The Riegle Report

U.S. Chemical and Biological Warfare-Related Dual Use Exports to Iraq and their Possible Impact on the Health Consequences of the Gulf War

A Report of Chairman Donald W. Riegle, Jr. and Ranking Member Alfonse M. D'Amato of the Committee on Banking, Housing and Urban Affairs with Respect to Export Administration

United States Senate, 103d Congress, 2d Session
May 25, 1994

Table of Contents

Introduction

Chapter 1. Iraqi Chemical and Biological Warfare Capability

Part 1

- Status of Iraqi Readiness to Use Chemical Weapons Against Coalition Force
- Destruction of Iraq's Chemicals and Chemical Weapons by the United Nations
- Chemical Warfare Doctrine and the Use of Combined Agent Warfare
- Chemical Nerve Agents
    - Sarin (GB)
    - Soman (GD)
    - Tabun (GA)
    - VX

Part 2

- Vesicants and Blood Agents
    - Lewisite
    - Cyanogen Chloride
    - Hydrogen Cyanide
- Blister Agents
    - Mustard Gas
- Related Chemical Agent Information
- Biotoxins

- Biological Warfare Capability
- U.S. Exports of Biological Materials to Iran
- UNSCOM Biological Warfare Inspections

Part 3

- Biological Warfare Defense
  - Types of Biological Agents
  - Dissemination of Biological Agents
  - Defensive Measures
- Iraq's Experience in the Use of Chemical Warfare Agents
- Gulf War Syndrome: The Case for Chemical/Biological Agent Exposure

Chapter 2. Group I Exposures: Reported Direct Exposure Events

Part 1.

Part 2.

Part 3.

Part 4: Reports by Coalition Forces of Iraqi Chemical Mines Located During Breaching Operations, Other Combat-Related Reports, and Conclusions

Chapter 3. Reports of Exposure of Coalition Forces Resulting from the Fallout of the Bombings of Iraqi Chemical, Biological, and Nuclear Facilities (Group II)

Part 1.

- The Czechoslovak Chemical Defense Unit in the Persian Gulf and the Results of the Investigations of the Military Use of Poisonous Gases
- Other Related Information
- U.S. Unofficial Reports of Downwind Exposure Due to Coalition
- Bombings of Iraqi Chemical and Biological Facilities
- Weather Reports, Climatic Information, and Imagery Smoke Plume Data

Part 2: Gulf War Weather.

Part 3: Gulf War Weather (Continued).

Part 4: Gulf War Weather (Continued).

Part 5: Conclusions.

Chapter 4. Other Identifiable Exposures

Part 1

- Chemical/Biological Warfare Pre-Treatment Drug Reaction
  - Anthrax and Botulinum Toxoid Vaccines
  - Pyridostigmine Bromide (Group III)
- Reported Contact with Iraqi Enemy Prisoners of War
- Chemical Agent Resistant Coating (CARC)>
- Depleted Uranium Ammunition
- Environmental Exposures
- Decontamination of Equipment Returned from the Persian Gulf
- Theater Operations
- Transmission

Part 2

- Conclusions

- Chemical/Biological Warfare Agent Exposure: Why Wasn't Everyone Affected?
- Chemical/Biological Warfare Agent Exposure: Did the Military Know or Suspect that Individuals Were Exposed to these Hazardous Substances?
- The Need for Immediate Primary Scientific Research and Advanced Medical Research
- Conclusions

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - Tabun (GA)
  - Sarin (GB)
  - Soman (GD)
  - VX
- Blister Agents
  - Sulfur Mustard (HD, THD)
  - Sulfur Mustard (HT) (No Data Available)

This report is derived entirely from unclassified sources.

Approved for public release. Unlimited distribution is authorized.

U.S. Senate Committee on Banking, Housing, and Urban Affairs.

INTRODUCTION

In October 1992, the Committee on Banking, Housing, and Urban Affairs, which has Senate oversight responsibility for the Export Administration Act (EAA), held an inquiry into the U.S. export policy to Iraq prior to the Persian Gulf War. During that hearing it was learned that U.N. inspectors identified many U.S. - manufactured items exported pursuant to licenses issued by the U.S. Department of Commerce that were used to further Iraq's chemical and nuclear weapons development and missile delivery system development programs.

On June 30, 1993, several veterans testified at a hearing of the Senate Committee on Armed Services. There, they related details of unexplained events that took place during the Persian Gulf War which they believed to be chemical warfare agent attacks. After these unexplained events, many of the veterans present reported symptoms consistent with exposure to a mixed agent attack. Then, on July 29, 1993, the Czech Minister of Defense announced that a Czechoslovak chemical decontamination unit had detected the chemical warfare agent Sarin in areas of northern Saudi Arabia during the early phases of the Gulf War. They had attributed the detections to fallout from coalition bombing of Iraqi chemical warfare agent production facilities.

In August 1993, Senate Banking Committee Chairman Donald W. Riegle Jr. began to research the possibility that there may be a connection between the Iraqi chemical, biological, and radiological warfare research and development programs and a mysterious illness which was then being reported by thousands of returning Gulf War veterans. In September 1993, Senator Riegle released a staff report on this issue and introduced an amendment to the Fiscal Year 1994 National Defense Authorization Act that provided preliminary funding for research of the illnesses and investigation of reported exposures.

When this first staff report was released by Senator Riegle, the estimates of the number of veterans suffering from these unexplained illnesses varied from hundreds, according to the Department of Defense, to thousands, according to the Department of Veterans Affairs. It is now believed that tens of thousands of U.S. veterans are suffering from a myriad of symptoms collectively labelled either Gulf War Syndrome, Persian Gulf Syndrome, or Desert War Syndrome. Hundreds and possibly thousands of servicemen and women still on active duty are reluctant to come forward for fear of losing their jobs and medical care. These Gulf War veterans are reporting muscle and joint pain, memory loss, intestinal and heart problems, fatigue, nasal congestion, urinary urgency, diarrhea, twitching, rashes, sores, and a number of other symptoms.

They began experiencing these multiple symptoms during and after -- often many months after -- their tour of duty in the Gulf. A number of the veterans who initially exhibited these symptoms have died since returning from the Gulf. Perhaps most disturbingly, members of veteran's families are now suffering these symptoms to a debilitating degree. The scope and urgency of this crisis demands an appropriate response.

This investigation into Gulf War Syndrome, which was initiated by the Banking Committee under the direction of Chairman Riegle, has uncovered a large body of evidence linking the symptoms of the syndrome to the exposure of Gulf War participants to chemical and biological warfare agents, chemical and biological warfare pre-treatment drugs, and other hazardous materials and substances. Since the release of the first staff report on September 9, 1993, this inquiry has continued. Thousands of government officials, scientists, and veterans have been interviewed or consulted, and additional evidence has been compiled. This report will detail the findings of this ongoing investigation.

Since the Banking Committee began its inquiry, the position of the Department of Defense regarding the possible causes of Gulf War Syndrome has altered only when challenged with evidence that is difficult to dispute. Yet, despite the vast resources of the Department of Defense, several independent and congressional inquiries with limited resources continue to uncover additional evidence of hazardous exposures and suspicious events.

The Department of Defense, when first approached regarding this issue by Committee staff, contended that there was no evidence that U.S. forces were exposed to chemical warfare agents. However, during a telephone interview on September 7, 1993 with Walter Reed Army Medical Center commander Major General Ronald Blanck, Committee staff was informed that the issue of chemical and biological warfare agent exposure had not been explored because it was the position of "military intelligence" that such exposure never occurred.

Then, during a November 10, 1993 press briefing at the Pentagon, the Department of Defense acknowledged that the Czech government did detect chemical agents in the Southwest Asia theater of operations. After analyzing the results of the Czech report, the Department of Defense concluded that the detections were unrelated to the "mysterious health problems that have victimized some of our veterans." According to former Secretary of Defense Les Aspin, in some cases the wind was wrong and the distances too great to suggest a link. For instance, Seabees serving to the south and east of the detection site have complained of persistent health problems; but according to the Pentagon, the wind was blowing in the other direction at the time of the detections and the concentrations were too low to do harm over that kind of a distance.

The fact is, no one has ever suggested that there was a link between the Czech detections and what occurred during the early morning hours of January 19, 1991 near the Port of Jubayl. (These two events will be described in detail in Chapter 2.) Former Defense Secretary Aspin said

at the briefing that this incident could not have been from the Coalition bombings of the Iraqi chemical weapons facilities because the winds were blowing to the northwest. Yet according to available Soviet documents, the dispersal of chemical agents and other hazardous substances is controlled by other factors in addition to surface wind direction and velocity, such as topography, temperature, precipitation, vertical temperature gradient, and atmospheric humidity. These factors all contribute to the size and type of dispersal that will be observed. Unclassified visual and thermal satellite imagery confirms that the fallout from the bombings of Iraqi targets during the air and ground war moved to the southeast, with the weather patterns and upper atmospheric wind currents, towards Coalition force positions. (See Chapter 3.)

