, canned foods. The bacteria grow and produce toxin while the food sits on the shelf. Growth requires a neutral to moderately alkaline medium. Acid conditions reduce the resistance of the bacteria to sterilization by heat, which helps explain why outbreaks never implicate preserved acid fruits. Large-scale production is possible.

Physical and Chemical Properties

Botulinum toxin is a large protein (molecular weight 900,000) that has smaller subunits of molecular weights from 70,000 to 150,000. There are seven known types of toxin (A through G); type A is of greatest military interest. The molecular weight of type A toxin is 150,000. The structures of botulinum and tetanus toxins are very similar.

Purified toxin may be a white powder or a colorless needlelike crystal. It readily dissolves in water when finely powdered. It is stable in solution up to seven days when protected from heat and light. It can be used in solutions or freeze-dried as a powder.

Route of Entry

This toxin normally enters the body through the digestive system in contaminated food. Fresh, well-cooked foods are not involved, as heat destroys the toxin. The bacteria do not grow or reproduce in the human body poisoning comes entirely from the toxin already formed in the ingested material. Botulinum toxin differs from other bacterial toxins in that digestive tract secretions do not destroy it. The toxin could possibly enter the body through breaks in the skin or by inhalation, as in the case of laboratory accidents. Botulinum toxin in its powder form lends itself to entry by inhalation or contamination of wounds.

Rate of Action

Symptoms usually begin 12 to 72 hours after ingestion of contaminated food; the delay may range from 2 hours to 8 days. The delay in symptoms depends upon the amount of toxin and its absorption from the digestive tract. If toxin dispersal is by aerosol, the onset is much more rapid (averaging 3 to 6 hours), although symptoms remain the same. Introduction of botulinum through the skin is unlikely unless the skin is broken.

Symptoms

Initial symptoms include weakness, malaise, dizziness, and in some cases nausea and profuse vomiting. Other

symptoms include difficulty swallowing and speaking, blurred or double vision, sensitivity to light, and muscular weakness progressing from the head downward. In severe cases, death results from respiratory paralysis. All personnel possibly exposed to the toxin should seek immediate medical attention, because it is difficult to treat once symptoms appear.

Treatment

Medical care consists of supportive measures, including mechanical respiration. Antitoxin is available; its administration should take place immediately upon suspecting botulism poisoning. Upon recognizing a case of botulism, immediately search for all other possibly exposed persons. Treatment after severe symptoms set in is usually ineffective; the antitoxin will not reverse existing paralysis. Recovery is very slow, and several months may pass before a victim regains certain muscle movements.

Mode of Action

Botulinum toxin inhibits acetylcholine release. The toxin is highly specific for the nerve-muscle junctions and synaptic ganglia. The toxin acts presynaptically. Botulinum toxin probably does not cross the blood-brain barrier.

Toxicity

This toxin is among the most potent biological toxins known. The exact lethal dose for humans is unknown, but it may be as low as 1 to 10 nanograms. Mortality rate is 60 percent or higher. Animal studies show an LD₅₀ of 0.001 to 1 ng/kg. The LD₅₀ in mice is about 0.3 ng/kg. Humans are less sensitive to botulinum toxin than mice are. The oral LD₅₀ for humans is about 1 µg/kg. Studies show that inhaled toxin is ten times to a hundred times more toxic than ingested toxin. The RTECS number is ED9300000.

Stability

The persistency of this toxin varies. Botulinum toxin decomposes within 12 hours in the air. It is stable for a week in nonmoving water. It may persist indefinitely in food when not exposed to air. This toxin is probably not UV-light sensitive; it is easily stabilized to environmental conditions. However, heat may destroy the toxin.

Decontamination

Basic skills decontamination for personnel would prove effective in neutralizing this toxin. If mustard may be present, use a 1-percent to 2-percent hypochlorite solution (from household bleach, STB, or HTH). The toxin can withstand acids, but bleach or other alkaline solutions can destroy it. This toxin is sensitive to heat. Boiling for 15 minutes or, when in food, cooking for 30 minutes at 80°C (176°F) will destroy it.

Comments

Because of its intense toxicity, water volubility, and difficulty in detection, this agent could present a particularly

great hazard, Immunization with botulinum toxoid is possible for types A through E.

Other Clostridium Species

Other *Clostridium* species, which are anaerobic spore-forming bacteria that produce toxins, include *Clostridium tetani* and *Clostridium perfringens*. Employment of these toxins is less likely. *Clostridium tetani* produces tetanus toxin, a lethal delayed-action paralytic neurotoxin, causing "lockjaw." Tetanus toxin is produced after introduction of the tetanus spores into the body. They grow at the site of an injury, usually a puncture wound contaminated with soil,

street dust, or feces. The purified (crystalline) toxin is relatively unstable and very sensitive to heat. Its toxicity is about the same as crystalline botulinum toxin. The lethal oral dose for humans is probably 0.2 to 0.3 mg. *Clostridium perfringens* toxin causes gas gangrene in tissues surrounding a wound. *Clostridium perfringens* is similar to tetanus in its mode of transmission.

Conotoxins

Conotoxins are small, proteinaceous neurotoxins from fish-hunting sea snails (*Conus*). The toxins are 13 to 14 amino acids long; they can easily be chemically synthesized or produced by genetic engineering. Conotoxins are water-soluble and highly stable; dissemination could be by

aerosols. Alpha-conotoxin blocks the acetylcholine receptors and produces extreme muscle weakness (flaccid paralysis) and respiratory and circulatory failure. The LD₅₀ in mice is 15 to 30 µg/kg. The estimated lethal dose for humans is 3 to 6 µg/kg.

Microcystin (Fast Death Factor; FDF)

Use

Microcystin is a lethal, rapid-acting cytotoxin.

Source

A freshwater, blue-green alga, *Microcystis (Polycystis) aeruginosa*, produces microcystin. Other freshwater, blue-green algae may also produce it. Lyophilized (freeze-dried) microcystin retains its toxicity. See comments.

Physical and Chemical Properties

Microcystin appears to be a family of small, cyclic peptides. The most common toxin in the family has a molecular weight of 994; others are similar. Microcystin is derived from a polypeptide with a molecular weight of 1,790 to 2,950. It is soluble in water, acids, bases, and some polar solvents, such as alcohol and acetone.

Route of Entry

Microcystin is potent in air-dried material. If Threat forces use it as a warfare agent, it would presumably enter the body through the respiratory system. There are reports of human exposure in which the victims drank water from contaminated reservoirs.

Rate of Action

See symptoms.

Symptoms

Microcystin causes severe and rapid liver damage with resulting shock, liver enlargement, and death in a matter of hours. Symptoms would vary with the dose received. Symptoms in test animals include shivering and increased breathing rate and depth. These symptoms precede muscle twitching, convulsions, and gasping respiration before death. In test animals given twice the lethal dose, death occurs one to three hours after exposure.

Treatment

Victims should receive general supportive care. No antidote or antitoxin is available. Substances that protect against the mushroom alkaloid phalloidin (for example, rifampicin and deoxycholate) reduce cell deformation.

Mode of Action

Microcystin deforms and disrupts cell membranes in the liver. Test animals have shown extensive liver damage leading to circulatory collapse.

Toxicity

Its LD₅₀ in mice is 25 to 100 µg/kg (ip) with a survival time of 30 to 90 minutes. The LD₅₀ is much higher by oral or dermal routes. Each gram of lyophilized cells contains about 1 to 4 milligrams of toxin; toxicity of cellular material is about 50 mg/kg. The RTECS number is XW5810000.

Stability

Microcystin is heat-stable when dry but unstable to heat when wet. It is stable to acid but sensitive to highly alkaline conditions.

Decontamination

Use large amounts of soap and water. Because microcystin is soluble in water and sensitive to alkalis, large amounts of water, STB, or DS2 will decontaminate supplies and equipment.

Comments

We know relatively little about the physical and chemical properties of this toxin. However, reports indicate the Soviets have done considerable research with it. Toxicity appears to be associated with a plasmid. If it is, it should be possible to clone the gene and have it expressed in another organism.

Palytoxin

Use

Palytoxin is a lethal, rapid-acting neurotoxin.

Source

A bacterium associated with soft corals of the genus *Palythoa*, which inhabit the digestive tract of filefish, produce palytoxin. It can be isolated from the corals. See comments.

Physical and Chemical Properties

Palytoxin is a relatively large, nonproteinaceous toxin; its molecular weight is 2,677. It is a polyhydroxy, long-chain macromolecule with a cyclic structure. It is soluble in water or alcohol. See Figure 4-3 for the structure.

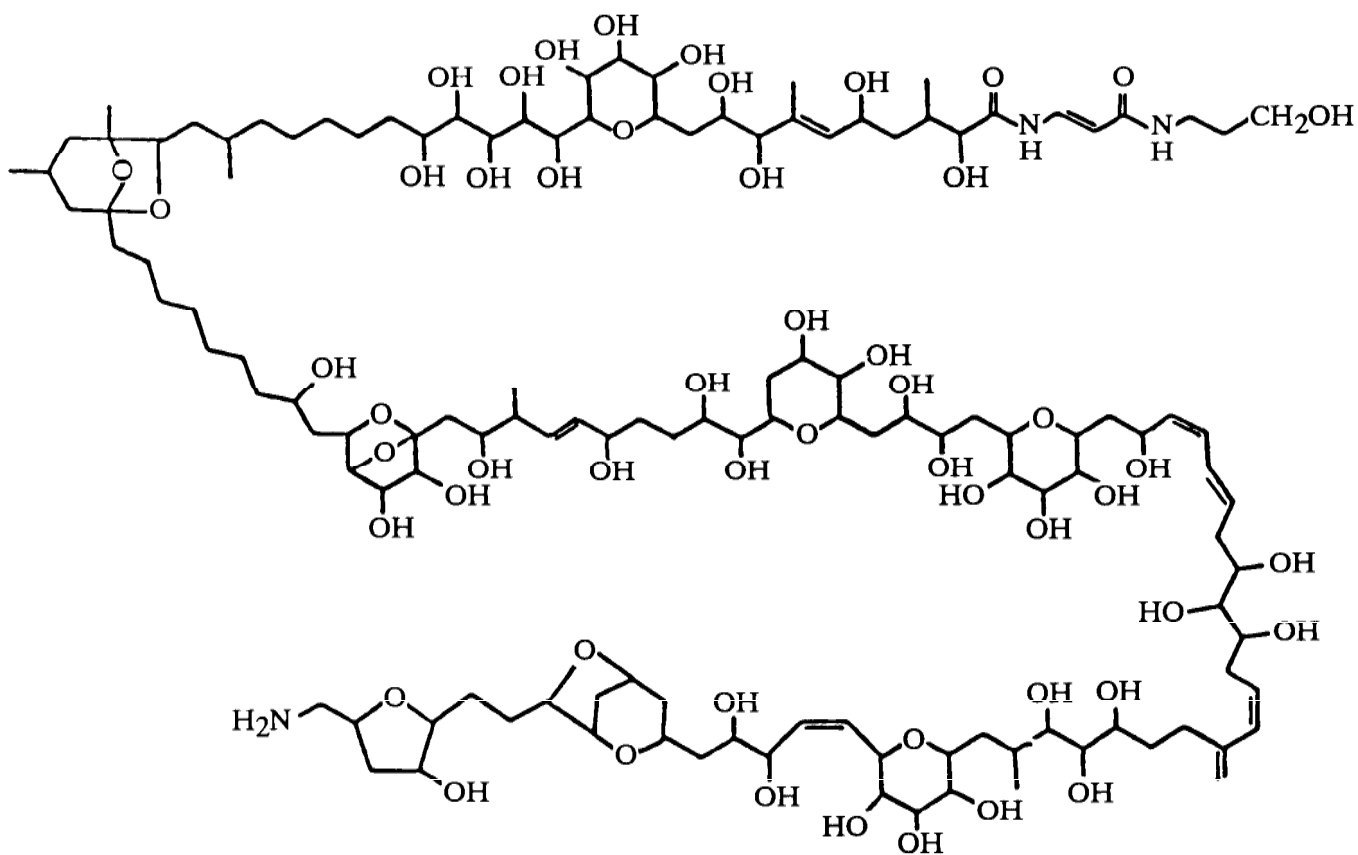


Figure 4-3. Palytoxin.

Route of Entry

This toxin could enter the body by inhalation (as aerosolized toxin), ingestion, or absorption through the skin or eyes. Absorption through intact skin requires high doses.

Symptoms

There have been no reported cases of human poisoning. Experimental animals show symptoms of drowsiness, weakness, vomiting, respiratory distress, diarrhea, convulsions, shock, low body temperature, and death within 30 to 60 minutes after intravenous injection. Death may result from constrictions of the blood vessels of the heart.

Treatment

Victims should receive general supportive care. There is no definitive human treatment. However, rapid administration of steroids has reduced the severity of effects.

Mode of Action

This toxin has a very potent effect on the coronary artery. It apparently causes irreversible depolarization of nerve and muscle tissue by an unknown mechanism, possibly affecting sodium channels. Palytoxin in high concentra-

tions exhibits delayed effects, causing the disintegration of red blood cells.

Toxicity

The LD₅₀ is about 0.08 µg/kg in monkeys, 0.2 µg/kg in cats, and 0.4 µg/kg in mice. Mouse LD₅₀ through the skin is 1,270 µg/kg. The CAS registry number is 11077-03-5, and the RTECS number is RT647500.

Stability

Palytoxin is stable to heat, acids, and alkalis.

Decontamination

Because of the stability of this toxin in a variety of conditions, decontamination should include large amounts of water.

Comments

The exotic nature of the biological source limits the possibilities for extraction of this toxin. However, advances in genetic engineering could make the manufacture of this toxin possible.

Ricin

Use

Ricin is a lethal, delayed-action cytotoxin; it is persistent in the environment.

Source

This toxin comes from the seeds of the castor bean plant, *Ricinus communis*. This relatively inexpensive, accessible, natural source allows easy preparation of large quantities of ricin; therefore, there is little motivation to produce it synthetically. Large-scale production of ricin by recombinant DNA techniques is probably possible.

Physical and Chemical Properties

Ricin is a lectin – a plant glycoprotein that binds and agglutinates animal cells. This toxin has a molecular weight of 65,000. It consists of two polypeptide chains linked by a disulfide bond. It is soluble and stable in water or dilute acid.

Route of Entry

Ricin normally enters the body by ingestion. Aerosolized ricin would enter the body by inhalation. The toxin attaches to cell surfaces of a variety of tissues, particularly the stomach lining if ingested or the moist, upper respiratory tissues if inhaled.

Rate of Action

Initial symptoms usually appear between 6 to 10 hours and 3 days. Clinical signs appear as early as 45 minutes after oral administration if the victim has an empty stomach.

Symptoms

The symptoms may include nausea, vomiting, bloody diarrhea, abdominal cramps, breathing difficulty, renal failure, and circulatory collapse. Victims may linger for 10 to 12 days before death or recovery, depending upon the dose received.

Treatment

Victims should receive general supportive care including fluid input and support of circulation and respiration. Antitoxin is available; its early administration is necessary to prevent severe tissue damage. Fluid input is critical, as fluid losses of up to 2-½ liters are probable.

