POISON GAS: AS A LETHAL WEAPON IN CHEMICAL WARFARE
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Abstract: Poison gas is any gas that is also a poison. Poison gases can kill or injure a person if present in a high enough concentration. There is a diverse range of different poison gases and each has unique properties. Many toxic liquids are also volatile and their vapors are poison gas. "Poison gas" can refer to poisons in chemical weapons, but most of them are in fact liquids, for example mustard gas and VX are viscous liquids that are dispersed into fine mists. In small amounts, corrosive poison gases usually cause irritation and may have a smell, but this is not universal. There are several gases that can kill insidiously, without a warning smell or irritation. All gases other than oxygen can displace air and cause death by asphyxiation. This does not make them poison gases. Nitrogen and carbon dioxide are two common examples. This has to be taken into account in handling of any compressed or liquified gas. Gases can be also dangerous because they are flammable (easily bursting into flame). Poison gases can cause injury more readily than condensed poisons because they diffuse (move) in the air, exposing skin and are easily breathed in. Immediate contact with corrosive gas leads to chemical burns of the skin and lungs. When absorbed into the inside of the human body and into the blood, either by breathing in or through skin, several poison gases cause a poisoning. In a poisoning, medical help is often necessary; simply removing the victim from the gas is not enough to prevent symptoms. Corrosive poison gases such as hydrogen chloride cause chemical burns into skin and inside the lungs. This causes lungs to fill up with liquid, which can kill. Alkylating poison gases such as methyl chloride are attacked by human DNA and proteins. In human cells, this causes cell death, cancer and a diverse set of symptoms caused by malfunctioning of the alkylated proteins. Mustard gas is an alkylating agent. Gases that release the fluoride ion into the human body can cause a loss of calcium in the blood, which leads to heart attack and death. Examples are hydrogen fluoride and chlorine trifluoride. Ammonia can cause corrosive burns. Hydrogen sulfide has a powerful smell, but it will easily desensitize the nose. After that it can shut down the breathing center in the brain, which stops the breathing reflex and kills the victim from asphyxiation. Nerve agents are poisonous liquids that easily evaporate. They shut down biochemical processes that allow muscles to relax. The result is that all muscles contract, including muscles required for breathing, causing death by asphyxiation. Poison gases are used in industry as chemical reagents. The chemical reactions they can be used for are more important than their toxicity. Today, chemists try to avoid the use of poisonous gases, but it is often not possible. Examples of large-volume industrial poisonous gases are hydrogen sulfide, cracked from oil, chlorine, in diverse chemical uses and to disinfect drinking water, and ammonia, which is a valuable fertilizer into itself and can be converted into a range of other fertilizers, in addition to chemical uses. Some fumigants (chemicals used to kill pests) are also poison gases, for example phosphine, which is used to kill pests living in grains. The advantage of a gas is that it evaporates after use, so that if food is treated with gas, there is no need to rinse it to remove the poison.

Keywords: Tear gas, phosgene, mustard gas, nerve gas, lethal gas, organophosphorus agents, holocaust, asphyxia.

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INTRODUCTION

A gas chamber is an apparatus for killing humans or animals with gas, consisting of a sealed chamber into which a poisonous or asphyxiant gas is introduced. The most commonly used poisonous agent is hydrogen cyanide; carbon dioxide and carbon monoxide have also been used. Gas chambers were used as a method of execution for condemned prisoners in the United States beginning in the 1920s and continue to be a legal execution method in 3 states. During the Holocaust, large-scale gas chambers designed for mass killing were used by Nazi Germany as part of their genocide program and also by the Independent State of Croatia at the Jasenovac concentration camp. The use of gas chambers has also been reported in North Korea. When executions by gas chambers are conducted in the United States, the general protocol is as follows. First, the executioner will place a quantity of potassium cyanide (KCN) pellets into a compartment directly below the chair in the chamber. The condemned person is then brought into the chamber and strapped into the chair, and the airtight chamber is sealed. At this point the executioner will pour a quantity of concentrated sulfuric acid (H₂SO₄) down a tube that leads to a small holding tank directly below the compartment containing the cyanide pellets. The curtain is then opened, allowing the witnesses to observe the inside of the chamber. The prison warden then asks the condemned individual if he or she wishes to make a final statement. Following this, the executioner(s) throws a switch/lever to cause the cyanide pellets to drop into the sulfuric acid, initiating a chemical reaction that generates hydrogen cyanide (HCN) gas:

\[ 2\text{KCN}(s) + \text{H}_2\text{SO}_4(aq) \rightarrow 2\text{HCN(g)} + \text{K}_2\text{SO}_4(aq) \]

The gas is visible to the condemned, and he/she is advised to take several deep breaths to speed unconsciousness in order to prevent unnecessary suffering. Accordingly, execution by gas chamber is especially unpleasant for the witnesses to the execution due to the physical responses exhibited by the condemned during the process of dying. These responses can be violent and can include convulsions and excessive drooling. Following the execution, the chamber is purged of the gas through special scrubbers and must be neutralized with anhydrous ammonia (NH₃) before it can be opened. Guards wearing oxygen masks remove the body from the chamber. Finally, the prison doctor examines the individual in order to officially declare that he or she is dead and release the body to the next of kin. One of the problems with the gas chamber is the inherent danger of dealing with such a toxic gas. Anhydrous ammonia is used to cleanse the chamber after cyanide gas has been used:

\[ \text{HCN} + \text{NH}_3 \rightarrow \text{NH}_4^+ + \text{CN}^- \]

The anhydrous ammonia used to clean the chamber afterwards, and the contaminated acid that must be drained and disposed of, are both very poisonous. Nitrogen gas or oxygen-
depleted air has been considered for human execution, as it can induce nitrogen asphyxiation. It has not been used to date.¹

![Gas chamber](image)

**Figure-1: Gas chamber**

Chemical weapons in World War I were primarily used to demoralize, injure and kill entrenched defenders, against whom the indiscriminate and generally slow-moving or static nature of gas clouds would be most effective. The types of weapons employed ranged from disabling chemicals, such as tear gas and the severe mustard gas, to lethal agents like phosgene and chlorine. This chemical warfare was a major component of the first global war and first total war of the 20th century. The killing capacity of gas was limited, with four percent of combat deaths caused by gas. Gas was unlike most other weapons of the period because it was possible to develop effective countermeasures, such as gas masks. In the later stages of the war, as the use of gas increased, its overall effectiveness diminished. The widespread use of these agents of chemical warfare, and wartime advances in the composition of high explosives, gave rise to an occasionally expressed view of World War I as "the chemists' war".