According to a knowledgeable source who has requested confidentiality, the Czechs believed that the detections were caused by the weather inversion which occurred that day (January 19, 1991) as the weather front moved southward. The Czechoslovak chemical detection unit reported this information to U.S. command officials immediately, but the responding units were unable to confirm their findings when they arrived, according to the Pentagon. Nonetheless, at the November 10, 1993 briefing, the Department of Defense admitted that the Czech detections were believed to be valid. The Department of Defense failed to disclose that the Czechoslovak chemical detection team also detected yperite (HD) that morning. The presence of both of these agents in such close proximity could only reasonably be the result of one of two possibilities: (1) direct Iraqi mixed agent attack, or (2) fallout from the Coalition bombings of Iraqi weapons facilities and storage bunkers.

Defense Department officials, having had possession of the Czech report for over a month, were at a loss to explain the chemical mustard agent detected by the Czechoslovak chemical detection team in the Saudi desert near King Khalid Military City on January 24, 1991. This despite the fact that both the Czechs and French claim that this detection was reported to U.S. command authorities during the Persian Gulf War. Additionally, during the Gulf War, the Czechs claimed that they detected Chemical nerve agent after a Scud missile attack. These statements, heretofore only reported in the press, have been confirmed by a member of the U.S. 1st Cavalry Division and by an entire platoon of a U.S. Army chemical detection unit who trained with the Czechoslovak Chemical detection unit near King Khalid Military City. These reports have not been addressed publicly by the Department of Defense and will be addressed in this report in Chapter 3.

The contents of this report supports the conclusion that U.S. forces were exposed to some level of chemical and possibly biological warfare agents during their service in the Gulf War. Any review conducted by the Pentagon must extend far beyond the information being reported by the Czech Ministry of Defense. The Czech information, while important, represents just a small fraction of the evidence currently available, only some of which will be detailed in this report.

It is now the position of the Department of Defense that it has no other evidence that U.S. forces were exposed to chemical agents. Yet this report contains descriptions and direct eyewitness

accounts that provide evidence which suggest that gas was detected, along with many other events which may have been actual attacks on U.S. forces.

This report supports the conclusion that U.S. forces were exposed to chemical agents. The assertion that the levels of nerve agents detected by the Czechs and others were not harmful is flawed. In subsequent requirements for chemical detection equipment, the Department of the Army acknowledges that the principal chemical agent detection alarm deployed during the war, the M8A1 was not sufficiently sensitive to detect sustained low levels of chemical agent and to monitor personnel for contamination. Further, U.S. Army Material Safety Data Sheets (MSDS) indicate that chronic exposures to levels of over .0001 mg/m3 for Sarin (GB) is hazardous and requires the use of protective equipment. (See appendix A.) The minimum level of chemical agent required to activate chemical agent detection alarm M8A1, commonly in use during the war, exceeds this threshold by a factor of 1,000. (See appendix A.) As the chemical agent alarms began to sound during the "air war," French, Czech, and many U.S. commanders confirmed that they were sounding from the fallout from the bombings. Over time, even at these levels, after repeatedly being told that there was no danger, many U.S. forces failed to take precautionary measures. Other report that the alarms were sounding so frequently that they were turned off. M8A1 alarms do not detect blister agents.

The findings of this report prepared at the request of Chairman Riegle detail many other events reported by U.S. servicemen and women that in some cases confirm the detection of chemical agents by U.S. forces. In other cases these reports indicate the need for further detailed investigation. But still the question remains: Is exposure to these and other chemical agents the cause of Gulf War Syndrome? We have received hundreds of reports that many of these symptoms are being experienced by family members. Numerous developments have taken place over the last several months which suggest that, while chemical agents and other environmental hazards may have contributed to the Gulf War illnesses, bacteriological, fungal, and possibly other biological illnesses may be the fundamental cause. This position is supported by the following.

First, Dr. Edward S. Hyman, a New Orleans bacteriologist, has treated a small number of the sick veterans and several of their wives for bacteriological infections, and has developed a protocol of treatment that has resulted in symptom abatement in many of his patients.

Second, during the November 10, 1993 unclassified briefing for Members of the U.S. Senate, in response to direct questioning, then Undersecretary of Defense John Deutch said that the Department of Defense was withholding classified information on the exposure of U.S. forces to biological materials. In a Department of Defense- sponsored conference on counter-proliferation, held at Los Alamos National Laboratory on 6-7 May, 1994, Dr. Deutch admitted that biological agent detection is a priority development area for the Department of Defense, since there currently is no biological agent detection system fielded with any U.S. forces anywhere in the world.

Third, the Department of Defense has named Dr. Joshua Lederberg to head its research team into the causes of Gulf War illnesses. Dr. Lederberg, among his other credits, is a Nobel Laureate and an expert in the fields of bacteriology, genetics, and biological warfare defenses.

Fourth, in detailed informational interviews conducted of 400 Gulf War veterans, it has been learned that over 3/4 of their spouses complain that they have begun to suffer from many of the same debilitating symptoms. (See Chapter 4.)

This report, like the one which preceded it, will discuss the relationship between the high rate of Gulf War illnesses among Group I individuals (those possibly exposed to a direct mixed agent event), and the lower rates among those in Group II (individuals exposed to the indirect fallout from coalition bombings of Iraqi chemical, biological, and nuclear targets) and Group III (individuals who suffered severe adverse reactions to the nerve agent pre-treatment pills). Despite the varying rates of illness between the groups, however, the symptoms are similar. While other possible causes of the Gulf War Syndrome such as petrochemical poisoning, depleted uranium exposures, and regionally prevalent diseases, have been discussed elsewhere and must be pursued, there is a great deal of compelling evidence which indicates that all of these possibilities must now be seriously considered. We believe, however, that no other explanations prove as compelling as the ones which will be presented in this report.

This report includes a great number of first-hand accounts and other documentary evidence in addition to the anecdotal information that appeared in the print and electronic media during the Gulf War. It establishes convincingly that the Department of Defense assertions are inaccurate. We believe there is reliable evidence that U.S. forces were exposed to chemical and possibly biological agents. But regardless of whether U.S. forces were exposed or not, the entire official body of information, including all classified or heretofore unpublished information, available research data sets, case histories, and diagnostic breakdown information must be made available to independent civilian medical researchers in order to further the research into the causes of and treatments for these illnesses. Absent a release of information by the Department of Defense of the science which forms the bases for their theories, the Department of Defense position must be viewed by qualified scientists as anecdotal and unsubstantiated.

Given that there is also a growing body of evidence indicating that spouses and children of Gulf War veterans are vulnerable to similar illnesses, the Department of Defense must now share all of its information with civilian, non-governmental researchers. These family members are civilians who may be at risk. This illness was first reported over three years ago.

On February 9, 1994, Chairman Riegle sent a letter to Secretary of Defense William Perry asking that he release all U.S. military personnel from any oath of secrecy they may have taken regarding classified information specifically pertaining to chemical or biological warfare agent exposure in the Persian Gulf theater. This request was based on a recommendation of the National Academy of Sciences, National Institute of Medicine in their 1993 publication Veterans at Risk: The Health Effects of Mustard Gas and Lewisite. On May 4, 1994, the Secretaries of

Defense, Health and Human Services, and Veterans Affairs responded to the Chairman's letter stating that there was no classified information on chemical or biological detections or exposures. This directly contradicts the statement of Deputy Secretary Deutch in his November 10, 1993 unclassified briefing to Members and staff.

Why isn't the Department of Defense aggressively pursuing the answers to the questions surrounding of the events which may have caused illnesses being suffered by many Gulf War veterans? One possible explanation lies in a 1982 article. Then Senate Armed Services Committee Chairman John Tower wrote, "Chemical training in the United States armed forces is, at best, perfunctory. It is rarely conducted in a simulated contaminated environment and stocks of individual protective equipment are too limited, and therefore too valuable, to risk them in the numbers necessary to allow troops to operate in them for realistic training. As a result, most U.S. personnel are relegated to a minimal and highly artificial exposure to the problems and hardships entailed in performing their respective combat missions should they have to 'button up'." As numerous U.S. General Accounting Office (GAO) reports have noted, the U.S. was not much better prepared prior to the Gulf War than it was when Senator Tower wrote in his article.