Mode of Action

Ricin inhibits protein synthesis.

Toxicity

The oral LD₅₀ for humans is 1 mg/kg; a single seed can be fatal. The LD₅₀ in mice is about 3 µg/kg by injection or aerosol. The CAS registry number is 9009-86-3, and the RTECS number is VJ262500.

Stability

Ricin is stable in water or dilute acid.

Decontamination

As for most other toxins, use soap and water to remove contamination from personnel, equipment, and supplies,

Comments

Immunizations are highly effective in animals.

Saxitoxin (Paralytic Shellfish Poison)

Use

Saxitoxin is a lethal, rapid-acting, paralytic neurotoxin.

Source

The first isolation of saxitoxin was from the toxic Alaska butter clam, contaminated by dinoflagellates of the genus *Gonyaulax*. (Shellfish and mussels become contaminated while feeding on them.) Recently puffer fish that had ingested *Protogonyaulax* revealed saxitoxin. The toxin has also been identified in a freshwater, blue-green alga. See comments.

Physical and Chemical Properties

This nonprotein compound is structurally related to tetrodotoxin. Saxitoxin normally appears as the dihydrochloride salt, a white powder that would lend to its use in an aerosol. The toxin is very soluble in water and slightly soluble in alcohols. Figure 4-4 shows the structure.

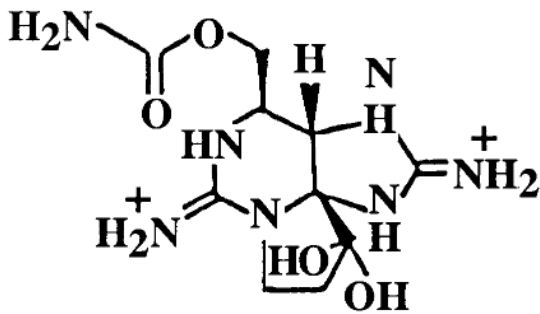


Figure 4-4. Saxitoxin.

Route of Entry

This toxin usually enters the body by ingestion, but inhalation is also possible. It also can enter the body through wounds.

Rate of Action

Symptoms occur between ten minutes and four hours (average 30 minutes) after ingestion of this toxin. Inhalation of the toxin will produce a more rapid onset. Injection may cause death in less than 15 minutes.

Symptoms

Ingested saxitoxin causes tingling or burning sensations of the lips, face, and tongue. The sensations occur in fingertips and gradually change to numbness, spreading to the arms, legs, and neck. Incoordination and associated symptoms may occur. These associated symptoms include a feeling of lightness, dizziness, vomiting, nausea, headache, drooling, rapid pulse, and abdominal pain. Severe, generalized muscle weakness (flaccid paralysis) can lead to death from respiratory failure in 1 to 24 hours. If the casualty survives 18 hours, recovery is usually rapid and complete.

Treatment

Induce vomiting and provide general supportive care. Artificial respiration may be necessary. **Faulty identification of this toxin as nerve gas with resultant use of atropine would increase fatalities.**

Mode of Action

Upon entry this toxin blocks transient sodium-ion channels and causes paralysis by blocking depolarization. Its action is similar to that of tetrodotoxin.

Toxicity

The LD₅₀ in mice is about 1 µg/kg through aerosol exposure, about 8 µg/kg by injection, and 322 µg/kg orally. Toxicity in humans (LD₅₀ 5.7 µg/kg) is similar to that in mice when introduction of saxitoxin is directly into the body. However, to humans it is much more toxic by mouth (LD₅₀ 7 µg/kg), equivalent to direct introduction into the body. A single contaminated mussel can contain up to 50 lethal human doses. If aerosolized, the estimated LC₅₀ by inhalation is 5 mg-min/m³. The estimated LC₅₀ by wound contamination is 0.05 mg/person. As the dihydrochloride salt, the CAS registry number is 35554-08-0. The RTECS number is UY8708600.

Stability

Saxitoxin is relatively persistent. It is resistant to heat and acid but very sensitive to alkaline solutions.

Decontamination

Decontaminate with soap and water. Any standard decontaminant is also acceptable, because saxitoxin is very sensitive to alkalis.

Comments

Although chemical synthesis of saxitoxin is feasible, the cost would probably prohibit production. Dinoflagellates are difficult to culture. However, production of the toxin by culturing dinoflagellates will probably become feasible in the future.

Scorpion Venom Toxins

Use

Scorpion toxins are delayed-action neurotoxins.

Source

Toxins can be extracted from various scorpions. Large-scale manufacture of scorpion toxins would require genetic engineering techniques.

Physical and Chemical Properties

Scorpion venom consists of a family of small, basic proteins that are rigid in structure. The chemical structures of over 30 distinct scorpion toxins have been identified. They are very similar with molecular weights of about 7,000. The toxin components are water-soluble and heat-stable.

Route of Entry

These toxins usually enter the body by injection. They could also be aerosolized and would enter the body by inhalation.

Rate of Action

This toxin has a somewhat delayed action; therefore, determining the nature of the attack could be difficult.

Symptoms

Localized pain and swelling usually occur upon injection. Mainly, scorpion toxins affect the cardiovascular and neuromuscular systems, similar to the effects of chemical nerve agents. Initial symptoms in cases of exposure by inhalation are unknown. Eyes may water and vision may dim. The pulse rate becomes rapid and irregular, and blood pressure increases. Breathing becomes difficult; respiratory or congestive heart failure may occur in about 20 hours. Rigid paralysis may also result from exposure.

Treatment

Atropine counters some toxic effects *in vitro* (cell cultures). Prevent and treat shock, and provide artificial respiration. Relieve pain with procaine. **Do not use narcotics.** Barbiturates will lessen patient stress. Antivenin are available, but maximum effectiveness requires administration within two hours of exposure.

Mode of Action

These toxins disseminate through the bloodstream to the nervous system. They modify the sodium-ion channels, resulting in continuous release of acetylcholine and other neurotransmitters.

Toxicity

The lethality of venom is 0.34 to 1.2 mg/kg in mice. The potency of the toxic components varies between species; the most potent toxins have LD₅₀ values of 0.02 mg/kg.

Stability

Scorpion venom would be relatively persistent. Scorpion toxins are very stable to heat, acids, bases, and denaturants. They also are stable to enzymes that digest proteins (proteases).

Decontamination

Use soap and water to remove contamination from personnel, equipment, and supplies.

Comments

If combined with batrachotoxin and related toxins, the venom is twenty times more toxic.

Snake Venoms and Toxins

Use

Snake venoms consist of a rapid-acting mixture of toxins.

Source

A variety of poisonous species could serve as sources for biological toxins. These species include the cobras, kraits,

and coral snakes (elapids) and the rattlesnakes, copperheads, and other vipers (crotalids). Whole snake venoms are relatively unavailable. The toxins might be more available. See comments.

Physical and Chemical Properties

Generally, snake venoms are extremely complex mixtures of water, low-molecular-weight protein toxins, enzymes, and salts. These venoms may contain a variety of water-soluble toxins, including neurotoxins, cardiotoxins, and toxins that cause severe tissue destruction and hemorrhage. The mixture of these toxins varies widely between species.

The toxic protein components of snake venom vary in size and stability. Some are small proteins (for example, cobra cardiotoxin with a molecular weight of 6,000 to 7,000). Others are much larger (for example, textilotoxin with a molecular weight of 80,000). Large proteins are usually less stable than small ones. Myotoxin is a small polypeptide (with a molecular weight of 4,600) and is very stable. Cobratoxin (molecular weight of about 7,000) is relatively heat-stable. However, cardiotoxins (basic proteins with a molecular weight of 6,000 to 7,000) lose their toxicity when heated to 90°C for 30 minutes. Exposure to ultraviolet light for 15 to 20 minutes will also cause them to lose their toxicity.

Route of Entry

These toxins usually enter the body by injection. However, they could possibly be aerosolized and enter the body by inhalation. These toxins disseminate throughout the body through the bloodstream and affect target tissues.

Rate of Action

Snake venoms and toxins produce effects rapidly.

Symptoms

Cobras, kraits, mambas, and coral snakes (elapids) produce venom that primarily affects the nervous system. The active compounds of these venoms are neurotoxins, cardiotoxin, and enzymes. Venoms of most pit vipers, such as rattlesnakes and copperheads, contain very small amounts of neurotoxins. These venoms tend to cause severe local tissue destruction (necrosis), including muscle destruction, and hemorrhage.

Upon injection of neurotoxic components, initial symptoms usually include pain and swelling at the site of injection. Inhalation would probably result in several symptoms. These symptoms include fluid accumulation in the lungs, painful and difficult breathing, drowsiness, drooping eyelids, blurred vision, vomiting, and difficulty in speaking. A severe drop in blood pressure and shock frequently follow. Convulsions and paralysis may occur. Death may result from cardiac or respiratory failure or shock.

Cardiotoxins reduce the blood pressure and heart rate, cause heart irregularities, and eventually stop the heartbeat; however, they have no direct neurotoxic activity.

Cobra, mamba, and coral snake venoms contain cardiotoxins.

Necrotic toxins from pit viper venoms cause severe local tissue destruction, including muscle destruction and hemorrhaging. Myotoxin destroys muscles but not blood vessels. Dozens of hemorrhagic toxins, zinc-containing enzymes (proteases) that act on proteins, have been isolated.

Treatment

Initial care for victims normally consists of measures for the treatment and prevention of shock. The victim should be calmed and evacuated. Circulation and/or respiration may require support. Antisera are available, and administration should take place as soon as possible. Experimentally, neostigmine and atropine have been used in the treatment of victims of cobra neurotoxin.

Mode of Action

The neurotoxins usually act by blocking transmission at the synapse. The other components have a variety of destructive (necrotic and hemolytic) effects against target tissues.

Snake neurotoxins inhibit transmission before (presynaptic) or after (postsynaptic) the synapse. Presynaptic inhibitors initially increase acetylcholine and then block acetylcholine release, causing a flaccid (limp) paralysis leading to circulatory and respiratory failure. Venomous snakes, such as the tiger snake and taipan from Australia, the Asiatic banded krait, and the South American rattlesnake produce presynaptic neurotoxins.

Postsynaptic inhibitors block the receptor for the neurotransmitter acetylcholine, almost irreversibly. Examples of postsynaptic inhibitors are cobratoxin from the Formosan cobra, erabutoxin from the sea snake, and alpha-bungarotoxin from the banded krait.

Toxicity

The potencies of snake venoms vary considerably. Variations range from 0.05 mg/kg for the brown snake, 0.1 for the Taipan, 0.16 for the sea snake, and 0.56 for the cobra to 11.4 for the Eastern diamondback rattlesnake. As little as 1 to 20 milligrams of a purified toxin component will usually prove fatal to the average human. The LD₅₀ in mice of some snake toxins are: taipoxin 1 µg/kg; beta-bungarotoxin 14 µg/kg; crotoxin 50 µg/kg; erabutoxin 150 µg/kg; cardiotoxin 1,500 µg/kg; myotoxin 5,000 µg/kg.

Stability

The complex nature of whole snake venoms should make them relatively nonpersistent in the environment. The stability of the components varies.

Decontamination

If required, decontaminate with soap and water.

Comments

The isolation or manufacture of individual components might produce these toxins. Genetic engineering techniques could enhance the process.

Staphylococcus Enterotoxin Type B (SEB)

Use

This toxin is a rapid-acting toxin. The vomiting, diarrhea, and painful cramps associated with staphylococcal toxins make them effective incapacitants. The incapacitating effects (about a day) would last longer than those of many potential chemical incapacitants.

Source

The bacteria *Staphylococcus aureus* produces Staphylococcus enterotoxin type B (SEB). Staphylococcal food poisoning usually results from ingestion of the toxins rather than ingestion of the bacteria. Foods contaminated with SEB have a normal appearance, odor, and taste. A potential natural hazard exists in situations involving mass feedings and lack of refrigeration; improper food handling is responsible for many natural outbreaks. Large-scale production of the enterotoxin appears possible by recombinant DNA techniques.

Physical and Chemical Properties

The staphylococcal enterotoxins are a group of globular proteins with molecular weights ranging from 27,000 to 35,000. There are at least eight types: A, B, C1, C2, C3, D, E, and F. Purified *Staphylococcus enterotoxin*, type B is a white, fluffy material. The toxin is water-soluble and stable in heat, cold, acids, and bases.

Route of Entry

Victims normally encounter this toxin as a food poisoning. However, inhalation of aerosolized toxin is possible.

Rate of Action

Symptoms usually occur within one-half to six hours (average three hours) after ingestion. Symptoms can appear within a few minutes after exposure to large doses by aerosol. The use of purified toxin would probably result in simultaneous incapacitation of all the troops. Use of the bacteria would produce a longer effect, with some troops recovering as others became ill.

Symptoms

Victims experience a sudden onset of vomiting, abdominal cramps, nausea, explosive watery diarrhea, and severe weakness. The symptoms usually continue 6 to 8 hours, rarely longer than 48 hours. Recovery usually occurs spontaneously within a day with no residual effects. However, the victim may be weak for another few days, especially

when the loss of body fluids is severe. Difficulty breathing, because of fluid accumulation in the lungs, may occur in severe inhalation cases.

Treatment

Rest and fluids will promote recovery. Seek medical care if there is respiratory distress. Production of an antitoxin or toxoid vaccine should be possible, although none is currently available.

Mode of Action

The toxin interacts with receptors in the gut, causing a massive loss of fluids. The toxin also stimulates intense vomiting.

Toxicity

Animals vary in susceptibility to this toxin. Humans appear to be more sensitive to SEB than laboratory animals (see Table 4-3). This toxin is unusual in that lethal doses by ingestion are hundreds of times higher than incapacitating doses. Deaths rarely occur in healthy individuals with normal exposure to this toxin. However, if exposed through tactical employment, victims could receive massive doses that could cause death.

Table 4-3. Susceptibilities to *Staphylococcus enterotoxin* type B.

	Rhesus monkey	Cynomolgus monkey	Human (estimated)
ED₅₀			
Intravenous	0.1 µg/kg	0.08 µg/kg	
Aerosol	6.0 µg/kg	3.0 µg/kg	30 ng/person
Oral	1.0	1.0 µg/kg	20 to 25 µg/person
LD₅₀			
Intravenous	24 µg/kg	11 µg/kg	
Aerosol	27 µg/kg	11 µg/kg	1.7 µg/person
Oral	—	—	—
IC_{t50}			0.1 to 1 mg-min/m ³
LC_{t50}			5 mg-min/m ³

Stability

The toxin resists acids, bases, and chlorine in amounts found in potable water. The toxin is quite stable to heat and

freezing its destruction in food or water requires boiling longer than 30 minutes. The organisms that produce the toxin remain viable after 67 days of refrigeration.

Decontamination

Use large amounts of soap and water to decontaminate personnel, equipment, and supplies. SEB is difficult to decontaminate with active chlorine (STB, HTH). Formaldehyde detoxifies SEB.

Tetrodotoxin (TTX); Fugu Poison

Use

Tetrodotoxin is a rapid-acting, lethal, neurotoxic agent.

Source

Tetrodotoxin comes primarily from the liver and ovary of puffer fish (*Arothron*). It also comes from some species of newt, octopus, frogs, and goby. Dinoflagellates (*Takifugu poecilonotus*) also may produce it.

