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Kimyasal Savaş Ajanları: Özellikleri, Etkileri ve Dekontaminasyon Stratejileri

Chemical Warfare Agents: Characteristics, Effects, and Decontamination Strategies

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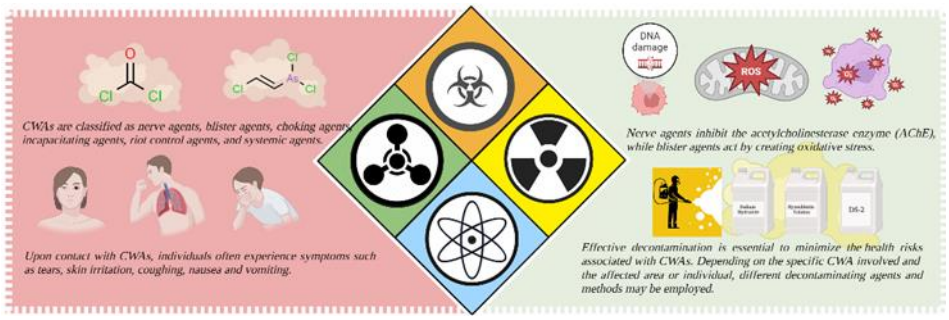
Önemli Noktalar

Chemical warfare agents (CWAs) encompass a broad array of chemicals strategically employed. A comprehensive understanding of chemical warfare agents and their corresponding decontamination protocols is critical.

Grafiksel Özet



Özet

Kimyasal savaş ajanları (KSA'lar), insanları etkisiz hale getirmek, yaralamak ve öldürmek, gıda kaynaklarını kirletmek ve yok etmek, kaos ve paniğe neden olmak için kullanılan tüm kimyasalları kapsar. KSA'lar çok eski zamanlardan beri kullanılmaktadır ancak en yoğun kullanımı I. Dünya Savaşı sırasında olmuştur. Kimyasal Silahlar Konvansiyonu'na göre, KSA'ların savaş alanında kullanımı yasaktır, ancak bu ajanların endüstriyel kullanımı tamamen yasaklanamaz. Bu nedenle KSA'lar her zaman ve her durumda panik yaratırlar. KSA'lar sinir ajanları, yakıcı ajanlar, boğucu ajanlar, kapasite bozucu ajanlar, isyan kontrol ajanları ve sistemik ajanlar olarak sınıflandırılır. Bu ajanların mekanizmaları birbirinden farklıdır; sinir ajanları asetilkolinesteraz enzimini (AChE) inhibe ederken, yakıcı ajanlar oksidatif stres yaratarak etki gösterir. Bu makalede KSA'nın tarihsel arka planı, insanlar ve çevre üzerindeki etkileri, etkili bir şekilde temizlenme stratejileri ve geleceğe yönelik olası çıkarımlar tartışılmaktadır. Sonuç olarak, KSA'ların ve bunlara karşılık gelen dekontaminasyon protokollerinin kapsamlı bir şekilde anlaşılması, KSA olayları durumunda insan yaşamının ve çevrenin korunması açısından kritik öneme sahiptir. Hazırlıklı olma ve etkili dekontaminasyon prosedürleri küresel güvenliği sürdürmenin temel taşını oluşturur.

Abstract

Chemical warfare agents (CWAs) encompass all chemicals used to incapacitate, injure, and kill people, contaminate, and destroy food sources, and create chaos and panic. Although CWAs have been used since ancient times, their most intense use was during World War I. According to the Chemical Weapons Convention, the use of CWAs on the battlefield is prohibited, but their industrial use cannot be completely banned. Therefore, CWAs always create panic in any situation. CWAs are classified as nerve agents, blister agents, choking agents, incapacitating agents, riot control agents, and systemic agents. These agents have different mechanisms; nerve agents inhibit the acetylcholinesterase enzyme (AChE), while blister agents act by creating oxidative stress. This review discusses the historical background of CWAs, their effects on humans and the environment, effective decontamination strategies, and possible implications for the future. Understanding CWAs and the corresponding decontamination protocols comprehensively is critical for protecting human life and the environment in CWA incidents. Preparedness and effective decontamination procedures are the maintaining global security.

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1. INTRODUCTION

The term CBRN, an acronym representing Chemical, Biological, Radiological, and Nuclear hazards, encapsulates a spectrum of risky scenarios with profound implications for human society and the natural environment. The increasing prevalence of CBRN risks is inextricably linked to advancements in modern warfare and industrial development. Furthermore, the deliberate application of CBRN materials in acts of terrorism, alongside their employment in industrial settings and research laboratories within the healthcare sector, inadvertently precipitates significant threats. The repercussions of these threats can manifest as the loss of countless lives and grievous injuries, often instigating widespread panic and chaos among the public. The management of CBRN events invariably demands meticulous attention and resources, accentuating the indispensability of personal protective equipment and rigorous decontamination protocols. In the annals of warfare, the deployment of chemical weapons has been historically classified as "chemical warfare." These insidious agents are meticulously crafted to induce both physical and psychological harm by harnessing the toxic attributes of chemical substances. Remarkably cost-effective and resilient to external factors, chemical weapons represent an ominous specter, their ease of development enabling a greater reach of devastation than initially anticipated. The use of chemical weapons gained prominence during and post-World War I, leading to the formulation of countermeasures referred to as "chemical defense." The use of chemical weapons came to the forefront during World War I and led to the taking of countermeasures called “chemical defense”. The First World War demonstrated the deadly and destructive nature of the use of chemicals in modern war. At the beginning of the war, the gas bombs used by the French were not very effective and this continued with bullets filled with tear gas by the Germans. During the war, the Germans continued to produce new bullets filled with chloro-sulfate using powerful chemical industries [1]. These bullets were used against the British in Neuve-Chapelle in October 1914 but had little influence [2]. These inadequate initiatives at the beginning of the war did not prevent the development of new agents on both fronts. The Germans attacked Ypres, Belgium in April 1915 with 168 tons of chlorine gas [3]. In the autumn of 1915, the use of chlorine and phosgene caused serious respiratory problems in soldiers who were exposed. However, because of the gas masks developed to reduce the effects of chlorine and phosgene, the Germans turned to mustard gas to use against the British [4]. Mustard gas was different from other gases, remained in the region for a long time, and exposure was more concerning. In addition, although 100 years have passed since the use of mustard gas on the battlefield, there is still no effective treatment, and research to develop therapeutic compounds continues [5]. In the First World War, chemical casualties were quite high, and it is estimated that approximately 1.2 million soldiers were injured and over 91.000 people died due to the use of chemical agents [6]. The Treaty of Versailles (1919), forbade Germans from producing and using chemical weapons [7]. With the Geneva Protocol signed in 1925 with the approval of 16 countries, the use of chemical

