War and Public Health

Second Edition

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Chemical Weapons

Ernest C. Lee and Stefanos N. Kales

Chemical warfare has existed for thousands of years. The Chinese used arsenical smoke as a weapon as early as 1000 B.C.E.[1] In the 20th century, chemical agents were used against military and civilian targets on numerous occasions. The world still remains vulnerable to the deliberate use of chemical weapons. To better appreciate the public health threats posed by chemical agents, a basic understanding of their properties is helpful.

The Basics of Chemical Agents

Chemical agents are compounds designed to kill or disable people through toxic or poisonous mechanisms. They are relatively simple to make and use. Their effects are often dramatic and immediate. Both combatant and noncombatant populations can be the targets of these weapons. Environmentally persistent chemical agents can also be used to deny terrain or to contaminate food and water. Like nuclear and biological weapons, chemical weapons have psychological, political, operational, and strategic impacts.

Chemical attacks can be delivered by almost any type of conventional weapon system or spray device, or by nontraditional means, such as the plastic bags used by the Aum Shinrikyo cult to launch sarin attacks in Japan in the mid-1990s.[2] Intentional release of industrial chemicals is also another possible means of chemical attack; information about the presence of specific chemicals may be available from workers in a chemical plant. In contrast, delivery by chemical *weapons* is more likely to involve unidentified substances.

Chemical agents have shorter latency periods between exposure and the onset of symptoms than do biological agents. Chemical exposures are quickly recognizable due to the rapid onset of similar symptoms in a group of persons or the close proximity of a group of persons to a chemical release.[3] However, real-time identification of specific chemicals through clinical, laboratory, or environmental testing is difficult. Given the rapid action of chemical agents, the window for effective therapy is often narrow if serious chemical intoxication has occurred. Therefore, empirical treatment of chemical casualties is of paramount importance and requires some understanding of toxic mechanisms, presenting symptoms, and principles of triage and emergency management.

General Properties and Exposure Variables

Chemical agents can be absorbed by several routes, depending on the physical state of the toxin (vapor, aerosol, liquid, or solid) and existing ambient conditions (temperature and humidity). In vapor or aerosol form, chemical agents usually enter the body via the respiratory tract through oral and nasal mucosa, large and small airways, and pulmonary alveoli. After inhalation, they may produce respiratory injury or be absorbed systemically, with subsequent toxic effects elsewhere. Vapors and liquid droplets can be absorbed through the skin and mucous membranes. Solid-state compounds can also produce harmful effects through skin exposure, or, if dispersed as fine powders, they can affect the respiratory tract and mucous membranes. Agents may penetrate the skin to form temporary reservoirs and spread systemically, causing adverse effects.

Chemical agents may be divided into two major physical categories based on their rate of environmental decomposition after release:

1. Persistent agents that present a danger for days to weeks by remaining a contact hazard or by vaporizing slowly to produce an inhalation hazard
2. Nonpersistent agents that rapidly disperse and present a hazard for minutes to hours.

The potential number of persons adversely affected during a chemical attack is determined by the setting (indoor or outdoor), the proximity of individuals to the release and their density, and ambient conditions such as wind, rainfall, humidity, and temperature. Wind can be exploited to spread airborne chemicals and thus increase the number of individuals exposed; wind can also disperse toxins more rapidly. Rain reduces the effectiveness of chemical agents by washing away, diluting, and promoting hydrolysis. High temperatures decrease the persistence of chemical agents but produce higher vapor concentrations; low temperatures increase persistence. Because of these variables, the prediction of direct and secondary effects of chemical weapons is complex.

Major Classes of Chemical Agents

Because of the many chemical agents and the difficulties in rapid identification, empirical assessment and management of those affected by recognition of syndromes is recommended. Within this framework of major "toxidromes," chemical agents can be divided into four major groups: asphyxiants, cholinesterase inhibitors, respiratory tract irritants, and vesicants/skin caustics (Table 8-1).[3] As a general principle for all significant chemical exposures, after extrication, exposed victims should be decontaminated. First responders should have appropriate personal protective equipment to avoid exposure from the environment as well as secondary exposures from their patients. Immediate management is directed at the ABCs of Airway, Breathing, and Circulation.