Physical and Chemical Properties

Tetrodotoxin is a water-soluble, three-ring nonprotein compound with a molecular weight of 319. Purified toxin may occur as colorless crystals or a white powder (lending itself to aerosol delivery). It is soluble in dilute acetic acid and slightly soluble in water or ether. Strong acids and in alkaline solutions destroy it. Figure 4-5 shows the structure.

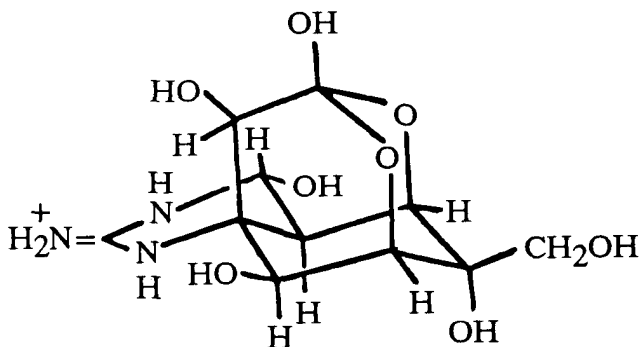


Figure 4-5. Tetrodotoxin.

Route of Entry

The toxin usually enters the body by ingestion, but inhalation or entry through abraded skin is also possible. See comments.

Rate of Action

The onset of symptoms occurs within minutes after injection (and presumably after inhalation) of the toxin.

Comments

The protective mask provides adequate protection from inhalation of SEB disseminated as an aerosol. In the context of biological warfare, several measures will reduce the likelihood of casualties. These measures include effective protection of food and water supplies (and containers for transportation of food to remote sites), and the boiling of all water (even if chlorinated). Measures also include the use of high temperatures for cooking followed by immediate serving.

Symptoms develop more slowly with ingested toxin, taking 10 to 45 minutes.

Symptoms

Symptoms include nausea, vomiting, dizziness, paleness, and malaise. The victim may experience tingling and prickling sensations that proceed to general numbness. Weakness, dilation of pupils, twitching, tremor, and loss of coordination follow. Severe, generalized muscle weakness leading to death by respiratory arrest may occur within minutes of the onset of symptoms. Symptoms and mechanisms are similar to saxitoxin poisoning, although tetrodotoxin also produces severe shock.

Treatment

Victims will require general supportive care with particular attention paid to maintaining respiration. Except at very high doses, this toxin does not normally affect cardiac function. No antidote or antitoxin is available. Anticholinergics, such as atropine, are not effective.

Mode of Action

Once inside the body, tetrodotoxin inhibits sodium-ion channels in nerves and muscles. As a result, the nerve impulse is lost and paralysis occurs. (It does not affect the neuromuscular junction.)

Toxicity

The inhaled toxin is extremely potent with an LD₅₀ of about 100 to 200 µg/person (1.5 to 3 µg/kg). Ingested tetrodotoxin requires a much larger dose (30 µg/kg) because of the destruction of the toxin by the acid in the stomach. The ingested toxin is still highly toxic, however. The oral LD₅₀ in mice is 435 µg/kg. Injected LD₅₀ is 8 to 14 µg/kg in mice, dogs, and rabbits. The CAS registry number for tetrodotoxin is 4368-28-9, and the RTECS number is 101450000.

Stability

The toxin is stable to heat, but strong acids and alkaline

Decontamination

Use water with STB to decontaminate equipment. DS2 will also break down tetrodotoxin. Decontaminate skin with soap and water.

Comments

The Japanese especially value puffer fish, called fugu in the Orient, as a delicacy. The sensations of eating fugu probably result from the narcotic effect caused by ingestion of low levels of tetrodotoxin. Tetrodotoxin is common in Haitian voodoo as a toxin that creates zombies by direct introduction into the blood through abraded skin.

Trichothecene (T-2) Mycotoxins

Use

Trichothecene (T-2) mycotoxin may be suitable as a nonlethal harassing agent. There is a marked difference between the very low, effective (incapacitating) dose and the high, lethal dose. Therefore, there may be many ill casualties who will not die.

Source

The trichothecenes are a family of about 40 rapidly acting, fungal toxins (mycotoxins). They are primarily isolated from molds of the genus *Fusarium* found on infected grain. Harvesting and extracting infected grain can produce large amounts of these toxins.

Physical and Chemical Properties

These cytotoxins are nonproteins. Volubility in water or other solvents depends on the structure of the toxin (that is, any hydroxyl, acetyl, and ester groups attached). Figure 4-6 shows the structure. These toxins are heat-stable, usually water-soluble, but sensitive to strong acids. Purified T-2 toxin may occur as colorless crystals or as a clear to yellowish oil. The molecular weight of T-2 is 466. Figure 4-6 shows the structure.

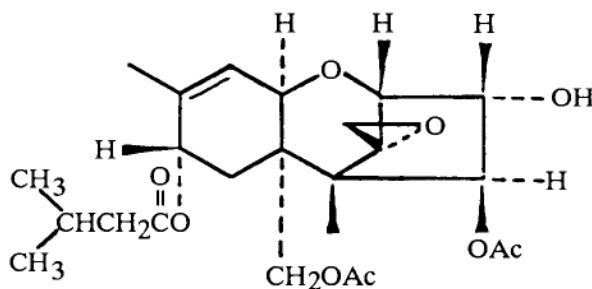


Figure 4-6. Trichothecene (T-2).

Route of Entry

Dissemination of trichothecenes is likely to be in powder or smoke aerosol form or as large, liquid drops as a ground contaminant. (They have sometimes been called “yellow rain.”) These toxins enter the body by absorption through

the skin or eyes, inhalation (aerosols), or ingestion in contaminated food or water.

Protection

Upon recognition of an attack or onset of symptoms, personnel should immediately mask and put on all protective equipment (MOPP4). Skin exposure to toxins may result in severe itching. This itching would make the protective mask uncomfortable to wear. If vomiting occurs, a damp cloth held over the nose and the mouth may help limit additional inhalation of toxins until the victim can control the vomiting and redon the protective mask.

Rate of Action

Time to effects after exposure depends on the dose. Initial symptoms may occur within 1 hour after inhalation or as long as 12 to 24 hours after skin contact. High doses produce vomiting, dizziness, rapid heart beat, and chest pain within 10 to 30 minutes. Skin irritation is delayed 12 to 24 hours; death may occur within a day. After low, single-dose exposure, peak effects tend to occur in 1 to 3 days; skin irritation may be the first symptom.

Symptoms

Low-dose symptoms include nausea; shortness of breath; dizziness; eye and skin irritation; formation of small, hard blisters; and chest pains. Although trichothecene symptoms may resemble those of blister agents, nausea commonly occurs with exposure to these toxins. Trichothecenes are “radiomimetics”; that is, they mimic the effects of ionizing radiation. High doses can result in additional symptoms, such as bloody vomit or diarrhea and blistering of the skin, within hours. Death may follow rapidly from high doses because of massive hemorrhaging and shock, or it may occur weeks later. The delayed death would result from bone marrow suppression (leading to anemia and reduction in immunity), liver failure, and/or internal bleeding.

Treatment

Victims require general supportive care. No antidote or antitoxin is available. For ingested toxin, repeated doses of oral charcoal can be helpful.

Mode of Action

The precise mode of action is unknown. One effect is inhibition of protein synthesis. The toxin also affects clotting factors in the blood, leading to hemorrhage. The most pronounced effects occur in rapidly dividing cells (that is, blood and bone marrow cells).

Toxicity

The best estimate for the human lethal dose is 3 to 35 milligrams. The ED₅₀ for vomiting is 0.1 mg/kg; and for skin irritation it is tenths of a microgram. Microgram doses can cause irreversible injury to the eyes. Doses as low as 5 nanograms (10⁻⁹ gram) may cause skin irritation. Doses as low as 14 µg/kg can cause sustained nausea for days. Aerosol doses may well be ten times more potent than parenteral doses. The CAS registry number for trichothecene mycotoxins is 21259-20-1, and the RTECS number is YD0100000. Table 4-4 shows susceptibilities to this toxin.

Note: Small repeated doses may accumulate to lethal levels.

Stability

The trichothecenes are very stable; storage for years at room temperature produces no loss of activity. Environmental persistency of T-2 is five to seven days. They are heat-stable with no loss of activity noted after heating at 100°C for one hour. They are quite stable in solution. Strong acids will abolish all toxic effects.

Decontamination

Rapid removal of toxins from the skin and eyes is essential. Use water or saline for the eyes; use soap and water with repeated flushing for the skin. Flushing the skin with water is an acceptable field expedient procedure for T-2 toxin if done within five minutes after exposure. The M258 and M258A1 skin decontamination kits are effective in

removing T-2 toxin from the skin. The use of absorbents, such as diatomaceous earth, is not effective.

Usually, soap and water will effectively remove toxin from equipment and supplies. Total destruction requires strong bleach and sodium hydroxide (NaOH; lye) or strong acids. STB and DS2 are effective decontaminants (30 minutes at 70°F [21°C] to 80°F [27°C] on nonporous surfaces). Household bleach diluted 50 percent with water or a mixture of bleach, vinegar, and water makes an effective field expedient decontaminating agent for T-2 for a four-hour contact time. T-2 resists decontamination even at very high temperatures. If contamination appears oily, fuels would effectively remove it.

Table 4-4. Susceptibilities to trichothecene (T-2).

	Mouse	Rat	Humans (estimated)
LD₅₀			
Aerosol	0.24 mg/kg	0.05 mg/kg	25 to 50 µg/kg
Intravenous	4.2 mg/kg	1.2 mg/kg	500 µg/kg
Intraperitoneal (ip)	5.2 mg/kg	2.6 mg/kg	
Oral	10.5 mg/kg	2.3 mg/kg	1.6 mg/kg
Dermal	> 67 mg/kg	4.3 mg/kg	2.4 to 8 mg/kg if in carrier
Incapacitation			
Skin reddening and burning			10 ng/cm ₂
Nausea and/or vomiting			50 to 100 µg/kg
Eye damage			1 µg/eye

Appendix A

Chemical Agent Physical Properties

Soldiers in the field do not need to know exact chemical composition of matter, including chemical agents. However, the behavior of these agents in the environment directly results from their composition. Therefore, the composition is important. Summary statements that describe the behavior of chemical agents use such terms as vapor density, melting point, boiling point, and latent heat of vaporization. Most of these terms relate to temperature. If you understand how temperature affects matter, you will understand these terms. You will thus better understand the behavior of agents.

Thermochemistry

The quantity of energy (calories) required to raise the temperature of one gram of material one degree Celsius is a characteristic property of the material known as the specific heat. Specific heat changes predictably with temperature so long as the material under observation does not undergo a change in physical state. As heat is supplied to ice, its temperature increases until it reaches the melting point. If the heat energy supply is very slow or if it stops altogether, an equilibrium occurs. An equilibrium is a condition in which the number of molecules per unit of time that are going from the solid to the liquid state equals the number going from the liquid to the solid state. Determina-

tion of the temperature of phase transition, such as that occurring when melting or boiling is taking place, always takes place under equilibrium conditions.

The number of calories of heat required to melt a gram of ice into water at 0°C is the latent heat of melting. As liquid water receives even more heat, its temperature increases until it reaches the boiling point. This is the temperature at which the vapor pressure of the water equals the pressure of the atmosphere on the water. Boiling points are normally established where the atmospheric pressure is equal to 760 mm Hg.

Molecular Weight

Molecular weight (MW) is the value represented by the sum of the atomic weights of all the atoms in the molecule. For example, the MW of ethyldichloroarsine, C₂H₅AsCl₂, is as follows:

$$\begin{aligned} \text{C (atomic weight = 12)} \times 2 &= 24.0 \\ \text{H (atomic weight = 1)} \times 5 &= 5.0 \\ \text{As (atomic weight = 74.9)} \times 1 &= 74.9 \\ \text{Cl (atomic weight = 35.5)} \times 2 &= 71.0 \\ \text{MW} &= 174.0 \end{aligned}$$

High molecular weights tend to indicate solids. Conversely, low molecular weights tend to indicate gasses. Agents easily broken down by heat often have very high molecular

weights. These very high molecular weights may indicate that decontamination by fire is practical.

The MW gives an indication of the persistency of an agent. Generally, the higher the number, the lower the rate of evaporation and the greater the persistency. Another use for the MW is in calculating the vapor density.

Molecular weight indicates the ability of a chemical to penetrate filters. Molecular weights of 29 or less are very difficult to stop with activated charcoal filters. Examples are ammonia (NH₃, MW 17) and carbon monoxide (CO, MW 28).

Vapor Density

Vapor density is the ratio of the density of any gas or vapor to the density of air, under the same conditions of

temperature and pressure. It is a measure of how heavy the vapor is in relation to the same volume of air. Vapor density

helps in estimating how long an agent will persist in valleys and depressions. The higher the vapor density, the longer the vapor will linger in low-lying areas.

To calculate the vapor density, divide the MW by 29 (the average MW of air). If the result is greater than 1.0, the agent is heavier than air. The agent will tend to collect in low-lying areas, such as foxholes and ditches, and in vehicles. If the result is less than 1.0, the agent almost invariably is nonpersistent. It will quickly dissipate into the atmosphere.

For example, phosgene, COC12, with an MW of 98.92, has a vapor density of 3.4 times that of air. The calculation follows:

Vapor	Molecular Weight	Vapor Density
Phosgene	$\frac{98.92}{29} = 3.4$	$\frac{3.4}{1} = 3.4$
Air		

Phosgene will persist five minutes or longer in the open in the summer. Hydrogen cyanide, MW 27.02, may persist only a minute under the same circumstances.

Agents with low vapor densities rise. Agent AC is the only militarily significant agent that is lighter than air. Chemical agent clouds with high vapor densities seek lower ground in much the same way as water poured on the ground. Agents with high vapor densities tend to evolve into long coherent streamers. For example, agent GB is almost five times as heavy as air. It can produce a streamer 40 meters wide by 22 kilometers long from a single round.

Diffusion is usually a minor factor in the dissemination of chemical agents. This is especially so after air dilutes the chemical agent. Air currents and other influences tend to offset any effects of diffusion or vapor density. Because very high vapor densities cause agents to seek lower ground, the agent can overcome the effects of wind to a limited degree. These densities can actually cause the agent cloud to go upwind if the upwind area is lower than the downwind area and the wind is not very strong. This explains the upwind radius on all chemical hazard predictions.

Liquid and Solid Densities

The density of a liquid chemical agent is the weight in grams of one cubic centimeter of the liquid at a specified temperature. The density of a solid chemical agent is the weight of one cubic centimeter of the solid at a specified temperature. Liquid density in this manual is a measure of an agent's weight in comparison to water (density 1.0). This comparison gives the specific gravity. Liquids form layers, and the one with the greatest density sinks to the bottom. The layer with the lowest density rises to the top.

If an agent is much denser than water, it will tend to sink to the bottom and separate out. An example is mustard-lewisite (liquid density 1.66). If an agent has about the same density as water, such as the nerve agents, it will tend to mix throughout the depth of the water. (However, GB is the only nerve agent that will actually dissolve in water.) The nerve agents are slightly denser than water. Therefore, the concentration of nerve agent will tend to increase with depth. Agents that are less dense than water, such as AC (liquid density 0.69), tend to float on water.