**1914: Tear gas**

The earliest military uses of chemicals were tear-inducing irritants rather than fatal or disabling poisons. During the First World War, the French army was the first to employ gas, using 26mm grenades filled with tear gas (ethyl bromoacetate, BrCH₂COOC₂H₅) in August 1914. The small quantities of gas delivered, roughly 19cm³ per cartridge, were not even detected by the Germans. The stocks were rapidly consumed and by November a new order was placed by the French military. As bromine was scarce among the Entente allies, the active ingredient was changed to chloroacetone (ClCH₂COCH₃). In October 1914, German troops fired fragmentation
shells filled with a chemical irritant against British positions at Neuve Chapelle, though the concentration achieved was so small that it was barely noticed. None of the combatants considered the use of tear gas to be a conflict with the Hague Treaty of 1899, which prohibited the launching of projectiles containing asphyxiating or poisonous gas.²

1915: Large-scale use and lethal gases

The first instance of large-scale use of gas as a weapon was on 31 January 1915, when Germany fired 18,000 artillery shells containing liquid xylyl bromide tear gas on Russian positions on the Rawka River, west of Warsaw during the Battle of Bolimov. However, instead of vaporizing, the chemical froze and failed to have the desired effect. The first killing agent employed by the German military was chlorine. Chlorine is a powerful irritant that can inflict damage to the eyes, nose, throat and lungs. At high concentrations and prolonged exposure it can cause death by asphyxiation. German chemical companies BASF, Hoechst and Bayer (which formed the IG Farben conglomerate in 1925) had been producing chlorine as a by-product of their dye manufacturing. In cooperation with Fritz Haber of the Kaiser Wilhelm Institute for Chemistry in Berlin, they began developing methods of discharging chlorine gas against enemy trenches.

Table-1: List of nerve gases

<table>
<thead>
<tr>
<th>Name</th>
<th>First use</th>
<th>Type</th>
<th>Usage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xylyl bromide</td>
<td>1914</td>
<td>Lachrymatory, toxic</td>
<td>Both</td>
</tr>
<tr>
<td>Chlorine</td>
<td>1915</td>
<td>Corrosive. Lung Irritant</td>
<td>Both</td>
</tr>
<tr>
<td>Phosgene</td>
<td>1915</td>
<td>Irritant, Corrosive, toxic</td>
<td>Both</td>
</tr>
<tr>
<td>Benzyl bromide</td>
<td>1915</td>
<td>Lachrymatory</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Chloromethyl chloroformate</td>
<td>1915</td>
<td>Irritant - Eyes, skin, lungs</td>
<td>Both</td>
</tr>
<tr>
<td>Trichloromethyl chloroformate</td>
<td>1916</td>
<td>Severe irritant, causes burns</td>
<td>Both</td>
</tr>
<tr>
<td>Chloropicrin</td>
<td>1916</td>
<td>Irritant, lachrymatory, toxic</td>
<td>Both</td>
</tr>
<tr>
<td>Stannic chloride</td>
<td>1916</td>
<td>Severe irritant, causes asphyxiating</td>
<td>Allies</td>
</tr>
<tr>
<td>Bromoacetone</td>
<td>1916</td>
<td>Lachrymatory, irritant</td>
<td>Both</td>
</tr>
<tr>
<td>Acrolein</td>
<td>1916</td>
<td>Lachrymatory, toxic</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Hydrogen cyanide (Prussic acid)</td>
<td>1916</td>
<td>Toxic, Chemical Asphyxiant</td>
<td>Allies</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>1916</td>
<td>Irritant, toxic</td>
<td>Allies</td>
</tr>
<tr>
<td>Diphenylchloroarsine</td>
<td>1917</td>
<td>Irritant/Sternutatory (causes sneezing)</td>
<td>Central Powers</td>
</tr>
<tr>
<td>α-chlorotoluene (Benzylic alcohol)</td>
<td>1917</td>
<td>Irritant, lachrymatory</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Mustard gas</td>
<td>1917</td>
<td>Vescicant (blistering agent), lung irritant</td>
<td>Both</td>
</tr>
<tr>
<td>Bis(chloromethyl) ether</td>
<td>1918</td>
<td>Irritant, can blur vision</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Ethyldichloroarsine</td>
<td>1918</td>
<td>Vescicant</td>
<td>Central Powers</td>
</tr>
<tr>
<td>N-Ethylcarbazole</td>
<td>1918</td>
<td>Irritant</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Blue Cross</td>
<td>1917</td>
<td>Toxic</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Green Cross</td>
<td>1915</td>
<td>Toxic</td>
<td>Central Powers</td>
</tr>
<tr>
<td>Yellow Cross</td>
<td>1916</td>
<td>Toxic</td>
<td>Central Powers</td>
</tr>
<tr>
<td>White Cross</td>
<td>1914</td>
<td>Toxic</td>
<td>Central Powers</td>
</tr>
</tbody>
</table>
1915: More deadly gases

The deficiencies of chlorine were overcome with the introduction of phosgene, which was prepared by a group of French chemists led by Victor Grignard and first used by France in 1915. Colourless and having an odor likened to "mouldy hay," phosgene was difficult to detect, making it a more effective weapon. Although phosgene was sometimes used on its own, it was more often used mixed with an equal volume of chlorine, with the chlorine helping to spread the denser phosgene. The Allies called this combination White Star after the marking painted on shells containing the mixture. Phosgene was a potent killing agent, deadlier than chlorine. It had a potential drawback in that some of the symptoms of exposure took 24 hours or more to manifest. This meant that the victims were initially still capable of putting up a fight; although this could also mean that apparently fit troops would be incapacitated by the effects of the gas on the following day. ³ Although phosgene was never as notorious in public consciousness as mustard gas, it killed far more people, about 85% of the 100,000 deaths caused by chemical weapons during World War I.

1917: Mustard Gas

The most widely-reported and, perhaps, the most effective gas of the First World War was mustard gas. It was a vesicant that was introduced by Germany in July 1917 prior to the Third Battle of Ypres. The Germans marked their shells yellow for mustard gas and green for chlorine and phosgene; hence they called the new gas Yellow Cross. It was known to the British as HS (Hun Stuff), while the French called it Yperite (named after Ypres). Mustard gas is not a particularly effective killing agent (though in high enough doses it is fatal) but can be used to harass and disable the enemy and pollute the battlefield. Delivered in artillery shells, mustard gas was heavier than air and it settled to the ground as an oily liquid resembling sherry. Once in the soil, mustard gas remained active for several days, weeks, or even months, depending on the weather conditions. The skin of victims of mustard gas blistered, their eyes became very sore and they began to vomit. Mustard gas caused internal and external bleeding and attacked the bronchial tubes, stripping off the mucous membrane. This was extremely painful. Fatally injured victims sometimes took four or five weeks to die of mustard gas exposure. ⁴
Nerve agents are a class of phosphorus-containing organic chemicals (organophosphates) that disrupt the mechanism by which nerves transfer messages to organs. The disruption is caused by blocking acetylcholinesterase, an enzyme that normally destroys acetylcholine, a neurotransmitter. As chemical weapons, they are classified as weapons of mass destruction by the United Nations according to UN Resolution 687 (passed in April 1991) and their production and stockpiling was outlawed by the Chemical Weapons Convention of 1993; the Chemical Weapons Convention officially took effect on April 29, 1997. The use of dangerous gases in warfare is forbidden by treaties already in the Hague Conventions of 1899 and 1907 and Geneva Protocol of 1925. Poisoning by a nerve agent leads to contraction of pupils, profuse salivation, convulsions, involuntary urination and defecation, and eventual death by asphyxiation as control is lost over respiratory muscles. Some nerve agents are readily vaporized or aerosolized and the primary portal of entry into the body is the respiratory system. Nerve agents can also be absorbed through the skin, requiring that those likely to be subjected to such agents wear a full body suit in addition to a respirator.\textsuperscript{5}

