CHAPTER 4 CHEMICAL AGENTS

4.1 INTRODUCTION

Throughout history there are descriptions of chemical compounds used as battle weapons. Fires producing overpowering smoke, chlorine and sulfur dioxide gases and special formulations such as "Greek Fire" have overpowered enemy forces. World War I launched modern chemical warfare with the use of tear gas, chlorine cylinders, mustard gas and mortar shells containing phosgene and chlorine. Advanced methods of formulating chemical compounds during World War II resulted in the production of various nerve agents able to seriously debilitate or kill opposing troops.

Chemical agents that are used as Weapons of Mass Destruction (WMD) include those that were formulated solely for purposes of weaponization and those that are used customarily for industrial, medicinal and other purposes. Any chemical that causes adverse health effects can potentially be used as a weapon. The ease of attainment, methods of distribution and likelihood of serious health consequences, including death, are all considered important factors in considering which chemical agents are most likely to be used as weapons. Industrial processes that routinely produce chemicals for manufacturing purposes can often be readily adapted to produce chemical warfare agents. Countries that choose to conceal chemical weapons production programs can readily do so. These weapons are often very inexpensive to produce and can be stored for many years. They can be used as single agents or in combinations with other chemical or biological agents.

There are multiple methods of delivering chemical weapons, including rockets, bombs, mines, explosive shells, spray devices and other vehicles. The weapons delivery can be covert, with the only indication of a chemical assault provided by the casualties. At times, the odor of an agent can inform those affected of the presence of a chemical agent. Odors can be useful but are not a dependable means of determining chemical exposure.

Chemical agents are generally classified by physiological effects or military background. The route of exposure to chemical agents can be through inhalation, ingestion, or contact with the skin. These agents can produce both immediate and long-term health effects. The adverse effects depend on the specific chemical agent and the route and dose of exposure. The health consequences of chemical agents generally occur sooner than those of biological agents, with some producing health effects within minutes. Combinations of chemicals or releases that include biological agents, as well, can produce such diverse health effects that it is virtually impossible to determine the responsible agent(s).

First aid and other health care providers need to assess the complicated clinical findings to determine the nature of the attack and optimal course of treatment. When the likely source of a chemical agent has been identified, descriptions of events and environmental conditions can provide helpful clues to health care providers in identifying specific agents.

The agents reviewed in this manual include the nerve and blistering (vesicant) agents. These are chemical warfare agents that are currently included in the weapons arsenals of some nations. Also reviewed are chemicals routinely used or produced in industry, including cyanide, chlorine and phosgene. The list of possible chemical warfare agents is endless. To be prepared for a chemical attack, it is important for emergency responders and health care providers to be knowledgeable about likely chemical agents and their health consequences.

CYANIDE SUMMARY
U.S. E.P.A. REGION 5 HOMELAND DEFENSE

Blood Agent	Hydrogen Cyanide (AC) (HCN) HCN is a gas that is lighter than air, possibly emitting a "bitter almond" odor. It is made by mixing cyanide salt with strong acid. The characteristic odor may not be detectable by individuals due to genetic differences and olfactory fatigue. CYANOGEN CHLORIDE (CK) (CNCl) CNCl is a gas or liquid and is heavier than air.
Chemical Action	Cyanide inhibits cytochrome A3; blocks tissue uptake and utilization of oxygen in body tissues. Cyanide is a cellular asphyxiant. Lactic acidosis results from cyanide toxicity.
Source and Weaponization Techniques	Cyanide compounds are ubiquitous in nature and the manufacturing world. Many foodstuffs contain low levels of cyanide. It is metabolized safely by humans when present at non-toxic levels. Cyanide is used in pesticides, plastics, electroplating, chemical syntheses and in multiple other industrial processes. Cyanide is a by-product of combustion of both natural and synthesized materials; it is found in cigarette smoke, vehicle exhaust and drinking water. Cyanide has been used as a poisoning agent since antiquity, through all routes of exposure. Hydrogen cyanide is an effective asphyxiant when used in confined spaces. Cyanide compounds have been used as WPM.
Clinical Manifestations	Cyanide can be absorbed through the lungs, GI tract, skin, eyes and mucous membranes. The central nervous (CNS) and cardiovascular systems are extremely sensitive to the asphyxiant effects of cyanide and related clinical signs present rapidly after exposure. Signs and symptoms of illness that victims can experience include: CNS – dizziness, euphoria, headache, weakness, nausea, vomiting, drowsiness, and eventually seizures, tetanic spasms, hallucinations, loss of consciousness and coma. Cardiovascular – cardiac arrhythmias, high and low blood pressure, rapid and slow heart beat. Cardiovascular collapse can occur. Respiratory – rapid, deep respirations, shortness of breath, chest tightness, pulmonary edema. Metabolic – anion gap acidosis with elevated blood levels of lactic acid. Dermal – cyanide can be absorbed through the skin. Ocular – eye inflammation. <u>Small exposure -</u> Effects depend on type of compound, route and dose of exposure and individual characteristics. Onset of effects may be within seconds. <u>Initial symptoms –</u> cardiac arrhythmia, increased heart rate, shortness of breath, excitement, headache, anxiety, agitations, personality. changes, sweating, weakness, vertigo. <u>Late symptoms</u> – convulsions, coma, respiratory compromise, cardiac failure. <u>Large exposure</u> – can be fatal within minutes.