Note: Sometimes density is not the only factor that determines how an agent will distribute in water. An example is HD. If agent HD falls on water, little globs of HD will scatter throughout the depth of the water. The larger ones tend to coagulate and sink, forming a layer at the bottom. The finer droplets or mist created by the explosion form a layer on top, one molecule thick, like an oil slick. Surface tension holds the layer on the surface. If the slick is very thin, it will be iridescent, reflecting rainbowlike colors. If it is a little thicker, it may not be noticeable.

Liquid density is of interest in computing the chemical efficiency of a munition, because we always express toxicities in units of weight. For example, a munition filled with CG (liquid density about 1.4 at 20°C) will contain twice as much chemical agent by weight as a munition of the same volume filled with AC (liquid density about 0.7 at 10°C). It will also have a much higher chemical efficiency. To find the chemical efficiency of a munition, divide the weight of the filling by the total weight of the filled munition.

Melting Point

Melting point is the temperature at which a solid changes to a liquid. White phosphorous (WP) presents an example of the importance of melting point. It has a low melting point. In temperatures above that melting point, you must

store any WP-filled munition on its end. When the WP melts, the center of gravity will remain unchanged and thus prevent instability of the munition in flight.

Freezing Point

Freezing point is the temperature at which a liquid changes to a solid. It is generally equivalent to the melting point. It is important to know the freezing point of a chemical agent, because dissemination characteristics vary markedly with physical state. For example, HD can freeze in a spray tank at low temperatures and cannot be dispensed.

Below their freezing points most agents become unreliable in creating casualties by direct effect. The effects result from secondary transfer. Warming the frozen agent upon entering an enclosure or area where the temperature is high enough will melt or vaporize the agent.

Boiling Point

Boiling point is the temperature at which the vapor pressure of a liquid equals the atmospheric pressure. The boiling point represents the highest usable temperature of a liquid agent. You can use it to estimate the persistency of a chemical (under a given set of conditions). The reason is that the vapor pressure and the evaporating tendency of a chemical agent vary inversely with its boiling point.

The higher the boiling point, the more slowly a liquid evaporates at ordinary temperatures. For example, HD boils at 217°C and evaporates relatively slowly at ordinary temperatures. CG boils at 7.5°C and evaporates rapidly at moderate temperatures. Thus, agents with low boiling points are normally nonpersistent. Those with high boiling points are persistent. The boiling point also gives an indication of the practicality for decontamination with hot air.

Vapor Pressure

Vapor pressure is the pressure exerted by a vapor when a state of equilibrium exists between the vapor and its liquid (or solid) state. It is the pressure in a closed space above a substance when no other gas is present. Vapor pressure varies with temperature, so the temperature must be stated to determine vapor pressure. At any temperature any liquid (or solid) will have some vapor pressure, however small.

Substances with high vapor pressure evaporate rapidly. Those with low vapor pressure evaporate slowly. The impact of vapor pressure on the rate of evaporation makes vapor pressure a very important property in considering

the tactical use and duration of effectiveness of chemical agents. A potential chemical agent is valuable for employment when it has a reasonable vapor pressure. One with exceptionally high vapor pressure is of limited use. It vaporizes and dissipates too quickly. Examples are arsine and carbon monoxide. On the other hand, mechanical or thermal means may effectively aerosolize and disseminate solid and liquid agents of very low vapor pressure. Vapor pressure and volatility are related. Translated into volatility, vapor pressure is most understandable and useful.

Volatility

Volatility is the weight of vapor present in a unit volume of air, under equilibrium conditions, at a specified temperature. It is a measure of how much material (agent) evaporates under given conditions. The volatility depends on vapor pressure. It varies directly with temperature. We express volatility as milligrams of vapor per cubic meter (mg/m^3). Calculate it numerically by an equation derived from the perfect gas law. This equation follows:

$$V = \frac{16020 \times MW \times VP}{K}$$

Where V = Volatility

MW = Molecular weight

VP = Vapor pressure in mm Hg at a specified temperature

K = Kelvin temperature = Celsius temperature plus 273

You need to know more than the vapor pressure or volatility to judge the effectiveness of a chemical agent. You must also consider the degree of toxicity of physiological action of the chemical agent. A highly toxic chemical agent of relatively low volatility, such as GB, may be far more lethal than a less toxic chemical agent of much higher volatility, such as CG.

Flash Point

The flash point is the temperature at which a chemical agent gives off enough vapors to be combustible upon application of a flame under controlled conditions. The

flash point is of interest with chemical agents that have a low enough flash point to cause them to burn when the containing munition bursts.

Decomposition Temperature

The decomposition temperature is that at which a chemical breaks down into two or more substances. This temperature can be used to evaluate candidate chemical agents. A low decomposition temperature (one that is

markedly lower than the boiling point) will usually mean that dissemination of the chemical agent will cause excessive decomposition.

Latent Heat of Vaporization

The latent heat of vaporization is the heat required to change one gram of liquid into vapor without a change in temperature. That is, it is the total heat in calories that disappears at any given temperature when one gram of liquid evaporates under an external pressure of one atmosphere. This property is important in determining the behavior of high-volatility chemical agents when released from shells or bombs. Most chemical agents are in liquid form within the munition. In some munitions the liquid is under considerable pressure. When pressurized munitions burst, the liquid may rapidly become vapor. This process

requires a quantity of heat equivalent to the latent heat of vaporization. The result is cooling of the chemical agent and its surroundings, causing the vapor to settle. This settling action produces the effect known as "pancaking," that is, a spreading downward and outward of the newly released agent. Some chemical agents show the desirable pancaking effect to a greater degree than others. The reason is their high latent heats of vaporization. Examples of agents that exhibit good pancaking effect are CG and CK. Chemical agents that are liquid at ordinary temperatures and pressures do not exhibit this effect.

Minimum Void

The theoretical minimum void is the minimum amount of space that must be left in a container during filling. It allows for the expansion of the filling with increase in temperature. Use the following formula to calculate the theoretical minimum void:

$$\text{Theoretical minimum void}(\%) = \frac{SV_f - SV_i}{SV_f} \times 100$$

Where SV_f = final specific volume of filling

SV_i = initial specific volume of filling

Note: Specific volume is the reciprocal of the density ($SV = 1/\text{density}$).

Use this formula to calculate the percent of increase in the volume of the filling. Base it on the volume at the highest temperature to which the filling will be subjected. Calculations with this formula will determine the minimum void for any given filling, independent of container size or type.

As an example, use a filling temperature of 60°F and calculate the theoretical minimum void for a container of mustard. The calculation follows:

Given SV_i of HD at 60°F = 0.7553 cc/g

SV_f of HD at 140°F = 0.8130 cc/g

$$\text{Theoretical minimum void} = \frac{(0.8130 - 0.7553)}{0.8130} \times 100 = 7.10\%$$

The safety factor (K_c) is the amount of void you should leave in a container in addition to the theoretical minimum void. The specific operation determines the safety factor (expressed in percent of container volume). It is based on the size and dependability of the container. (Allowance is made for decreased quality of container metals under wartime procurement.) The safety factor is determined for each specific case. Calculate the actual void directly for any temperature, using the following formula:

Actual void = theoretical minimum void + safety factor (K_c)

Appendix B

Table of Chemical Agent Properties

Table B-1, on this and the next two pages, provides a quick reference for Chemical Agent Properties. The extreme left column of each page shows the general classes

of agents. The next column shows specific agents. The remaining columns across the page show selected properties.

Table B-1. Properties of chemical agents and compounds. (Part 1 of 3)

Agents	Chemical Agent; Formula; Symbol	Molecular Weight	State at 20°C	Odor	Vapor Density (Air = 1)	Liquid Density (g/cc)	Freezing/ Melting Point (°C)	Boiling Point (°C)
Choking Agents	Phosgene; COCl ₂ ; CG	98.92	Colorless gas	New-mown hay; green corn	3.4	1.37 at 20°C	- 128	7.6
	Diphosgene; ClCOOCCl ₃ ; DP	197.85	Colorless liquid	New-mown hay; green corn	6.8	1.65 at 20°C	- 57	127 to 128
Nerve Agents	Tabun; C ₂ H ₅ OPO(CN)N(CH ₃) ₂ ; GA	162.3	Colorless to brown liquid	Faintly fruity; none when pure	5.63	1.073 at 25°C	- 5	240
	Sarin; CH ₃ PO(F)OCH(CH ₃) ₂ ; GB	140.1	Colorless liquid	Almost none when pure	4.86	1.0887 at 25°C 1.102 at 20°C	- 56	158
	Soman; CH ₃ PO(F)OCH(CH ₃)C(CH ₃) ₃ ; GD	182.178	Colorless liquid	Fruity; camphor when impure	6.33	1.0222 at 25°C	- 42	198
	GF; CH ₃ PO(F)OC ₆ H ₁₁	180.2	Liquid	Sweet; musty; peaches; shellac	6.2	1.1327 at 20°C	- 30	239
	VX (C ₂ H ₅ O)(CH ₃ O)P(O)S(C ₂ H ₄)N[C ₂ H ₂ (CH ₃) ₂] ₂	267.38	Colorless to amber liquid	None	9.2	1.0083 at 20°C	Below - 51	298
	"V-Sub X" V _x	211.2	Colorless liquid	None	7.29	1.062 at 20°C	-	256
Blood Agents	Hydrogen cyanide HCN AC	27.02	Colorless gas or liquid	Bitter almonds	0.990 at 20°C	0.687 at 20°C	- 13.3	25.7
	Cyanogen chloride CNCl CK	61.48	Colorless liquid or gas	Pungent, biting; can go unnoticed	2.1	1.18 at 20°C	- 6.9	12.8
	Arsine AsH ₃ SA	77.93	Colorless gas	Mild garlic	2.69	1.34 at 20°C	- 116	- 62.5

Vapor Pressure (mm. Hg)	Volatility (mg/m ³)	Flash Point	Decomposition Temperature (°C)	Heat of Vaporization (cal/g)	Median Lethal Dosage (mg-min/m ³)	Median Incapacitating Dosage (mg-min/m ³)	Rate of Detoxification	Eye and Skin Toxicity	Rate of Action
1.173 at 20°C	4,300,000 at 7.6°C	None	800	59	3,200	1,600	Not detoxified — cumulative	None	Immediate to 3 hr, depending on concentration
4.2 at 20°C	45,000 at 20°C	None	300 to 350	57.4	3,200	1,600	Not detoxified — cumulative	Slightly lacrimatory	Immediate to 3 hr, depending on concentration
0.037 at 20°C	610 at 25°C	78°C	150	79.56	400 for a resting person	300 for a resting person	Slight; but definite	Very high	Very rapid
2.9 at 25°C 2.10 at 20°C	22,000 at 25°C 16,090 at 20°C	Nonflammable	150	80	100 for a resting person	75 for a resting person	Cumulative	Very high	Very rapid
0.4 at 25°C	3,900 at 25°C	High enough not to interfere with military use	130	72.4	GB, GA range	GB, GA range	Low; essentially cumulative	Very high	Very rapid
0.044 at 20°C	438 at 20°C	94°C	—	90.5	—	—	Low	Very high	Very rapid
0.0007 at 20°C	10.5 at 25°C	159°C	Half life 36 hr at 150	78.2 at 25°C	100	50	Low; essentially cumulative	Very high	Very rapid
0.007 at 25°C 0.004 at 20°C	75 at 25°C 48 at 20°C	—	—	67.2	—	—	Low; essentially cumulative	Very high	Rapid
742 at 25°C 612 at 20°C	1,080,000 at 25°C	0°C; ignited 50% of time when disseminated by artillery shells	> 65.5	233	Varies widely with concentration	Varies with concentration	Rapid — 0.017 mg/kg/min	Moderate	Very rapid
1,000 at 25°C	2,600,000 at 12.8°C 6,132,000 at 25°C	None	> 100	103	11,000	7,000	Rapid 0.02 to 0.1 mg/kg/min	Low; lacrimatory and irritating	Very rapid
11,100 at 20°C	30,900,000 at 0°C	Below shell detonation temp; mixtures w/air may explode spontaneously	280	53.7 at — 62.5°C	5,000	2,500	Low	None	Delayed action — 2 hr to 11 days

Physiological Action	Protection Required	Stability	Decontamination	Means of Detection in Field	Use
Damages and floods lungs	Protective mask	Stable in steel if dry	None needed in field; aeration in closed spaces	M18A2; odor	Delayed-action casualty agent
Damages and floods lungs	Protective mask	Unstable; tends to convert to CG	None needed in field; steam, ammonia, and aeration in closed spaces	Odor	Delayed- or immediate-action casualty agent
Cessation of breath—death may follow	Protective mask and clothing	Stable in steel at normal temperatures	Bleach slurry, dilute alkali, or DS2; steam and ammonia in confined area; M258A1, M280	M18A2, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Cessation of breath—death may follow	Protective mask and clothing	Stable when pure	Steam and ammonia in confined area; hot soapy water; M258A1, M280	M18A2, M256, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Cessation of breath—death may follow	Protective mask and clothing	Less stable than GA or GB	Bleach slurry, dilute alkali; in confined area, hot soapy water; M258A1, M280	M18A2, M256, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Cessation of breath—death may follow	Protective mask and clothing	Relatively stable in steel	Bleach slurry, dilute alkali, or DS2; steam and ammonia in confined area; M258A1, M280	M18A2, M256, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Produces casualties when inhaled or absorbed	Protective mask and clothing	Relatively stable at room temperature	STB slurry or DS2 solution; hot soapy water; M258A1, M280	M18A2, M256, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Produces casualties when inhaled or absorbed	Protective mask and clothing	Relatively stable	STB slurry or DS2 solution; hot soapy water; M258A1	M18A2, M256, M256A1, M8 and M8A1 alarms, M8 and M9 paper	Quick-action casualty agent
Interferes w/body tissues' oxygen use; accelerates rate of breathing	Protective mask; protective clothing in unusual situations	Stable if pure; can burn on shell explosion	None needed in field	M18A2, M256, M256A1, M8 alarm	Quick-action casualty agent
Chokes, irritates, causes slow breathing rate	Protective mask	Tends to polymerize; may explode	None needed in field	M18A2, M256, M256A1, M8 alarm	Quick-action casualty agent
Damages blood, liver, and kidneys	Protective mask	Not stable in uncoated metal containers	None needed	None	Delayed-action casualty agent

Table B-1 continued. (Part 2 of 3)