agents was completely banned in the war. The fate of chemical wars changed with the discovery of Germans' organophosphorus-based nerve agents before the Second World War [8]. In 1936, a very toxic organophosphate compound called Tabun was developed by Gerhard Schrader. Thus, Tabun became the first member of nerve agents. Later, Schrader and the research team discovered another organophosphate compound, which is like the tabun, but was more deadly than the tabun. In 1943, German planes sank several American ships on the coast of Italy, and the presence of mustard gas to be used as retaliation on ships was noticed. Throughout the 1950s and 1960s, advances have been made to include sarin and VX in the production and distribution of chemical weapons [9]. While the work on protective masks continued, the concern of not being able to detect nerve agents increased again. In the mid-1950s, major progress was made in the therapeutic of agents inhibiting acetylcholinesterase enzyme, and atropine was developed as an automatic injector [10]. Egyptians were claimed to have used mustard gas and nervous agents in the civil war in Yemen. Thus, this claim was the first use of a nervous agent reported in armed clashes. During the Vietnam War, the United States used tear gas [11]. The Soviet Union was accused of using chemicals in wars in Afghanistan. In addition, during the Jewish Genocide, the Nazis used the medication of insecticide containing carbon monoxide and hydrogen cyanide known as Zyklon-B to kill millions of people in their camps [12]. In 1943, during the Warsaw Ghetto uprising, poisonous gases were used against the Jews. In the 1980s, the use of chemical weapons during the war was once again important. In 1980, Iraq occupied Iran and used nervous and burning agents. In many ways, this war was similar to the First World War. In 1983, Iran protested Iraq's use of chemical agents, thus proving to use Iraq's mustard agent and tabun. In 1988, the Iraqi army used more than one chemical agent against the Kurdish minority in Northern Iraq (Halabja), and at the end of this incident, about 5.000 people died and many more people were influenced by chemical agents [13]. In the late 1980s, other war agents were developed by the US, such as the M40 gas mask. In addition, progress in collective protection, decontamination, and detection were recorded. In 1984, US President Ronald Reagan issued a statement that called for an international prohibition of chemical weapons. In 1990, President George H.W. Bush and Soviet Union leader Mikhail Gorbachev signed an agreement that prohibited the production of chemical weapons and started the destruction of stocks of both countries. In 1993, the Convention on Chemical Weapons was convened and signed. As of 2008, the majority of the United Nations member states participated in the Chemical Weapons Convention [14]. In 1990, the Iraqi army invaded its neighbor Kuwait. Although chemical war agent was not used on the battlefield, these gases spread to the environment as a result of the destruction of 8.5 tons of sarin and cyclosarin chemicals and rockets where some nerve agents were stored. Thus, many soldiers were influenced by this situation and their long-term effects have continued to the present day. This was called “Gulf War Syndrome” [15].

The potential use of chemical agents by terrorists has become a worldwide concern. The attractiveness of these agents to terrorists is because most of the chemical agents are cheap, and their production and transport are relatively easy. These features made chemicals an ideal weapon to create terrorism. In 1994, members of the Japanese religious sect, known as Aum Shinrikyo, carried out several attacks using sarin. The so-called Matsumoto and Tokyo Metro event includes housing and subway exposure [16]. A total of 19 people were killed and more than 6.000 people needed medical assistance. In the 21st century, chemical war agents re-emerged as contemporary threats. In the fall of 2006, Al Qaeda and the relevant groups used chlorine to spread panic in Iraq [17]. These attacks followed similar attacks in the following months. In August 2013, chemical weapons attacks occurred in the Guta region of Syria. In this case, many people were influenced by chemical agents.

Today, many countries and many different terrorists have chemical war agents. If there are legitimate uses for chemical substances in our society, the risk of conflict and terrorism of chemical agents will always be present. Worldwide research is continuing for better detection, protection, and treatment of chemical war agents. Many countries have signed various agreements to limit the use and production of chemical war agents, but terrorist organizations are not under such restrictions. In addition, the effects of chemical war agents can be seen because of accidents in addition to deliberate use. Accidents during the production or distribution of such substances result in a negative impact on many places and creatures. For example, as a result of an accident in 1984 in the city of Bhopal (India), 40 tons of methyl isocyanate spread from a factory that produces insecticide [18]. As a result of this factory accident, more than 500.000 people were affected by the incident and thousands of people lost their lives. Accidentally exposed to chemical war agents can be exemplified in many ways in both national and international terms. There have been many toxicological accidents in the history of Türkiye. In Mersin (2011), there was poisoning because of exposure to gas leaking from the tank sold as scrap. In 2014, letters containing yellow powder sent to the Consulate General and Ankara Courthouse aroused danger. Chlorine gas leaking from the chlorine tank in Siirt in 2017 negatively affected 135 people. In Turkey, events based on real chemical terrorist incidents are rare, but due to their geographical location, CBRN events in neighboring countries also affect our country negatively. For this reason, it is very important for any exposure to know what chemical agents are by taking advantage of the past and to investigate the possible effects. Within the scope of this comprehensive review, we delve into the fundamental facets of chemical warfare agents, encompassing their diverse classifications and the intricate procedures requisite for effective decontamination.

2. CHEMICAL WARFARE AGENTS

Chemical warfare agents (CWAs) encompass a broad array of chemicals strategically employed to neutralize, injure, and lethally affect individuals, contaminate food sources, and sow chaos and panic. The deployment of CWAs disrupts economic and strategically vital targets, compelling both military and civilian personnel

to don protective clothing and utilize specialized equipment, significantly limiting their mobility. Gaseous chemical agents are favored in CWA arsenals due to their volatile nature, which enables rapid dispersion across diverse environments. These agents can infiltrate the body through inhalation in gaseous form, absorption through the skin in solid or liquid states, or ingestion if they contaminate foodstuffs. Notably, certain chemical combinations, such as Lewisite with sulfur mustard, have been observed to alter key properties of the latter, including vapor pressure reduction, lowered freezing points, and increased stability. Additionally, sulfur mustard can be combined with phenyldichloroarsine, commonly referred to as "Winterlost" or "winter mustard" [19]. CWAs exhibit a diverse range of characteristics and are classified based on several factors, including their physiological effects, physical state (solid, liquid, or gas), toxicity, and intended use. For instance, some substances, like sulfur mustard, exist in solid form with a freezing point of 14.4°C under cold conditions but transition into a liquid state with a boiling point of 219°C in high-temperature environments [20]. Certain CWAs are colorless and odorless, posing diagnostic challenges. Moreover, the detectability of chemical agents varies, with some becoming evident within short intervals, typically 1-2 hours, while others may manifest their effects only after approximately 48 hours. The lethality of CWAs is contingent upon factors such as application methods, exposure dosage, and duration of exposure. Table 1 comprehensively overviews commonly employed chemical substances used as CWAs. CWAs are divided into six different groups according to their chemical properties and mechanism of action as shown in Table 1. Accordingly, while tabun is a nerve agent, phosgene is a choking agent. A detailed explanation of the classification of the CWas was examined in detail in the text.

Table 1: Common Chemical Warfare Agents [21].