	Sequelae –survivors can experience personality changes, memory loss, abnormal muscle movements, and brain damage. Effects of chronic exposure include fatigue, chest pain, loss of appetite, nosebleeds, shortness of breath, nausea / vomiting, palpitations and headache.
Medical Treatment	Provide the victim supportive care, including oxygen and respiratory support. Cyanide antidotes must be administered immediately. Use amyl nitrite perle until IV sodium nitrite is initiated. This should be followed by IV sodium thiosulfate.[1] Treatment recommendations may change and also might be altered if multiple chemical exposures are suspected. Anticonvulsants should be administered for seizures. Supportive care must be provided.
Medical Prophylaxis	None available
Emergency Action Plan	If cyanide exposure is possible: Emergency responders must be appropriately trained and attired in PPE to protect from contamination until they have decontaminated victims and departed from the contaminated area. Immediately remove the individual from exposure and contact emergency medical care. Clothing or other items contaminated with liquid cyanide compounds pose the greatest risk for secondary contamination by off-gassing vapors or by direct contact. Avoid direct contact with victims or their gastric contents. Follow basic first aid directives for working in a contaminated area, remembering ABCs and spine stabilization. If individual is not breathing, administer artificial respiration with a bag valve mask device. A barrier device must be utilized for resuscitation. Emergency responders trained to administer antidotes to cyanide poisoning should evaluate and treat the victim. Remove clothing and decontaminate as quickly as possible, with extent of decontamination determined by suspected agent and extent of contact. Flush skin and hair with water for at least 20 minutes, followed by washing with soap and water twice and rinsing with water. Do not induce emesis if ingestion has occurred. Irrigate eyes with large quantities of tepid water for at least 15 minutes. The individual's head should be tilted to the side, the eyelids gently pulled apart with fingers and water poured slowly into the eyes. Inform emergency responders and medical personnel of chemicals involved; take protective measures to avoid secondary exposure. Contact FOH physician and maintain contact during course of therapy, as the situation allows. Complete EPA post exposure occupational health evaluation through FOH clinic after completion of therapy.

NERVE AGENT SUMMARY
U.S. E.P.A. REGION 5 HOMELAND DEFENSE

Nerve Agent	Military: Sarin (GB), Tabun (GA), Soman (GD), VX Commercial: Parathion, Sevin Nerve Agents are clear liquids at room temperature that can evaporate to gases. GA –fruity odor, GB – odorless, most volatile nerve agent, GD – camphor odor, VX- odorless, amber –colored, least volatile. Agents are miscible in water; VX at temperatures < 48.9^0 F.
Chemical Action	Nerve agents inhibit an enzyme known as acetylcholinesterase as well as other enzymes. Acetylcholinesterase is an enzyme that stops the action of the neurotransmitter, acetylcholine. When nerve agents inhibit acetylcholinesterase, the agents bind irreversibly to the enzyme. The action of acetylcholine is subsequently not blocked by acetylcholinesterase, and a buildup of acetylcholine results at the nerve receptors. The continued exposure to acetylcholine at the nerve receptor site results in overstimulation of end organs, including exocrine glands, skeletal muscles, smooth muscles and the central nervous system. This overstimulation causes the signs and symptoms of nerve agent toxicity. Nerve warfare agents are similar to organophosphate pesticides in chemical action. Acetylcholinesterase is similar to an enzyme in red blood cells known as red blood cell (RBC)cholinesterase. Measurements of RBC cholinesterase can be used as some correlate to acetylcholinesterase activity in the nervous system. GA decomposition can result in HCN, CO, hydrogen cyanide, oxides of nitrogen and oxides of phosphorus. GB and GD can hydrolyze under acid conditions to produce HF. VX hydrolysis produces a class B poison.[1]
Weaponization Techniques	Agents can be aerosolized and incorporated into munitions. Agents are extremely toxic, including by ingestion. Loss of consciousness and convulsions can occur within seconds of exposure to nerve agents; respiratory failure and death can occur within minutes. "As little as one drop of VX on skin can be fatal and 1 to 10 mL of GA,GB, or GD can be fatal."[2] Nerve agents have been used as WPM by Iraq against Iran and by terrorists.
Clinical Manifestations Inhalational (Vapor)	Nerve agents are readily absorbed through inhalation, ingestion, dermal or ocular contact. Clinical effects may be experienced within minutes or delayed up to 18 hours, depending on the agent, dose and route of exposure Most inhalational effects occur within seconds to minutes of initial exposure. <u>Very small exposure</u> – miosis, visual disturbances, runny nose, may be respiratory symptoms. <u>Small exposure</u> – Eyes, nose and lungs most involved, with bronchoconstriction, bronchosecretions, dyspnea and progressively worsening symptoms. Symptoms can include salivation, lacrimation,