Sarin, or GB, is an organophosphorus compound with the formula \((\text{CH}_3)_2\text{CHO}\)\text{CH}_3\text{P(O)F}. It is a colorless, odorless liquid, used as a chemical weapon owing to its extreme potency as a nerve agent. It has been classified as a weapon of mass destruction in UN Resolution 687. Production and stockpiling of sarin was outlawed by the Chemical Weapons Convention of 1993, and it is classified as a Schedule 1 substance. Sarin can be lethal even at very low concentrations, with death following within 1 to 10 minutes after direct inhalation due to suffocation from lung muscle paralysis, unless some antidotes, typically atropine or biperiden and pralidoxime, are quickly administered to a person. People who absorb a non-lethal dose, but do not receive immediate medical treatment, may suffer permanent neurological damage.
Like other nerve agents, sarin attacks the nervous system by interfering with the re-absorption of neurotransmitters at neuromuscular junctions. Death will usually occur as a result of asphyxia due to the inability to control the muscles involved in breathing function. Specifically, sarin is a potent inhibitor of acetylcholinesterase, an enzyme that degrades the neurotransmitter acetylcholine after it is released into the synaptic cleft. In vertebrates, acetylcholine is the neurotransmitter used at the neuromuscular junction, where signals are transmitted between neurons from the central nervous systems to muscle fibres. Normally, acetylcholine is released from the neuron to stimulate the muscle, after which it is degraded by acetylcholinesterase, allowing the muscle to relax. A build-up of acetylcholine in the synaptic cleft, due to the inhibition of cholinesterase, means the neurotransmitter continues to act on the muscle fibre, so that any nerve impulses are effectively continually transmitted. Sarin acts on cholinesterase by forming a covalent bond with the particular serine residue at the active site. Fluoride is the leading group, and the resulting phosphoester is robust and biologically inactive. Its mechanism of action resembles that of some commonly used insecticides, such as malathion. In terms of biological activity, it resembles carbamate insecticides, such as Sevin, and the medicines pyridostigmine, neostigmine, and physostigmine.

Figure-3: Bombing of nerve gas shells

Soman, or GD (systematic name: O-Pinacolyl methylphosphonofluoridate), is an extremely toxic chemical substance. It is a nerve agent, interfering with normal functioning of the mammalian nervous system by inhibiting the cholinesterase enzyme. It is an inhibitor of both
acetylcholinesterase and butyrylcholinesterase. As a chemical weapon, it is classified as a weapon of mass destruction by the United Nations according to UN Resolution 687. Its production is strictly controlled, and stockpiling is outlawed by the Chemical Weapons Convention of 1993 where it is classified as a Schedule 1 substance. Soman was the third of the so-called G-series nerve agents to be discovered along with GA (tabun), GB (sarin), and GF (cyclosarin). It is a volatile, corrosive, and colorless liquid with a faint odor when pure. More commonly, it is a yellow to brown color and has a strong odor described as similar to camphor. The LC₅₀ for soman is 70 mg·min/m³ in humans. It is both more lethal and more persistent than sarin or tabun, but less so than cyclosarin. GD can be thickened for use as a chemical spray using an acryloid copolymer. It can also be deployed as a binary chemical weapon; its precursor chemicals are methylphosphonyl difluoride and a mixture of pinacolyl alcohol and an amine. Soman is an organophosphorous nerve agent with a mechanism of action similar to Tabun.

\[
\begin{align*}
\text{H}_3\text{C} & \quad \text{CH}_3\text{O} \\
\text{H}_3\text{C} & \quad \text{P} \quad \text{CH}_3 \\
\text{H}_3\text{C} & \quad \text{O} \quad \text{F}
\end{align*}
\]

3,3-dimethylbutan-2-yl \((R)\)-methylphosphonofluoridate

Nerve agents inhibit acetylcholine esterase (AChE) by forming an adduct with the enzyme via a serine residue on that enzyme. These adducts may be decomposed hydrolytically or, for example, by the action of some oximes and thereby regenerate the enzyme. A second reaction type, one in which the enzyme–organophosphate (OP) complex undergoes a subsequent reaction, is usually described as “aging”. Once the enzyme–OP complex has aged it is no longer regenerated by the common, oxime reactivators. The rate of this process is dependent on the OP. Soman is an OP that stimulates the rate of aging most rapidly decreasing the half-life to just a few minutes.⁶

Figure-4: Nerve gas shells
AChE is an enzyme involved with neurotransmission. Because of the severe decrease of the half-life of this enzyme, neurotransmission is abolished in a matter of minutes. Soman is a very effective compound that has severe health implications at very low doses. The LC$_{50}$ of soman in air is 70mg min per m$^3$. For compounds such as soman, which may also be used as a weapon, often a fraction of the LC$_{50}$ dose is where the first effects appear. Miosis, is one of the first symptoms of soman intoxication and can be seen in doses of less than 1% of the LC$_{50}$. By using animal models, it is able to predict the LD$_{50}$ value of soman. Most LD$_{50}$ values via the same administration route give somewhat different lethal doses, which means the organisms metabolize the compounds differently.$^7$

**Tabun** or GA is an extremely toxic chemical substance. It is a clear, colorless, and tasteless liquid with a faint fruity odor. It is classified as a nerve agent because it fatally interferes with normal functioning of the mammalian nervous system.

![Structure of Tabun or GA](image)

**Tabun** or GA is an extremely toxic chemical substance. It is a clear, colorless, and tasteless liquid with a faint fruity odor. It is classified as a nerve agent because it fatally interferes with normal functioning of the mammalian nervous system. Its production is strictly controlled and stockpiling outlawed by the Chemical Weapons Convention of 1993. Tabun is the first of the so-called G-series nerve agents along with GB (sarin), GD (soman) and GF (cyclosarin). Although pure tabun is clear, less-pure tabun may be brown. It is a volatile chemical, although less so than either sarin or soman. Tabun can be destroyed with bleaching powder, though the poisonous gas cyanogen chloride is produced.