Chemical Warfare Agent	Chemical Substance
Nerve Agents	Tabun ($C_5H_{11}N_2O_2P$)
	Sarin ($C_4H_{10}FO_2P$)
	Soman ($C_7H_{16}FO_2P$)
	VX ($C_{11}H_{26}NO_2PS$)
Blister Agent	Sulfur mustard ($C_4H_8Cl_2S$)
	Nitrogen mustard ($C_5H_{11}Cl_2NO$)
	Lewisite ($C_2H_2AsCl_3$)
Choking Agent	Chlorine (Cl_2)
	Chloropicrin (CCl_3NO_2)
	Phosgene ($COCl_2$)
	Diphosgene ($C_2Cl_4O_2$)
Capacity Disrupting Agents (Incapacitating)	BZ ($C_{21}H_{23}NO_3$)
	LSD ($C_{20}H_{25}N_3O$)
Riot Control Agents	Chloroacetophenone (C_8H_7ClO)
	Chlorobenzylidenemalononitrile ($C_{10}H_5ClN_2$)
	Dibenzoxazepine ($C_{13}H_9NO$)
Systemic Poisons (Asphyxiant/Blood)	Hydrogen Chloride (HCl)
	Hydrogen Cyanide (HCN)
	Hydrogen Sulfide (H_2S)

2.1. Nerve Agents

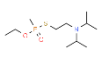
Nerve agents are notorious for their ability to disrupt the functioning of the nervous system, often leading to paralysis or even death. These agents do not occur naturally but are derived from organophosphate (OP) compounds, functioning as potent inhibitors of the acetylcholinesterase enzyme [22]. Remarkably, cholinesterase inhibitors, which are instrumental in treating certain diseases and controlling pests, can also be harnessed as terrorist weapons [21]. Nerve agents are categorized by NATO codes, succinct two-

letter designations, with two principal series, V series and G series agents. The G series, representing the origin of "Germany," encompasses GB (sarin), GD (Soman), and GA (Tabun). On the other hand, the V Series, possibly denoting "Venomous," includes VE, VG, VM, and VX [23]. Though all nerve agents share common traits of being liquid and colorless, impurities can impart a yellow or brown hue to these substances. The physical properties of various nerve agents can markedly differ from one another (see Table 2). VX, characterized by lower volatility compared to G agents, exhibits enhanced stability. In terms of volatility among G agents, the order stands as GB > GD > GA [24].

Exposure to nerve agents can result in a spectrum of physiological effects, including headaches, profuse sweating, impaired balance, respiratory distress, nausea, and vomiting. Additionally, as depicted in Figure 1a, nerve agents can impact the brain, leading to loss of consciousness, coma, and eye constriction, affect the lungs by causing respiratory difficulties, influence blood pressure, and alter respiratory rates. These agents obstruct the nervous system by inhibiting the acetylcholinesterase enzyme (AChE) within the body [25, 26]. By binding to acetylcholinesterase, nerve agents disrupt signal transmission, as acetylcholine breakdown at nerve transmission points is impeded (see Figure 1b) [27]. Acetylcholine is typically hydrolyzed into choline and acetate [28], resulting in the accumulation of acetylcholine, which can trigger a cholinergic crisis—a state characterized by overstimulation of end organs. It's worth noting that VX, unlike other agents, has the potential to induce acute lung injury [21].

Atropine, pralidoxime chloride, and diazepam therapeutics are used under the exposure of nerve agents [29]. Since atropine is effective against all nerve agents, it is one of the most important treatment methods and should be used immediately in case of exposure. Atropine competes with AChE and prevents AChE accumulation. Oximes should be applied in combination with atropine because they are quickly excreted from the body. Diazepam should be used to reduce convulsions against brain damage caused by nerve agents. There are no prophylactic antidots that can be used before exposure to the nerve agent. The most effective treatment is the application of atropine and pralidoxime chloride as automatic injectors. Pyridostigmine is approved for military use by the FDA as a pretreatment for GD but requires rapid use with atropine and pralidoxime (2-PAM) after exposure to the chemical agent to be effective [30]. In addition to antimuscarinic drugs, reactivators such as HI-6 [31], obidoxime (OBI), and NOX-6 can be used to treat airway shrinkage after exposure to organophosphate compounds [32]. Another alternative approach is to use non-oxime compounds such as Bispyridinium MB327, which reverses the neuromuscular blocking effect of therapeutic OP compounds [33].

Table 2: Physical And Chemical Properties Of Common Nerve Agents [28].

Property	GB	GD	GA	VX
Chemical Structure				
Common name	Sarin	Soman	Tabun	VX

Chemical name	Isopropyl methyl-phosphonic-fluoridate	Pinacolyl methyl-phosphonic-fluoridate	Ethyl dimethyl amido pyrophosphate	*S*-(2-diisopropyl aminoethyl) *O*-ethyl methyl phosphorothioate
Molecular weight, MW (Da)	140.10	182.18	162.13	267.38
Specific gravity at 25°C	1.009	1.022	1.073	1.008
Boiling point °C	147	167	246	300
Melting point °C	-56	-80	-49	-20

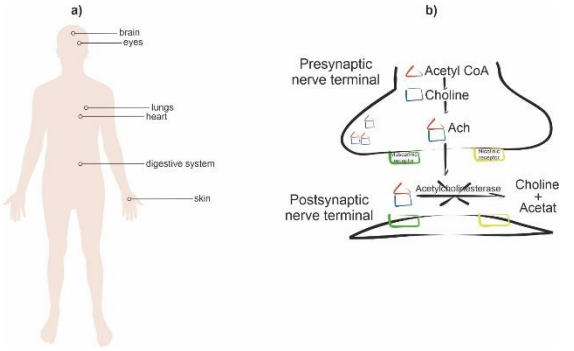


Figure 1: a) Organs Or Systems In The Body That Nerve Agents Affect. **b)** The Mechanism Of Action Of Acetylcholinesterase.

2.1.1. Sarin (GB): An Artificial Chemical Warfare Agent

Sarin, commonly known as "GB," is a synthetic chemical warfare agent with a unique historical origin. Initially developed as an insecticide in Germany in 1938, its transformation into a deadly weapon marked a significant turning point in its history. In its pure form, Sarin exists as a liquid that is entirely devoid of odor, color, and taste [34]. However, it's worth noting that Sarin possesses the ability to evaporate into the environment. Notably, Sarin stands out as the most volatile among nerve agents, characterized by its rapid evaporation and dispersion into the atmosphere. The intoxicating effects of Sarin are contingent on several factors, including the amount of exposure, the manner of exposure, and the duration of exposure. Fortunately, antidotes are available for Sarin poisoning and are most effective when administered promptly. The deployment of Sarin for terrorist purposes gained notoriety with incidents occurring in Japan in 1994 and 1995 [35]. These events served as grim reminders of the potent threat posed by this chemical agent and underscored the importance of vigilance and preparedness in dealing with such hazards.