	urination, gastrointestinal (GI) distress, bradycardia, bronchospasm, abdominal cramps, miosis, (SLUGBAM). Large exposure – all of the above and loss of consciousness, severe breathing difficulty, cessation of breathing, convulsions, muscular fasciculations, paralysis, death.
Clinical Manifestations: Dermal (Liquid on Skin)	Dermal exposure effects can be delayed. Most effects occur within 30 minutes, but can be delayed as long as 18 hours. Small exposure – sweating, localized muscle twitch, nausea, vomiting, diarrhea, weakness. Large exposure – all of above and loss of consciousness, severe breathing difficulty, cessation of breathing, convulsions, muscular fasciculations, paralysis, death.
Clinical Signs/Symptoms	Other signs and symptoms caused by nerve agent exposure include: Neurological – behavioral changes, irritability, fatigue, insomnia, memory loss, slurred speech, depression. These changes can be noted for 6 weeks after recovery from nerve agent exposure. [3] Respiratory – excessive secretions, breathing difficulty. Cardiovascular – bradycardia, tachycardia, arrhythmias, hypertension. Gastrointestinal – abdominal pain, diarrhea, nausea, vomiting, fecal incontinence. Ocular – dim and blurry vision, eye pain, conjunctivitis. Skeletal muscles – muscle twitching and fasciculations. Metabolic – excessive sweating.
Medical Treatment	Immediate medical treatment may include antidotes, as atropine and pralidoxime chloride (Mark I Kit), diazepam and other medications. Supportive care must be provided as required, including oxygenation and respiratory support. Seizures must be monitored for and treated. For ingestion exposure in alert patients able to swallow, an activated charcoal slurry should be administered. Emesis should not be induced. Atropine and homatorpine eye drops can be used for eye complaints. Any individual who might have exposure to a nerve agent or organophosphate pesticide must have baseline RBC cholinesterases (2 blood samples within 14 days) drawn. RBC cholinesterase samples at the time of an exposure event can be compared with baseline results to assess level of RBC cholinesterase inhibition. RBC cholinesterase levels revealing >70%inhibition from baseline usually correlate with signs of severe toxicity. [4] However, patient clinical signs and symptoms of exposure to nerve agent will dictate medical treatment, not results of the RBC cholinesterase test.
Medication Prophylaxis	Pyridostigmine bromide has been used for pre-exposure prophylaxis. It has been administered over a 7 day period prior to anticipated nerve agent exposure. Other prophylactic therapies are being developed.

Emergency Action Plan

If exposure to a nerve agent is possible:
Immediately remove the individual from exposure and contact emergency medical care.
Follow basic first aid directives for working in a contaminated area, remembering ABCs and spine stabilization.
Antidotes to nerve agents and other medical therapies must be administered by emergency responders with appropriate training.
Antidotes must be administerered quickly, before the nerve agent has irreversibly bound to acetylcholinesterase. If individual is not breathing, administer artificial respiration with a bag valve mask device.
Contaminated skin, clothing or other material can contaminate other individuals by direct contact or off-gassing vapor; contaminated clothing should be removed and sealed in a plastic bag.
Extent of decontamination must be determined by suspected agent and extent of contact. Vapor exposure only may not require decontamination. If only vapor exposure has occurred, remove and isolate clothing.
For direct skin exposure, wash skin and hair with soap and water. Skin can also be decontaminated with 0.5% sodium hypochlorite solution or absorbent powders as flour, talc or fuller's earth.
Decontaminate skin with soap and water or 0.5% sodium hypochlorite solution for 20 minutes.
Eyes exposed to nerve agents must be immediately decontaminated. Irrigate eyes with large quantities of water or sterile saline. The individual's head should be tilted to the side, the eyelids gently pulled apart with fingers and water poured slowly into the eyes for 20 minutes.
Do not induce emesis.
Avoid spreading chemical to unexposed areas.
Isolate clothing, optimally with double plastic bags and avoid secondary contamination from chemical in environment.
Inform emergency responders and medical personnel of chemicals involved; take protective measures to avoid secondary exposure.
Contact FOH physician.
Complete EPA post exposure occupational health evaluation through FOH clinic after completion of medical evaluation and therapy.

CHLORINE SUMMARY
U.S. E.P.A. REGION 5 HOMELAND DEFENSE

Pulmonary Agent	CHLORINE (Cl_2) Yellow-green gas that is non-combustible. Chlorine has a distinctive and irritating odor. It reacts explosively with common compounds and is heavier than air.
Chemical Action	Chlorine causes tissue damage by oxidizing action that produces oxygen and hydrochloric acid. Tissue irritation and inflammation result.
Sources and Weaponization Techniques	Produced commercially by electrolysis of sodium chloride brine. One of the top ten greatest volume chemicals produced annually in the United States. Chlorine is used as a bleach in the processing of paper and cloth. It is also used frequently in the manufacturing of other chemicals. Chlorine can be aerosolized and incorporated into munitions.
Clinical Manifestations	Chlorine is a severe respiratory tract and skin irritant. The odor of chlorine can indicate the level of exposure, however, olfactory sensation can be lost with prolonged, low-level exposures. Chlorine can cause severe burns and cell death with exposure by inhalation, ingestion, or direct contact to skin or eyes. Contact with chlorine in liquid form can cause frostbite. Chlorine is most likely to be ingested in a solution; if ingested it can cause injury by tissue corrosion. Inhalational Exposure: Low concentration exposure (<10 ppm) – Individuals exposed can experience stinging and burning of the eyes and nose, sore throat, sneezing, coughing, and bloody nose or sputum. Higher concentration exposure (>15 ppm) – Individuals exposed experience progressively worsening symptoms, including rapid breathing, wheezing, coughing blood, respiratory distress, airway constrictions, pulmonary edema, bluish discoloration of skin and possibly lung collapse. Large, acute exposure to chlorine (as 430 ppm over 30 minutes) can result in rapid death.[1] Chlorine exposure can cause extensive signs/ symptoms of illness; some of these include: skin / ocular burns, headache, dizziness, muscle weakness, choking, syncope, epigastric pain, anxiety, apprehension, dyspnea, pneumonia, respiratory distress and respiratory failure. Chronic chlorine exposure can result in a higher incidence of upper respiratory infections, sore throats, chest pain, reactive airways dysfunction syndrome and asthma. The effects of acute chlorine exposure can be noted immediately or delayed by several hours. This can occur with inhalational exposures resulting in pulmonary complications as pulmonary edema and congestion.