Chapter 1. Iraqi Chemical and Biological Warfare Capability (Part 1)

Status of Iraqi Readiness to Use Chemical Weapons Against Coalition Force
Destruction of Iraq's Chemicals and Chemical Weapons by the United Nations
Chemical Warfare Doctrine and the Use of Combined Agent Warfare
Chemical Nerve Agents

Over the last ten years, Iraq, a signatory to both the Geneva Protocols of 1925 (prohibiting the use of poisoned gas) and the Biological Warfare Convention of 1972 (banning biological weapons), has expended an enormous amount of research and energy in developing these and other prohibited weapons.

Iraq was believed to have been manufacturing mustard gas at a production facility in Samarra since the early 1980s. It also began an extensive program to produce nerve agent precursor chemicals, taking advantage of its own natural resources. Phosphate mines/industries are at Akashat, Al Qaim, and Rutbah. The Iraqi Al Fallujah gas warfare complex was believed to be capable of producing up to 1,000 tons per month of Sarin, as well as the nerve agent VX. In addition, with the assistance of foreign firms, Iraq developed the capability to experiment with hydrogen cyanide, cyanogen chloride, and lewisite. By the start of the Gulf War, Iraqi forces had developed chemical delivery capabilities for rifle grenades, 81mm mortars, 152mm, 130mm, and 122mm artillery rounds; bombs; 90mm air-to-ground rockets; 216 kilogram FROG and 555 kilogram SCUD warheads; and possibly land mines and cruise missiles.

On July 30, 1991, Ambassador Rolf Ekeus, director of the United Nations Special Commission on Iraq (UNSCOM), charged with overseeing the elimination of Iraq's chemical and nuclear arsenals, told the Security Council that the U.N. inspectors had found chemical warheads armed with nerve gas. Mr. Ekeus claimed that some warheads found were already fitted onto the SCUD missiles.

Iraq's chemical warfare capability was known to the U.S. government before the war. A month before the war began, then Central Intelligence Agency (CIA) director William Webster estimated that Iraq possessed 1,000 tons of poisonous chemical agents, much of it capable of being loaded into two types of missiles: the FROG (Free Rocket Over Ground) and the SCUD B(SS-1). Jane's Strategic Weapons Systems lists warhead capabilities for the FROG-7 as high explosive (HE), chemical, or nuclear, and for the Iraqi versions of the SCUD as probably HE or chemical.

Status of Iraqi Readiness to Use Chemical Weapons Against Coalition Forces

In March 1991, Molly Moore reported from Jubayl, Saudi Arabia that Marine Commanders found no indications of chemical weapons stockpiles on the battlefields of Kuwait. According to a Washington Post report that day, (March 7, 1991), U.S. intelligence analysts claimed that these weapons "never got distributed down to the battlefield" from storage sites north of the Euphrates River. A U.S. military intelligence source stated in March 1991 that "it was a matter of not deploying chemical weapons, rather than not having them, ...my guess .... is they never managed to get it down to division level."

Regarding the presence of chemical weapons and Iraqi readiness to use them against Coalition forces, Committee staff has received the following information:

Dale Glover, of the 1165th Military Police Company, was with the 7th Corps, approximately 75 miles inside Iraq, when they came upon a destroyed artillery site. They entered a bunker that was half uncovered by the bombing. Inside there was a very strong ammonia smell. They discovered leaking chemical munitions inserts packed inside aluminum casings. A test confirmed a blister agent. They went back to their unit and reported what they had found. Mr. Glover recalled that "they didn't get back to us for 2- 3 hours, then told us it was a false positive, nothing to be concerned about." However, he said, within hours they were ordered to move from the location where they were camped, about three miles from the bunker. Mr. Glover recalled that they had been at that position only a couple of weeks and had not expected to move that soon. When questioned if the site they discovered was south of the Euphrates, he confirmed that it was.

Another source who identified himself to the Committee but wishes to remain anonymous has informed Committee staff that he also was with the 7th Corps in southern Iraq. Somewhere between As Salman and Bashra (in a position south of the Euphrates River), his unit came upon bunkers containing crates of substances that "made you choke, made you want to throw up, burned your eyes. It smelled like ammonia, only a lot stronger." He could not approach the crates without experiencing immediate breathing problems. He said these crates were leaking.

Chris Alan Kornkven was a Staff Sergeant with the 340th Combat Support Company during the Persian Gulf War. He reported to Committee staff that a U.S. military doctor at the 312th Evacuation Hospital told him that doctors at the hospital had been speaking with Iraqi officers. According to these doctors, the Iraqi officers said that they had chemical weapons at the front, and had authorization to use them, but that the winds in their area were blowing the wrong way.

Several press sources carried reports of encounters with chemical mines by the 2nd Marine Division during the initial mine field breaching operation early on February 24, 1991. According to the Chicago Tribune, which interviewed officers and enlisted Marines involved in the operation, a FOX vehicle confirmed positive readings for a nerve agent and for a mustard gas. A second detecting device gave the same positive reading. General Keys, the 2nd Division commander, and Colonel Livingston, commander of the 6th Marine Regiment, told reporters they believed it was possible that a chemical mine was blown up or hit. General Schwarzkopf told reporters he considered the reports "bogus."

British troops also discovered Iraqi chemical mines on the Gulf battlefield, according to Gannett News Service. A British official (not further identified) said the incident was reported to Prime Minister John Major's war cabinet; no details were given.

Press reports indicate Iraqi readiness to use these weapons against Coalition forces. The British Sunday Times reported on January 27, 1991, that American intelligence detected greatly increased activity at Iraq's main chemical plant at Samarra in the last week of December, and the British Ministry of Defense said that the Allies believe that Iraq "may have as many as 100,000 artillery shells filled with chemicals and several tons (of bulk agent) stored near the front line." According to the Times report, a British Ministry of Defense official said: "The plant was at peak activity and the chemicals were distributed to the troops in Kuwait and elsewhere in theatre." The Times reported that an unnamed Pentagon source said that Hussein had given front-line commanders permission to use these weapons at their discretion, and that "it was no longer a question of if, but when."

Iraqi soldiers captured by the British units also informed the allies that before the war started, Iraq distributed substantial supplies of chemical weapons along the front lines to be held for the ground war. According to Newsweek, U.S. intelligence sources had reported that Saddam Hussein had ordered his commanders to fire chemical weapons as soon as the allies launched a ground offensive. A British signals officer was reported to have said that "we were tuned into the Iraqi command radio net. We heard them give the release order to their front-line troops to use chemical weapons against Rhino Force if it crossed the border."

Destruction of Iraq's Chemicals and Chemical Weapons by the United Nations

In April 1993, weapons inspectors from the United Nations charged with locating all of Iraq's nuclear, chemical and biological weapons by U.N. Resolution 687, confirmed that in Muthanna, 65 miles northwest of Baghdad, Iraq manufactured a form of mustard gas as well as Sarin and Tabun, both nerve agents. This vast desert complex was the nucleus of Iraq's chemical weapons program. During the allied bombing in the early days of the Gulf War, Muthanna was a priority

target. It was repeatedly attacked and production sites were destroyed. As United Nations inspectors attempted to destroy Iraq's chemical weapons arsenal, they discovered bombs, missiles, and chemical weapons of mass destruction spread out across this immense complex. Of particular concern were the chemical warheads of Al-Hussein modified SCUD missiles, each filled with five gallons of Sarin. Twenty-eight of these warheads have been drained and destroyed by the U.S. inspectors. These weapons were not destroyed during the bombings at Muthanna because they had been removed to other locations before the Gulf War started. Their relocation and transfer back to Muthanna was described by U.N. inspectors as a painstaking process. According to Brigadier General Walter Busbee, U.S. Army Chemical and Material Destruction Agency, Aberdeen Proving Grounds, these warheads were exported to Iraq from the former Soviet Union.

Chemical warfare agents which either survived the allied bombing or were inventoried and returned to the Muthanna facility for destruction include:

- 13,000 155-mm artillery shells loaded with mustard gas;
- 6,200 rockets loaded with nerve agent;
- 800 nerve agent aerial bombs;
- 28 SCUD warheads loaded with Sarin;
- 75 tons of the nerve agent Sarin;
- 60-70 tons of the nerve agent tabun; and,
- 250 tons of mustard gas and stocks of thiodiglycol, a precursor chemical for mustard gas.

U.N. inspectors have concluded that the Muthanna plant was capable of producing two tons of Sarin and five tons of mustard gas daily. The plant was also capable of manufacturing VX, a nerve gas and one of the most toxic chemicals ever produced.