Asphyxiants

Asphyxiants may be classified as either simple or chemical. Simple asphyxiants, such as nitrogen, carbon dioxide, and inert gases, physically displace oxygen in air when released in sufficient concentrations in relatively closed or confined spaces. Inhalation results in oxygen deficiency and hypoxemia. Chemical asphyxiants, such as cyanides, carbon monoxide, and hydrogen sulfide, interfere with oxygen transport or cellular respiration or both, causing subsequent tissue hypoxia. Asphyxiants are absorbed via inhalation; cyanides can also be readily absorbed by the mucous membranes and skin. Mild symptoms include fatigue, headache, nausea, and dizziness. Severe symptoms range from dyspnea to cardiac ischemia, altered mental status, seizure, syncope, and coma. Asphyxiants cause prominent cardiovascular and neurological signs. Respiratory failure may occur from central nervous system depression. Standard military protective masks equipped with charcoal impregnated with metal salts provide adequate protection against field concentrations of cyanide vapors[4] (Figure 8-1) Specific management of asphyxiants starts with extrication to fresh air and the administration of 100 percent oxygen. Cyanide poisoning is additionally treated with antidotes: sodium nitrite and thiosulfate (used in the United States) or hydroxocobalamin (used in Europe).

Table 8-1. Features of Selected Major Chemical Exposures

Features	Asphyxiants	Cholinesterase Inhibitors	Respiratory Tract Irritants	Vesicants
Most likely agent in accidental release	Carbon monoxide	Organophosphorus pesticides	Chlorine and its derivatives, ammonia	—
Most likely agent in act of terrorism	Cyanide	Sarin and VX	Chlorine, phosgene	Sulfur mustard
Hallmark	Tissue hypoxia in cardiovascular system and central nervous system; usually, absence of respiratory tract irritation; no increase in secretions	Cholinergic syndrome with pupil constriction (miosis) and increased exocrine secretions; increasing effects on central nervous system with increasing exposure	Respiratory tract irritation and symptoms, usually more prominent than irritation of eyes and skin	Eye injuries and skin burns with vesicle formation, followed by respiratory tract irritation and, in the case of exposure to high concentrations, systemic effects
Typical Presentations				
Mild symptoms	Headache, fatigue, anxiety, irritability, dizziness, nausea	Miosis, dim vision, eye pain, rhinorrhea, irritability, headache, chest tightness, sweating	Nose and throat irritation, sore throat, cough, chest tightness, eye irritation	Conjunctivitis, limited erythema, epistaxis, sore throat, cough

Moderate-to-severe symptoms	Dyspnea, altered mental status, cardiac ischemia, syncope, coma, seizure	Salivation, Lacrimation, Urination, Defecation, Gastrointestinal cramping, and Emesis (SLUDGE); wheezing, muscle weakness, fasciculations, cognitive impairment, incontinence, coma, seizure	Laryngitis, wheezing, stridor, laryngeal edema, acute lung injury	Corneal damage, vesicles and bullae, nausea, wheezing, stridor, laryngeal edema, acute lung injury
Hyperacute onset—sudden collapse	High concentrations of cyanide or hydrogen sulfide and oxygen deficiency within a confined space	Exposure to VX or high vapor concentrations of other nerve agents	—	—
Acute onset—typically within minutes to hours after exposure	Most exposures to asphyxiant gases (carbon monoxide, cyanide) and oxygen deficiency	Vapor exposure, ingestion of liquid form, or moderate-to-large dermal exposure	Riot-control agents, irritants highly and intermediately water soluble (ammonia, hydrochloric acid, chlorine)	Lewisite, phosgene oxime, high concentrations of sulfur mustard
Delayed onset—typically 4 to 6 hours after exposure	Low-to-moderate concentrations of substances that metabolize to primary asphyxiants—methylene chloride (carbon monoxide), acrylonitrile, and propionitrile (cyanide)	Limited exposure of skin to droplets but not vapor	Poorly soluble gases (phosgene, nitrogen dioxide)	Sulfur mustard

Source: Kales SN, Christiani DC. Acute chemical emergencies. N Engl J Med 2004;350:801.