Medical Treatment	There is no specific antidote for chlorine poisoning. Specific problems require usual medical care, including treatment for bronchospasm and respiratory support as needed. Treatment is supportive.
Medical Prophylaxis	None effective.
Emergency Action Plan	If exposure to chlorine gas is possible: Immediately remove the individual from exposure and contact emergency medical care. Emergency responders must be appropriately trained and attired in PPE to protect from contamination until they have decontaminated victims and departed from the contaminated area. Follow basic first aid directives for working in a contaminated area, remembering ABCs and spine stabilization. Remove clothing and decontaminate as quickly as possible, with extent of decontamination determined by extent of contact. Individuals should assist with decontamination to the extent that they are able. They should walk out of the contaminated zone to a safe zone if they are able. Do not induce emesis. If frostbite has not occurred, exposed skin and hair should be washed with plain water for 3 to 5 minutes, and then washed with mild soap twice. Afterwards the areas should be rinsed thoroughly with water. If there is frostbite, do not try to remove clothing, flush skin with water or rub area. Place frostbitten skin in warm water, about 108^{o}F . If warm water is not available, the affected part can be wrapped gently in a blanket. The victim should gently exercise the affected area during this process.[2] Eyes that have suffered frostbite cannot be irrigated. If eyes have been exposed and not affected by frostbite, irrigate with water or saline for 15 minutes. If possible, remove contact lenses. Contact FOH physician and maintain contact during course of therapy, as the situation allows. Complete EPA post exposure occupational health evaluation through FOH clinic after completion of therapy.

PHOSGENE SUMMARY
U.S. E.P.A. REGION 5 HOMELAND DEFENSE

Pulmonary Agent	Phosgene(CG) – Carbonyl chloride($COCl_2$), Colorless gas that may have odor of newly mown hay or sharp odor, depending on concentration. Appears as white cloud during battlefield release. Odor not reliable for detection, and the odor threshold is 5 times greater than the OSHA PEL. Phosgene gas is heavier than air and has low water solubility. At cooler temperatures, (below 47 degrees) phosgene is found in liquid form. Phosgene reacts with water to form hydrochloric acid and carbon dioxide.
Chemical Action	Reaction of phosgene chemicals with other compounds produces acid which results in corrosive damage to exposed tissue.
Sources	Phosgene is formed as a by-product of volatile chlorinated compound combustion and decomposition. It is produced when materials containing solvents, household substances and dry cleaning fluids are heated or burned. Phosgene is used in processing pharmaceuticals, pesticides, polyurethane, isocyanates and other products.
Weaponization Techniques	Phosgene was used as a toxic inhalant during World War I and is currently available as a weapon of mass destruction.
Clinical Manifestations	Phosgene can cause tissue damage to deep lung tissue. It can be initially non irritating, allowing victims to maintain normal respiration and thus further exposure. Clinical effects depend on intensity of exposure. Initial effects can precede a relatively symptom-free period during the first 24 – 48 hours and then progress to severe non-cardiogenic pulmonary edema. The latency period shortens in relation to the intensity of the exposure. <u>Initial effects</u> - cough, chest tightness, difficulty breathing, headache, vomiting, irritation of eyes, nose and skin. Exertional dyspnea may precede pulmonary edema. Phosgene causes tissue damage within minutes of exposure. Lacrimation and an odd taste occur with moderate exposure. High concentration exposures may cause rapid onset of cough, laryngospasm and sudden death. When pulmonary edema does occur, it can progress rapidly. It is caused by the damage that has occurred to the lung tissue. Coughing productive of large amounts of frothy sputum and cyanosis develop quickly. Fluid in the lungs can lead to vascular instability, heart failure and death. Phosgene can also cause nausea and vomiting, renal and hepatic failure. Patient survival for the first 48 hours post exposure is a good indicator that of eventual recovery. Pulmonary and other problems may persist after initial recovery.

Medical Treatment	Provide supportive care including respiratory support and oxygen. Keep patient warm and quiet and monitor for signs of pulmonary edema and other complications. Treat other clinical effects as needed
Medical Prophylaxis	None
Emergency Action Plan	If exposure to phosgene is possible: Immediately remove the individual from exposure and contact emergency medical care. Emergency responders must be appropriately trained and attired in PPE to protect from contamination until they have decontaminated victims and departed from the contaminated area. Clothing or other items contaminated with liquid phosgene pose the greatest risk for secondary contamination by off-gassing vapors or by direct contact. Follow basic first aid directives for working in a contaminated area, remembering ABCS and spine stabilization. If artificial resuscitation is necessary, use a resuscitation mask equipped with a one way valve or other appropriate device. Keep victims quiet and warm, as post-exposure activity can cause further harm and increase the possibility of death. If frostbite has developed on exposed areas, do not flush with water. If frostbite has not developed, flush skin and eyes with large amounts of water. For decontamination, remove clothing as quickly as possible and continue flushing skin with water for at least 20 minutes.. If eye exposure has occurred, flush eyes with water for at least 20 minutes. Tilt head and occasionally pull eyelids apart with fingers to flush with water. If frostbite has developed, do not remove clothing or flush area with water. All individuals with possible phosgene exposure should undergo medical evaluation. This is essential as clinical effects may not be noted for up to 48 hours. Contact FOH physician as soon as possible and maintain contact through course of treatment.. Complete EPA post exposure occupational health evaluation through FOH clinic after completion of therapy.