In addition to Muthanna, chemical agents were destroyed at two airbases: one located 40 miles west of Baghdad and the other located near An Nassiriyah, where a number of 122mm rockets loaded with Sarin (GB) were blown in place. According to UNSCOM sources, many of these weapons were hastily deployed prior to the air war and later returned for destruction. The U.N. has destroyed hundreds of tons of bulk chemical agents and tens of thousands of chemical munitions. In addition, hundreds of thousands of liters of key chemical precursors which have been identified and destroyed include: 14,600 liters of DF; 121,000 liters of D4 and 153,983 liters of thiodiglycol. According to UNSCOM, the Iraqis were capable of employing both binary and mixed agent weapons. Binary weapons identified used DF. When combined with appropriate chemicals, GB and GF are produced.

UNSCOM also discovered, at various locations, evidence of research into certain biological agents, including botulinus toxin, anthrax, an organism responsible for gas gangrene (clostridium perfringens) and others as identified below. The evidence discovered by the group suggested that this was primarily an offensive biological warfare program.

On February 13, 1994, a clandestine radio service in Iraq, the Voice of the Iraqi People, reported that Saddam Hussein's government was still attempting to hide chemical and biological weapons from international inspectors by repeatedly relocating them. Citing unidentified individuals, the radio reported that the banned weapons were being hidden in the oil pipelines that have been "out of operation because of the international embargo."

Chemical Warfare Doctrine and the Use of Combined Agent Warfare

There is substantial evidence to suggest that in the use of chemical weapons, as in other military areas, the Iraqi military adhered, at least in part, to Soviet military doctrine. Soviet military doctrine suggested that chemical warfare should be conducted with mixed agents. Mixed agents, often referred to as "cocktails," are intended to enhance the capabilities of nerve agents and defeat the precautions taken by the enemy. Use of mixed agents could account for the wide variety of symptoms displayed by the Gulf War veterans. Mixed agents can be made by combining a variety of biotoxins, nerve agents, vesicants, blister agents and some biological agents -- such as bacteria and fungi, and others described briefly below.

According to some sources, Iraq used mixed agent weapons combining cyanogen, mustard gas, and tabun against the Kurds. Saddam Hussein stated on April 2, 1990, that Iraq had "double combined chemical" weapons since the last year of the Iran-Iraq War. It was also believed that in 1984 Iraq may have used mixed agent weapons with biological tricothecenes and mycotoxins against Majnoon Island during the Iran-Iraq War.

The utility of chemical weapons and the possibility of exposing one's own troops to indirect chemical weapons effects is an issue which has been seriously debated by both U.S. and Soviet military planners. Soviet doctrine questions the utility of initiating chemical warfare, since chemical weapons produce secondary effects that could obstruct troop advances. U.S. Military doctrine warns that according to its calculations, the use of a nerve agent against a target area of no more than a dozen hectares (a hectare is about 2.47 acres) can, under certain weather conditions, create a hazard zone downwind of up to 100 kilometers in length. Within this downwind area, friendly military units would have to take protective measures.

According to the official military announcements made in the last half of January 1991 and based on the quantity of chemical agents observed by UN inspectors after the war, the scope of coalition bombing against these facilities involved hundreds -- if not thousands -- of tons of bulk chemical nerve agents, mustard gas, as well as tens of thousands of pieces of chemical

munitions. This quantity of chemical warfare agents vastly exceeds the amounts that might be expected to be deployed by a military force in a single chemical attack.

The dispersal of the chemical agents and other hazardous substances is controlled by factors such as topography, wind velocity, direction, temperature, precipitation, vertical temperature gradient and atmospheric humidity. These factors all contribute to the size and type of dispersal pattern which will be observed. In addition, as confirmed by unclassified U.S. satellite imagery, debris from the Coalition bombings were upwardly dispersed, rather than downwardly dispersed as would occur in offensive use, causing chemical agents to be carried by upper atmospheric currents and distributed as "traces" of chemical fallout over "down weather" positions. Czech and French officials confirmed the detections of these chemicals during the war. (See Chapter 3.)

In considering the consequences of the placement of troops in areas downwind (where non-lethal exposure to chemical warfare agents might be expected), it must be remembered that chemical nerve agents, such as Sarin and Soman and other agents, have cumulative effects -- often explained as slow rates of detoxification.

Chemical Nerve Agents

Nerve agents kill by disrupting the metabolic processes, causing a buildup of a chemical messenger (acetylcholine) by inhibiting the production of acetylcholine-esterase, a key regulator of neurotransmission. Lethal exposure to chemical nerve agents is generally characterized by drooling, sweating, cramping, vomiting, confusion, irregular heart beat, convulsions, loss of consciousness and coma.

According to a material safety data sheet (MSDS) for Soman (GD), and VX prepared by the U.S. Army Chemical Research, Development and Engineering Center, Aberdeen Proving Grounds, Maryland, "the inhibition of cholinesterase enzymes throughout the body by nerve agents is more or less irreversible so that their effects are prolonged. Until the tissue cholinesterase enzymes are restored to normal activity, probably by very slow regeneration over a period of weeks or 2 to 3 months if damage is severe, there is a period of increased susceptibility to the effects of another exposure to any nerve agent. During this period the effects of repeated exposures are cumulative; after a single exposure, daily exposure to concentrations of nerve agent insufficient to produce symptoms may result in the onset of symptoms after several days. Continued daily exposure may be followed by increasingly severe effects. After symptoms subside, increased susceptibility persists for one to several days. The degree of exposure required to produce recurrence of symptoms, and the severity of these symptoms depend on duration of exposure and time required to produce recurrence of symptoms, and the severity of these symptoms depend on the duration of exposure and the time intervals between exposures. Increased susceptibility is not specific to the particular nerve agent initially absorbed." (See appendix A for MSDS on Soman, Sarin, Tabun, and VX).

Some of the symptoms commonly associated with acute exposure to chemical nerve agents include myosis, frontal headaches, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting, runny nose, tightness in chest, sometimes with prolonged wheezing, expiration suggestive of broncho-constriction or increased secretion and coughing. Following systemic absorption, these symptoms are identified as typical: tightnesss in chest, wheezing, anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness, heartburn, diarrhea, involuntary defecation, increased sweating, increased salivation, increased tearing, slight bradycardia, myosis, blurring vision, urinary urgency and frequency, fatigue, mild weakness, muscular twitching, cramps, generalized weakness, including muscles of respiraton, with dyspnea and cyanosis, pallor and occasional elevation of blood pressure; giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremors, withdrawal and depression; bursts of slow waves of elevated voltage in EEG (especially on over ventilation), drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia, coma (with absence of reflexes), Cheyne-Stokes respirations, convulsions, depression of the respiratory and circulatory centers, with dyspnea, cyanosis and fall in blood pressure.

The majority of automatic chemical agent detection alarms (M8A1) deployed during the war were not sufficiently sensitive for detecting sustained low levels of chemical agent and monitoring personnel for contamination. U.S. Army Material Safety Data Sheets (MSDS) indicate that chronic exposure to levels of over .0001 mg/m3 for Sarin (GB) is hazardous and required the use of protective equipment. (See appendix A). The minimum level of chemical agent required to activate the automatic chemical agent detection alarm M8A1, commonly in use during the war, exceeds this threshold by a factor of 1,000. As the chemical agent alarms began to sound during the "air war," French, Czech, and many U.S. commanders confirmed that they were sounding from the fallout from the bombings. Over time, even at these levels, after repeatedly being told that there was no danger, U.S. forces failed to take precautionary measures. Others report that the alarms were sounding so frequently that they were turned off.

This increased susceptibility associated with prolonged exposures to non-lethal dosages of nerve gases, suggests that the synergistic effects of the fallout from the bombings of the chemical warfare agent facilities and the administration of the cholinesterase inhibiting drug, pyridostigmine bromide, should be further researched as factors contributing to the symptoms being described by the Gulf War veterans.

The following is a listing of a number of agents which the Iraqi government could have combined or which could have been combined in the atmosphere as a result of Coalition bombings:

Sarin (GB) - A colorless and practically odorless liquid, Sarin dissolves well in water and organic solvents. The basic military use of Sarin is as a gas and a persistent aerosol. A highly toxic agent with a clearly defined myopic effect, symtoms of intoxication appear quickly without any period of latent effect. Sarin has cumulative efffects -- that is, a slow rate of detoxification

independent of its method of entry into the body. According to Joachim Krause and Charles K. Mallory in Chemical Weapons in Soviet Military Doctrine: Military and Historical Experience, 1915-1991, the progressive signs of initial Sarin intoxication include myosis (contraction of the pupil), photophobia, difficulty breathing and chest pain.