Figure 8-1. A member of the U.S. Air Force Security Forces talks through his MCU-2P chemical/biological protective mask as he communicates with other team members via radio during a combat employment readiness exercise. (Source: Department of Defense photograph by Tech. Sgt. Lance Cheung, U.S. Air Force.)

Cholinesterase Inhibitors

Carbamate pesticides, organophosphorus pesticides, and weaponized organophosphorus compounds (such as sarin, soman, tabun, and VX) all inhibit acetylcholinesterase, resulting in cholinergic overstimulation and subsequent muscarinic and nicotinic effects.[3,5–7] Cholinesterase inhibitors may be absorbed by inhalation, by ingestion, and through the skin. If nerve agent vapor exists alone, a specialized mask may provide adequate protection; however, if liquid agent is present, a mask, chemical protective suit, gloves, and overboots are required.

Muscarinic symptoms include rhinorrhea, salivation, bronchorrhea, and ophthalmic symptoms such as tearing, miosis, dim vision, and headaches. Large does may cause abdominal cramping, nausea, emesis, diarrhea, and fecal or urinary incontinence. Nicotinic symptoms include muscle weakness, fasciculations, and paralysis. Initially, tachycardia and hypertension may occur. Central nervous systems effects can range from irritability and mild cognitive impairment to convulsions and coma. Multiple mechanisms can contribute to respiratory failure, which can be fatal. Although depression of erythrocyte and serum cholinesterase activity confirms intoxication, treatment should not await these results, because they are not rapidly available. Antidotes include atropine, pralidoxime (or other oxime drugs), and benzodiazepines. Atropine works primarily at muscarinic sites, with dosing adjusted to minimize dyspnea, airway resistance, and respiratory secretions. Pralidoxime reactivates acetylcholinesterase. Benzodiazepines, such as diazepam, are the only effective anticonvulsant drugs for the treatment of persons poisoned with cholinesterase inhibitors.[3,5–7]

Organophosphorus chemical weapons (nerve agents) differ from organophosphorus insecticides, to which they are structurally related. Nerve agents are watery and volatile and act rapidly, but their effects are of shorter duration and require a smaller total dose of atropine. In contrast, insecticides are oily and less volatile. They have a slower onset of toxicity but longer duration of effects and require a large cumulative dose of atropine.[3,6,8,9]

Over time, the organophosphorus component of a nerve agent irreversibly forms a covalent bond with acetylcholinesterase, in a process known as "aging," and the enzyme becomes resistant to reactivation by pralidoxime.[1,7] Therefore, after appropriate decontamination, pralidoxime must be given promptly to prevent aging. Aging time can range form minutes (soman) to hours (sarin). In contrast, aging is not clinically relevant for organophosphorus insecticides, because these agents age at a very slow rate; however, oximes are still given to reactivate cholinesterases.[10]

Respiratory Tract Irritants (Choking Agents)