2.1.2. Soman (GD): An Odoriferous Artificial Toxin

Soman, also known as "GD," is a colorless and tasteless artificial toxin with an odor similar to mothballs or rotten fruit. Soman was first discovered in Germany as an insecticide in 1944. Soman is normally liquid but evaporates when heated. Soman is highly volatile. However, its severe exposure is dangerous enough to result in death [36]. It is known that soman or other nerve agents were used in the Iran-Iraq Chemical War in the 1980s. It is known that soman or other nerve agents were used in the Iran-Iraq Chemical War in the 1980s.

2.1.3. Tabun (GA): The Silent Threat from Germany

Tabun, also recognized as "GA," emerged as a significant chemical compound initially developed as a pesticide in Germany in 1936. Unlike its menacing effects, Tabun is nearly imperceptible to the senses. It presents as a tasteless toxin with a subtly fruity fragrance. The

degree of its purity determines its appearance, transitioning from a colorless liquid to a brownish hue. Tabun's peril lies in its capacity to disrupt neural synapses. Even minute doses of Tabun prove lethally toxic, capable of inducing fatality [35]. Its mechanism of action involves the overstimulation of the nervous system, culminating in organ damage. Those exposed to Tabun are at risk of experiencing permanent neurological impairment. Similar to other nerve agents, Tabun can evaporate when subjected to heat. In terms of volatility, Tabun falls between Sarin and VX, being less volatile than Sarin but more so than VX. Its relatively higher density causes it to accumulate in lower areas, further amplifying its hazardous potential. Tabun found infamous application alongside other nerve agents during the Iran-Iraq War in the 1980s, underscoring the grave repercussions of its deployment in conflict scenarios [37].

2.1.4. VX: The Stealthy Lethal Agent from the UK

VX, an acronym that does not belie its ominous nature, is an insidious chemical developed in the United Kingdom during the 1950s. It is characterized by being completely devoid of odor and taste, rendering it exceptionally discreet as a toxic substance. VX presents itself as an oily liquid that exhibits an unusual trait—it is exceedingly slow to evaporate. VX, perhaps the most perilous of all nerve agents, poses an exceptional threat. One distinctive feature is its protracted breakdown within the human body, giving rise to a cumulative effect upon repeated exposures [38, 39]. Notably, VX's exceptionally slow evaporation contributes to the creation of enduring hazards on contaminated surfaces, making thorough decontamination a critical priority [24]. The sinister history of VX reveals its sole application in chemical warfare, with documented use during the Iran-Iraq War in the 1980s. This exclusive deployment underscores its extraordinary lethality and its resolute place in the annals of chemical warfare agents.

2.2. Blister Agent: Skin and Respiratory Hazards

The damage inflicted by blistering agents, or vesicants, on the skin, bears a resemblance to injuries caused by burns. These noxious substances not only impact the skin but also wreak havoc on the upper respiratory tract and lungs, often leading to the development of pulmonary edema. When these agents come into contact with the skin, they induce burning and swelling. Their harmful effects extend beyond the skin, affecting the eyes, lungs, mucous membranes, and blood-forming organs. Inhalation of blister agents damages the respiratory system, while ingestion can result in symptoms such as vomiting and diarrhea. Although these injuries are typically non-lethal, they tend to heal slowly.

Blister agents encompass several compounds, including arsenic, sulfur mustard (commonly known as mustard gas), nitrogen mustards (HN1, HN2, and HN3), and Lewisite (L1, L2, and L3). Among them, sulfur mustard stands out as one of the most potent blister agents. Pure sulfur mustard has a distinct garlic-like odor and is a colorless liquid. It readily dissolves in organic solvents but exhibits limited solubility in water. Importantly, it has low volatility. Although more resilient than sulfur mustards, nitrogen mustards are comparatively less stable during storage [40,

41]. A common feature shared by blister agents is their ability to induce oxidative stress. Oxidation of thiol groups is ubiquitous in all blister agents due to oxidative stress [42, 43]. This oxidative stress disrupts the redox balance, which governs critical processes such as oxidant production, apoptosis, and the activation of redox-related transcription factors. Additionally, oxidative stress inflicts damage upon cellular organelles and weakens antioxidant defenses, culminating in either necrosis [44] or apoptosis [45].

There is no specific antidote used to treat mustard, but their general treatment is like burn injuries. Blepharospasm can be relieved with an atropine solution (1%), and eye drops such as ciprofloxacillin can be used. Additionally, systemic analgesics and antihistamines can be used to relieve itching and pain. However, 2,3-dithiocaptopropanol, known as British Anti Lewisite (BAL), is used against lewisite poisoning and is a therapeutically powerful anaerobic glycolysis inhibitor [46].

2.2.1. Sulfur mustard: A Pungent Menace with Lingering Effects

Sulfur mustard, often referred to by a variety of names such as mustard gas, mustard agent, H, HT, and HD, is a chemical compound that merits careful consideration. While the term "mustard gas" is commonly used, it's important to note that sulfur mustard is, in fact, a liquid under normal conditions. This potent substance is unmistakably characterized by its pungent aroma, akin to garlic, onion, or mustard. In its liquid or solid form, sulfur mustard exhibits a distinct color spectrum, ranging from clear yellow to brown. Its remarkable permanence in the environment is owed to its oily composition and limited solubility in water. Even at low concentrations, sulfur mustard poses a severe threat to the eyes and skin, possibly inflicting substantial damage. Due to its lipophilic nature, sulfur mustard possesses a swift penetration capability into tissues and cells, effectively corroding these vital structures. The initial chemical reaction involves intramolecular cyclization, culminating in the formation of an electrophilic ethylene sulfonium intermediate. This intermediate can bind to a multitude of biological molecules, including sulfhydryl, carboxyl, aliphatic amino groups, and heterocyclic nitrogen atoms [47, 48]. This process results in alkylation and cross-linking of critical biomolecules such as nucleic acids, proteins, lipids, and various membrane components [41, 49]. Notably, sulfur mustard induces DNA damage and hampers the production of blood cells. Moreover, it exhibits a pronounced affinity for the epithelial layers of the cornea and lungs, eliciting biochemical changes such as apoptosis or necrosis [50, 51]. Sulfur mustard initiates an inflammatory response in the tissues surrounding the eyes and leads to skin reddening. In the absence of prompt decontamination, the affected red skin progresses to ulceration, transforming into watery boils within a relatively short timeframe, typically 4 to 6 hours [52].

2.2.2. Nitrogen Mustard: Versatile Compounds with Dual Roles

Nitrogen mustards, akin to sulfur mustards, belong to the category of blister agents and possess a rich history that dates back to the 1920s when they were first explored as potential chemical warfare agents. These compounds typically present as oily liquids but can also

manifest as solids and gases. Nitrogen mustard is known for its amber or yellow appearance and is characterized by its versatility, with three primary variants: HN-1, HN-2, and HN-3. HN-1, originally developed by the German and Czech pharmaceutical industries, initially served as a pharmaceutical agent intended for the treatment of malignant tumors [52]. Subsequently, it found a darker application as a chemical warfare agent. In contrast, HN-2, designed as a warfare agent, transformed and was later repurposed as an antineoplastic agent. HN-3, developed with military intent, continues to be employed as a chemical warfare agent.