The symptoms of exposure include: nervousness/restlessness, miosis (contraction of the pupil), rhinorrhea (runny nose), excessive salivation, dyspnea (difficulty in breathing due to bronchoconstriction/secretions), sweating, bradycardia (slow heartbeat), loss of consciousness, convulsions, flaccid paralysis, loss of bladder and bowel control, apnea (breathing stopped) and lung blisters. The exact symptoms of overexposure are similar to those created by all nerve agents. Tabun is toxic even in minute doses. The number and severity of symptoms which appear vary according to the amount of the agent absorbed and rate of entry of it into the body. Very small skin dosages sometimes cause local sweating and tremors accompanied with characteristically constricted pupils with few other effects. Tabun is about half as toxic as sarin by inhalation, but in very low concentrations it is more irritating to the eyes than sarin. Also,
tabun breaks down slowly, which after repeated exposure can lead to build up in the body. The effects of tabun appear slowly when tabun is absorbed through the skin rather than inhaled. A victim may absorb a lethal dose quickly, although death may be delayed for one to two hours. A person's clothing can release the toxic chemical for up to 30 minutes after exposure. Inhaled lethal dosages kill in one to ten minutes, and liquid absorbed through the eyes kills almost as fast. However, people who experience mild to moderate exposure to tabun can recover completely, if treated almost as soon as exposure occurs. The LC₅₀ for tabun is about 400 mg-min/m³. Treatment for suspected tabun poisoning is often three injections of a nerve agent antidote, such as atropine. Pralidoxime chloride (2-PAM Cl) also works as an antidote; however, it must be administered within a period of from minutes to a few hours following exposure to be effective.

![Figure-5: Poisoning effect of chemical warfare](image)

**Biological effects**

As their name suggests, nerve agents attack the nervous system of the human body. All such agents function the same way: by inhibiting the enzyme acetylcholinesterase, which is responsible for the breakdown of acetylcholine (ACh) in the synapse. ACh gives the signal for muscles to contract, preventing them from relaxing. Initial symptoms following exposure to nerve agents (like sarin) are a runny nose, tightness in the chest, and constriction of the pupils. Soon after, the victim will then have difficulty breathing and will experience nausea and drooling. As the victim continues to lose control of their bodily functions, they will involuntarily salivate, lacrimate, urinate, defecate, and experience gastrointestinal pain and vomiting. Blisters and burning of the eyes and/or lungs may also occur. This phase is followed by initially myoclonic jerks followed by status epilepticus. Death then comes via complete respiratory depression, most likely via the excessive peripheral activity at the neuromuscular junction of the diaphragm. The effects of nerve agents are very long lasting and increase with successive exposures. Survivors of nerve agent poisoning almost invariably suffer chronic neurological damage. This neurological damage can also lead to continuing psychiatric effects.⁸
Mechanism of action

Pralidoxime (2-pyridine aldoxime methyl chloride,) or 2-PAM, usually as the chloride or methiodide salts, belongs to a family of compounds called oximes that bind to organophosphate-inactivated acetylcholinesterase. It is used to combat poisoning by organophosphates or acetylcholinesterase inhibitors (nerve agents) in conjunction with atropine and diazepam.

\[
\text{N}^+ \text{CH}_3 \text{N} \overset{\text{OH}}{\text{O}} \text{H} \quad 2-[(E)-(hydroxyimino)methyl]-1-methylpyridinium
\]

Pralidoxime is typically used in cases of organophosphate poisoning. The acetylcholinesterase enzyme has two parts to it. An acetylcholine molecule, bound at both ends to both sites of the enzyme, is cleaved in two to form acetic acid and choline. In organophosphate poisoning, an organophosphate binds to just one end of the acetylcholinesterase enzyme [the esteric site], blocking its activity. Pralidoxime is able to attach to the other half [the unblocked, anionic site] of the acetylcholinesterase enzyme. It then binds to the organophosphate, the organophosphate changes conformation, and loses its binding to the acetylcholinesterase enzyme. The conjoined poison / antidote then unbind from the site, and thus regenerate the enzyme, which is now able to function again. After some time though, some inhibitors can develop a permanent bond with cholinesterase, known as aging, where oximes such as pralidoxime cannot reverse the bond. Pralidoxime is often used with atropine (a muscarinic antagonist) to help reduce the parasympathetic effects of organophosphate poisoning. Pralidoxime is only effective in organophosphate toxicity (i.e. it does not have an effect if the acetylcholinesterase enzyme is carbamylated, as occurs with neostigmine or phystostigmine). Pralidoxime has an important role in reversing paralysis of the respiratory muscles but due to its poor blood–brain barrier penetration, it has little effect on centrally-mediated respiratory depression. This is why atropine, which has excellent blood–brain barrier penetration, is concomitantly administered with pralidoxime during the treatment of organophosphate poisoning. While the efficacy of atropine has been well-established, clinical experience with pralidoxime has led to widespread doubt about its efficacy in treatment of Organophosphorus poisoning. When a normally functioning motor nerve is stimulated, it releases the neurotransmitter acetylcholine, which transmits the impulse to a muscle or organ. Once the impulse is sent, the enzyme acetylcholinesterase immediately breaks down the acetylcholine in order to allow the muscle or organ to relax. Nerve agents disrupt the nervous system by
inhibiting the function of the enzyme acetylcholinesterase via forming a covalent bond where acetylcholine would break down (undergoes hydrolysis). Acetylcholine thus builds up and continues to act so that any nerve impulses are continually transmitted and muscle contractions do not stop. This same action also occurs at the gland and organ levels, resulting in uncontrolled drooling, tearing of the eyes (lacrimation) and excess production of mucus from the nose (rhinorrhea). The structures of the complexes of Soman (one of the most toxic nerve agents) with acetylcholinesterase from Torpedo californica have been solved by X-ray crystallography (PDB codes: 2wfz, 2wg0, 2wg1, and 1som). The mechanism of action of soman could be seen on example of 2wfz.