Respiratory tract irritants primarily attack the airways and lungs, causing respiratory tract inflammation, bronchospasm, and lung injury (noncardiogenic pulmonary edema). This group includes phosgene, diphosgene, chlorine, and chloropicrin as well as "tear gas" (lacrimogenic agents).[3,11–13] (Tear gas, usually considered nonlethal, was used by U.S. military forces in Vietnam to force into the open people who had been hiding.) Appropriate chemical masks can protect against these agents. Highly water-soluble irritants, such as ammonia, are absorbed in the upper respiratory tract, triggering symptoms that give early warning of toxicity. Less water-soluble irritants, such as phosgene, are able to penetrate more deeply with minimal or no symptoms, causing lung injury with a delayed onset.[3] In water, phosgene is hydrolyzed, forming hydrochloric acid and carbon dioxide. Phosgene causes acute lung injury, which interferes with gas exchange and ultimately leads to hypoxia. It can also cause irritation of the eyes and upper respiratory tract.[3,4,14] During the acute phase, exposed personnel may exhibit only minimal signs and symptoms; however, acute lung injury can later develop suddenly. Diuretics should be avoided, because they can exacerbate intravascular hypovolemia.[4] After decontamination of the skin and eyes, initial treatment consists of rest and oxygen. Rest is crucial, because physical exertion exacerbates lung inflammation. Bronchodilators should be used to treat bronchospasm, if present.[4] The use of corticosteroids, other than for the treatment of severe bronchospasm, is controversial.

Vesicants/Skin Caustics (Blister Agents)

There are three major families of vesicants: mustard, arsenical vesicants such as lewisite, and the halogenated oximes. Vesicants burn and blister any part of the body they contact.[4] Ophthalmic and cutaneous effects of exposure are the most prominent. Ophthalmic effects include conjunctivitis, corneal damage, and vision loss.[3,15,16] Skin lesions include vesicles and bullae, which are fragile and can rupture, promoting wound infection. Blister fluid, however, is not contaminated with the vesicant agent. Moist skin areas, such as the groin and axillae, are more susceptible to lesions. Inhalation of vapors can lead to respiratory epithelial necrosis, with complications including hemorrhagic edema and secondary pneumonia. These complications usually occur within 48 hours after exposure and are the most common cause of death.[3,17]

Mustard is an alkylating agent that affects DNA chains and is an inflammatory activator. Mustard agents can cause vomiting and diarrhea when ingested, and hematopoietic suppression, including bone marrow failure, may occur within days to weeks after exposure.[3]

Vesicants can penetrate the skin by contact with either vapor or liquid. Latency depends on the class of the agent: several hours for mustard, shorter duration for lewisite, and negligible for oximes.[4] A specialized mask, chemical protective suit, gloves, and overboots are required for protection. Because mustard is absorbed by many materials, protective equipment must be changed regularly. Treatment consists of rapid decontamination (preferably within 2 minutes) before irreversible chemical reactions with the skin occur. Airway protection is required for moderate to severe exposures. Additionally, specialized ophthalmic, burn, and critical care may be required. Ophthalmic treatment consists of topical anticholinergic agents, antibiotics, and petrolatum to prevent eyelid adhesion. Burn care includes debridement, topical antibiotics, and analgesics.[3,15,18,19]

Basic Management

Public Health Preparedness

Unlike military personnel, who can focus preparation on the relatively few chemicals agents capable of meeting military requirements, civilian health personnel may face attacks by non-state entities whose agent selection principles could differ from military ones. Furthermore, the timing of attacks on civilian populations may be more unpredictable. Most major U.S. cities have a Metropolitan Medical Response System (MMRS), which is usually better equipped to respond to chemical agent attacks than typical emergency medical service response teams.[20]

A variety of chemical agent detectors have been designed to alert first responders to imminent danger. Detectors must function in real-world environments where price, portability, and time are critical factors. Often, the most challenging aspect for chemical agent identification is differentiating weapon agents of interest from other chemicals in the environment. Various technologies employed include spectroscopy, flame photometry, photoionization, and use of calorimetric indicators, electrochemical detectors, acoustical wave sensors, and immunoassays.[20] Detectors must be subjected to extensive scrutiny, because excessive false-positive results can lead to response fatigue; in contrast, a single false-negative finding can result in the loss of human life.