Agents	Chemical Agent; Formula; Symbol	Molecular Weight	State at 20°C	Odor	Vapor Density (Air = 1)	Liquid Density (g/cc)	Freezing/ Melting Point (°C)	Boiling Point (°C)
Blister Agents	Distilled mustard; (CICH ₂ CH ₂) ₂ S; HD	159.08	Colorless to pale yellow liquid	Garlic or horseradish	5.4	1.268 at 25°C 1.27 at 20°C	14.45	217
	Nitrogen mustard; (CICH ₂ CH ₂) ₂ NC ₂ H ₅ ; HN-1	170.08	Dark liquid	Fishy or musty	5.9	1.09 at 25°C	- 34	194
	Nitrogen mustard; (CICH ₂ CH ₂) ₂ NCH ₃ ; HN-2	156.07	Dark liquid	Soapy in low concentrations; fruity in high concentrations	5.4	1.15 at 20°C	- 65 to - 60	75 at 15 mmHg
	Nitrogen mustard; N(CH ₂ CH ₂ Cl) ₃ ; HN-3	204.54	Dark liquid	None if pure	7.1	1.24 at 25°C	- 3.7	256
	Phosgene oximedichlorofoxime; CCl ₂ NOH; CX	113.94	Colorless solid or liquid	Sharp, penetrat- ing	3.9	-	35 to 40	53 to 54 at 28 mmHg
	Lewisite; CICHCHAsCl ₂ ; L	207.35	Colorless to brownish	Variable; may resemble geraniums	7.1	1.89 at 20°C	- 18	190
	Mustard-lewisite mixture; HL	186.4	Dark, oily liquid	Garlic	6.5	1.66 at 20°C	- 25.4 (pure) - 42 (plant purity)	< 190
	Phenyldichloroarsine; C ₆ H ₅ AsCl ₂ ; PD	222.91	Colorless liquid	None	7.7	1.65 at 20°C	- 20	252 to 255
	Ethyldichloroarsine; C ₂ H ₅ AsCl ₂ ; ED	174.88	Colorless liquid	Fruity, but biting; irritating	6.0	1.66 at 20°C	- 65	156
Methyldichloroarsine; CH ₃ AsCl ₂ ; MD	160.86	Colorless liquid	None	5.5	1.836 at 20°C	- 55	133	
Vomiting Agents	Diphenylchloroarsine; (C ₆ H ₅) ₂ AsCl; DA	264.5	White to brown solid	None	Forms little vapor	1.387 at 50°C	41 to 44.5	333
	Adamsite; C ₆ H ₄ (AsCl)-(NH)C ₆ H ₄ ; DM	277.57	Yellow to green solid	None	Forms little vapor	1.65 (solid) at 20°C	195	410
	Diphenylcyanoarsine; (C ₆ H ₅) ₂ AsCN; DC	255.0	White to pink solid	Bitter almond- garlic mixture	Forms little vapor	1.3338 at 35°C	31.5 to 35	350

Vapor Pressure (mm Hg)	Volatility (mg/m ³)	Flash Point	Decomposition Temperature (°C)	Heat of Vaporization (cal/g)	Median Lethal Dosage (mg-min/m ³)	Median Incapacitating Dosage (mg-min/m ³)	Rate of Detoxification	Eye and Skin Toxicity	Rate of Action
0.072 at 20°C	610 at 20°C	105°C; ignited by large explosive charges	149 to 177	94	1,500 by inhalation; 10,000 by skin exposure	200 by eye; 2,000 by skin; 150 inhaled	Very low; cumulative	Eyes very susceptible; skin less so	Delayed – hours to days
0.24 at 25°C	1,520 at 20°C	High enough not to interfere with military use	Decomposes before boiling point is reached	77	1,500 by inhalation; 20,000 by skin exposure	200 by eye; 9,000 by skin	Not detoxified – cumulative	Eyes susceptible to low concentration; less toxic to skin	Delayed – 12 hr or longer
0.29 at 20°C	3,580 at 25°C	High enough not to interfere with military use	Below boiling point; polymerizes w/heat generation	78.8	3,000 by inhalation	< IIN-1; > IIN-3; 100 by eye	Not detoxified – cumulative	Toxic to eyes; blisters skin	Skin – delayed 12 hr or more; eyes – faster than HD
0.0109 at 25°C	121 at 25°C	High enough not to interfere with military use	Below boiling point	74	1,500 by inhalation; 10,000 by skin exposure (est)	200 by eye; 2,500 by skin (est)	Not detoxified – cumulative	Eyes very susceptible; skin less so	Serious effects same as for HD; minor effects sooner
11.2 at 25°C (solid) 13 at 40°C (liquid)	1,800 at 20°C	–	Decomposes slowly at normal temperature	101 at 40°C	3,200 (est)	Very low	–	Powerful irritant to eyes and nose; liquid corrosive to skin	Immediate effects on contact
0.394 at 20°C	4,480 at 20°C	None	> 100	58 at 0° to 190°C	1,200–1,500 by inhalation; 100,000 by skin exp	< 300 by eye; > 1,500 by skin	Not detoxified	1,500 mg min/m ³ severely damages eyes; skin less so	Rapid
0.248 at 20°C	2,730 at 20°C	High enough not to interfere with military use	> 100	Value between HD and L	1,500 by inhalation; over 10,000 by skin exposure	200 by eye; 1,500–2,000 by skin	Not detoxified	Very high	Prompt stinging; blistering delayed about 13 hr
0.033 at 25°C	390 at 25°C	High enough not to interfere with military use	Stable to boiling point	69	2,600 by inhalation	16 as vomiting agent; 1,800 as blister	Probably rapid	633 mg min/m ³ produces eye casualty; less toxic to skin	Immediate eye effect; skin effects ½ to 1 hr
2.09 at 20°C	20,000 at 20°C	High enough not to interfere with military use	Stable to boiling point	52.5	3,000–5,000 by inhalation; 100,000 by skin exposure	5 to 10 by inhalation	Rapid	Vapor harmful on long exposure; liquid blisters < L	Immediate irritation; delayed blistering
7.76 at 20°C	74,900 at 20°C	High enough not to interfere with military use	Stable to boiling point	49	3,000–5,000 (est)	25 by inhalation	Rapid	Eye damage possible; blisters < HD	Immediate irritation; delayed blistering
0.0036 at 45°C	48 at 45°C	350°C	300	56.6	15,000 (est)	12 (> 10 minutes)	Moderate	Irritating; not toxic	Very rapid
Negligible	Negligible	None	> boiling point	80	Variable – average 11,000	22 (1 min exposure) 8 (60 min exp)	Rapid in small amounts	Irritating; relatively nontoxic	Very rapid
0.0002 at 20°C	2.8 at 20°C	Low	300 (25% decomposed)	71.1	10,000 (est)	30 (30 sec exposure) 20 (5 min exp)	Rapid	Irritating; not toxic	More rapid than DM or DA

Physiological Action	Protection Required	Stability	Decontamination	Means of Detection in Field	Use
Blisters; destroys tissue; injures blood cells	Protective mask and clothing	Stable in steel or aluminum	Bleach, fire, DS2, M258A1, M280	M18A2, M256, M256A1, M8 and M9 paper	Delayed-action casualty agent
Blisters; affects respiratory tract; destroys tissue; injures blood cells	Protective mask and clothing	Adequate	Bleach, fire, DS2, M258A1, M280	M18A2, M256, M256A1, M8 and M9 paper	Delayed-action casualty agent
Similar to HD Bronchopneumonia possible after 24 hr	Protective mask and clothing	Unstable	Bleach, fire, DS2, M258A1, M280	M18A2, M256, M256A1, M8 and M9 paper	Delayed-action casualty agent
Similar to HN-2	Protective mask and clothing	Stable	Bleach, fire, DS2, M258A1, M280	M18A2, M256, M256A1, M8 and M9 paper	Delayed-action casualty agent
Violently irritates mucous membrane of eyes & nose; forms wheals rapidly	Protective mask and clothing	Decomposes slowly	None entirely effective; wash w/large amounts of water or DS2	M18A2, M256, M256A1, M8 alarm	Rapid-action casualty agent
Similar to IID, plus may cause systemic poisoning	Protective mask and clothing	Stable in steel and glass	Bleach, fire, DS2, caustic soda, M258A1, M280	M18A2, M256, M256A1	Moderately delayed-action casualty agent
Similar to HD, plus may cause systemic poisoning	Protective mask and clothing	Stable in lacquered steel	Bleach, fire, DS2, caustic soda, M258A1, M280	M18A2, M256, M256A1	Delayed-action casualty agent
Irritates; causes nausea, vomiting, and blisters	Protective mask and clothing	Very stable	Bleach, DS2, caustic soda, M258A1, M280	M18A2	Delayed-action casualty agent
Damages respiratory tract; affects eyes; blisters; can cause systemic poisoning	Protective mask and clothing	Stable in steel	None needed in field; bleach, caustic soda or DS2 in closed spaces; M258A1, M280	M18A2	Delayed-action casualty agent
Irritates respiratory tract; injures lungs & eyes; causes systemic poisoning	Protective mask and clothing	Stable in steel	Bleach, DS2, caustic soda, M258A1, M280	M18A2	Delayed-action casualty agent
Like cold symptoms, plus headache, vomiting, nausea	Protective mask	Stable if pure	None needed in field; caustic soda or chlorine in closed spaces	None	Former training and riot control agent
Like cold symptoms, plus headache, vomiting, nausea	Protective mask	Stable in glass or steel	None needed in field; bleach or DS2 in closed spaces	None	Former training and riot control agent
Like cold symptoms, plus headache, vomiting, nausea	Protective mask	Stable at normal temperatures	None needed in field; alkali solution or DS2 in closed spaces	None	Former training and riot control agent

Table B-1. continued. (Part 3 of 3)

Agents	Chemical Agent; Formula; Symbol	Molecular Weight	State at 20°C	Odor	Vapor Density (Air = 1)	Liquid Density (g/cc)	Freezing/ Melting Point (°C)	Boiling Point (°C)	Vapor Pressure (mm Hg)	
Tear Agents	Chloroacetophenone; C ₆ H ₅ COCH ₂ Cl; CN	154.59	Solid	Apple blossoms	5.3	1.318 (solid) at 20°C	54	248	0.0041 at 20°C	
	Chloroacetophenone in chloroform; CNC	128.17 on basis of components	Liquid	Chloroform	4.4	1.40 at 20°C	0.23	Variable 60 to 247	127 at 20°C	
	Chloroacetophenone and chloropicrin in chloroform; CNS	141.78 on basis of components	Liquid	Flypaper	Approx 5	1.47 at 20°C	2	Variable 60 to 247	78 at 20°C	
	Chloroacetophenone in benzene and carbon tetrachloride; CNB	119.7 on basis of components	Liquid	Bensene	Approx 4	1.14 at 20°C	- 7 to - 30	Variable 75 to 247	Variable - mostly solvent vapor	
	Bromobenzylcyanide; BrC ₆ H ₄ CH ₂ CN; CA	196	Yellow solid or liquid	Soured fruit	6.7	1.47 at 25°C	25.5	Decom- poses at 242	0.011 at 20°C	
	O-chlorobenzylmalononitrile; ClC ₆ H ₄ CHC(CN) ₂ ; CS	188.5	Colorless solid	Pepper	-	1.04 at 20°C (solid)	93 to 95	310 to 315 (w/ decomp)	0.00034 at 20°C	
	CR; (C ₆ H ₄) ₂ (O)(N)CH;	195.25	Yellow pow- der in solution	Burning sensation	6.7	-	72	335	0.00059 at 20°C	
	Chloropicrin; Cl ₃ CNO ₂ ; PS	164.38	Liquid	Stinging, pungent	5.6	1.66	- 69	112	18.3 at 20°C	
Incapac- itating Agents	BZ	337.4	White crystal	None	11.6	Bulk 0.51 solid Crystal 1.33	167.5	320	0.03 at 70°C	
Binary Compounds	GB2	Methylphosphonicdifluoride; CH ₃ POF ₂ ; DF	100.1	Liquid	Pungent	3.45	1.359 at 25°C	- 37.1	100	36 at 25°C
		Isopropylamine and 2-propanol; OPA	59.82 on basis of components	Liquid	Alcohol and ammonia	2.1	0.744 at 25°C	< - 88	60	197 at 25°C
	VX2	QL C ₁₁ H ₂₆ NO ₂ P	235	Liquid	Fishy	8.1	0.908 at 25°C 0.9125 at 20°C	None	232	0.01 at 25°C 0.01 at 20°C
		Sulfur; S ₈ ; NE	256.48	Yellow solid	Odorless to vile	8.8	2.07 at 20°C	112.8	444.6	-

Volatility (mg/m ³)	Flash Point	Decomposition Temperature (°C)	Heat of Vaporization (cal/g)	Median Lethal Dosage (mg-min/m ³)	Median Incapacitating Dosage (mg-min/m ³)	Rate of Detoxification	Eye and Skin Toxicity	Rate of Action
34.3 at 20°C	High enough not to interfere w/military use	Stable to boiling point	98	7,000 to 14,000	80	Rapid	Temporary severe eye irritation; mild skin irritation	Instantaneous
Indeterminate	None	Stable to boiling point	NA	11,000 (est)	80	Rapid	Temporary severe eye irritation; mild skin irritation	Instantaneous
610,000 at 20°C (includes solvent)	None	Stable to boiling point	NA	11,400	60	Slow because of effect of PS	Irritating; not toxic	Instantaneous
Indeterminate	< 4.44	> 247	NA	11,000 (est)	80	Rapid unless large amounts of solvent inhaled	Temporary severe eye irritation; mild skin irritation	Instantaneous
115 at 20°C	None	60 to 242	79.5 at 20°C	8,000 to 11,000 (est)	30	Rapid in low dosage	Irritating; not toxic	Instantaneous
0.71 at 25°C	197°C	–	53.6	61,000	10 to 20	Rapid	Highly irritating; not toxic	Instantaneous
0.63 at 25°C	188°C	–	–	–	0.15	Moderate	Highly irritating; not toxic	Instantaneous
165,000 at 20°C	Not flammable	> 400	–	2,000	9	Slow	Highly irritating	Instantaneous
0.5 at 70°C	246°C	Begins at 170°C	62.9	High 200,000 (est)	112	–	–	Delayed action – 1 to 4 hr, depending on exposure
147,926 at 19.5°C	Not flammable	–	18.7	NA	NA	–	Highly toxic	Rapid
–	– 9.4°C	–	–	NA	NA	–	Toxic, irritant	Rapid
126 at 25°C 130 at 20°C	95°C	–	–	NA	NA	–	Low toxicity; skin irritant	–
–	–	> 120	–	NA	NA	–	Low toxicity	–

Physiological Action	Protection Required	Stability	Decontamination	Means of Detection in Field	Use
Causes tearing; irritates eyes and respiratory tract	Protective mask	Stable	Aeration in open; sodium carbonate solution or alcoholic caustic soda in closed spaces	None	Training and riot control agent
Causes tearing; irritates respiratory tract	Protective mask	Adequate	Aeration in open; sodium carbonate solution or alcoholic caustic soda in closed spaces	None	Training and riot control agent
Acts as vomiting and choking agent as well as tear agent	Protective mask	Adequate	None needed in field; hot solution of soda ash and sodium sulfite in closed spaces	None	Former training and riot control agent
Powerfully lacrimatory	Protective mask	Adequate	Aeration in open; sodium carbonate solution or alcoholic caustic soda in closed spaces	None	Former training and riot control agent
Irritates eyes and respiratory passages	Protective mask	Fairly stable in glass, lead, or enamel	20% alcoholic caustic soda	None	Former training and riot control agent
Highly irritating; not toxic	Protective mask & clothing, secured at neck, wrists, & ankles; gloves to handle	Stable	Aeration, soap & water; Do not use bleach or STB.	None	Training and riot control agent
Irritates skin, eyes, nose, and throat	Protective mask and clothing	Stable	Aeration, soap & water; Do not use bleach, detergents, or peroxide.	None	Riot control agent
Acts as tear, vomiting, and choking agent	Protective mask and clothing	Adequate, unstable in light	Large amounts of water or rinse with a 5% solution of bisulfite	None	Former riot control agent
Fast heart beat, dizziness, vomiting, dry mouth, blurred vision, stupor, increasing random activity	Protective mask	Adequate	Soap & water; shake or brush; hypochlorite or caustic alcoholic solution; M258A1	None	Former delayed-action incapacitating agent
Highly irritating to eyes, nose, throat, and lungs	Protective mask and clothing	Stable, but reactive	Water or STB	NA	Forms GB when combined
Vapor irritating to eyes, nose, & throat; liquid highly irritating to skin and eyes	Protective mask and clothing	Flammable	Water	NA	
Skin irritant; headaches; nausea	Protective mask, gloves, and clothing	Unstable in air; flammable	10% sodium hydroxide, alkali	NA	Forms VX when combined
May irritate skin, eyes, nose, and throat	—	Stable; keep away from bleach and water	Water	NA	