**Antidotes**

Atropine and related anticholinergic drugs act as antidotes to nerve agent poisoning because they block acetylcholine receptors, but they are poisonous in their own right. (Some synthetic anticholinergics, such as biperiden may counteract the central symptoms of nerve agent poisoning better than atropine, since they pass the blood–brain barrier better than atropine.) While these drugs will save the life of a person affected with nerve agents, that person may be incapacitated briefly or for an extended period, depending on the amount of exposure. The endpoint of atropine administration is the clearing of bronchial secretions. Atropine for field use by military personnel is often loaded in an autoinjector, for ease of use in stressful conditions. Pralidoxime chloride, also known as 2-PAM chloride, is also used as an antidote. Rather than counteracting the initial effects of the nerve agent on the nervous system like atropine, pralidoxime chloride reactivates the poisoned enzyme (acetylcholinesterase) by scavenging the phosphoryl group attached on the functional hydroxyl group of the enzyme. Though safer to use, it takes longer to act. Revival of acetylcholinesterase with pralidoxime chloride works more effectively on nicotinic receptors while blocking acetylcholine with atropine is more effective on muscarinic receptors. Often, severe cases of the poisoning are treated with both drugs.9

**Xylyl bromide**, also known as methylbenzyl bromide or T-stoff, is a poisonous organic chemical compound with the molecular formula C₈H₇Br, formerly used as a tear gas. Physically it is a colourless liquid (melting point 21°C) with a pleasant aromatic odour.
Xylyl bromide is highly toxic, irritant and lachrymatory and has been incorporated in chemical weapons since the early months of World War I. Some commentators say the first use was in August 1914, when the French attacked German soldiers with tear gas grenades, but the agent used in that incident was more likely to be ethyl bromoacetate, which the French had tested before the war. The first extensive use of xylyl bromide was the firing by German forces of 18,000 "T-shells" at Russian positions in the Battle of Bolimów in January 1915. The shells were modified 15 cm (6 inch) artillery shells containing an explosive charge and c. 3 kg (7 lb) xylyl bromide. The attack was a complete failure because the winter weather was too cold to permit an effective aerosol, and the agent was either blown back towards the German lines, fell harmlessly to the ground, or was insufficiently concentrated to cause damage. A similar attack at Nieuwpoort in March 1915 was also unsuccessful.¹⁰

**Chlorine** (Cl₂) is a toxic gas that irritates the respiratory system. Because it is heavier than air, it tends to accumulate at the bottom of poorly ventilated spaces. Chlorine gas is a strong oxidizer, which may react with flammable materials. Chlorine is detectable with measuring devices in concentrations of as low as 0.2 parts per million (ppm), and by smell at 3 ppm. Coughing and vomiting may occur at 30 ppm and lung damage at 60 ppm. About 1000 ppm can be fatal after a few deep breaths of the gas. Breathing lower concentrations can aggravate the respiratory system, and exposure to the gas can irritate the eyes. The toxicity of chlorine comes from its oxidizing power. When chlorine is inhaled at concentrations above 30 ppm, it begins to react with water and cells, which change it into hydrochloric acid (HCl) and hypochlorous acid (HClO). When used at specified levels for water disinfection, the reaction of chlorine with water is not a major concern for human health. Other materials present in the water may generate disinfection by-products that are associated with negative effects on human health, however, the health risk is far lower than drinking undisinfected water.¹¹

![Figure-6: Chlorine shells](image-url)
Phosgene (COCl₂) was synthesized by the British chemist John Davy (1790–1868) in 1812 by exposing a mixture of carbon monoxide and chlorine to sunlight. He named it "phosgene" in reference of the use of light to promote the reaction; from Greek, phos (light) and gene (born). It gradually became important in the chemical industry as the 19th century progressed, particularly in dye manufacturing.¹²

**Chemical warfare**

Following the extensive use of phosgene gas in combat during World War I, it was stockpiled by various countries as part of their secret chemical weapons programs. In May 1928, eleven tons of phosgene escaped from a war surplus store in central Hamburg. 300 people were poisoned of whom 10 died. US Army phosgene identification poster from World War II. Phosgene was then only frequently used by the Imperial Japanese Army against the Chinese during the Second Sino-Japanese War. Gas weapons, such as phosgene, were produced by Unit 731 and authorized by specific orders given by Hirohito (Emperor Showa) himself, transmitted by the chief of staff of the army. For example, the Emperor authorized the use of toxic gas on 375 separate occasions during the battle of Wuhan from August to October 1938.

![Figure-7: Death valley of nerve gas](image)

**Safety**

Phosgene is an insidious poison as the odor may not be noticed and symptoms may be slow to appear. The odor detection threshold for phosgene is 0.4 ppm, four times the threshold limit value. Its high toxicity arises from the action of the phosgene on the proteins in the pulmonary alveoli, the site of gas exchange: their damage disrupts the blood-air barrier, causing suffocation. It reacts with the amines of the proteins, causing cross linking by formation of
urea-like linkages, in accord with the reactions discussed above. Phosgene detection badges are worn by those at risk of exposure. Sodium bicarbonate may be used to neutralize liquid spills of phosgene. Gaseous spills may be mitigated with ammonia.

**Benzyl bromide**, or α-bromotoluene, is an organic compound consisting of a benzene ring substituted with a bromomethyl group. It can be prepared by the bromination of toluene at room temperature in air, using manganese (IV) oxide as a heterogeneous catalyst. It is a colorless liquid that is decomposed slowly in water.

![Benzyl bromide](image)

(Bromomethyl)benzene

Benzyl bromide is used in organic synthesis for the introduction of the benzyl protecting group for alcohols and carboxylic acids. Benzyl bromide is a strong lachrymator and is also intensely irritating to skin and mucous membranes. Because of these properties, it has been used as a war gas.\(^\text{13}\)

**Chloromethyl chloroformate** (ClCOOCH\(_2\)Cl) is a chemical compound developed for use in chemical warfare in World War I. It is a tearing agent designed to cause temporary blindness. It is a colorless liquid with a penetrating, irritating odor.\(^\text{14}\)

**Diphosgene** is a chemical compound with the formula ClCO\(_2\)CCl\(_3\). This colorless liquid is a valuable reagent in the synthesis of organic compounds. Diphosgene is related to phosgene but is more conveniently handled because it is a liquid, whereas phosgene is a gas. Diphosgene was originally developed as a pulmonary agent for chemical warfare, a few months after the first use of phosgene. It was used as a poison gas in artillery shells by Germany during World War I. The first recorded battlefield use was in May 1916. Diphosgene was developed because the vapors could destroy the filters in gas masks in use at the time.\(^\text{15}\)

**Chloropicrin**, also known as **PS** and **nitrochloroform**, is a chemical compound currently used as a broad-spectrum antimicrobial, fungicide, herbicide, insecticide, and nematicide. Its chemical structural formula is Cl\(_2\)CNO\(_2\). Chloropicrin can be absorbed systemically through inhalation, ingestion, and the skin. At high concentrations it is severely irritating to the lungs, eyes, and skin. In World War I German forces used concentrated chloropicrin against Allied forces as a tear gas. While not as lethal as other chemical weapons, it caused vomiting and forced Allied
soldiers to remove their masks to vomit, exposing them to other, more toxic chemical gases used as weapons during the war.\textsuperscript{16}

\textbf{Stannic chloride} (SnCl\textsubscript{4}) was used as a chemical weapon in World War I, as it formed an irritating (but non-deadly) dense smoke on contact with air: it was substituted for by a mixture of silicon tetrachloride and titanium tetrachloride near the end of the War due to shortages of tin. It is also used in the glass container industry for making an external coating containing tin (IV) oxide which toughens the glass. It is a starting material for organotin compounds. Stannic chloride is used in chemical reactions with fuming (90\%) nitric acid for the selective nitration of activated aromatic rings in the presence of unactivated ones.\textsuperscript{17}

\textbf{Bromoacetone} is an organic compound with the formula CH\textsubscript{3}COCH\textsubscript{2}Br. This colorless liquid is a lachrymatory agent.