Special Populations

Chemical attacks on civilian populations pose a unique challenge to public health workers. Many emergency response plans have been largely based on military chemical casualty care doctrines, which are designed to protect a healthy adult combatant in a battlefield scenario. However, the general population also contains groups that are more vulnerable to chemical effects, including children, older people, and individuals with underlying illness of varying types and degrees of severity. Although management of chemical effects on pediatric patients does not differ markedly from that for adult patients, physiological differences between children and adults must be considered. Children's smaller mass reduces the dose of chemical agent required to cause detrimental effects, while their higher respiratory rates and minute volumes increase the dose of chemical agent delivered at a constant concentration of toxic vapor. Children also have less mature metabolic systems for detoxification. In addition, children exposed to a chemical agent may present to a health care provider in a different manner that an adult. For example, children in cholinergic crisis induced by nerve agents may not necessarily manifest miosis. Finally, because children, on average, have more years of life left than their adult counterparts, there is more time for latent effects of chemical agent exposure to become manifest; therefore, children are theoretically more vulnerable to the longer-term effects of alkylating agents, such as mustard, which is mutagenic as well as carcinogenic.[21,22]

Older populations should be considered when planning for response to a chemical attack. On average, older persons have a higher prevalence of underlying chronic diseases. Additionally, liver volume, hepatic blood flow, and hepatic clearance capacity decline with age.

Delayed and Long-term Effects

The probability of delayed effects in persons exposed to certain chemical agents depends on the dose, exposure duration, and individual susceptibility. Delayed effects include mutations, cancer, and birth defects; however, only limited research is available concerning these adverse effects. Public health planning should also include measures designed to mitigate long-term psychological sequelae among attack survivors.

Contingency Planning

Given the potential magnitude of harm that can be inflicted on a population, advanced preparation needs to be made for a large-scale chemical attack. Military and federal government resources can be valuable to local emergency planners. The Defense Threat Reduction Agency (DTRA) has developed software tools to model nuclear, chemical, biological, and radiological releases. Such simulation technology provides a fast, effective, and inexpensive means to prepare plans for dealing with potential attacks.

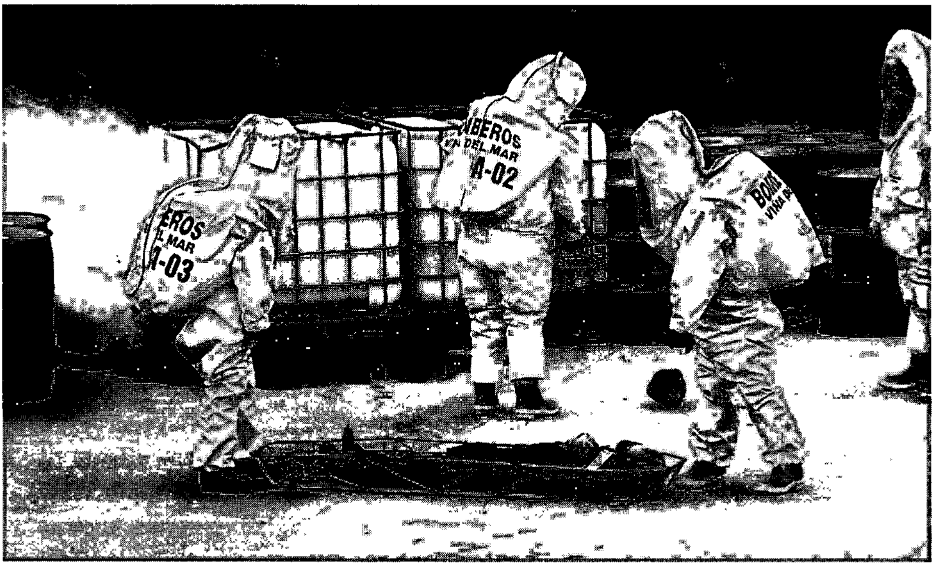

Figure 8-2. Exercise in protection from chemical weapons in Chile. (Source: Organization for the Prohibition of Chemical Weapons.)