Notably, nitrogen mustards are DNA alkylating agents capable of inducing cross-linking between guanine N-7 atoms on different DNA strands. This cross-linking is irreversible and triggers a process known as apoptosis, leading to programmed cell death [53]. Due to these distinctive properties, nitrogen mustard derivatives have earned recognition in cancer prevention. Several of these derivatives, such as cyclophosphamide, chlorambucil, and melphalan, have secured approval from the US FDA and are deployed in cancer therapy [54]. This dual role—first as chemical warfare agents and then as vital tools in cancer treatment—illustrates nitrogen mustards' complex history and versatility. Lewisite: The Silent Menace with a Geranium-like Odor

Lewisite, known by its military designation "L," contains arsenic and is infamous for its potent toxicity, particularly to the skin. In its pure form, Lewisite is a colorless and odorless oily liquid accompanied by a geranium-like scent. This insidious substance readily dissolves in organic solvents, underscoring its dangerous nature. Exposure to pure Lewisite can lead to severe consequences, including blindness, systemic blood poisoning, and destructive effects on lung tissues. Remarkably, despite its development as a chemical warfare agent, Lewisite was not deployed on the battlefield. This choice may be attributed to its heightened reactivity compared to mustard gas. Lewisite's toxic effects can be devastating and may culminate in death due to severe fluid loss and systemic toxicity resulting from capillary leakage—a condition recognized as "Lewisite shock" [55]. This toxic substance inflicts damage on various organs, including the heart, lungs, liver, kidneys, and gastrointestinal system. Furthermore, Lewisite has been implicated in causing damage to multiple organs [56]. The toxicity of Lewisite stems from its ability to inhibit pyruvate oxidation and its tendency to bind to sulfhydryl-containing proteins or enzymes, leading to cell death. Moreover, Lewisite can induce DNA damage and promote carcinogenic processes [56]. These multifaceted detrimental effects underscore the peril of Lewisite exposure in terms of acute and long-term health consequences. Choking Agents: Unleashing Havoc on the Respiratory System

Choking agents, aptly named for their ability to assail the lungs and induce pulmonary edema, fall within the category of lung irritants. These chemical assailants inflict extensive damage on the respiratory tract, with particular emphasis on the nose, throat, and, most significantly, the lungs [57]. In severe cases, their noxious effects can lead to a harrowing outcome—fluid buildup in the lungs, culminating in oxygen deprivation and death, hence the term "choking agents." Choking

agents comprise a roster of perilous substances, including chlorine, phosgene, diphosgene, chlorpicrin, and perfluoroisobutylene (PFIB). Among them, phosgene reigns as the most menacing member of this class [58] and gained notoriety during World War I in 1915, accounting for a staggering 80% of all chemical-related fatalities. Physiologically, exposure to choking agents triggers a cascade of distressing effects, ranging from nose and throat irritation to respiratory distress, a choking sensation, cyanosis (bluish skin discoloration), nausea, vomiting, pulmonary edema (fluid buildup in the lungs), and ultimately, respiratory failure. Choking agents manifest in various forms—liquid, gas, or aerosol—all of which are exceptionally effective. Gaseous choking agents, in particular, are notorious for their capacity to irritate the respiratory tract and induce swelling in these areas. For instance, chlorine, appearing as a greenish gas, does not readily dissolve in water under normal conditions. Consequently, when chlorine is inhaled, it reacts with the water in the body to produce hypochlorous acid (HClO), which penetrates cells by interacting with proteins. On the other hand, phosgene exists as a colorless gas and reacts with water and ammonia to generate hydrochloric acid and urea, respectively [59]. Unfortunately, there exists no antidote for choking agents, making treatment a challenging endeavor. Available options often encompass oxygen therapy and the administration of high-dose steroids [60]. These measures aim to mitigate the devastating impact of choking agents on the respiratory system, underscoring the urgency of early intervention and protective measures in cases of exposure.

2.2.3. Phosgene: The Stealthy and Deadly Gas

Phosgene is a colorless gas in its natural state, exuding a faint odor reminiscent of moldy or freshly mown straw at low concentrations [61]. However, this seemingly innocuous fragrance masks a highly toxic nature that often goes unnoticed by those exposed to it. When released in liquid form into the environment, phosgene swiftly transforms into a gas that hovers close to the ground and disperses rapidly. Generally, the liquid form of phosgene is used for storage and transportation. Phosgene holds the dubious distinction of being one of the most lethal choking agents and gained infamy for its widespread use during the First World War. Beyond its malevolent history, phosgene plays a pivotal role in industrial processes, contributing to the production of pesticides, isocyanates, dyes, pharmaceuticals, and various other industrial chemicals [62] (Figure 2a). The synthesis of phosgene involves the reaction of chlorine and carbon monoxide at high temperatures in the presence of activated charcoal [63].

Exposure to phosgene initiates a sequence of adverse effects, beginning with eye irritation. Interestingly, these effects often accumulate within the body. Phosgene primarily targets lung capillaries, gaining direct access to the bloodstream and causing the lungs to fill with fluid. Even at low concentrations, inhaled phosgene induces a decrease in arterial oxygen partial pressure and bradycardia. However, the precise concentration at which phosgene exerts toxic effects varies among individuals. For instance, approximately 1 ppm of phosgene reduces respiratory volume and capacity, while 3

ppm of phosgene undergoes hydrolysis, yielding HCl and irritating the upper respiratory tract [64]. Phosgene's insidious mode of action involves reactions with amine, thiol, hydroxyl, and sulfhydryl groups found on proteins, carbohydrates, and lipids through an acylation mechanism. Consequently, excessive oxidative damage ensues, depleting glutathione stores [65, 66]. This multifaceted assault underscores the dire consequences of phosgene exposure, emphasizing the importance of preventative measures and swift medical intervention in the event of contact with this deadly gas.

2.2.4. Chlorine: A Versatile Chemical with Disinfectant Properties

Chlorine serves a vital role primarily in drinking water disinfection and the food industry, and it finds its way into various household products [67]. In its gaseous state, chlorine gas typically appears yellow or green and emits a pungent and distinctive odor. The olfactory threshold for detecting chlorine gas is remarkably low, as concentrations as low as 0.2-0.4 ppm can be easily detected thanks to its strong odor, although the perception of this odor may diminish over time. For storage and transportation, chlorine is often pressurized and cooled to transition into a liquid form [68]. In this liquid state, chlorine accumulates near the ground and can spread rapidly, rendering it highly toxic. Although not inherently flammable, chlorine gas can react with explosive substances, introducing another hazard layer. Upon contact with water, chlorine gas undergoes a chemical transformation, giving rise to hydrochloric acid (HCl) or hypochlorous acid (HOCl) (Figure 2b).