LEWISITE SUMMARY
U.S.E.P.A. REGION 5 HOMELAND DEFENSE

Vesicant Agent	Lewisite ($C_2H_2AsCl_{3)}$ Lewisite is designated L, also known as 2-chlorovinyldichloroarsine and by other chemical names. Lewisite is an arsenical vesicant. It is an oily, colorless liquid with the odor of geraniums. Impure lewisite, as from industrial production, is an amber to dark brown liquid. It remains fluid at lower temperatures and can be used in cold weather.
Chemical Action	Lewisite inhibits many enzymes. It binds with thiol groups on some enzymes but the exact mechanisms of action of lewisite are not well-defined. Some of the enzymes affected include pyruvic oxidase, alcohol dehydrogenase, succinic oxidase, hexokinase and succinic dehydrogenase.
Sources and Weaponization Techniques	Lewisite was first formulated in 1918. The United States had plans to use lewisite in WWI, but they were never realized. Lewisite has been developed for WPM purposes by some countries, but there is no definitive information as to whether it has actually been used. Lewisite is mixed with mustard to lower the freezing point of mustard and formulate a compound with a broader range of use. The Mustard / Lewisite mixture has a garlic odor. Lewisite or the mixture can be dispersed on the ground or by aerial spraying. Both the vapor and liquid phases of these agents are extremely toxic.
Clinical Manifestations	Lewisite is absorbed through the skin, eyes, respiratory and gastrointestinal tracts. It produces immediate harmful effects including pain and burning in the eyes, nose and skin. Skin blisters start within several minutes of liquid exposure, later with vapor. Exposure to liquid results in skin erythema developing within 15 to 30 minutes and blisters forming over the next 12 to 18 hours. The ocular effects after exposure to lewisite vapor include immediate pain and blepharospasm. The eyes swell, and can swell shut because of this within the hour. With inhalational exposure, the victim can experience violent sneezing, profuse nasal discharge, bloody nose, cough, shortness of breath, sinus pain and laryngitis. Airway obstruction can result from pseudomembrane formation. Pulmonary edema can develop after significant exposure. Severe skin, eyes and airway damage can occur later. Lewisite has systemic effects, but no bone marrow suppression Nausea, vomiting and diarrhea occur. Bloody diarrhea can develop approximately 20 minutes after exposure. “Lewisite shock” caused by vascular instability secondary to increased capillary permeability results from large exposures. Death can occur. Other possible systemic effects include hepatic necrosis, decreased renal

	function, bone marrow suppression, chronic respiratory and eye conditions. Chronic exposure can cause arsenical poisoning.
Medical Treatment	Supportive care must be provided, including respiratory, burn and eye care. Monitor for infections. Treatment must be based on the individual's clinical course. Antidote—British Anti-Lewisite (BAL) - decreases internal damage (hospital-based treatment).
Medical Prophylaxis	None
Emergency Action Plan	If exposure to Lewisite or the Mustard / Lewisite mixture is possible: Emergency responders must be appropriately trained and attired in PPE to protect from contamination until they have decontaminated victims and departed from the contaminated area. Immediately remove the individual from exposure and contact emergency medical care. Rescuers are at serious risk of secondary contamination if lewisite or lewisite / mustard has contaminated clothing or victim. Clothing or other items contaminated with liquid agent pose the greatest risk for secondary contamination by off-gassing vapors or by direct contact. Decontamination must be accomplished immediately for it to be effective at stopping the progression of lewisite damage, eyes and skin must be flushed with water immediately. Follow basic first aid directives for working in a contaminated area, remembering ABCs and spine stabilization. Artificial respiration should be provided with a bag-valve-mask device that includes a canister or air filter, if needed. A pocket mask equipped with a one way valve can be used for resuscitation, depending on contamination concerns. Remove clothing and decontaminate as quickly as possible with water and then wash with soap and water or 0.5% sodium hypochlorite solution. If water is not easily available, decontamination can be performed with adsorbent powders including flour, talcum powder or Fuller's earth. The powder should be applied to the skin, allowed time to adsorb the mustard and removed with moist cloth or towel. Do not induce emesis. Irrigate eyes with large quantities of water. The individual's head should be tilted to the side, the eyelids gently pulled apart with fingers and water poured slowly into the eyes for at least 15 minutes. Avoid spreading chemical to unexposed areas. Isolate clothing, optimally with double plastic bags and avoid secondary contamination from chemical in environment. Later decontamination necessary to possibly prevent further spread and prevent exposure to others. Clinical symptoms may be delayed for 24 hours. Victims should remain

Emergency Action Plan (continued)	under medical management for greater than 24 hours and instructed to inform medical management team immediately if signs or symptoms are experienced. Contact FOH physician and maintain contact during course of therapy, as the situation allows.Complete EPA post exposure occupational health evaluation through FOH clinic after completion of therapy.