Soman (GD) - A neuro-paralytic toxic agent. Soman is a transparent, colorless, involatile liquid smelling of camphor. Soluble in water to a limited degree, Soman is absorbed into porous and painted surfaces. Soman is similar to Sarin in its injurious effects, but more toxic. When it acts on the skin in either droplet or vapor form, it causes a general poisoning of the organism.

Tabun (GA) - A neuro-paralytic toxic agent. Tabun is a transparent, colorless liquid. The industrial product is a brown liquid with a weak sweetish smell; in small concentrations, it smells of fruit, but in large concentrations, it smells of fish. Tabun dissolves poorly in water but well in organic solvents; it is easily absorbed into rubber products and painted surfaces. Injury occurs upon skin contact with Tabun vapor and droplets. The symptoms of injury appear almost immediately . Marked myosis occurs.

VX - This colorless, ordorless, liquid has a low volatility and is poorly soluble in water, but dissolves well in organic solvents. The danger of pulmonary VX intoxication is determined by meteorological conditions and the delivery method used. VX is thought to be very effective against respiratory organs when in the form of a thinly dispersed aerosol. The symptoms of VX intoxication are analogous to those of other nerve agents, but their development is markedly slower. As with other nerve agents, VX has a cumulative effect.

Chapter 1, Part 2

[Vesicants and Blood Agents]
[Blister Agents]
[Related Chemical Agent Information]
[Biotoxins]
[Biological Warfare Capability]
[U.S. Exports of Biological Materials to Iraq]
[UNSCOM Biological Warfare Inspections]

Vesicants and Blood Agents

Lewisite - A vesicant toxic agent, industrial lewisite is a dark brown liquid with a strong smell. Lewisite is a contact poison with practically no period of latent effect. Lewisite vapors cause irritation to the eyes and upper respiratory tract. According to the Center for Disease Control, lewisite would cause stinging and burning. Its smell, generally characterized as the strong smell of geraniums, could be confused with the smell of ammonia (the reaction to which is regulated by pain fibers rather than smell). Iraqi stores of lewisite were not located after the war according to the Department of Defense.

Cyanogen Chloride - The French first suggested the use of cyanogen chloride as a toxic agent. U.S. analysts have reported that it is capable of penetrating gas mask filters. Partially soluble in water, it dissolves well in organic solvents. It is absorbed easily into porous materials; its military state is a gas. Cyanogen chloride is a quick acting toxic agent. Upon contact with the eyes or respiratory organs, it injures immediately. Lethal exposures result in loss of consciousness, convulsions and paralysis.

Hydrogen Cyanide - A colorless liquid smelling of bitter almonds, hydrogen cyanide is a very strong, quick acting poison. Hydrogen cyanide affects unprotected humans through the respiratory organs and during the ingestion of contaminated food and water. It inhibits the enzymes which regulate the intra-cell oxidant-restorative process. As a result, the cells of the nervous system, especially those affecting breathing -- are injured, which in turn leads to quick death. An important feature of hydrogen cyanide is the absence of a period of latent effect. The military state of hydrogen cyanide is a gas. The toxic and physiologic properties of hydrogen cyanide permit it to be used effectively in munitions -- predominantly in rocket launched artillery. Death occurs after intoxication due to paralysis of the heart. Non-lethal doses do not cause intoxication.

Blister Agents

According to the material safety data sheet (MSDS) for sulfur mustard gas (HD) prepared by the U.S. Army Chemical Research, Development and Engineering Center, Aberdeen Proving Ground, Maryland, "Chronic exposure to HD can cause skin sensitization, chronic lung impairment, cough, shortness of breath, chest pain, and cancer of the mouth, throat, respiratory tract, skin, and leukemia. It may also cause birth defects. (See appendix A for MSDS sheets on sulfur mustard agents HD and T.) The U.S. Army Chemical and Biological Defense Command lists the current detector sensitivity threshold for the M256A1 kits, a commonly used piece of chemical agent detection equipment in the Gulf War, as 2.0 mg/m3. According to the Material Data Safety Sheets for sulfur mustard, total weight average exposures of greater than .003mg/m3 over an 8 hr. period requires the use of protective equipment. (See appendix A.) Therefore, the detection kit would not detect the agent until the amount of agent present exceeded the safety threshold by a factor of over 660. The M8A1 automatic alarms do not detect blister agent.

Mustard Gas - This is a colorless, oily liquid which dissolves poorly in water, but relatively well in organic solvents, petroleum, luricant products, and other toxic agents. The injurious effect of mustard gas is associated with its ability to inhibit many enzyme systems of the body. This, in turn, prevents the intra-cell exchange of chemicals and leads to necrosis of the tissue. Death is associated mainly with necrosis of the tissue of the central nervous system. Mustard gas has a period of latent effect (the first signs of injury appear after 2-12 hours), but does not act cumulatively. It does not have any known antidotes. In military use it can come in gas, aerosol, and droplet form. It therefore acts through inhalation, cutaneously, perorally and directly through the blood stream. The toxic and physico-chemical properties of mustard gas allow it to be used in all types of munitions.

Related Chemical Agent Information

Committee staff has learned that Iraq may have acquired any one of a number of the Soviet binary novachok ("newcomer") series of chemical warfare agent compounds or information relevant to the development of those compounds. This series of chemical warfare agents reportedly contains both lethal and debilitating agents. According to a confidential Committee source, if the Iraqis had obtained samples of these compounds they could be easily analyzed and produced with readily available materials. Several of these compounds are described as agents that even in microdoses can have long lasting effects. These agents are described as inducing myosis, vomiting, memory loss, involuntary motions and internal organ dysfunction. Many of these materials are also described as having mutagenic effects. These materials are, according to

the source, stored in the lipids (body fats) and have no known antidotes. In addition, according to the Committee source, the Soviets were believed to have conducted research in a number of dioxin-based chemical warfare agents, and on at least one agent that could be used to contaminate drinking water supplies. Committee staff is conducting further inquiries to determine if Iraq may have had access to any of these compounds.

Biotoxins

Biotoxins are natural poisons, chiefly of cellular structure. A distinction is made between exotoxins which are given off by an organism while it is alive, and endotoxins which are given off after a cell's death. The exotoxins cause the injurious effects of biological weapons, but endotoxins guarantee the effects of chemical weapons and do not cause the widespread disease outbreaks associated with biological warfare. Some examples of biotoxins include botulinus toxin and staphylococcic enterotoxin.

Biological Warfare Capability

According to the U.N., the Iraqi biological warfare program was initiated in mid- 1986 at Salman Pak. UNSCOM inspectors discovered evidence of research into certain biological agents including botulinus toxin and anthrax -- as well as organisms responsible for gas gangrene, tetanus and brucellosis, components of a biological weapons program which was not defensive in nature. In four years of work prior to the war, only 10 papers were published. These research programs focused on Iraqi efforts to isolate the most pathogenic spores. They also did research on the aerosolization and on the environmental survivability of some of these biological materials according to the United Nations.

While the Department of Defense maintains that the Iraqi military did not weaponize its biological warfare program, UNSCOM is less certain, reporting that their degree of confidence that weaponization did not occur is low. In fact, readily high performance agricultural aerosol generators could easily be converted to both decontaminate areas in which chemicals are used and to aerosolize biological and chemical warfare agents.

Other ways in which biological materials could have been weaponized include the use of Iraqi 250 and 500lb bombs, aerial rockets, unmanned aerial vehicles, FAW ground-to-ground missiles, helicopters and Iraqi aircraft. The Committee has received several reports of Iraqi helicopters

penetrating Saudi airspace during the war by flying at low levels through the wadis and of Iraqi aircraft penetrating the area over the northern Persian Gulf.

According to UNSCOM, indications that suggested that the program was offensive in nature include:

- No declared links between the BW defense program and medical corps research.
- No links between aerosolization research and research on defensive filters.

The United Nations said that the Biological Inspection was initiated on August 8, 1991 at Salman Pak. The inspection was delayed because of the need to extensively immunize the members of the inspection team. The Salman Pak facility was razed one week prior to the arrival of the inspection team.

The United States is aware of the Iraqi potential for using biological weapons. The employment of biological agents in a "cocktail" mix with chemical warfare agents is consistent with Soviet military doctrine. It is clear that biological weapons are much more difficult than chemical weapons to detect and defend against. Some of the symptoms experienced by veterans suffering from Persian Gulf Syndrome are consistent with biological warfare agent use. Verification will require sophisticated medical diagnosis, which to date has not been publicly undertaken.