Exposure to chlorine gas can lead to a spectrum of adverse effects. Mild exposure can result in symptoms such as vomiting and diarrhea, while more severe exposure triggers inflammation of the bronchial tubes and lungs [69]. This inflammation leads to sputum production, bleeding, and the filling of the lungs with fluid, often culminating in permanent lung damage or death due to suffocation. The toxicity of chlorine gas varies significantly depending on the dose and duration of exposure; exposure to concentrations as low as 1-3 ppm can irritate the eyes and oral mucous membranes, while exposure to 15 ppm initiates pulmonary symptoms. Remarkably, exposure to 430 ppm of chlorine gas can be fatal within 30 minutes [21]. Given its broad applications and potential dangers, the handling and storage of chlorine require meticulous care and adherence to safety protocols to prevent accidental exposure.

2.2.5. Chloropicrin: From Choking Agent to Riot-Control Agent and Beyond

Chloropicrin, also known as PS, occupies a unique place in the realm of chemical agents as it straddles multiple classifications. While it is primarily categorized as a choking agent due to its impact on the upper respiratory tract, chloropicrin has also been use as a riot-control agent. This versatile chemical presents as an oil, ranging in color from colorless to light green, and boasts a sharp, pungent odor akin to anise. Chloropicrin readily dissolves in organic solvents like chloroform, acetone, and ethyl acetate.

Historically, chloropicrin was employed in various capacities. It was mixed with sulfur mustard during the First World War to lower the freezing point. Additionally, the British utilized

chloropicrin in combination with chlorine, giving rise to designations like White Star (comprising 50% chlorine and 50% phosgene), Yellow Star (30% chloropicrin and 70% chlorine), and Green Star (65% chloropicrin and 35% hydrogen sulfide) [70]. Chloropicrin's utility expanded beyond warfare applications when its biocidal and fungicidal properties were recognized towards the end of the First World War, establishing it as a valuable fumigant [71]. However, the use of chloropicrin as a fumigant has waned in contemporary times. Chloropicrin exhibits a notable propensity to disperse in soil and plants, making exposure most likely through the consumption of drinking water disinfected via chlorination (1987). The chemical's initial impact targets the respiratory tract, while ingestion can result in corrosive effects on the anterior stomach tissue [72]. Chloropicrin's multifaceted history highlights its adaptability and underscores the importance of responsible handling and appropriate safety measures when dealing with this substance.

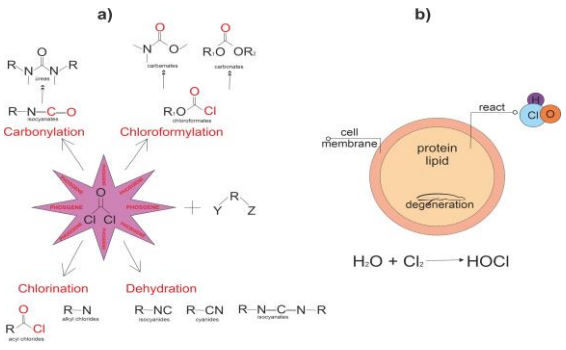


Figure 2: a) Reaction Of Organic Substrates With Phosgene **b)** If Chloropicrin Is Inhaled, Chlorine Gas Is Oxidized With The Water Present In The Body, And Hypochlorous Acid (Hclo) Is Produced. Hclo Damages Cellular Structures By Reacting With Proteins.

2.3. Riot Control Agents: Maintaining Order through Temporary Disability

Riot control agents, often called irritant, vomiting, and tear agents, serve a crucial role in crowd control [73]. These compounds induce temporary disability by irritating the eyes and upper respiratory tract, making them valuable tools for law enforcement and peacekeeping efforts. RCAs are employed to halt coordinated activities, disperse gatherings, temporarily neutralize demonstrators, and restore order. Their use typically results in the cessation of individual or group actions, the dispersal of communities, and the minimization of injuries. RCAs exert their effects primarily by acting as potent irritants on peripheral chemosensors. Their limited duration of action leads to short-term toxic effects that tend to subside within minutes after exposure [74]. Upon contact with RCAs, individuals often experience symptoms such as tears, eye pain, and skin irritation (Figure 3). Tear agents, for instance, stimulate tear gland nerves, promoting tear production. The intensity of skin irritation and sensations like tearing is so overwhelming that victims may struggle to act rationally, impeding and neutralizing the coordinated activities of those exposed [75].

Modern RCAs typically exist in crystalline solid forms, commonly administered as aerosol sprays, fine particles, or solutions. They include agents like chloroacetophenone (CN), o-chlorobenzylidene malononitrile (CS), and dibenzoxazepine (CR), collectively known as tear agents. These substances promptly affect nerve endings in mucous membranes, the cornea, and the skin, although their physical effects are short-lived. Additionally, vomiting agents such as

adamcide (10-chlor-5, 10-dihydrophenarcazine) (DM), diphenilarsin chloride (DA), and diphenilarcin cyanide (DC) typically irritate the nose and upper respiratory tract. Solid agents, like these, may also be used to enhance the effects of other toxic gases in certain situations. When treating lachrymator exposure, eyes and clothing should be decontaminated immediately.

2.3.1. Chloroacetophenone: A Former Riot Control Agent with Stringent Toxicity

Chloroacetophenone, known by its military designation CN and its trade name Mace™, was once a prevalent choice among law enforcement agencies as a standard riot control agent. However, due to its elevated toxicity compared to other riot control agents, CN gradually fell out of favor and was supplanted by CS over time [74, 76, 77]. Exposure to CN is associated with more pronounced pathological effects in the lungs, leading to the development of edema. Additionally, CN has been linked to early bronchopneumonia and possesses potent sensitizing properties for the skin. Its acute toxicity stems from its interactions with enzymes containing sulfhydryl (SH) groups, inhibiting certain sulfhydryl-based enzymes, including Lactic dehydrogenase [78]. CN is characterized by an aromatic scent often described as reminiscent of apple blossoms and presents itself in crystalline solid form. CN can find application in various scenarios, including riots, military operations, and exercises. It can be dispersed into indoor or outdoor environments, water sources, food items, and agricultural products, typically delivered in vapor or liquid spray (aerosol) form [73]. It is commonly deployed in the form of powder, smoke, or liquid, discharged from grenades or similar devices. Despite its former prominence, the inherent toxicity of CN has prompted the adoption of less toxic alternatives for maintaining public order and safety.