MUSTARD SUMMARY
U.S. E.P.A. REGION 5 HOMELAND DEFENSE

Vesicant Agent	Mustard (Blistering) Sulfur mustards include multiple compounds designated H, HD and HT. The sulfur mustards have a garlic or mustard odor, and are yellow /brown oily liquids. Nitrogen mustards include multiple compounds designated HN-1, HN-2 and HN-3.The nitrogen mustards have odors that vary and are colorless to yellow, oily liquids. The mustards are persistent chemicals with low volatility and this increases as ambient temperature increases. The mustard vapors are heavier than air.
Chemical Action	The mustards are vesicants and alkylating agents with multiple biochemical effects that are not well-defined. There may be delayed effects, including DNA and bone marrow damage.
Weaponization Technique	Sulfur mustards were initially synthesized in the early / mid 1800s. They were used as warfare agents in World War I, causing temporary blindness in a high percentage of affected troops. They have been frequently developed as chemical warfare agents and can be incorporated into munitions. Nitrogen mustards were produced initially in the 1920s and 1930s. Some of the nitrogens have been used for medicinal purposes for wart removal and chemotherapy; none have been used as chemical warfare agents. Mustard gas was used in the Iran / Iraq war.
Clinical Manifestations	The mustard compounds are vesicants and irritants; they cause damage and necrosis with direct tissue contact. They rapidly bind to proteins in the body and cannot be isolated from tissue or body fluids, even shortly after exposure. The sulfur mustards stimulate the cholinergic system. The degree of exposure determines the clinical symptom presentation. Mustards are absorbed through the skin, eyes, respiratory and gastrointestinal tracts. Effects are delayed, noted 1-24 hours after exposure, usually felt 4-8 hours after. Some of the clinical signs and symptoms victims may experience include the following: Ocular – the eye is most sensitive to sulfur mustard effects, resulting in conjunctival and scleral pain and inflammation, lacrimation, blepharospasm, photophobia, corneal edema and perforation, blindness and scarring. Dermal- redness, rash usually develops within 4 – 8 hours, with blistering from 2 to 18 hours afterwards. Contact with liquid mustards can cause first second or third degree skin burns, depending on the agent and dose. If ≥ 25 % of the body surface area is burned, death may result. Respiratory –airway inflammation develops within hours after

	exposure, with timing and severity dependent on the exposure dose. Nose bleeds, nasal and sinus pain, loss of smell and taste, cough, laryngitis, wheezing and shortness of breath can result from mustard exposure. Airway obstruction, lung fibrosis and frequent respiratory infections can occur. Gastrointestinal –nausea and vomiting can occur early and resolve. GI tract chemical burns can occur; nausea, vomiting and diarrhea occurring days after exposure can be secondary to GI burn damage. CNS – insomnia, hyperexcitability, seizures and other effects can occur. Hematopoietic – bone marrow suppression, anemia, hemorrhage and increased susceptibility to infection can occur. <u>INITIAL EFFECTS:</u> Small exposure—eye tearing, itching, burning, runny nose, nosebleeds, hoarseness, mucous membrane irritation, coughing, sneezing, skin inflammation. Large exposure—above effects plus eye pain, lid swelling, eye redness, possible corneal damage and blindness, productive cough, mild to severe respiratory difficulty, airway and lung blistering, skin blistering, nausea, diarrhea. <u>LATER EFFECTS:</u> Above plus additional hematopoietic, gastrointestinal and central nervous system effects. Death is usually caused by massive pulmonary damage with ensuing infection and sepsis after inhalational exposure. Death typically occurs 1 week after an acute exposure. Tissue damage occurs as with radiation exposure.
Medical Treatment	Supportive care, including respiratory support as needed. Burn treatment, eye care and pain treatment must be provided. Monitor for infections. Treatment will be based on the individual clinical course.
Medical Prophylaxis	None
Emergency Action Plan	If exposure to mustard is possible: Emergency responders must be appropriately trained and attired in PPE to protect from contamination until they have decontaminated victims and departed from the contaminated area. Immediately remove the individual from exposure and contact emergency medical care. Rescuers are at serious risk of secondary contamination if liquid mustard has contaminated clothing or victim. Clothing or other items contaminated with liquid phosgene pose the greatest risk for secondary contamination by off-gassing vapors or by direct contact.

Emergency Action Plan (continued)

Decontamination must be accomplished immediately for it to be effective at stopping the progression of mustard damage.

Follow basic first aid directives for working in a contaminated area, remembering ABCs and spine stabilization.

Artificial respiration should be provided with a bag-valve-mask device that includes a canister or air filter, if needed.

Remove clothing and decontaminate as quickly as possible with water and then wash with soap and water or 0.5% sodium hypochlorite solution. Warm water at low pressure should be used for showering. If water is not easily available, decontamination can be performed with adsorbent powders including flour, talcum powder or Fuller's earth. The powder should be applied to the skin, allowed time to adsorb the mustard and removed with a moist cloth or towel.

Do not induce emesis.

The eyes should be irrigated with large quantities of water. The individual's head should be tilted to the side, the eyelids gently pulled apart with fingers and water poured slowly into the eyes.

Avoid spreading chemical to unexposed areas.