Appendix C

Table of Equivalents

Distance

1 inch	= 2.54 centimeters	= 25.4 millimeters
1 foot	= 0.305 meter	= 30.48 centimeters
1 yard	= 0.9144 meter	
1 mile	= 1.61 kilometers	= 5,280 feet
1 kilometer	= 1,000 meters	= 0.6214 mile
1 meter	= 100 centimeters	= 1,000 millimeters
	= 3.28 feet	
1 centimeter	= 0.3937 inch	= 10 millimeters
1 millimeter	= 0.039 inch	= 0.1 centimeter
1 micron	= 10 ⁻⁴ centimeter	= 10 ⁻⁶ meter
	= 1 micrometer (μm)	

Volume

1 kiloliter	= 1,000 liters	= 1 cubic meter
1 liter	= 1,000 milliliters	= 1,000 cubic centimeters (cc)
1 milliliter	= 1 cubic centimeter (exactly 1.000027 cc)	
1 fluid ounce	= 29.57 milliliters	
1 US gallon	= 3.785 liters	
1 Imperial gallon	= 4.546 liters	

Weight

1 kilogram	= 1,000 grams	= 2.2 pounds
1 gram	= 1,000 milligrams	= 0.035 ounce
1 milligram	= 1,000 micrograms	= 1/1000 gram
1 microgram	= 10 ⁻³ grams	= 1/1,000 milligram
1 nanogram	= 10 ⁻⁹ grams	= 1/1,000 microgram
1 pound	= 0.45 kilogram	= 16 ounces
1 ounce	= 28.34 grams	

Appendix D

Temperature Conversions

To convert Celsius to Fahrenheit, use this formula:

$$^{\circ}\text{F} = 1.8 (^{\circ}\text{C}) + 32^{\circ}$$

To convert Fahrenheit to Celsius, use this formula

$$^{\circ}\text{C} = (^{\circ}\text{F} - 32^{\circ}) / 1.8$$

Use the following table as a quick reference.

$^{\circ}\text{C}$	$^{\circ}\text{F}$
-60	-76
-55	-67
-50	-58
-45	-49
-40	-40
-35	-31
-30	-22
-25	-13
-20	-4
-15	5
-10	14
-5	23
0	32
2	36
4	39
6	43
8	46
10	50
12	53
14	57
16	61
18	64
20	68
22	72
24	75

$^{\circ}\text{C}$	$^{\circ}\text{F}$
26	79
28	82
30	86
32	90
34	93
36	97
38	100
40	104
45	113
50	122
55	131
60	140
65	149
70	158
75	167
80	176
85	185
90	194
95	203
100	212
105	221
110	230
115	239
120	248
125	257

$^{\circ}\text{C}$	$^{\circ}\text{F}$
130	266
135	275
140	284
145	293
150	302
155	311
160	320
165	329
170	338
175	347
180	356
185	365
190	374
195	383
200	392
205	401
210	410
215	419
220	428
225	437
230	446
235	455
240	464
245	473
250	482

Appendix E

Technical Aspects of Toxins

This appendix presents further information about the technical aspects of toxins. These aspects include the chemical nature of toxins and their mechanisms of action.

Chemical Nature of Toxins

The chemical nature of toxins affects their stability, volatility, and potential for chemical synthesis. A distinction exists between those toxins that are proteinaceous (proteinlike) and those that are nonproteinaceous, low-molecular-weight compounds. Table E-1 identifies some toxins by their chemical nature.

Table E-1. Chemical nature of selected toxins.

High-Molecular-Weight Proteins	Low-Molecular-Weight Nonproteins
Abrin	Lipid-Soluble
Botulinum	Aconitine
Ricin	Batrachotoxin
Scorpin toxin	Grayanotoxin
Sea anemone toxin	Veratridine
Tetanus toxin	Some mycotoxins
Low-Molecular-Weight Proteins	Water-Soluble
Bungarotoxin	Tetrodotoxin
Conotoxin	Saxitoxin
Microcystin	Palytoxin
SEB	Some mycotoxins

Proteinaceous Toxins

The usual sources of proteinaceous toxins are unicellular organisms, such as bacteria. Also, snake and spider venoms contain a mixture of proteinaceous as well as low-molecular-weight toxins. Ricin from castor beans and abrin from the tropical legume *Abrus precatorius* are also proteins. The proteinaceous toxins are solids when purified but are soluble in water-based solutions. These toxins may be fluids in nature (for example, venoms). Large proteinaceous toxins are heat-sensitive. Smaller proteins tend to be stable. In the case of protein toxins, vaccination with an inactivated form of the toxin (toxoids) can induce immunity in some instances.

Proteinaceous toxins may be cytotoxins or neurotoxins. Proteinaceous cytotoxins produce several naturally occurring diseases. These diseases include anthrax, diphtheria, dysentery, pertussis, and plague. Chapter 4 describes the cytotoxins microcystin from freshwater algae and ricin from castor beans. Proteinaceous neurotoxins include botulinum and tetanus toxins. These neurotoxins also include toxins from snake and scorpion venoms (Chapter 4).

Low-Molecular-Weight Non-Proteinaceous Toxins

Major sources of nonproteinaceous toxins are marine organisms, freshwater blue-green algae, and fungi. Most are neurotoxins. However, tricothecene mycotoxins inhibit protein synthesis. Obtaining sufficient quantities from natural sources to pose a serious threat would severely limit military exploitation of these toxins.

Peptides

A number of small peptides (5 to 20 amino acids) appear to act as neurotransmitter or "hormones" in the brain. They integrate or modify responses, information, mood, awareness, and consciousness.

The biological half-life of most small peptides and peptide hormones (bioregulators) in the body is measured in minutes. Peptides have low volatility and difficulty in passing through natural barriers, such as the skin. Chemical analogues could reduce some of these limitations. The greatest threat could be from production inside a human, following infection with a new biological agent. Peptides are hydrolyzed by acid solutions.

Synthesis of Toxins and Peptides

The possibility exists for toxin or peptide production by genetic engineering of microorganisms or by chemical synthesis. Nonprotein, low-molecular-weight toxins or peptides are more subject to manipulation and/or production by genetic engineering than are the more complex proteins. However, the potential exists for synthesis of some smaller components of proteins. Several protein toxins have had a gene cloned and/or have a known sequence. These toxins

include the heat-stable *Escherichia coli* and *Staphylococcus* enterotoxins, anthrax lethal factor, diphtheria toxin, ricin, and tetanus toxin.

Mechanisms of Action

Classes of toxins according to their mechanisms of action are neurotoxins and cytotoxins.

Neurotoxins

Neurotoxins can fall into subclasses according to the mechanism by which they create their toxic effects as presynaptic neurotoxins, postsynaptic neurotoxins, ion-channel-binding toxins, and ionophores.

Table E-2 shows the effects of action of selected neurotoxins.

Table E-2. Effects of action of selected neurotoxins.

Toxin	Action
Botulinum	Block neurotransmitter release, depolarization, or ion-transport
Tetrodotoxin	
Saxitoxin	
Beta-bungarotoxin	
Conotoxin	
Tetanus	
Alpha-latrotoxin	Cause depolarization or inhibit inactivation
Batrachotoxin	
Brevetoxin	
Grayanotoxin	
Palytoxin	
Anatoxin A (VFDF)	
Scorpion toxin	
Sea anemone toxin	
Batrachotoxin	Produce seizures and tremors
Anatoxin A (VFDF)	
Batrachotoxin	Produce shock and death from cardiotoxic effects
Anatoxin A	
Tetrodotoxin	
SEB	
Botulinum	
Maitotoxin	

Symptoms and treatments for neurotoxins vary. The reason is the variety of mechanisms by which they produce their effects. Medical assistance for casualties is vital.

Chemical synthesis is possible for low-molecular-weight toxins, such as batrachotoxin, saxitoxin, and tetrodotoxin, or peptides.

Presynaptic Neurotoxins

Presynaptic neurotoxins include microbial paralytic toxins, such as botulinum and tetanus toxins, and snake phospholipases. All can be lethal by aerosol. Botulinum and tetanus neurotoxins block the release of acetylcholine. A variety of snake venoms also contain toxins that act presynaptically. They block release of acetylcholine from nerve terminals, apparently through the activity of an enzyme, phospholipase (PLA). Presynaptic neurotoxins may lead to a rigid or a flaccid (limp) paralysis, depending on the mechanism of action. Botulinum produces flaccid paralysis. Tetanus produces rigid paralysis.

Postsynaptic Neurotoxins

Postsynaptic neurotoxins competitively block the acetylcholine receptor. They include snake alpha-toxins and conotoxin. The snake alpha-toxins include cobra neurotoxin, alpha-bungarotoxin from the banded krait, and erabutoxin from sea snakes. Marine fish-hunting cone snails (*Conus*) are the natural source of conotoxin.

Ion-Channel-Binding Toxins

Channel-binding toxins interfere with the movement of ions, such as sodium or potassium, through membranes. The activity of nerve and muscle cells requires a balance of these ions on each side of a membrane. These toxins come from scorpion, snake, and bee venoms. These may not appear highly toxic because of the low doses normally encountered in nature. However, the toxicities based on LD₅₀ values make them potential biological warfare agents.

Sodium-ion channel-binding toxins include scorpion toxins and toxins from rattlesnakes and copperheads, such as myotoxin A, gyrotoxin, and crotamine. These snake channel-binding toxins are small, stable proteins. Their dissemination as aerosols is possible. (Some low-molecular-weight nonproteinaceous compounds, such as batrachotoxin, also produce neurotoxic effects by binding sodium-ion channels.)

Potassium-ion channel-binding toxins include apamin. Apamin is in bee venom. It is a small, neurotoxic protein (18 amino acids with 2 disulfide bonds). This toxin blocks potassium flow by binding to calcium-dependent potassium-ion channels.

Ionophores

Ionophores are molecules that promote the transfer of ions across membranes. Alpha-latrotoxin and diam-

phototoxin are ionophores. Alpha-latrotoxin is the neurotoxic component of black widow spider venom. Diamphotoxin comes from a beetle pupa found in Africa. The Kung tribesmen use it for hunting.

Cytotoxins

Cytotoxins literally affect all cell types in the body. They cause cellular destruction or interfere with metabolic processes such as cell respiration or protein synthesis, common to all cells. Table E-3 identifies some cytotoxins

by their effects. Those toxins that affect only selected tissues or systems are not literally cytotoxins. However, for purposes of this manual, these toxins fall into this group. Examples of toxins that have primary effects in a single system include those that—

- Affect the digestive tract (enterotoxins).
- Cause bleeding (hemorrhagic toxins).
- Cause liver or kidney damage (hepatotoxins or nephrotoxins).
- Inflammate skin and mucous membranes.

Table E-3. Effects of action of selected cytotoxins.

Effect	Cytotoxin
Damage skin (dermatotoxins)	T-2 and other tricothecenes
Cause hemorrhage (hemorrhagic toxins)	T-2 and aflatoxins Palytoxin Abrin
Cause liver damage and/or jaundice (hepatotoxins)	Aflatoxin and other mycotoxins
Damage membranes and/or tissues (necrotic toxins)	Snake venoms Acidic phospholipase (PLA) from snakes Crotoxin Microcystin
Damage muscles (myotoxins)	Crotamine; myotoxin (from snake venom)
Affect intestinal membrane (enterotoxins)	Staphylococcal enterotoxins A and B <u>Escherichia coli</u> enterotoxins
Affect heart membranes (cardiotoxins)	Cardiotoxin Sea wasp toxin
Destroy red blood cells	Staphylococcal hemolytic toxins Ricin Tricothecene
Interfere with regulation/metabolic processes	<u>Escherichia coli</u> enterotoxins Pertussis toxin Plague toxin Anthrax toxin Cholera toxin
Block protein synthesis	Abrin Diphtheria toxin Ricin <u>Shigella</u> desentry toxin Tricothecenes

Appendix F

Tables of Toxin Properties

Tables on the following pages provide a quick reference to toxin characteristics. Table F-1 includes sources and toxicological information. Table F-2 summarizes the physical and chemical properties. The extreme left column of

each table shows the general classes of agents. The next column gives specific agents. The remaining columns across the page present selected properties.

Table F-1. Summary of toxin physical and chemical characteristics.

Toxin	Chemical Nature	Molecular Weight	State at 20°C (Pure)	Solubility	Stability/Persistency
Cytotoxins					
Microcystin (FDF)	Polypeptide	994		Soluble in water, alcohols, ketones, polar organics	Unstable (depends on purification); sensitive to alkali (STB)
Ricin	Glycoprotein	65,000	Powder (freeze-dried)	Water-soluble	Very stable; stable in water or dilute acids; persistent
Staphylococcus enterotoxin (SEB)	Small protein	28,500	White, fluffy powder	Water-soluble	Stable in heat, acids, alkali; relatively nonpersistent
Tricothecene mycotoxins	Nonproteins	400–700	Colorless crystal; clear-yellow oil	T-2: not very soluble in water; soluble in organic solvents	Very stable; resist heat, acids; persistent
Mixed Toxin Types					
Snake Venoms/Toxins	Small proteins	6,000–80,000	Powder (freeze-dried toxin)	Water-soluble	Relatively nonpersistent
Neurotoxins	Proteins	80,000			
Cardiotoxin	Small protein	6,800–8,000			
Necrotic toxins	Small protein	4,600			
Neurotoxins					
Anatoxin A (VFDF)	Alkaloid	165		Water-soluble	Sensitive to heat, light, and alkali
Batrachotoxin	Small non-protein	538		Insoluble in water; soluble in organic compounds (fats, oils, alcohols, and fuels)	Somewhat unstable; sensitive to alkali; relatively nonpersistent
Botulinum, Type A	Large protein	150,000	White powder, colorless crystals	Water-soluble	Stable but nonpersistent; stable 7 days in water, 12 hours in air; destroyed by bases or boiling 15 minutes
Palytoxin	Large non-protein	2,677		Water-soluble	Persistent; stable in heat, acids, alkali
Saxitoxin	Small non-protein	370 (294--489)	White powder (absorbs water)	Water-soluble	Relatively persistent; sensitive to alkali; stable to heat and acids
Scorpion Venom/Toxins	Small basic proteins	7,000	Liquid venom; powdered toxin	Water-soluble	Stable; relatively persistent
Tetrodotoxin	Small non-protein	320	Colorless crystals; white powder	Soluble in acidic solution	Heat stable; sensitive to strong acids and alkalies
Chemical Nerve Agents					
GB	Organophosphate	140	Colorless liquid	Soluble in organics	Nonpersistent

Table F-2. Summary of toxin characteristics.