The question of whether U.S. forces were attacked with a biological agent is problematic. According to Chemical/Biological Program: A Department of Defense Perspective, "it has been recognized that our biological defense program was inadequate. Creditble analysis indicated that optimal employment of biological agents could result in a significantly large hazard area." It further cites a memo from the Chairman of the Joint Chiefs of Staff to the SECDEF (Secretary of Defense) noting: "inadequate ability to counter BW (biological warfare) attack/BW defense is a priority requirement. The inadequacy of the current biological defense and detection program was also supported by Deputy Secretary of Defense John Deutch in an unclassified May 6, 1994 address delivered at a Department of Defense sponsored counterproliferation conference at the Los Alamos National Laboratory. According to Deputy Secretary Deutch, the United States has "no biological detection capability deployed with any forces, anywhere."

Novel BW agents created by altering DNA plasmids and vectors are specifically intended to avoid detection. As noted below, several shipments of biological materials that might have been used to carry out such a program were licensed for export from the United States to the Iraq Atomic Energy Commission. In such a program, common intestinal flora such as e. coli could be altered to produce viral, bacterial, or other toxins and would be difficult to treat. If Iraq was successful in developing such agents, diagnosis will continue to elude physicians testing for traditional illnesses. Novel BW agents would certainly elude biological detection devices. There is evidence, based on the nature of the materials imported, that this type of research was being conducted. Since the Iraqi government managed to dismantle much of its biological warfare

program prior to the UNSCOM inspections, we can only speculate on how advanced this program might have been.

It has been suggested that if these problems the veterans are experiencing are Gulf War related, then we should be seeing even more serious problems among the Iraqis. Since beginning this investigation we have learned that many Iraqi enemy prisoners of war (EPW) suffered skin rashes, sores, nausea, vomiting, coughing and other medical problems while they were being detained in Saudi Arabia. Many members of units who had close contact with these individuals are now reporting to the Committee symptoms consistent with those being suffered by other Gulf War veterans. In addition, Iraq has claimed a dramatic rise in reported cases of communicable diseases since the end of the Gulf War including typhoid, brucellosis, hepatitis and cholera.

Further, reports of Gulf War illnesses being reported are no longer limited to veterans of the Gulf War. Others reporting manifestation of these symptoms include:

- Department of Defense civilians who served in the Persian Gulf War.
- Department of Defense civilians working at the Anniston (AL) Army Depot and the Sharpsite (CA) Army Depot decontaminating equipment which was returned from the Persian Gulf.
- Spouses, particularly the spouses of male veterans, are reporting the following symptoms: chronic or recurring vaginal yeast infections, menstrual irregularities (excessive bleeding and severe cramping), rashes, fatigue, joint and muscle pain, and memory loss.
- Children born to veterans prior to the Gulf War. In many cases both male and female children born prior to the war have experienced symptoms similar to those of the veterans and their spouses.
- Children born following the Gulf war. Some reports have been published which suggest a high rate of miscarriages in the families of Gulf War veterans. Further, several reports have surfaced which suggest that there has been a high rate of physical abnormalities in children born to Gulf War veterans since the war.

U.S. Exports of Biological Materials to Iraq

The Senate Committee on Banking, Housing, and Urban Affairs has oversight responsibility for the Export Administration Act. Pursuant to the Act, Committee staff contacted the U.S. Department of Commerce and requested information on the export of biological materials during the years prior to the Gulf War. After receiving this information, we contacted a principal supplier of these materials to determine what, if any, materials were exported to Iraq which might have contributed to an offensive or defensive biological warfare program. Records available from the supplier for the period from 1985 until the present show that during this time, pathogenic (meaning "disease producing"), toxigenic (meaning "poisonous"), and other biological research materials

were exported to Iraq pursuant to application and licensing by the U.S. Department of Commerce. Records prior to 1985 were not available, according to the supplier. These exported biological materials were not attenuated or weakened and were capable of reproduction. According to the Department of Defense's own Report to Congress on the Conduct of the Persian Gulf War, released in April 1992: "By the time of the invasion of Kuwait, Iraq had developed biological weapons. It's advanced and aggressive biological warfare program was the most advanced in the Arab world... The program probably began late in the 1970's and concentrated on the development of two agents, botulinum toxin and anthrax bacteria... Large scale production of these agents began in 1989 at four facilities in Baghdad. Delivery means for biological agents ranged from simple aerial bombs and artillery rockets to surface-to-surface missiles."

Included in the approved sales are the following biological materials (which have been considered by various nations for use in war), with their associated disease symptoms:

Bacillus Anthracis: anthrax is a disease producing bacteria identified by the Department of Defense in The Conduct of the Persian Gulf War: Final Report to Contress, as being a major component in the Iraqi biological warfare program.

Anthrax is an often fatal infectious disease due to ingestion of spores. It begins abruptly with high fever, difficulty in breathing, and chest pain. The disease eventually results in septicemia (blood poisoning), and the mortality is high. Once septicemia is advanced, antibiotic therapy may prove useless, probably because the exotoxins remain, despite the death of the bacteria.

Clostridium Botulinum: A bacterial source of botulinum toxin, which causes vomiting, constipation, thirst, general weakness, headache, fever, dizziness, double vision, dilation of the pupils and paralysis of the muscles involving swallowing. It is often fatal.

Histoplasma Capsulatum: causes a disease superfically resembling tuberculosis that may cause pneumonia, enlargement of the liver and spleen, anemia, an influenza like illness and an acute inflammatory skin disease marked by tender red nodules, usually on the shins. Reactivated infection usually involves the lungs, the brain, spinal membranes, heart, peritoneum, and the adrenals.

Brucella Melitensis: a bacteria which can cause chronic fatique, loss of appetite, profuse sweating when at rest, pain in joints and muscles, insomnia, nausea, and damage to major organs.

Clostridium Perfringens: a highly toxic bateria which causes gas gangrene. The bacteria produce toxins that move along muscle bundles in the body killing cells and producing necrotic tissue that is then favorable for further growth of the bacteria itself. Eventually, these toxins and bacteria enter the bloodstream and cause a systemic illness.

In addition, several shipments of Escherichia Coli (E. Coli) and genetic materials, as well as human and bacterial DNA, were shipped directly to the Iraq Atomic Energy Commission.

The following is a detailed listing of biological materials, provided by the American Type Culture Collection, which were exported to agencies of the government of Iraq pursuant to the issueance of an export licensed by the U.S. Commerce Department:

```
Date      : February 8, 1985
Sent To   : Iraq Atomic Energy Agency
Materials Shipped:

          Ustilago nuda (Jensen) Rostrup

Date      : February 22, 1985
Sent To   : Ministry of Higher Education
Materials Shipped:

          Histoplasma capsulatum var. farciminosum (ATCC 32136)
          Class III pathogen

Date      : July 11, 1985
Sent To   : Middle and Near East Regional A
Material Shipped:

          Histoplasma capsulatum var. farciminosum (ATCC 32136)
          Class III pathogen

Date      : May 2, 1986
Sent To   : Ministry of Higher Education
Materials Shipped:

     1.   Bacillus Anthracis Cohn (ATCC 10)
           Batch # 08-20-82 (2 each)
           Class III pathogen

     2.   Bacillus Subtilis (Ehrenberg) Cohn (ATCC 82)
           Batch # 06-20-84 (2 each)

     3.   Clostridium botulinum Type A (ATCC 3502)
           Batch # 07-07-81 (3 each)
           Class III pathogen

     4.   Clostridium perfringens (Weillon and Zuber) Hauduroy, et al
(ATCC 3624)
           Batch # 10-85SV (2 each)

     5.   Bacillus subtilis (ATCC 6051)
           Batch # 12-06-84 (2 each)

     6.   Francisella tularensis var. tularensis Olsufiev (ATCC 6223)
```

Batch # 05-14-79 (2 each)
Avirulent, suitable for preparations of diagnotic antigens

7. Clostridium tetani (ATCC 9441)
Batch # 03-84 (3 each)
Highly toxigenic

8. Clostridium botulinum Type E (ATCC 9564)
Batch # 03-02-79 (2 each)
Class III pathogen

9. Clostridium tetani (ATCC 10779)
Batch # 04-24-84S (3 each)

10. Clostridium perfringens (ATCC 12916)
Batch #08-14-80 (2 each)
Agglutinating type 2

11. Clostridium perfringens (ATCC 13124)
Batch #07-84SV (3 each)
Type A, alpha-toxigenic, produces lecithinase C.J. Appl.