Various emergency response agencies should communicate and work together in formulating contingency plans for chemical attacks and a chain of command that is mutually agreed upon. To this end, joint training exercises are essential. Resources, such as decontamination equipment, personal protective equipment, and antidotes, must be prepositioned to strategic locations. Maps of major industrial sites that could be targets of attacks should be maintained by hazardous material response teams and MMRSs, along with information regarding treatment for respective hazardous material or energy exposures. Emergency contingency plans should be logistically and economically feasible as well as sustainable. Hospitals as well as first-response units should have decontamination equipment, personal protective equipment, and adequate training in the use of this equipment (Figure 8-2). Panic that may ensue after an attack will likely lead to many people seeking medical attention that is not needed, overwhelming triage personnel if they are not adequately trained. Large-scale, multimodality patient simulation can be used to train clinicians and nonclinicians for potential attack scenarios.[23]

The Use of Chemicals Agents in War and Terrorist Attacks

Although guns and conventional explosives have been the terrorist weapons of choice, some terrorist groups show interest in acquiring the capability to use chemical, biological, radiological, or nuclear materials. Terrorism attacks have become more lethal and are often designed to kill as many people as possible. Some terrorist groups are driven by ethnic hatred, political beliefs, or religious ideology. Certain groups may lack a concrete goal other than to punish their enemies by killing as many of them as possible.

The Aum Shinrikyo religious cult launched two attacks in Japan using sarin. In the first attack, a truck was used to release an aerosol cloud of sarin into a residential neighborhood of Matsumoto in June 1994.[1,2] As a result, 7 people died and another 200 required hospitalization for at least one night. In a second attack, terrorists carried diluted sarin solution in plastic bags into subway trains and punctured the bags, releasing sarin vapor into three convergent lines of the Tokyo subway system. This attack was the largest disaster ever caused by nerve gas in peacetime. It was a failure in many respects; Aum Shinrikyo had used many highly skilled technicians and spent tens of millions of dollars developing a chemical attack that killed fewer people than conventional explosives could have. However, examination of the aftermath illustrates how even a botched attack easily overwhelmed an ill-prepared disaster management system. Although only 12 people died, approximately 1,000 were injured. In addition, because of the panic that the attack caused, 4,971 patients who had no signs of adverse effects were evaluated by health care facilities on the day of the attack.[25,26]

Several problems with hospital plans and disaster management were revealed. The main hospital involved had not established a definite plan of how to channel large numbers of affected people through its three entrances. As a result, affected people, family members, media crew, and onlookers streamed into the hospital from all three entrances, creating a chaotic situation. Many medical records were lost. Because the cause of the illness was not known until about 3 hours after the release, many hospital staff members were secondarily exposed. Hospitals lacked decontamination facilities and proper ventilation. Staff members did not have immediate access to chemically resistant personal protective equipment. One hospital alone expended 700 ampules of pralidoxime chloride and 2,800 ampules of atropine sulfate; because its original stockpile of antidote was depleted, the hospital had to airlift in additional supplies.[24]

Although use of chemical agents against noncombatants has only recently drawn attention, they have been used by military forces for centuries. The ancient Spartans used noxious smoke and flame against cities during the Peloponnesian War. Leonardo da Vinci proposed a powder of arsenic sulfide. During the Russo-Japanese War, Japanese soldiers used arsenical rag torches.[27]

Most chemical agents used in World War I were discovered during the 18th and 19th centuries. Both the French and the British tested various chemicals weapons on the battlefield. The French used ethyl bromoacetate grenades against the Germans during the German invasion of Belgium and France.[27] The Germans pursued offensive chemical weapons. In October 1914, German forces fired 3,000 projectiles filled with dianisidine chlorosulfate, a lung irritant, at the British in Neuve-Chapelle; however, the explosion of the shells nullified their chemical activity. The Germans later developed munitions containing xylyl bromide and fired more than 18,000 of them at Russian positions near Bolimov, located in the plains west of Warsaw; in this case, cold temperatures prevented vaporization of the gas, and the attack was largely unsuccessful.[28–30]

The first successful German chemical attack occurred in April 1915 in Ypres, Belgium. German forces waited for favorable wind conditions and then released large amounts of chlorine gas from cylinders.[31] The Allies responded with chlorine attacks. Thus began a deadly competition to develop better protective masks, more potent chemicals, and more effective delivery systems (Figure 8-3). The Germans escalated to the use of phosgene and diphosgene, while the French resorted to hydrogen cyanide and cyanogen chloride. In order to bypass protection rendered by masks, the Germans introduced mustard, a persistent vesicant capable of harming body areas not protected by gas masks.