2.3.2. *Ortho*-chlorobenzylidene malononitrile: A Widely Used Riot Control Agent

Ortho-chlorobenzylidene malononitrile, better known by its military designation CS, owes its name to its originators, chemists Corson and Stoughton, who synthesized this agent. While CS exhibits minimal solubility in ethyl alcohol, it undergoes rapid hydrolysis upon contact with water. Among riot control agents, CS stands out as the most widely utilized. CS follows two known metabolic pathways. The first involves hydrolysis, leading to the formation of 2-chlorobenzaldehyde and malononitrile, while the secondary pathway results in 2-chlorobenzyl malononitrile without reduction [79]. When CS reacts with water, it produces 2-chlorobenzaldehyde and malonitrile. Notably, the half-life of CS in water is approximately 14 minutes, whereas its reactions with thiols and amines occur much more rapidly. CS possesses a scent reminiscent of pepper and presents itself in the form of white crystalline powder. Exposure to CS can lead to various health effects, including lung, heart, and liver damage, as well as gastrointestinal symptoms [80]. Despite its efficacy as a riot control agent, its use necessitates careful consideration due to its potential health impacts and the need for appropriate safety measures.

2.3.3. Dibenzoxazepine: A Riot Control Agent with Moderate Reactivity

Dibenzoxazepine, also known as dibenz(b, f)-1:4-oxazepine or CR, is a riot control agent with lower reactivity compared to CS or CN, and it undergoes hydrolysis at a relatively slower rate [81]. Consequently, CR is associated with comparatively lower toxicity than other tear agents. Exposure to CR typically results in effective irritation of the eyes, nose, and skin; however, these effects are not permanent. The respiratory tract rapidly absorbs CR, and it follows a metabolic pathway that includes oxidation to the lactam, ring hydroxylation, sulfate conjugation, and subsequent renal excretion [82]. CR has been used in the form of aerosol sprays and capsules to suppress prison riots in Northern Ireland. Notably, CR can be 5-10 times more potent than CS, causing greater irritation even at lower concentrations [83]. This heightened irritant effect can be a valuable asset in crowd control situations where it is essential to deter and disperse individuals without causing lasting harm.

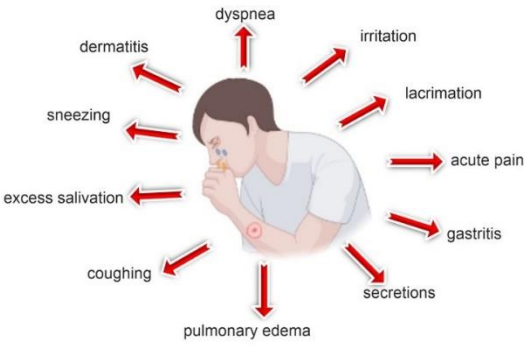


Figure 3: Physiological effects of riot control agents

2.4. Capacity Disrupting Agents (Incapacitating Agents): Altered Perception and Mood without Autonomic Nervous System Effects

Incapacitating agents, also known as psychomimetic substances, induce thought, perception, and mood changes without affecting the autonomic nervous system. Even at very low doses, these agents often elicit effects akin to psychotic disorders or other symptoms related to the central nervous system, such as sensory loss, paralysis, or delusions [84]. Unlike lethal chemical warfare agents, incapacitating agents cause temporary incapacitation and disorders. They are preferred in wartime scenarios because they induce temporary effects on individuals. For incapacitating agents to be effective, they must be highly potent and, when administered in appropriate doses, induce effects that can persist for hours to days without causing permanent disability or fatality [85]. Quinuclidinyl benzilate (BZ) is an example of a central nervous system incapacitating agent. BZ leads to physiological effects such as heart palpitations, dizziness, confusion, and a slowing of mental activities. Lysergic acid diethylamide (LSD) is another chemical agent categorized as a central nervous system stimulant among incapacitating agents. It stands out as one of the most potent agents in this category. Exposure to LSD leads to a profound alteration in an individual's perception, rendering them unable to accurately perceive time or their surroundings. This unique effect underscores the distinctive nature of incapacitating agents in altering cognitive and emotional states without causing permanent harm or death.

2.4.1. Quinuclidinyl benzilate: A Central Nervous System Incapacitating Agent

BZ, also known as QNB, 3-quinuclidyl benzilate, and benzylic acid, is a synthetic glycolic ester with high solubility in polar organic solvents. It crystallizes into a compound with a bitter taste. Unlike lethal agents, BZ serves as a competitive muscarinic cholinergic receptor antagonist, emphasizing its intent to create temporary incapacitation rather than causing fatality [86]. BZ exhibits effectiveness through various administration routes, including oral, parenteral, and inhalation, and undergoes metabolism in the liver before being excreted by the kidneys. By interfering with cholinergic nerve conduction in the brain, spinal cord's peripheral autonomic nervous system, and muscarinic regions, BZ induces an acetylcholine (ACh) deficiency. Notably, BZ readily crosses the blood-brain barrier and binds to subtypes of muscarinic receptors (M1-M5) in ACh [87]. The long-lasting effects of BZ may be attributed to its strong absorption by mitochondria, resulting in reduced oxygen consumption by nerve cells [88]. After exposure to an effective dose of BZ, environmental effects may manifest within 1 hour, while central effects might occur approximately 4 hours post-exposure. The primary effects can last for about 24-48 hours [89]. These distinctive properties render BZ an invaluable tool for temporary incapacitation without causing permanent harm.

2.4.2. Lysergic acid diethylamide (LSD): An Effective Incapacitating Agent

LSD, in its pure form, is a colorless and odorless chemical warfare agent with a distinct bitter taste. It can enter the body through ingestion or inhalation, and even at low doses, it profoundly alters human perception, behavior, and mood. However, these effects are temporary, making LSD one of the most potent incapacitating agents. Exposure to LSD leads to a significant shift in an individual's perception, resulting in the loss of the sense of time and place, effectively isolating them from reality. Some behaviors exhibited after LSD exposure bear resemblance to symptoms of mental disorders like schizophrenia. The effects of LSD primarily stem from its agonist activity at receptor subtypes such as 5-HT2A, 5-HT2C, and 5-HT1A [90], with LSD also displaying a high affinity for dopamine receptors [91, 92]. Notably, individuals affected by LSD typically do not require first aid, as spontaneous recovery typically occurs within 24 hours. This remarkable characteristic underscores its potential as an agent for temporary incapacitation without causing lasting harm.

2.5. Systemic Agents: Disrupting Oxygen Utilization and Rapidly Affecting Body Tissues

Systemic agents, also known as blood poisoning agents or cyanide chemicals, have a profound impact on bodily functions by impeding the regular utilization of oxygen in body tissues. These agents disrupt the production of blood components and exert toxicity at the cellular level, specifically by interrupting the electron transport chain within the inner mitochondrial membranes. This disruption leads to a swift depletion of oxygen throughout all body tissues, with the central nervous system being particularly vulnerable [93]. Due to the rapid action of cyanide, immediate treatment is imperative when exposed to systemic agents. These agents

typically enter the body via respiration and are expelled as gas or vapor. They are characterized by their high volatility, lighter-than-air properties, and gaseous nature. Exposure to systemic agents can result in death within a matter of seconds. Common systemic agents include arsine, hydrogen cyanide (HCN), and cyanogen chloride (ClCN). Physiological effects of systemic agents encompass respiratory distress, a choking sensation, tachycardia, respiratory irritation, nausea, and vomiting.