Toxin	Natural Source	Rate of Action*	LD ₅₀ ** (μ g/kg)	Mechanism***	Effect
Cytotoxins					
Microcystin (FDF)	<u>Microcystis</u> blue-green algae	Rapid	25–200	Deforms/damages liver cell membranes	Shivering, stupor, prostration, shock, liver enlargement
Ricin	<u>Ricinus communis</u> castor bean	Delayed or rapid	0.1–3.7; 1,000 (oral, humans)	Inhibits protein synthesis	Nausea, vomiting, cramps, bloody nose, diarrhea, difficulty breathing, twitching
Staphylococcus enterotoxin (SEB)	<u>Staphylococcus aureus</u> bacteria	Rapid	0.023 (aerosol, humans)	Binds to linings of gut and lungs	Vomiting, cramps, nausea, diarrhea, severe weakness
Tricothecene mycotoxins	<u>Fusarium</u> molds on infected grains	Rapid	500 (humans)	Block protein synthesis	Itching, tingling, vomiting, hemorrhaging, bloody diarrhea
Mixed Toxin Types					
Snake Venoms/Toxins	Snakes	Rapid	1–5,000	Varied	Paralysis or hemorrhaging
Neurotoxins	Elapids and crotalids	Rapid	1–300; 0.2–100 (humans)	Block ACh release or ACh receptor	Weakness, circulatory and respiratory failure
Cardiotoxins	Cobra, mamba, coral snakes	Rapid	1,500; 300 (humans)	Affect membranes	Heart irregularities, lower blood pressure
Necrotic toxins	Pit vipers	Delayed	5,000; 1,000 (humans)	Destroy tissues	Hemorrhage, muscle destruction
Neurotoxins					
Anatoxin A (VFDF)	<u>Anabaena flos-aquae</u> blue-green algae	Very rapid	170–250; 5,000; (oral)	Binds ACh receptor (mimics ACh); may inhibit AChE	Nerve agent effects: incoordination, tremors, paralysis, respiratory arrest
Batrachotoxin	<u>Phyllobates</u> South American frog	Rapid	0.1–2	Increases sodium + channel permeability	Loss of coordination, numbness, headache, irregular heart rate, respiratory paralysis
Botulinum	<u>Clostridium botulinum</u> bacteria	Delayed	0.0003; 0.01 (humans)	Blocks ACh release	Dilated pupils, double vision, dry mouth, weakness, paralysis
Palytoxin	<u>Palythoa</u> soft corals	Very rapid	0.45; 0.08 (humans)	Increases sodium + channel permeability	Muscle contractions, heart irregularities, rigid paralysis
Saxitoxin	<u>Gonyaulax</u> dinoflagellates in shellfish	Rapid	8; 7 (oral, humans)	Blocks sodium + channels	Tingling, numbness, weakness, flaccid (limp) paralysis
Scorpion Venom/Toxins	<u>Centruroides</u>	Delayed	300–1,200	Modify sodium + channel	Irregular, increased heart rate and breathing; vomiting; excess tears, sweat, and saliva
Tetanus	<u>Clostridium tetani</u> bacteria	Delayed	0.001	Blocks ACh release	Muscle spasms, frequently of the jaw muscles
Tetrodotoxin	<u>Takifugu</u> puffer fish	Rapid	8–9; 30 (oral, humans)	Blocks sodium + channels	Vomiting; tingling; numbness; lack of muscle control; loss of voice; paralysis, especially arms and legs
Chemical Nerve Agents					
GB		Very rapid	1,000	Blocks AChE	Rigid paralysis
*Rate of Action: Very rapid = 5 minutes; rapid = 5 minutes to 1 hour; delayed = 1 to 12 hours					
**LD ₅₀ is based on injection in mice. When available, doses for humans are included. For details see Chapter 4, Section III.					
***ACh = acetylcholine; AChE = acetylcholinesterase.					

G l o s s a r y

A

AC — hydrogen cyanide.

AChE — acetylcholinesterase.

acetylcholine — a chemical neurotransmitter produced by nerve cells predominantly outside the central nervous system. It is a chemical “messenger,” stimulating the heart, skeletal muscles, and numerous secretory glands.

acetylcholinesterase — an enzyme that normally hydrolyzes acetylcholine, thereby stopping its activity. Acetylcholinesterase is inhibited by organophosphates, carbamates, and glycolates.

acid — a chemical compound having a pH less than 7. Acids usually have a sour taste and a propensity to react with bases to form salts.

acute — having a short and relatively severe course; arising quickly, as acute symptoms.

aerosol — a liquid or solid composed of finely divided particles suspended in a gaseous medium. Examples of common aerosols are mist, fog, and smoke.

alogen — a substance that produces pain.

alkali — a class of bases that neutralize acids and form salts. Sodium hydroxide (lye) and ammonium hydroxide are alkalis.

alkaline — having the properties of an alkali, for example, sodium hydroxide; opposed to acid. Having hydroxyl ions (OH⁻); basic.

alkaloids — a group of basic organic substances of plant origin. Many have important physiological actions and are used in medicine, for example, cocaine.

amino acids — basic building-block units that can be chemically linked together to form larger molecules, such as peptides and proteins.

analgesic — substance used in medicine to relieve pain.

analogue — a chemical compound similar in structure to another chemical compound and having the same effect on body functions.

antibiotics — substances produced by and obtained from living cells, frequently those of lower plants, such as bacteria and molds; they are antagonistic to certain other forms of life, including pathogenic organisms. Examples are penicillin and streptomycin. Some antibiotics may also be produced synthetically.

antibody — a specific protein substance produced by the body in reaction to an antigen (a specific foreign material), such as a bacterium or a toxin; examples are antitoxins and agglutinins.

antigen — any foreign substance that, when introduced into the body, stimulates the formation of an antibody and that, when mixed with that antibody, reacts with it in some observable way. Antigens are usually protein in nature. They frequently consist of products produced by microorganisms.

antiplant — herbicide.

antiserum; antisera (plural) — a serum containing an antibody or antibodies. It is obtained from humans or animals that have survived exposure to an antigen.

antitoxin — a substance found in the blood serum or other body fluids that is specifically antagonistic to a toxin (antibody developed against a toxin) and that acts to neutralize it.

antivenin — a blood serum containing antibodies against venom, particularly snake venom.

aqueous — watery; prepared with water.

arrhythmia — any variation from the normal rhythm of the heartbeat.

arsenical — a chemical compound containing arsenic.

atropine — an alkaloid obtained from *Atropa belladonna*. It is used as an antidote for nerve agent poisoning. It inhibits the action of acetylcholine at the muscle junction by binding to acetylcholine receptors.

autonomic nervous system — that part of the nervous system that governs involuntary functions, such as heart rate, reflexes, and breathing. It consists of the sympathetic and parasympathetic nervous systems.

B

basic — relating to a base; having a pH greater than 7.

binary chemical munition — a munition in which chemical substances, held in separate containers, react when mixed or combined as a result of being fired, launched, or otherwise initiated to produce a chemical or antimateriel agent.

binary components — the component chemicals that combine to produce binary chemical agents. Examples of two common binary chemical agent components are as follows:

a. The components for binary GB (GB2) are methylphosphonic difluoride (DF) and isopropyl alcohol with an amine added (OPA).

b. The components for binary VX (VX2) are ethyl 2-didopropyl aminoethyl methylphosphonite (QL) and dimethylpolysulfide (NM).

biological agent — a microorganism that causes disease in people, plants, or animals or causes the deterioration of materiel.

biological operation — employment of biological agents to produce casualties in man or animal and damage to plants or materiel; or defense against such employment.

biological warfare — see biological operation.

botulism — poisoning by toxin derived from the microorganism *Clostridium botulinum*.

BRM — bioregulator/modulator.

BW — biological warfare.

BZ — a central nervous system depressant.

C

C — average concentration of an agent in the atmosphere; Celsius.

CA — bromobenzylcyanide.

carbamates — organic chemical compounds that can be neurotoxic by competitively inhibiting acetylcholinesterase binding to acetylcholine.

CAS — Chemical Abstracts Service.

casualty — any person who is lost to the organization by reason of having been declared dead, wounded, injured, diseased, interned, captured, retained, missing, missing in action, beleaguered, besieged, or detained.

catalyst — a material that increases or decreases the rate of a chemical reaction without being changed by the reaction.

central nervous system — consists of the brain and spinal cord. The CNS controls mental activity and voluntary muscular activity. It also coordinates the body's involuntary functions indirectly.

CG — phosgene.

chemical agent — a chemical substance that is intended for use in military operations to kill, seriously injure, or incapacitate people through its physiological effects. Excluded from consideration are riot control agents, chemical herbicides, and smoke and flame materials. Included are blood, nerve, choking, blister, and incapacitating agents.

chemical agent casualty — a person who has been affected sufficiently by a chemical agent to prevent or seriously degrade his or her ability to carry out the mission.

chemical agent symbol — the military Army code designation of any chemical agent. This is a combination of one to three letters or letter and number combinations. Do not confuse the symbol with the chemical formula.

chemical contamination — the presence of an agent on a person, object, or area. Contamination density of an agent is usually expressed either in milligrams or grams per square meter (mg/m^2 , g/m^2) or in pounds per hectare (lb/ha). A hectare is 10,000 square meters.

CK — cyanogen chloride.

CMPF — cyclohexyl methylphosphonofluoridate

CN — chloroacetophenone.

CNOH — cyanic acid.

CNS — central nervous system.

CO — carbon monoxide.

compound — In chemical terms, a uniform substance formed by the stable combination of two or more chemical elements, as distinct from a mixture.

concentration — the amount of an agent present in a unit volume. Usually expressed in milligrams per cubic meter (mg/m^3) of air.

contaminate — to introduce an impurity; for instance, foreign microorganisms developing accidentally in a pure culture. Clothing containing microorganisms is said to be contaminated.

coronary — pertaining to the heart.

covert — hidden, concealed, insidious.

CR — dibenz-(b,f)-1,4-oxazepine.

CS — O-chlorobenzylidene malononitrile, a tear agent.

Ct — vapor dosage.

cutaneous — pertaining to the skin.

CX — phosgene oxime.

cyanosis — blueness of the skin owing to insufficient oxygen in the blood.

cytotoxin — toxin that directly damages and kills the cell with which it makes contact.

D

DA — diphenylchloroarsine, a vomiting agent.

DC — diphenylcyanoarsine, a vomiting agent.

decay rate — the predictable rate at which microorganisms die.

decontaminating material — any substance used to destroy chemically or by other means, to physically remove, seal, or otherwise make the agents harmless.

decontamination — the process of making any person, object, or area safe by absorbing, destroying, neutralizing, making harmless, or removing chemical or biological agents, or by removing radioactive material clinging to or around it.

defoliant — an agent that, when applied to plants, kills or damages them or causes them to shed their leaves.

dehydrate — to remove water from.

depolarize — to remove the polarity, or difference in electrical charge, as on opposite sides of a cell membrane. When a nerve or muscle cell is stimulated it becomes depolarized.

desiccant — a substance that has an affinity (attraction) for water. When used as defoliants, desiccants remove water from plant tissue causing it to dry and shrivel.

desiccate — to dry completely.

detection — the determination of the presence of an agent.

detoxification rate — rate at which the body's own actions overcome or neutralize (detoxify) chemicals or toxins. Agents that the body cannot easily break down and neutralize and that accumulate in the body are called "cumulative."

DF — methylphosphonic difluoride.

dilate — to make wider or larger.

dilute solution — chemical agents that have been reduced in strength by dilution.

disease — a deviation from the normal state of function of a cell, an organ, or an individual.

disinfect — to free from pathogenic organisms or to destroy them.

disinfectant — an agent, usually chemical, that destroys infective agents.

dissemination — distribution or spreading.

DM — diphenylaminochloroarsine (Adamsite), a vomiting agent.

DMSO — dimethylsulfoxide.

DNA — deoxyribonucleic acid.

dosage — cumulative exposure equivalent to the concentration of chemical agent to which an individual is exposed integrated over the time of exposure.

dose — quantity of agent having entered the body.

DP — diphosgene.

DS2 — decontaminating solution No. 2.

dysentery — a disorder marked by inflammation of the intestines, particularly the colon, accompanied by pain in the abdomen, straining, and frequent stools containing blood and mucus. Dysenteries are caused by bacteria, protozoa, or parasitic worms or by some chemical irritant.

dyspnea — difficult or labored breathing.

E

ECT — effective dosage of an aerosol.

ED — ethyldichloroarsine.

edema — excessive accumulation of fluid in body tissues or body cavities.

EDMP — O, O'-ethyl (2-diisopropylaminoethyl) methylphosphonite, one of the binary components of VX.

endemic — native to, or prevalent in, a particular district or region. An endemic disease has a low incidence but is constantly present in a given community.

endogenous — produced or originating from within.

endogeneous biological regulators — naturally occurring, biological regulators with potential for chemical and biological warfare applications.

endotoxin — a toxin produced in an organism and liberated only when the organism disintegrates.

enterotoxins — toxins of bacterial origin that affect the intestines, causing diarrhea. Examples include toxins from *Vibrio cholera*, *Staphylococcus*, *Shigella E. coli*, *Clostridium perfringens*, *Pseudomonas*.

environment — the external surroundings and influences.
enzyme — organic substance capable of causing chemical changes to take place quickly at body temperature by catabolic action as in digestion.

eruption — a rash, visible lesion, or injury of the skin characterized by redness, prominence, or both.

eutectic mixture — a mixture of two or more substances in proportions that give the lowest freezing or melting point. The minimum freezing point attainable is termed the eutectic point.

exotoxin — a toxin excreted by a microorganism into the surrounding medium.

F

fatigue — weariness from labor or exhausting conditions where cells or organs have undergone excess activity so that they respond to stimulation with less than normal activity.

FDF — fast death factor.

fever — abnormally high body temperature; characterized by marked increase of temperature, acceleration of the pulse, increased tissue destruction, restlessness, and sometimes delirium.

flaccid — soft and limp; flabby.

flame — burning gas or vapor that causes lethal or incapacitating effects by means of direct burn wounds, depletion of oxygen, carbon monoxide poisoning, heat, or a combination of these factors. Flame can function secondarily as an incendiary.

flash point — the lowest temperature at which a substance gives off enough combustible vapors to produce momentary ignition when a flame is applied under controlled conditions.

FM — field manual; also symbol for titanium tetrachloride.

G

GA — tabun.

ganglia — a knot-like mass of neurons located outside the central nervous system.

GB — sarin.

GB2 — a binary nerve agent.