In 1943, a U.S. freighter that was carrying 100 tons of mustard gas in 100-pound bombs was bombed by German planes while it was waiting to be

Figure 8-3. Members of the 108th Field Artillery firing in mission-oriented protective posture, Argonne, France, October 1918. This battery was under fire of enemy gas shells at the time this photograph was taken. (Source: National Archives.)

unloaded at the seaport of Bari, Italy. As a result, 628 people were affected, of whom 69 died within two weeks of the bombing.[32]

In the remainder of the 20th century, there were other instances in which chemical agents were used with devastating consequences (Table 8-2). In addition to the 1994 and 1995 sarin attacks in Japan, other attacks involved the following chemical agents[33]:

- Adamsite, diphenylchlorarsine, and mustard gas in Russia (1919)
- Bromomethyl ethyl ketone, chloropicrin, and mustard gas in Morocco (1923–1926)
- Chlorine, chloroacetophenone, mustard gas, phenyldichlorarsine, diphenylchlorarsine, and phosgene in Abyssinia (1935–1945)
- Chloroacetophenone, diphenylcyanoarsine, hydrogen cyanide, lewisite, mustard gas, and phosgene in Manchuria (1937–1945)
- Chloroacetophenone, mustard gas, and phosgene in Yemen (1963–1967)
- 2-Chlorobenzalmalononitrile in Vietnam (1965–1975)
- 2-Chlorobenzalmalononitrile in Iraq (1982–1988)
- Mustard gas, sarin, and tabun in Iran (1982–1988).

Table 8–2. A Timeline of Chemical Weapons History

1899, 1907: First and second peace conferences at The Hague. In 1899, European nations prohibited "the use of projectiles whose sole purpose is the release of asphyxiating or harmful gases." In 1907, the Conference added the use of poison or poisoned weapons to the prohibition.

1914–1918: The first large-scale attack with chemical weapons occurred on April 22, 1915, at Ieper in Belgium, during World War I.

1925: The Protocol for the Prohibition of the Use in War of Asphyxiating, Poisonous or Other Gases, and of Bacteriological Methods of Warfare was signed at Palais Wilson in Geneva.

1939–1945: Chemical weapons were deployed on a large scale in almost all theaters in World War II, leaving behind a legacy of old and abandoned chemical weapons.

1946–1991: During the Cold War, many nations produced and stockpiled chemical weapons, amounting to tens of thousands of tons, enough to kill much of human and animal life worldwide.

1988: Iraq used chemical weapons against Iran during the 1980-1988 conflict. Iraq also used mustard gas and nerve agents against Kurdish residents in northern Iraq.

1995: In Japan, the Aum Shinrikyo cult released the chemical agent sarin in a terrorist attack on the Tokyo subway. About 5,000 people became sick, and 12 died.

1997: With the entry into force of the Chemical Weapons Convention on April 29, 1997, The Organization for the Prohibition of Chemical Weapons immediately began its work to implement the Convention.

Source: Organization for the Prohibition of Chemical Weapons. Available at: http://www.opcw.org/29april/page02.html (accessed June 12, 2007).

Today, dozens of chemical agents are stockpiled in many countries, threatening combatants and noncombatants.

Chemical Agents and International Law

Since at least the early 1600s, international law has condemned what would today be regarded as chemical warfare. Subsequent development of such law can be seen in the Brussels Declaration of 1874 and at the Hague Peace Conference of 1899. Following the extensive use of chemical weapons during World War I, the international community strengthened the existing legislation restricting these weapons, leading to the Protocol for the Prohibition of the Use in War of Asphyxiating, Poisonous or Other Gases, and of Bacteriological Methods of Warfare. This treaty, known as the Geneva Protocol of 1925, entered into force in 1928.