12. Bacillus Anthracis (ATCC 14185)
Batch #01-14-80 (3 each)
G.G. Wright (Fort Detrick)
V770-NP1-R. Bovine Anthrax
Class III pathogen

13. Bacillus Anthracis (ATCC 14578)
Batch #01-06-78 (2 each)
Class III pathogen

14. Bacillus megaterium (ATCC 14581)
Batch #04-18-85 (2 each)

15. Bacillus megaterium (ATCC 14945)
Batch #06-21-81 (2 each)

16. Clostridium botulinum Type E (ATCC 17855)
Batch # 06-21-71
Class III pathogen

17. Bacillus megaterium (ATCC 19213)
Batch #3-84 (2 each)

18. Clostridium botulinum Type A (ATCC 19397)
Batch # 08-18-81 (2 each)
Class III pathogen

19. Brucella abortus Biotype 3 (ATCC 23450)
Batch # 08-02-84 (3 each)
Class III pathogen

20. Brucella abortus Biotype 9 (ATCC 23455)
Batch # 02-05-68 (3 each)
Class III pathogen

21. Brucella melitensis Biotype 1 (ATCC 23456)
Batch # 03-08-78 (2 each)
Class III pathogen

22. Brucella melitensis Biotype 3 (ATCC 23458)
Batch # 01-29-68 (2 each)
Class III pathogen

23. Clostribium botulinum Type A (ATCC 25763)
Batch # 8-83 (2 each)
Class III pathogen

24. Clostridium botulinum Type F (ATCC 35415)
Batch # 02-02-84 (2 each)
Class III pathogen

Date : August 31, 1987
Sent To : State Company for Drug Industries
Materials Shipped:

1. Saccharomyces cerevesiae (ATCC 2601)
Batch # 08-28-08 (1 each)

2. Salmonella choleraesuis subsp. choleraesuis Serotype typhi (ATCC 6539)
Batch # 06-86S (1 each)

3. Bacillus subtillus (ATCC 6633)
Batch # 10-85 (2 each)

4. Klebsiella pneumoniae subsp. pneumoniae (ATCC 10031)
Batch # 08-13-80 (1 each)

5. Escherichia coli (ATCC 10536)
Batch # 04-09-80 (1 each)

6. Bacillus cereus (11778)
Batch #05-85SV (2 each)

7. Staphylococcus epidermidis (ATCC 12228)
Batch # 11-86s (1 each)

8. Bacillus pumilus (ATCC 14884)
Batch # 09-08-80 (2 each)

Date : July 11, 1988
Sent To : Iraq Atomic Energy Commission
Materials Shipped

1. Escherichia coli (ATCC 11303)
   Batch # 04-875
   Phase host

2. Cauliflower Mosaic Caulimovirus (ATCC 45031)
   Batch # 06-14-85
   Plant Virus

3. Plasmid in Agrobacterium Tumefaciens (ATCC 37349)
   (Ti plasmid for co-cultivation with plant integration vectors in E. Coli)
   Batch # 05-28-85

Date : April 26, 1988
Sent To: : Iraq Atomic Energy Commission
Materials Shipped:

1. Hulambda4x-8, clone: human hypoxanthine phosphoribosyltransferase
   (HPRT) Chromosome(s): X q26.1 (ATCC 57236) Phage vector
   Suggest host: E coli

2. Hulambda14-8, clone: human hypoxanthine phosphoribosyltransferase
   (HPRT) Chromosome(s): X q26.1 (ATCC 57240) Phage vector
   Suggested host: E coli

3. Hulambda15, clone: human hypoxanthine phosphoribosyltransferase
   (HPRT) Chromosome(s): X q26.1 (ATCC 57242) Phage vector
   Suggested host: E. coli

Date : August 31, 1987
Sent To : Iraq Atomic Energy Commission
Materials Shipped:

1. Escherichia coli (ATCC 23846)
   Batch # 07-29-83 (1 each)

2. Escherichia coli (ATCC 33694)
   Batch # 05-87 (1 each)

Date : September 29, 1988
Sent To : Ministry of Trade
Materials Shipped:

1. Bacillus anthracis (ATCC 240)
   Batch # 05-14-63 (3 each)
   Class III pathogen

2. Bacillus anthracis (ATCC 938)
   Batch # 1963 (3 each)
   Class III pathogen

3. Clostridium perfringens (ATCC 3629)
Batch # 10-23-85 (3 each)

4. Clostridium perfringens (ATCC 8009)
Batch # 03-30-84 (3 each)

5. Bacillus anthracis (ATCC 8705)
Batch # 06-27-62 (3 each)
Class III pathogen

6. Brucella abortus (ATCC 9014)
Batch # 05-11-66 (3 each)
Class III pathogen

7. Clostridium perfringens (ATCC 10388)
Batch # 06-01-73 (3 each)

8. Bacillus anthracis (ATCC 11966)
Batch #05-05-70 (3 each)
Class III pathogen

9. Clostridium botulinum Type A
Batch # 07-86 (3 each)
Class III pathogen

10. Bacillus cereus (ATCC 33018)
Batch # 04-83 (3 each)

11. Bacillus ceres (ATCC 33019)
Batch # 03-88 (3 each)

Date : January 31, 1989
Sent To : Iraq Atomic Energy Commission
Materials Shipped:

1. PHPT31, clone: human hypoxanthine phosphoribosyltransferase (HPRT)
Chromosome(s): X q26.1 (ATCC 57057)

2. Plambda500, clone: human hypoxanthine phosphoribosyltransferase
pseudogene (HPRT) Chromosome(s): 5 p14-p13 (ATCC 57212)

Date : January 17, 1989
Sent To : Iraq Atomic Energy Commission
Materials Shipped:

1. Hulambda4x-8, clone: human hypoxanthine phosphoribosyltransferase
(HPRT) Chromosomes(s): X q26.1 (ATCC 57237) Phage vector;
Suggested host: E. coli

2. Hulambda14, clone: human hypoxanthine phosphoribosyltransferase
(HPRT) Chromosome(s): X q26.1 (ATCC 57540), Cloned from human lymphoblast, Phase vector
Suggested host: E. coli

3. Hulambda15, clone: human hypoxanthine phosphoribosyltransferase
(HPRT) Chromosome(s): X q26.1 (ATCC 57241) Phage vector;
Suggested host: E. coli

Additionally, the Centers for Disease Control has compiled a listing of biological materials shipped to Iraq prior to the Gulf War. The listing covers the period from October 1, 1984 (when the CDC began keeping records) through October 13, 1993. The following materials with biological warfare significance were shipped to Iraq during this period.

Date : November 28, 1989
Sent To : University of Basrah, College of Science, Department of Biology
Materials Shipped:

1. Enterococcus faecalis

2. Enterococcus faecium

3. Enterococcus avium

4. Enterococcus raffinosus

5. Enteroccus gallinarium

6. Enterococcus durans

7. Enteroccus hirae

8. Streptococcus bovis
(etiologic)

Date : April 21, 1986
Sent To : Officers City Al-Muthanna, Quartret 710, Street 13, Close 69, House 28/I,
Baghdad, Iraq
Materials Shipped:

1. 1 vial botulinum toxoid
(non-infectious)

Date : March 10, 1986
Sent To : Officers City Al-Muthanna, Quartret 710, Street 13, Close 69 House 28/I,

Baghdad, Iraq
Materials Shipped:

1. 1 vial botulinum toxoid #A2 (non-infectious)

Date : June 25, 1985
Sent To : University of Baghdad, College of Medicine, Department of Microbiology
Materials Shipped:

1. 3 years cultures (etiologic) Candida sp.

Date : May 21, 1985
Sent To : Basrah, Iraq
Materials Shipped:

1. Lyophilized arbovirus seed (etiologic)

2. West Nile Fever Virus

Date : April 26, 1985
Sent To : Minister of Health, Ministry of Health, Baghdad, Iraq
Materials Shipped:

1. 8 vials antigen and antisera (r. rickettsii and r. typhi) to diagnose rickettsial infections (non-infectious)

UNSCOM Biological Warfare Inspections

UNSCOM inspections uncovered evidence that the government of Iraq was conducting research on pathogen enhancement on the following biological warfare related materials:

- bacillus anthracis
- clostridium botulinum
- clostridium perfringens
- brucella abortis
- brucella melentensis
- francisella tularensis
- clostridium tetani

In addition, the UNSCOM inspections revealed that biological warfare related stimulant research was being conducted on the following materials:

- bacillus subtillus
- bacillus ceres
- bacillus megatillus

UNSCOM reported to Committee staff that a biological warfare inspection (BW3) was conducted at the Iraq Atomic Energy Commission in 1993. This suggests that the Iraqi government may have been experimenting with the materials cited above (E. Coli and rDNA) in an effort to create genetically altered microorganisms (novel biological warfare agents).