\textbf{Acrolein} (systematic name: propenal, CH\textsubscript{2}=CH-CHO) is the simplest unsaturated aldehyde. It is a colourless liquid with a piercing, disagreeable, acrid smell. The smell of burnt fat (as when cooking oil is heated to its smoke point) is caused by glycerol in the burning fat breaking down into acrolein. It is produced industrially from propylene and mainly used as a biocide and a building block to other chemical compounds, such as the amino acid methionine. Acrolein is toxic and is a strong irritant for the skin, eyes, and nasal passages. The main metabolic pathway for acrolein is the alkylation of glutathione. The WHO suggests a "tolerable oral acrolein intake" of 7.5\(\mu\)g/day per kilogram of body weight. Although acrolein occurs in French fries, the levels are only a few micrograms per kilogram. In response to occupational exposures to acrolein, the US Occupational Safety and Health Administration has set a permissible exposure limit at 0.1ppm (0.25mg/m\textsuperscript{3}) at an eight-hour time-weighted average.\textsuperscript{18}

\textbf{Hydrogen cyanide} (HCN), sometimes called \textit{prussic acid}, is an inorganic compound with the chemical formula HCN. It is a colorless, extremely poisonous liquid that boils slightly above room temperature, at 25.6\(^\circ\)C (78.1\(^\circ\)F). HCN is produced on an industrial scale and is a highly valuable precursor to many chemical compounds ranging from polymers to pharmaceuticals. A hydrogen cyanide concentration of 300mg/m\textsuperscript{3} in air will kill a human within 10–60minutes. A hydrogen cyanide concentration of 3500ppm (about 3200mg/m\textsuperscript{3}) will kill a human in about 1minute. The toxicity is caused by the cyanide ion, which halts cellular respiration by acting as a non-competitive inhibitor for an enzyme in mitochondria called cytochrome c oxidase. Hydrogen cyanide has been absorbed into a carrier for use as a pesticide. Under IG Farben's brand name \textit{Zyklon B} (German >\textit{Cyclone B}, with the B standing for \textit{Blausäure} - "Prussic Acid"), it was used in the German concentration camp mass killing during the World War II (esp. in the Holocaust). The same product is currently made in the Czech Republic under the trademark "Uragan D2." Hydrogen cyanide was also the agent used in gas chambers employed in judicial
execution in some U.S. states, where it was produced during the execution by the action of sulfuric acid on an egg-sized mass of potassium cyanide. Hydrogen cyanide is commonly listed amongst chemical warfare agents known as blood agents. As a substance listed under Schedule 3 of the Chemical Weapons Convention as a potential weapon which has large-scale industrial uses, manufacturing plants in signatory countries which produce more than 30 tonnes per year must be declared to, and can be inspected by, the Organisation for the Prohibition of Chemical Weapons. During the First World War, USA and Italy used hydrogen cyanide against the Central Powers in 1918. France had used it in combat already in 1916, but this proved to be ineffective due to physical conditions. Under the name prussic acid, HCN has been used as a killing agent in whaling harpoons. Hydrogen cyanide gas in air is explosive at concentrations over 5.6%. This is far above its toxicity level.¹⁹

**Hydrogen sulfide** (H₂S) is considered a broad-spectrum poison, meaning that it can poison several different systems in the body, although the nervous system is most affected. The toxicity of H₂S is comparable with that of hydrogen cyanide or carbon monoxide. It forms a complex bond with iron in the mitochondrial cytochrome enzymes, thus preventing cellular respiration. Since hydrogen sulfide occurs naturally in the body, the environment and the gut, enzymes exist in the body capable of detoxifying it by oxidation to (harmless) sulfate. Hence, low levels of hydrogen sulfide may be tolerated indefinitely. At some threshold level, believed to average around 300–350ppm, the oxidative enzymes become overwhelmed. Many personal safety gas detectors, such as those used by utility, sewage and petrochemical workers, are set to alarm at as low as 5 to 10ppm and to go into high alarm at 15ppm. A diagnostic clue of extreme poisoning by H₂S is the discoloration of copper coins in the pockets of the victim. Treatment involves immediate inhalation of amyl nitrite, injections of sodium nitrite or administration of 4-dimethylaminophenol in combination with inhalation of pure oxygen, administration of bronchodilators to overcome eventual bronchospasm, and in some cases hyperbaric oxygen therapy (HBOT). HBOT has clinical and anecdotal support. Exposure to lower concentrations can result in eye irritation, a sore throat and cough, nausea, shortness of breath, and fluid in the lungs (pulmonary edema). These effects are believed to be due to the fact that hydrogen sulfide combines with alkali present in moist surface tissues to form sodium sulfide, a caustic. These symptoms usually go away in a few weeks. Long-term, low-level exposure may result in fatigue, loss of appetite, headaches, irritability, poor memory and dizziness.
Chronic exposure to low level H₂S (around 2ppm) has been implicated in increased miscarriage and reproductive health issues among Russian and Finnish wood pulp workers, but the reports have not (as of circa 1995) been replicated. Short-term, high-level exposure can induce immediate collapse, with a high probability of death. If death does not occur, studies have shown that, in humans, high exposure to hydrogen sulfide can lead to cortical pseudo laminar necrosis and degeneration of the basal ganglia. It has also been shown that Cerebral edema has been the cause of death in some fatal exposures with hydrogen sulfide.

- 0.00047ppm or 0.47ppb is the odor threshold, the point at which 50% of a human panel can detect the presence of the compound.

- 0.0047ppm is the recognition threshold, the concentration at which 50% of humans can detect the characteristic odor of hydrogen sulfide, normally described as resembling "a rotten egg".

- OSHA has established a permissible exposure limit (PEL) (8hour time-weighted average (TWA)) of 10ppm.

- 10–20ppm is the borderline concentration for eye irritation.

- 20ppm is the acceptable ceiling concentration established by OSHA.

- 50ppm is the acceptable maximum peak above the ceiling concentration for an 8hour shift, with a maximum duration of 10minutes.

- 50–100ppm leads to eye damage.
At 100–150ppm the olfactory nerve is paralyzed after a few inhalations, and the sense of smell disappears, often together with awareness of danger.