2.5.1. Hydrogen Cyanide (Prussic Acid, Hydrocyanic Acid) - NATO Code: AC

Hydrogen cyanide, also known as prussic acid and hydrocyanic acid, is assigned the NATO code AC. This chemical warfare agent exists in various forms, including solid, liquid, and gas, and is characterized by its colorless appearance and bitter almond-like scent. It is highly volatile and can readily transition between these forms. Hydrogen cyanide is soluble in a range of solvents, including water, ethanol, chloroform, and benzene, and retains its properties when dissolved in water. Hydrogen cyanide is lighter than air in its gaseous form, facilitating its rapid dispersion in the atmosphere. Exposure to hydrogen cyanide can occur through inhalation, absorption through skin openings, contact with the skin or eyes, or ingestion [94]. Hydrogen cyanide typically leads to eye and upper respiratory tract irritation, hampering enzyme systems [95]. Once hydrogen cyanide enters the bloodstream, it interferes with the activity of the cytochrome oxidase enzyme, preventing the utilization of oxygen by cells [96, 97]. This disruption results in respiratory failure and eventual fatality.

2.5.2. Cyanogen chloride (ClCN)

Cyanogen chloride (CK), also referred to as chlorine cyanide or chlorocyanogen, is a chemical warfare agent that exhibits different physical states depending on its temperature. It appears as a colorless liquid at temperatures below 12.8°C and a gaseous substance above this threshold. CK is characterized by its high volatility and systemic toxicity, disrupting the body's oxygen utilization mechanisms. Upon exposure, CK transforms hydrogen cyanide, which then deactivates crucial enzyme systems within the body. This results in systemic effects that affect various bodily systems, including the cardiovascular, central nervous, and pulmonary systems. CK exposure poses a significant risk and can lead to severe consequences, including fatality. CK can be combined with stabilizing agents like sodium pyrophosphate to enhance its shelf life. It also finds applications in commercial settings, particularly in chemical synthesis and fumigation processes [98].

2.5.3. Arsine

Arsine is a highly toxic chemical compound that exists as a colorless, flammable gas and emits an odor reminiscent of garlic, a characteristic common to many arsenic compounds. Unlike some other chemicals, arsenic is not naturally occurring but is produced commercially through specific chemical reactions. The industrial synthesis of arsine involves the reaction between aluminum arsenide and water or hydrochloric acid. Alternatively, it can be generated through the electrochemical reduction of arsenic compounds in an acidic solution [99]. Arsine has the potential to accumulate within the body,

leading to harmful effects. It can replace calcium in bones, inhibiting the formation of new blood cells in the bone marrow. When arsine enters the bloodstream via the respiratory system, it can cause liver and kidney damage and give rise to symptoms associated with hemolytic anemia, a condition characterized by the premature breakdown of red blood cells [100].

3. DECONTAMINATION OF CWAS

Decontamination of chemical warfare agents (CWAs) is a critical process aimed at removing these hazardous substances from affected areas or individuals to minimize their harmful effects. Rapid and effective decontamination is crucial for reducing the potential harm caused by CWAs. Decontamination typically involves chemical modification of the CWA through methods such as hydrolysis, catalysis, or oxidation [101, 102]. Here are some key points regarding the decontamination of CWAs:

Importance of Time: Time is a critical factor in the decontamination process. Decontamination should occur as quickly as possible to prevent further exposure and harm. For skin exposure, immediate decontamination (within one minute) is recommended using water, soap, talcum powder, or specialized decontaminants [103].

Contaminated Clothing: If clothing is contaminated with CWAs, it should be removed with care to prevent skin contact with the agent. Cutting off contaminated clothing may be necessary.

Decontaminating Agents:

Hypochlorite Solution: A 0.5% hypochlorite solution (household bleach diluted at 1:10) is considered one of the most effective decontaminating agents for CWAs. It works through physical removal, oxidation, or hydrolysis of the agent. It can be used on skin and soft tissue injuries, including open lacerations. After using hypochlorite solution on wounds, they should be washed with sterile saline solution.

M291 Resin: Dry wipes, known as M291 resin, composed of dry black carbon material, are used for point decontamination on the skin of contaminated individuals.

Sodium Hydroxide: Sodium hydroxide dissolved in an organic solvent can be used in emergencies. It is effective against V agents and mustard agents. However, it should be used with caution.

DS-2 (Decontamination Solution-2): DS-2 is a decontaminating solution consisting of 2% sodium hydroxide, 28% ethylene glycol monomethyl ether, and 70% diethylene triamine. It is used to decontaminate equipment that has been exposed to nerve or blister agents. DS-2 can neutralize these chemicals within 30 minutes [104].

Effective decontamination is essential to minimize the health risks associated with CWAs. Depending on the specific CWA involved and the affected area or individual, different decontaminating agents and methods may be employed.

4. CONCLUSION

In this comprehensive examination of chemical warfare agents (CWAs) and their associated characteristics, we have delved into a range of

toxic substances, each designed for distinct purposes on the battlefield. These agents, from nerve agents like Sarin and VX to choking agents like Chlorine and phosgene, serve as a chilling reminder of the potential for human-made devastation. Nerve agents, with their unparalleled toxicity, leave little room for error. Immediate decontamination, coupled with timely medical intervention, becomes paramount when confronting the severe consequences of exposure. Likewise, blister agents like Sulfur Mustard Nitrogen Mustards and Lewisite emphasize the dire need for swift response and effective decontamination strategies, given their ability to induce organ damage and promote cellular disruption. Choking agents, Riot control agents, and Incapacitating agents, while less lethal, still pose significant risks and necessitate rapid mitigation and supportive care measures. A thorough understanding of these agents and their potential for harm is crucial for ensuring effective decontamination and minimizing health impacts. Systemic agents, with their swift and devastating effects on the body's oxygen utilization, leave little time for response. Immediate decontamination and medical intervention are paramount to counter the severe consequences of exposure. Decontamination methods, discussed extensively in this manuscript, are the first line of defense against the devastating effects of CWAs. From hypochlorite solutions to specialized decontaminants like M291 resin, the choice of decontaminating agents is vital and depends on the nature of the agent and the affected area. From the past to the present, CWAs have always been a source of concern. In line with the developing technological approaches, it is clear that traditional weapons can be replaced by new-generation chemicals produced in the laboratory. International agreements such as the Chemical Weapons Convention should be increased. Thus, these agreements should help control the spread of chemical weapons and safely destroy existing weapons. In conclusion, a comprehensive understanding of chemical warfare agents and their corresponding decontamination protocols is critical for safeguarding human lives and the environment in the event of CWA incidents. Preparedness, rapid response, and effective decontamination procedures form the cornerstone of mitigating the catastrophic effects of these toxic substances and upholding global security.

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AUTHORSHIP CONTRIBUTION STATEMENT

İrem Mukaddes Bilgiseven: Writing- review & editing, Writing-original draft, Investigation, Conceptualization.

Serdar Karakurt: Writing- review & editing, Validation.

CONFLICTS OF INTEREST

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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