Isolate clothing, optimally with double plastic bags and avoid secondary contamination from chemical in environment.
Later decontamination necessary to possibly prevent further spread and prevent exposure to others.

Most casualties require medical care.

Clinical symptoms may be delayed for 24 hours. Victims should remain under medical management for greater than 24 hours and instructed to inform medical management team immediately if signs or symptoms are experienced.

Contact FOH physician as soon as possible and maintain contact through course of treatment.

Complete EPA post exposure occupational health evaluation through FOH clinic after completion of therapy.

4.6 CHEMICAL AGENT FOOTNOTES

Chlorine

1. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry: Chlorine CAS 7782-50-5; UN 1017. P. 5.
2. Ibid. P. 8.

Cyanide

1. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Hydrogen Cyanide (HCN) CAS 74-90-8; UN 1051. P. 12.

Nerve

1. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Nerve Agents. Tabun (GA) CAS 77-81-6; Sarin (GB) CAS 107-44-8; Soman (GD) CAS 96-64-0; and VX CAS 5078269-9. P. 1.
2. Ibid. P. 2.
3. Ibid. P. 7.
4. Sidell, FR. Nerve Agents. In: Zajtchuck R, Bellamy RF, Sidell FR, Takafuki ET, Franz DR, editors. Textbook of Military Medicine Part I. Warfare, Weaponry, and the Casulaty. Medical Aspects of Chemical and Biological Warfare. 1st Edition. Washington, DC: Borden Institute, Walter Reed Medical Center: 1997: P. 136.

4.7 REFERENCES: CHEMICAL AGENTS

1. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Hydrogen Cyanide (HCN) CAS 74-90-8; UN 1051.

2. Hazardtext Managements. Hydrogen Cyanide. MICROMEDEX Thomson Healthcare Series Vol. 112 expires 6/2002.

3. Baskin, SI, Brewer, TG, Cyanide Poisoning. In: Zajtchuck R, Bellamy RF, Sidell FR, Takafuki ET, Franz DR, editors. Textbook of Military Medicine Part I. Warfare, Weaponry, and the Casualty. Medical Aspects of Chemical and Biological Warfare. 1st Edition. Washington, DC: Borden Institute, Walter Reed Medical Center: 1997.

4. Hydroxocobalamin: Improved Public Health Readiness for Cyanide Disasters. Annals of Emergency Medicine; 37: (6) 635-641.

5. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Chlorine CAS 7782-50-5; UN 1017.

6. Hazardtext Management. Chlorine. MICROMEDEX Thomson Healthcare Series Vol. 112 expires 6/2002.

7. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Nerve Agents. Tabun (GA) CAS 77-81-6; Sarin (GB) CAS 107-44-8; Soman (GD) CAS 96-64-0; and VX CAS 5078269-9.

8. Sidell, FR. Nerve Agents. Zajtchuck R, Bellamy RF, Sidell FR, Takafuki ET, Franz DR, editors. Textbook of Military Medicine Part I. Warfare, Weaponry, and the Casualty. Medical Aspects of Chemical and Biological Warfare. 1st Edition. Washington, DC: Borden Institute, Walter Reed Medical Center: 1997: 129-179.

9. Centers for Disease Control and Prevention. FAQs about Nerve Agents. Last reviewed November 5, 2001. Available at: www.bt.cdc.gov/Documents…FAQNerveAgents.asp?link=1&page=che.

10. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Phosgene ($COCl_2$) CAS 75-44-5; UN 1076.

11. .Urbanetti, JS. Toxic Inhalational Injury. In: Zajtchuck R, Bellamy RF, Sidell FR, Takafuki ET, Franz DR, editors. Textbook of Military Medicine Part I. Warfare, Weaponry, and the Casualty. Medical Aspects of Chemical and Biological Warfare. 1st Edition. Washington, DC: Borden Institute, Walter Reed Medical Center: 1997: 247-270.

12. Centers for Disease Control and Prevention. FAQs about Pulmonary Agents. Last reviewed November 5, 2001. Available at: www.bt.cdc.gov/Docum…FAQPulmonaryAgents.asp?link=3&page=che.

13. Hazardtext Managements. Phosgene. MICROMEDEX Thomson Healthcare Series Vol. 112 expires 6/2002.

14. Hazardtext Managements. Lewisite. MICROMEDEX Thomson Healthcare Series Vol. 112 expires 6/2002.

15. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Blister Agents. Lewisite (L) $C_2H_2AsCl_3$) CAS 541-25-3, UN 1556; and Mustard-Lewisite Mixture (HL) CAS Number not available, UN 2810.

16. MeditextManagements. Mustard Gas. MICROMEDEX Thomson Healthcare Series Vol. 112 expires 6/2002.

17. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Blister Agents. Sulfur Mustard Agent H or HD
$C_4H_8Cl_2S$) CAS 505-60-2, UN 2927; and Sulfur Mustard Agent HT CAS 6392-89-8.

18. Sidell, FR, Urbanetti, JS, Smith WJ, Hurst CG, Vesicants. In: Zajtchuck R, Bellamy RF, Sidell FR, Takafuki ET, Franz DR, editors. Textbook of Military Medicine Part I. Warfare, Weaponry, and the Casualty. Medical Aspects of Chemical and Biological Warfare. 1st Edition. Washington, DC: Borden Institute, Walter Reed Medical Center: 1997: 197-228.