320–530ppm leads to pulmonary edema with the possibility of death.

530–1000ppm causes strong stimulation of the central nervous system and rapid breathing, leading to loss of breathing.

800ppm is the lethal concentration for 50% of humans for 5 minutes exposure (LC50).

Concentrations over 1000ppm cause immediate collapse with loss of breathing, even after inhalation of a single breath. Cortical pseudo laminar necrosis, degeneration of the basal ganglia and cerebral edema have also been shown.

Although respiratory paralysis may be immediate, it can also be delayed up to 72 hours.

Adamsite or DM is an organic compound; technically, an arsenical diphenylaminechlorarsine, that can be used as a riot control agent. DM belongs to the group of chemical warfare agents known as vomiting agents or sneeze gases. First synthesized in Germany by Heinrich Otto Wieland in 1915, it was independently developed by the US chemist Roger Adams (for whom it is named) at the University of Illinois in 1918.

DM was produced and stockpiled by the United States at the end of World War I, but not deployed on the battlefield then. It was used against the Bonus Army who demonstrated in Washington, DC, in 1932, reportedly causing the death and serious injury of several children who had accompanied their parents on the protests. It was again used in the Vietnam War. DM is an odourless crystalline compound with a very low vapour pressure. The colour of the crystals ranges from bright yellow to dark green depending on the purity. It is readily soluble in some organic solvents (e.g., acetone, dichloromethane), but nearly insoluble in water. In vaporous form it appears as a canary yellow smoke. Adamsite is usually dispersed as an aerosol, making the upper respiratory tract the primary site of action. Although the effects are similar to those caused by typical riot control agents (e.g. CS), they are slower in onset and longer in duration, often lasting several hours. After a latency period of 5–10 minutes irritation of the eyes, lungs and mucous membranes develops followed by headache, nausea and persistent vomiting. DM is now regarded as obsolete. It has been widely replaced by riot control agents such as CS which...
are less toxic and more rapid in onset of symptoms. Early battlefield use was intended to be via "Adamsite candles". These were large metal cans or tubes (weighing approximately 5 pounds) which contained a smoke composition made of Adamsite plus a slow burning pyrotechnic composition. A series of candles were lit and the Adamsite-laden smoke allowed to drift towards the enemy. In 2003, North Korea was reportedly producing Adamsite at its Aoji-ri Chemical Complex for stockpiling.\(^{21}\)

**Benzyl chloride**, or α-chlorotoluene, is an organic compound with the formula \(\text{C}_6\text{H}_5\text{CH}_2\text{Cl}\). This colourless liquid is a reactive organ chlorine compound that is a widely used chemical building block. Benzyl chloride is an alkylating agent. Indicative of its high reactivity (relative to alkyl chlorides), benzyl chloride reacts with water in a hydrolysis reaction to form benzyl alcohol and hydrochloric acid. Since benzyl chloride is quite volatile at room temperature, it can easily reach the mucous membranes where the hydrolysis takes place with production of hydrochloric acid. This explains why benzyl chloride is a lachrymator and has been used as a war gas. It is also very irritating to the skin.\(^{22}\)

The **sulfur mustards**, or **sulphur mustards**, \((\text{CICH}_2\text{CH}_2\text{SCH}_2\text{CH}_2\text{Cl})\) commonly known as **mustard gas**, are a class of related cytotoxic and vesicant chemical warfare agents with the ability to form large blisters on the exposed skin and in the lungs. Pure sulfur mustards are colorless, viscous liquids at room temperature. When used in impure form, such as warfare agents, they are usually yellow-brown in color and have an odor resembling mustard plants, garlic, or horseradish, hence the name. Mustard gas was originally assigned the name **LOST**, after the scientists Wilhelm Lommel and Wilhelm Steinkopf, who developed a method for the large-scale production of mustard gas for the Imperial German Army in 1916. Mustard agents are regulated under the 1993 Chemical Weapons Convention (CWC). Three classes of chemicals are monitored under this Convention, with sulfur and nitrogen mustard grouped in Schedule 1, as substances with no use other than in chemical warfare. Mustard agents could be deployed on the battlefield by means of artillery shells, aerial bombs, rockets, or by spraying from warplanes. The compound readily eliminates a chloride ion by intramolecular nucleophilic substitution to form a cyclic sulfonium ion. This very reactive intermediate tends to permanently alkylate the guanine nucleotide in DNA strands, which prevents cellular division and generally leads directly to programmed cell death, or, if cell death is not immediate, the damaged DNA may lead to the development of cancer. Oxidative stress would be another pathology involved in sulfur mustard toxicity. Sulfur mustard is not very soluble in water but is very soluble in fat, contributing to its rapid absorption into the skin. In the wider sense, compounds with the structural element \(\text{BCH}_2\text{CH}_2\text{X}\), where \(X\) is any leaving group and \(B\) is a Lewis base are known as **mustards**. Such compounds can form cyclic "onium" ions (sulfonium, ammoniums, etc.) that are good alkylating agents. Examples are bis(2-chloroethyl)ether, the (2-
haloethyl)amines (nitrogen mustards), and sulfur sesquimustard, which has two α-chloroethyl thioether groups \((\text{ClH}_2\text{C-CH}_2\text{-S-})\) connected by an ethylene \((-\text{CH}_2\text{CH}_2\-)\) group. These compounds have a similar ability to alkylate DNA, but their physical properties, e.g. melting point, vary.

![Figure-9: Toxic effect of sulfur mustard gas](image)