As written, the Geneva Protocol prohibits "the use in war of asphyxiating, poisonous or other gases, and of all analogous liquids, materials or devices."

However, it does not prohibit possession of these weapons. In effect, the treaty was a "no-first-use" agreement. Additionally, some states parties (countries) reserved the right to use the weapons against states not party to the protocol. For these reasons, a more comprehensive prohibition of weapons was negotiated in the 1993 Chemical Weapons Convention (CWC). This agreement, which entered into force in 1997, is of "unlimited duration."

The CWC established the Organization for the Prohibition of Chemical Weapons (OPCW), a permanent international body whose membership consists of all states parties to the Convention, which oversees the implementation of the CWC. The CWC "reaffirms principles and objectives of and obligations assumed under the Geneva Protocol of 1925." Each state party to the CWC commits to never (1) develop, produce, otherwise acquire, stockpile, or retain chemical weapons or transfer, directly or indirectly, chemical weapons to anyone; (2) use chemical weapons; (3) engage in any military preparations to use chemical weapons; or (4) assist, encourage, or induce anyone, in any way, to engage in any activity prohibited to a state party under the Convention.

The CWC also commits each state party to "destroy chemical weapons it owns or possesses, or that are located in any place under its jurisdiction or control, in accordance with the provisions of this Convention" and "to destroy all chemical weapons it abandoned on the territory of another state party, in accordance with the provisions of this Convention." Any such destruction must ensure the safety of the population and the protection of the environment. The CWC incorporates an elaborate regimen to ensure compliance and specifies how its obligations are to be implemented. Although the CWC makes no direct reference to the concept of universality, the objective of universal adherence follows from the goal in its preamble to exclude the use of chemical weapons "for the sake of all mankind."

Despite the elaborate measures in the CWC, several challenges remain. First, not all countries have joined the treaty, challenging the concept of universality. Second, the CWC allows each country "to withdraw from this Convention if it decides that extraordinary events, related to the subject-matter of this Convention, have jeopardized the supreme interests of its country." Such discretion could potentially be exploited out of self-interest. Third, export/import controls remain underdeveloped. For example, some mustard and nerve agent precursors are not listed in the schedules of controlled chemicals. Fourth, the effectiveness of compliance monitoring systems has not truly been tested. Finally, prohibitions under the CWC are directed primarily to the actions of states and only marginally address the matter of individual responsibility. With the emergence of non-state actors with interest in these and other weapons, amendments to the existing CWC or a new treaty is needed to require a country to establish criminal jurisdiction applicable to foreign nationals who commit chemical weapons offenses either on its own territory or elsewhere, regardless of nationality.

Control of Chemical Weapons Proliferation

Despite the many attempts to limit the spread of chemical weapons, the ease with which certain classes can be developed and their sheer destructive potential make them attractive to any government or non-state entity that is seeking military advantage. Multilateral commitment is critical in controlling proliferation of such weapons. Critical also are intelligence gathering, challenge inspections, and monitoring of chemical transfers and technologies used in the development and manufacture of chemical weapons (Figure 8-4). When destroying existing chemical weapons, countries must exercise extreme caution so as to avoid adverse effects on local populations as well as the environment. To these ends, the CWC states that the following processes may not be used in the destruction of chemical weapons: "dumping in any body

Figure 8-4. Organization for the Prohibition of Chemical Weapons (OPCW) inspectors inventory artillery munitions. (Source: Organization for the Prohibition of Chemical Weapons.)

of water, land burial, or open-pit burning." To augment the legal framework designed to control the spread of these weapons, national self-interest must not be underestimated in any multilateral agreement.

It is useful to remember that Napoléon Bonaparte once said, "Treaties are observed as long as they are in harmony with interests."

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