19. U.S. Department of Health and Human Services. Agency for Toxic Substances and Disease Registry. Blister Agents. Nitrogen Mustard (HN-1)
$C_6H_{13}Cl_2N$) CAS 538-07-8, UN 2810; Nitrogen Mustard (HN-2) ($C_5H_{11}Cl_2N$) CAS 51-75-2, UN 2927; and Nitrogen Mustard (HN-3) ($C_6H_{12}Cl_3N$)
CAS 555-77-1, UN 2810.

SOME POTENTIAL CHEMICAL TERRORISM AGENTS AND SYNDROMES (including biologic toxins)

Agents	Symptom Onset	Symptoms	Signs	Clinical Diagnostic Tests	Decon-tamin-ation	**Exposure route** and treatment (adult dosages)	Differential diagnostic considerations
Nerve agents	Vapor: seconds Liquid: minutes to hours	**Moderate exposure** : Diffuse muscle cramping, runny nose, difficulty breathing, eye pain, dimming of vision, sweating, **High exposure** : The above plus sudden loss of consciousness, flaccid paralysis, seizures	Pinpoint pupils (miosis) Hyper-salivation Diarrhea Seizures	Red Blood Cell or serum cholinesterase (whole blood) **Treat for signs and symptoms; lab tests only for later confirmation** Collect urine for later confirmation and dose estimation	Rapid disrobing Water wash with soap and shampoo	**Inhalation & dermal absorption** Atropine (2mg) iv or im (titrate to effect up to 6 to 15 mg) 2-PAMCI 600mg injection or 1.0 g infusion over 20-30 minutes Additional doses of atropine and 2-PAMCI depending on severity, Diazepan or lorazepam to prevent seizures if >4 mg atropine given Ventilation support	Pesticide poisoning from organophosphorous agents and carbamates cause virtually identical syndromes
Cyanide	Seconds to minutes	**Moderate exposure:** Dizziness, nausea, headache, eye irritation **High exposure** : Loss of consciousness	**Moderate exposure:** non-specific findings **High exposure** : convulsions, cessation of respiration	Cyanide (blood) or thiocyanate (blood or urine) levels in lab. **Treat for signs and symptoms; lab tests only for later confirmation**	Clothing removal	**Inhalation & dermal absorption** Oxygen (face mask) Amyl nitrite Sodium nitrite (300mg iv) and sodium thiosulfate (12.5g iv)	Similar CNS illness results from: Carbon monoxide (from gas or diesel engine exhaust fumes in closed spaces) H_2S (sewer, waste, industrial sources)
Blister Agents	2-48 hours	Burning, itching, or red skin Mucosal irritation (prominent tearing, and burning and redness of eyes) Shortness of breath Nausea and vomiting	Skin erythema Blistering Upper airway sloughing Pulmonary edema Diffuse metabolic failure	Often smell of garlic, horseradish, and mustard on body Oily droplets on skin from ambient sources No specific diagnostic tests	Clothing removal Large amounts of water	**Inhalation & dermal absorption** Thermal burn type treatment Supportive care For Lewisite and Lewisite/Mustard mixtures: British Anti-Lewisite (BAL or Dimercaprol)	Diffuse skin exposure with irritants, such as caustics, sodium hydroxides, ammonia, etc., may cause similar syndromes. Sodium hydroxide (NaOH) from trucking accidents
Pulmo-nary agents (pho-sgene etc)	1 – 24 (rarely up to 72 hours)	Shortness of breath Chest tightness Wheezing Mucosal and dermal irritation and redness	Pulmonary edema with some mucosal irritation (more water solubility = more mucosal irritation)	No tests available but source assessment may help identify exposure characteristics (majority of trucking incidents generating exposures to humans have labels on vehicle)	None usually needed	**Inhalation** Supportive care Specific treatment depends on agents	Inhalation exposures are the single most common form of industrial agent exposure (eg: HCl, Cl_2, NH_3) Mucosal irritation, airways reactions, and deep lung effects depend on the specific agent, especially water-solubility
Ricin (castor bean toxin)	18 – 24 hours	**Ingestion:** Nausea, diarrhea, vomiting, fever, abdominal pain **Inhalation:**, chest tightness, coughing, weakness, nausea, fever	Clusters of acute lung or GI injury; circulatory collapse and shock	ELISA (from commercial laboratories) using respiratory secretions, serum, and direct tissue	Clothing removal Water rinse	**Inhalation & Ingestion** Supportive care For ingestion: charcoal lavage	Tularemia, plague, and Q fever may cause similar syndromes, as may CW agents such as Staphylococcal enterotoxin B and phosgene
T-2 myco-toxins	2-4 hours	Dermal & mucosal irritation, blistering, and necrosis Blurred vision, eye irritation Nausea, vomiting, and diarrhea Ataxia Coughing and dyspnea	Mucosal erythema and hemorrhage Red skin, blistering Tearing, salivation Pulmonary edema Seizures and coma	ELISA from commercial laboratories Gas chromatography/Mass spectroscopy in specialized laboratories	Clothing removal Water rinse	**Inhalation & dermal contact** Supportive care For ingestion: charcoal lavage Possibly high dose steriods	Pulmonary toxins (O_3, NO_x, phosgene, NH_3) may cause similar syndromes though with less mucosal irritation.

Produced by the Employee Education System for the Office of Public Health and Environmental Hazards, Department of Veterans Affairs