Mustard gas has extremely powerful vesicant effects on its victims. In addition, it is strongly mutagenic and carcinogenic, due to its alkylating properties. It is also lipophilic. Because people exposed to mustard gas rarely suffer immediate symptoms, and mustard-contaminated areas may appear completely normal, victims can unknowingly receive high dosages. Within 24 hours of exposure to mustard agent, victims experience intense itching and skin irritation, which gradually turns into large blisters filled with yellow fluid wherever the mustard agent contacted the skin. These are chemical burns and are very debilitating. Mustard gas vapour easily penetrates clothing fabrics such as wool or cotton, so it is not only the exposed skin of victims that gets burned. If the victim's eyes were exposed then they become sore, starting with conjunctivitis, after which the eyelids swell, resulting in temporary blindness. Miosis may also occur, which is probably the result from the cholinomimetic activity of mustard. At very high concentrations, if inhaled, mustard agent causes bleeding and blistering within the respiratory system, damaging mucous membranes and causing pulmonary edema. Depending on the level of contamination, mustard gas burns can vary between first and second degree burns, though they can also be very bit as severe, disfiguring and dangerous as third degree burns. Severe mustard gas burns (i.e. where more than 50% of the victim's skin has been burned) are often fatal, with death occurring after some days or even weeks have passed. Mild or moderate exposure to mustard agent is unlikely to kill, though victims require lengthy periods of medical treatment and convalescence before recovery is complete. The mutagenic and carcinogenic effects of mustard agent mean that victims who recover from mustard gas burns have an increased risk of developing cancer in later life. Typical appearance of bullae on arm caused by blister agent burns. Skin damage can be reduced if povidone-iodine in a base of glycofurol is
rapidly applied, but since mustard agent initially has no symptoms, exposure is usually not recognised until skin irritation begins, at which point it is too late for countermeasures. The vesicant property of mustard gas can be neutralised by oxidation or chlorination, using household bleach (sodium hypochlorite), or by nucleophilic attack using e.g. decontamination solution "DS2" (2% NaOH, 70% diethylenetriamine, 28% ethylene glycol monomethyl ether). After initial decontamination of the victim's wounds is complete, medical treatment is similar to that required by any conventional burn. The amount of pain and discomfort suffered by the victim is comparable as well. Mustard gas burns heal slowly, and, as with other types of burn, there is a risk of sepsis caused by pathogens such as *Staphylococcus aureus* and *Pseudomonas aeruginosa*. A British nurse treating soldiers with mustard gas burns during World War I commented: They cannot be bandaged or touched. We cover them with a tent of propped-up sheets. Gas burns must be agonizing because usually the other cases do not complain, even with the worst wounds, but gas cases are invariably beyond endurance and they cannot help crying out. \(^{23}\)

**Bis(chloromethyl) ether** (ClCH\(_2\)OCH\(_2\)Cl) is carcinogenic. It is one of 13 chemicals considered an OSHA-regulated occupational carcinogen. Chronic exposure can increase the incidence of oat cell carcinoma, a type of lung cancer.

**Ethyldichloroarsine**, sometimes abbreviated "ED", is an organoarsenic compound with the formula CH\(_3\)CH\(_2\)AsCl\(_2\). This colourless volatile liquid is a highly toxic obsolete vesicant or blister agent that was used during World War I in chemical warfare. The molecule is pyramidal with the Cl-As-Cl and C-As-Cl angles approaching 90°. Its toxic action is similar to lewisite. \(^{24}\)

**Blue Cross (Blaukreuz)** is a World War I chemical warfare agent consisting of diphenylchloroarsine (DA, Clark I), diphenylcyaanoarsine (CDA, Clark II), ethyldichloroarsine (Dick), and/or methyl dichloroarsine (Methyldick). Clark I and Clark II were the main agents used. Clark I was used with Green Cross munition earlier; however for the first time it was used as a standalone agent in the night from July 10 to July 11 1917 at Nieuwpoort, Belgium, during the operation Strandfest. The artillery munition used as a delivery vehicle contained a large amount of glass spheres closed with a cork and sealed with trinitrotoluene. Later N-ethylcarbazole was added. Depending on the caliber, the munition contained between 7 and 120 kilograms of the agent. Blue Cross is also a generic World War I German marking for artillery shells with chemical payload affecting the upper respiratory tract.

**Green Cross (Grünkreuz)** is a World War I chemical warfare pulmonary agent consisting of chloropicrin (PS, Aquinite, Klop), phosgene (CG, Collongite) and/or trichloromethyl chloroformate (Surpalite, Perstoff). Green Cross is also a generic World War I German marking for artillery shells with pulmonary agents (chemical payload affecting the lungs). The tip of the
projectile with the fuse end painted green and a green cross at the bottom of the cartridge. Other Green Cross mixtures were based on phosgene and/or diphosgene. The first use of Green Cross was on May 31, 1915 in a German offensive in Ypres. The mixture was chlorine-phosgene, with 95% and 5%.

Yellow Cross (Gelbkreuz) is a World War I chemical warfare agent usually based on sulfur mustard (HS, Yperite, Lost). The original Gelbkreuz was a composition of 80-90% of sulfur mustard and 10-20% of tetra chloromethane or chlorobenzene as a solvent which lowered its viscosity and acted as an antifreeze, or, alternatively, 80% sulfur mustard, 10% bis(chloromethyl) ether, and 10% tetra chloromethane. A later formulation, Gelbkreuz 1, was a mixture of 40% ethyldichloroarsine, 40% ethyldibromoarsine, and 20% of bis(chloromethyl) ether. In some cases nitrobenzene was used to mask the material's characteristic odor. French "ypérite no.20" was a similar mixture of 80% sulfur mustard and 20% tetra chloromethane. Yellow Cross is also a generic World War I German marking for artillery shells with chemical payload affecting exposed surfaces of the body.

White Cross (Weisskreuz) is a World War I chemical warfare agent consisting of one or more lachrymatory agents: bromoacetone (BA), bromobenzyl cyanide (Camite), bromomethyl ethyl ketone (homomartonite, Bn-stoff), chloroacetone (Tonite, A-stoff), ethyl bromoacetate, and/or xylol bromide. During World War I, White Cross was also a generic code name used by the German Army for artillery shells with an irritant chemical payload affecting the eyes and mucous membranes.25

CONCLUSION

Chemical warfare (CW) involves using the toxic properties of chemical substances as weapons. This type of warfare is distinct from nuclear warfare and biological warfare, which together make up NBC, the military acronym for nuclear, biological, and chemical (warfare or weapons), all of which are considered "weapons of mass destruction" (WMDs). None of these fall under the term conventional weapons which are primarily effective due to their destructive potential. With proper protective equipment, training, and decontamination measures, the primary effects of chemical weapons can be overcome. Many nations possess vast stockpiles of weaponized agents in preparation for wartime use. The threat and the perceived threat have become strategic tools in planning both measures and counter-measures. A chemical weapon (CW) is a device that uses chemicals formulated to inflict death or harm on human beings. They are classified as weapons of mass destruction though they are separate from biological weapons (diseases), nuclear weapons, and radiological weapons (which use radioactive decay of elements). Chemical weapons can be widely dispersed in gas, liquid and solid forms and may easily afflict others than the intended targets. Nerve gas, tear gas and pepper spray are three
modern examples. Lethal, unitary, chemical agents and munitions are extremely volatile and they constitute a class of hazardous chemical weapons that are now being stockpiled by many nations. (Unitary agents are effective on their own and require no mixing with other agents.) The most dangerous of these are nerve agents GA, GB, and VX, and vesicant (blister) agents which are formulations of sulfur mustard such as H, HT, and HD. All are liquids at normal room temperature, but become gaseous when released. Widely used during the First World War, the effects of so-called mustard gas, phosgene gas and others caused lung searing, blindness, death and maiming. Pepper spray is of common use today. It is potentially lethal. There are no recent records of pepper spray being used in war despite the fact that it inflicts fewer injuries and has less side-effect if compared with impact and explosive weapons.

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