GD — soman.

gene — a segment of a chromosome definable in operational terms as a unit of genetic (inheritable) information.

genetic engineering — a variety of methods by which genetic material can be altered including recombination of genetic material to change or improve the hereditary properties of microorganisms, plants, or animals.

genetic material — the chemical compounds in each cell that contain the information. The major genetic material in all organisms is deoxy-ribonucleic acid (DNA); Ribonucleic acid (RNA) is the genetic material in certain viruses.

G-series nerve agents — a series of nerve agents developed by the Germans, that includes Tabun (GA), Sarin (GB), and Soman (GD).

H

H — Levinstein mustard, a blister agent.

H-series agents — a series of persistent blister agents, that include distilled mustard (HD) and the nitrogen mustards (HN-1, HN-2, and HN-3).

half-life — time required for half a material to decompose.

harassing concentration — a concentration of an agent that requires masking or other protective measures. Such concentration may be insufficient to kill but sufficient to interfere with normal operations.

HC1 — hydrogen chloride.

HCN — hydrogen cyanide.

HD — distilled mustard, a blister agent.

hemolysis — the destruction of red blood cells followed by release of the hemoglobin they contain.

hemorrhage — bleeding.

hepatitis — inflammation of the liver.

herbicide — a chemical compound that will kill or damage plants.

HF — hydrogen fluoride.

Hg — mercury.

HL — mustard-lewisite mixture.

HN — nitrogen mustard (HN-1, HN-2, and HN-3).

HT — mustard-T mixture.

HTH — calcium hypochlorite.

hydrolysis — interaction of a chemical agent with water to yield a less toxic product or products.

hydrolyze — to subject to hydrolysis; to split a chemical bond with water.

hygiene — the science of health and the preservation of good health.

I

IC₅₀ — median incapacitating dosage of a chemical agent vapor or aerosol.

ID₅₀ — median incapacitating dosage of a liquid chemical agent.

identification — can be subdivided into the following two levels:

Definitive identification — the determination of the exact identity of a compound or organism through the establishment of a group of unique characteristics.

Classification — the determination that a compound or organism is a member of a chemical or biological

class without knowledge of the exact identity of the compound or organism.

incapacitation — disablement.

incendiary — primarily an antimateriel compound that generates sufficient heat to cause destructive thermal degradation or destructive combustion of materiel.

industrial chemicals — chemicals developed or manufactured for use in industrial operations or research, by industry, government, or academia. These chemicals are not primarily manufactured for the specific purpose of producing human casualties or rendering equipment, facilities, or areas dangerous for use by man. Hydrogen cyanide, cyanogen chloride, phosgene, and chloropicrin are industrial chemicals that also can be military chemical agents (AC, CK, CG, and PS).

inflammation — reaction of tissues to injury; characterized by pain, heat, redness, or swelling of the affected parts.

intoxication — poisoning.

intracellular — inside, or within, the cell.

intraperitoneal — within the abdominal cavity.

intravenous — within the vein.

ion — an atom that has acquired an electrical charge because of gain or loss of electrons.

ion-channel — a passage that allows particular charged particles, such as sodium ions, potassium ions, or calcium ions, to pass through a membrane. Ions do not cross cell membranes through simple pores.

ionophore — a substance which creates a passage through membranes for ions.

ip — intraperitoneal.

J

jaundice — a disease symptom characterized by yellowing of the skin and eyes and by a deep yellow color of the urine.

K

K agents — incapacitating agents.

K_c — safety factor in addition to the theoretical minimum void.

Kg — kilogram(s).

L

L — lewisite.

lacrimator — a compound that causes a large flow of tears and irritates the skin.

latent period — a period of seeming inactivity.

LC₅₀ — median lethal dosage of a chemical agent vapor or aerosol.

LD₅₀ — median lethal dosage of a liquid chemical agent.

lesion — injury, diseased area or pathological change in an organ or tissue.

lethal chemical agent — an agent that maybe used effectively in field concentrations to kill.
 lipid — a fat or fat-like substance.
 lipophilic — fat-soluble.
 LSD — d-lysergic acid diethylamide.
 lyophilization — the process of drying substances, including microorganisms, in the frozen state under a vacuum; sometimes referred to as freeze drying.
 lysis — splitting.

M

malaise — a feeling of bodily discomfort.
 malignant — tending to go from bad to worse; capable of spreading from one site within the tissues to another.
 MD — methylchloroarsine.
 membrane — a thin layer of tissue that covers a surface or divides a space or organ.
 meningitis — inflammation of the meninges or certain membranes that envelop the brain and the spinal cord.
 MF — methylphosphono-fluoridic acid.
 mg — milligram(s).
 mg/kg — milligram(s) per kilogram (of body weight).
 mg-min/m³ — milligram-minute(s) per cubic meter.
 microencapsulate — to make, form, or place in an extremely small or microscopic capsule.
 micron — a unit of measurement: 1/1000 mm. Usually designated by the Greek letter μ .
 military chemical compound — chemical substance that has become accepted generally by the public for use in conventional war. Included are riot control agents, smoke and flame materials, and military herbicides. Excluded are chemical agents.
 miosis — excessive contraction of the pupil.
 miscible — capable of being mixed.
 mm — millimeter(s).
 mm Hg — millimeters of mercury; a unit used to describe atmospheric pressure.
 molecular weight — sum of the atomic weights of each atom in a molecule.
 molecule — a chemical combination of two or more atoms that form a specific chemical substance.
 monitoring — the act of detecting the presence of radiation and the measurement thereof with radiation measuring instruments.
 MOPP — mission-oriented protective posture, level 4.
 mortality rate — the ratio of the number of deaths from a given disease to the total number of cases of that disease.
 MPOD — Another designation for DF and DC.
 MW — molecular weight.

N

N — normality.
 NaOH — sodium hydroxide.

nausea — tendency to vomit; sickness of the stomach.
 NE — powdered elemental sulfur mixture.
 neat chemical agent — a nondiluted, full-strength (as manufactured) chemical agent. A chemical agent manufactured by the binary synthesis route will also be considered a neat agent regardless of purity.
 necrosis — death of a cell or group of cells.
 necrotic — capable of destroying living tissue.
 neural — relating or pertaining to nerves.
 neuron — a nerve cell. Neurons are characterized by their ability to become excited and to transmit their excitation onto other cells.
 neurotoxic — poisonous to nerve tissue.
 neuropeptide — a peptide produced by certain nerve cells, particularly in the CNS. Some may act as neurotransmitters or neurohormones. Acetylcholine, norepinephrine, serotonin, and histamine are neuropeptides.
 neurotransmitters — chemical substances released by neurons into the synapse and causing an effect on the postsynaptic cell. More than 50 compounds have been identified as neurotransmitters, including acetylcholine.
 neutralize — to render neutral.
 ng — nanogram(s).
 NIOSH — National Institute of Occupational Safety and Health.
 NM — dimethyl-polysulfide mixture.
 normality — in chemistry, a solution concentration designated by the number of gram-equivalents of solute per liter of solution.

O

off-target attack — an instance in which residual agent clouds drift onto positions or where personnel moving across country encounter toxic clouds or surface contamination.
 on-target attack — an instance in which agents are delivered directly into the target area where a position is the target for a direct attack by one or more agents delivered by air or ground means.
 OPA — isopropylamine and isopropyl alcohol.
 organic solvent — an organic chemical compound that dissolves another to form a solution. Examples of organic solvents are alcohols, turpentine, kerosene, benzene, chloroform, acetone, carbon tetrachloride, and toluene. Degreasers, paint thinners, antifreeze, and dry-cleaning compounds contain organic solvents.
 organophosphate — a phosphate-containing organic compound. Organophosphates inhibit cholinesterase enzymes. G-series and V-series nerve agents are organophosphates, as are certain common insecticides.
 oxidative processes — chemical reactions requiring oxygen.

oxime — a chemical compound containing one or more oxime groups (NOH). Although the chloroformoximes are blister agents, some oximes are beneficial. 2-PAM chloride (trade name protopam chloride or pralidoxime chloride) is used in treatment of nerve agent poisoning. This drug increases the effectiveness of drug therapy in poisoning by some, but not all, cholinesterase inhibitors (nerve agents). It reactivates the inhibited enzyme at skeletal muscles as well as at parasympathetic sites (glands and intestinal tract) and therefore relieves the skeletal neuromuscular block that causes the paralysis associated with the nerve agents.

P

2-PAM chloride — 2-pralidoxime chloride. See oxime.
 parasympathetic nervous system — the part of the autonomic nervous system that decreases pupil size, heart rate, and blood pressure and increases functions, such as secretion of saliva, tears, and perspiration.
 parenteral — in some manner other than by the intestinal tract.
 passive immunity — immunity acquired by introduction of antibodies produced in the body of another individual animal.
 pathogen — a disease-producing microorganism.
 pathogenic — causing disease.
 PB — pyridostigmine bromide.
 PD — phenyldichloroarsine.
 peptide — an organic compound of amino acids linked together by peptide bonds.
 percutaneous — effected or performed through the skin.
 persistency — in biological or chemical warfare, the characteristics of an agent which pertains to the duration of its effectiveness under determined conditions after its dispersal.
 pH — the chemist's measure of acidity and alkalinity. It is a scale in which the number 7 indicates neutral; numbers below 7 indicate acidity; and numbers above 7 indicate an alkaline substance.
 physostigmine — an alkaloid from the calabar bean *Physostigma*. Physostigmine salicylate is used to relieve symptoms of BZ and other glycolate exposure.
 phytotoxin — a toxin derived from a plant. An example is ricin from the castor bean.
 plasma — the fluid portion of the blood in which the cells are suspended.
 plasmid — a general term for all types of inclusions in a cell that can be considered as having genetic functions.
 polypeptide — a polymer of numerous amino acid residues (usually more than 20) linked together chemically by peptide bonds.

polyvalent vaccine — a vaccine made up of a number of strains of the same organism or of different organisms.
 postmortem — occurring or performed after death.
 postsynaptic — after a synapse.
 potable — fit or suitable for drinking.
 potassium — a chemical element important along with sodium in maintaining cell volume and an electrical gradient across cell membranes. Like sodium, potassium is important in nerve and muscle function.
 presynaptic — before a synapse.
 prostration — extreme exhaustion.
 proteins — a class of organic compounds of very high molecular weights which compose a large part of all living matter.
 PS — chloropicrin, a vomiting agent.
 pulmonary — pertaining to the lungs.
 pyridostigmine bromide — an antidote enhancer that blocks acetylcholinesterase, protecting it from nerve agents. When taken in advance of nerve agent exposure, PB increases survival provided that atropine and oxime (Mark I NAAK) and other measures are taken.

Q

QL (EDMP) — an organophosphorous ester, one of the components of VX.

R

RCA — riot control agent.
 reagent — a chemical substance used to produce a chemical reaction.
 receptor — a component of cell membranes where specific compounds bind, causing a change in the biological activity of the cell. Cells have receptors that can bind neurotransmitters, toxins, viruses, and other agents.
 recombinant DNA — a technique of genetic engineering by which units of genetic material are manipulated into new combinations or relationships.
 respiration — the act or function of using oxygen.
 respiratory — pertaining to respiration.

S

SA — arsine.
 S-4 smoke acid — sulfur trioxide chlorosulfonic acid solution.
 slurry — a thin, watery mixture.
 sodium — one of the two chemical elements in table salt (the other is chlorine). In the body, sodium is one of the most important constituents of blood and other body fluids. Its balance inside and outside cells is important in proper cell function including nerve and muscle activity.

specific gravity — the weight of a particular volume of a substance compared with the weight of an equal volume of water.

spores — resistant, dormant cells of some bacteria; primitive reproductive bodies of fungi.

STB — supertropical bleach.

sternutator — vomiting compound.

stupor — partial or nearly complete unconsciousness.

survey — the directed effort to determine the location and the nature of the agent in an area.

suspension — a mixture of fine particles and a liquid. If the mixture is allowed to stand, the fine particles will settle.

sympathetic nervous system — a network of nerves that trigger certain involuntary and automatic bodily functions, such as constricting blood vessels, widening the pupils, and speeding up the heartbeat.

symptoms — functional evidence of disease; a change in condition indicative of some mental or bodily state.

synapse — site at which neurons make functional contacts with other neurons or cells.

synergistic — working together; having combined cooperative action that increases the effectiveness of one or more of the components' properties.

synthesize — to build up a chemical compound from its elements or other compounds.

systemic — relating to the entire organism instead of a part.

systemic action — action affecting many systems. It includes the movement of the agent through the organism and its effect on cells and processes remote from the point of application.

T

t — time.

T — sulfur, oxygen, chlorine compound.

TGD — thickened Soman.

THC — tetrahydro cannabinol.

thickened agent — an agent to which a polymer or plastic has been added to retard evaporation and cause it to adhere to surfaces.

TM — technical manual.

TOF — trioctylphosphite.

toxemia — a general poisoning or intoxication owing to absorption of products (toxins) of microorganisms formed at a local source of infection.

toxic — poisonous; effects ranging from harmful to lethal depending on the dose and resistance of the individual.

toxicity — the quality of being poisonous.

toxin — generally, any poisonous substance of microorganism, plant, or animal origin.

toxoid — a chemically altered toxin changed so that it is no longer poisonous but still produces active immunity when injected into an animal or man.

training agent — an agent authorized for use in training to enhance proficiency for operating under NBC conditions.

T-2 — trichothecene, a mycotoxin.

TR — o,o'-diethylmethylphosphonite.

TTX — tetrodotoxin.

U

ug — microgram(s).

ultraviolet — light waves shorter than the visible blue-violet waves but longer than X rays. Ultraviolet radiation is very effective in killing unprotected microorganisms.

urticant — a substance which produces a stinging sensation, as if with nettles. Phosgene oxime (CX) is an urticant.

US — United States.

UV — ultraviolet.

V

V-agents — persistent, highly toxic nerve agents developed in the mid-1950s and absorbed primarily through the skin.

vaccine — a preparation of killed or attenuated (weakened) infective or toxic agent used as an inoculation to produce active artificial immunity.

vapor pressure — the pressure exerted at any temperature by a vapor when a state of equilibrium has been reached between it and its liquid or solid state.

vector — a carrier, especially the animal or intermediate host that carries a pathogen from one host to another, as the malaria-carrying mosquito.

venom — poisonous mixture of toxins and other natural chemicals produced by animals, including snakes, spiders, and scorpions.

vesicant — agent that acts on the eyes and lungs, capable of producing blisters, and blisters the skin.

VFDF — very fast death factor.

viable — capable of living.

virulence — capacity of a microorganism to produce disease.

viscosity — the resistance of a liquid to flow, resulting from the combined effects of internal friction and friction between the liquid and its surroundings.

viscous — resisting flow.

volatile — passing readily into a vapor; having a high vapor pressure.

volatility — the tendency of a chemical to vaporize or give off fumes. It is directly related to vapor pressure.

VX — a persistent chemical agent, the US standard V-agent.

Vx — a persistent chemical agent.

W

weapon system — an integrated relationship of agents, munitions, or spraying devices and their mode of delivery to the target.

WP — white phosphorous.

Z

Zone I — an area of major operational concern in the predicted biological downwind hazard area. Casualties may exceed 30% in unprotected personnel.

zootoxin — a toxin or poison of animal, such as the venom of snakes, spiders, and scorpions.

Symbols

µg — microgram(s).

µm — micrometer(s).

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