Chapter 1, Part 3

Biological Warfare Defense
Types of Biological Agents
Dissemination of Biological Agents
Defensive Measures
Iraq's Experience in the Use of Chemical Warfare Agents
Gulf War Syndrome: The Case for Chemical/Biological Agent Exposure

Biological Warfare Defense

The following section, describing the types, dissemination, and defensive measures against biological agents, is quoted verbatim from a United States Marine Corps Institute document, Nuclear and Chemical Operations, MCI 7711B, used in the Command and Staff College's nonresident program. It is clear from this document that the Department of Defense recognizes both the threat and U.S. vulnerability to biological weapons. This document also outlines the Department's understanding of what actions should be taken in the event that a biological weapon has been or is suspected to have been employed.

"Biological agents cannot be detected by the human senses. A person could become a casualty before he is aware he has been exposed to a biological agent. An aerosol or mist of biological agent is borne in the air. These agents can silently and effectively attack man, animals, plants, and in some cases, material. Agents can be tailored for a specific type of target.

Methods of using antipersonnel agents undoubtedly vary so that no uniform pattern of employment or operation is evident. It is likely that agents will be used in combinations so that the disease symptoms will confuse diagnosis and interfere with proper treatment. It is also probable that biological agents would be used in heavy concentrations to insure a high percentage of infection in the target area. The use of such concentrations could result in the breakdown of individual immunity because the large number of micro-organisms entering the body could overwhelm the natural body defenses.

Types of Biological Agents

Different antipersonnel agents require varying periods of time before they take effect, and the periods of time for which they will incapacitate a person also vary. Most of these diseases having

antipersonnel employment potential are found among a group of diseases that are naturally transmitted between animals and man. Mankind is highly vulnerable to them since he has little contact with animals in today's urban society. The micro-organisms of possible use in warfare are found in four naturally occurring groups -- the fungi, bacteria, rickettsiae, and viruses.

1. Fungi. Fungi occur in many forms and are found almost everywhere. They range in size from a single cell, such as yeast, to multicellular forms, such as mushrooms and puffballs. Their greatest employment potential is against plants, although some forms cause disease in man. A fungus causes the disease coccidioidomycosis in man. Other common infections caused by Fungi include ringworm and "athletes foot."
2. Bacteria. Bacteria comprises a large and varied group of organisms. They occur in varying shapes, such as rods, spheres, and spirals, but they are all one-celled plants. Some bacteria can assume a resistant structure called a spore, which enables them to resist adverse environmental conditions. Others may produce poisonous substances called toxins. Examples of human disease caused by bacteria are anthrax, brucellosis, tularemia, staphylococcus, and streptococcus.
3. Rickettsiae. Rickettsiae organisms have the physical appearance of bacteria and the growth characteristics of viruses. Members of this group must have living tissue for growth and reproduction, whereas most fungi and bacteria can be grown on artificial material. Another characteristic of rickettsiae is that most diseases caused by this group are transmitted by the bite of an insect, such as the mosquito, mite, or tick. Rocky Mountain Spotted Fever, Q fever, and typhus are diseases of mankind caused by rickettsiae.
4. Virus. The smallest living things known to mankind are viruses. Viruses are so small that an electron microscope is required to see them. Viruses cannot be grown in the absence of living tissue. Diseases which are caused by viruses cannot normally be treated with antibiotics. Viruses cause yellow fever, rabies, and poliomyelitis.

Dissemination of Biological Agents

1. Aerosol. Biological agents may be disseminated on, or over, the target by many means, such as aircraft, missiles, and explosive munitions. These devices produce a biological aerosol, and, if antipersonnel biological agents are ever used, they will probably be disseminated in the form of biological mists or aerosols. This method of dissemination would be extremely effecftive because the micro-organisms would be drawn into the lungs as a person breathes, and there they would be rapidly absorbed into the blood stream. The hours from dusk until dawn appear to be the best time for dissemination of biological agents. The weather conditions are most favorable for these agents at night, since sunlight will destroy many of them. In field trials, using harmless biological aerosols, area coverages of thousands of square miles have been accomplished. The aerosol particles were carried for long distances by air currents. (emphasis added)

2. Living Hosts. Personnel may be infected by disease carrying vectors, such as insects, rats, or other animals. Mosquitos may spread malaria, yellow fever, or encephalitis; rats spread plague (any mammal may carry rabies). Militarily, specific vectors may be selected, infected as required, and then released in the target area to seek out their human victims and pass on the disease. Since infection is transmitted through a bite in the skin, protective masks offer no protection. A vector borne agent may remain in the target area for as long as there are live hosts; thus, a major disadvantage results. The vector borne agent can become a permanent hazard in the area as the host infects others of his species.
3. Food and Water Contamination. Biological agents could also be delivered to target personnel by placing the agent in food and water supplies (sabotage). This type of attack would probably be directed against small targets, such as industrial complexes, headquarters, or specific individuals. The methods of delivering the attack are many and varied.

Defensive Measures

The United States carries out research aimed at improved means of detection of biological agents and treatment and immunization of personnel. Both of these are essential to biological defense.

1. Before an Attack: The inability of the individual to detect a biological attack is perhaps the greatest problem. Contributing factors are the delay experienced before the onset of symptoms and the time required to identify specific agents. Without an adequate means of detection, complete defensive measures may not be taken since an attack must first be detected before you can defend against it. Diseases caused by biological agents do not appear until a few days to weeks after contact with the agent. Personnel are protected against biological agents in aerosol form by the protective mask. Ordinary clothing protects the skin from contamination by biological agents. Other means of protection include immunizations; quarantining contaminated areas; cleanliness of the body, clothing, and living quarters; stringent rodent and pest control; proper care of cuts and wounds; and education of troops to eat and drink only from approved sources.
2. After an Attack: After a biological agent attack has occurred, it will be necessary to identify the agent used in the attack so that proper medical treatment may be given to exposed personnel. To perform this identification, it is necessary to collect samples or objects from the contaminated area and send them to a laboratory or suitable facility for processing. Samples may be taken from the air, from contaminated surfaces, or from contaminated water. After the sample is taken, laboratory time will be required to identify the suspected biological agent. The length of time for identification is being significantly shortened through the use of new medical and laboratory techniques. Proper defensive actions taken during a biological attack depend upon the rapid detection of the attack. Biological defense is continuous. You must always be prepared for the employment of these weapons. (emphasis added)

Iraq's Experience in the Use of Chemical Warfare Agents

The fears and the precautions taken prior to the Gulf War were not the product of excessive hysteria. Five United Nations reports have confirmed the use of chemical warfare agents in the Iran-Iraq War. Use of chemical weapons against both the Kurds and Shiite Moslems within Iraq is well documented. Press reports also document Iraqi readiness to use these weapons against Coalition forces during the Persian Gulf War.

In April 1993, two U.S. based human rights organizations confirmed that they had found residues of chemical weapons used by the Iraqi government of Saddam Hussein against Kurdish village in northern Iraq in 1988. These groups, Physicians for Human Rights and Human Rights Watch, said they had used advanced analytical techniques to discover the presence of mustard gas and the nerve gas Sarin. Those chemical weapons reportedly were dropped by aircraft on August 25, 1988 and killed four people in the Kurdish village of Birjinni. Testimony from survivors of the Birjinni bombing, who said victims of the raids died writhing and coughing blood, led to accusations that Iraq had gassed its own citizens as part of a campaign against rebellious Kurds that killed tens of thousands. This was the first time that scientists had been able to prove the use of chemical weapons, and especially a nerve gas, through the analysis of environmental residue acquired years after such an attack occurred.

Soil samples were gathered from the 1988 bombing sites and then delivered to a British laboratory. Chemists at Porton Down found traces of mustard gas and Sarin. Dr. Graham Pearson, director of the British Chemical and Biological Defense Establishment, verified these results and confirmed the samples were taken from bomb craters near the northern Iraqi village of Birjinni in June 1992. The byproducts of the breakdown of these poisons are so specific that they provide a "unique fingerprint" in chemical analysis that points directly to a poison gas attack.

An earlier attack had been reported on March 17, 1988 on the village of Halabja. Amnesty International reported that chemical weapons were used in an attack by Iraq, in which "some 5,000 Kurds were killed within an hour." A U.N. team sent to investigate the attack found evidence of chemical weapons, although they did not rule on who carried out the attack on the town, which had been occupied by Iran since mid-March.

On September 26, 1993, Shiite rebels living in the southern Iraqi marshlands reported an early morning shelling attack by Iraqi forces. The eyewitnesses, who spoke with a New York Times reporter, mentioned that the shells landed with a thud "and not the usual explosion" sending up white clouds. The artillery attack was followed by a ground assault by Iraqi troops who were equipped with gas masks.

A Shiite rebel claimed that upon entering one of the Iraqi armored personnel carriers they found battle orders calling for a chemical attack. Rebel leaders provided a copy of the captured orders. Written in Arabic on the twenty-sixth of September, the orders, numbered 1-15, instructed the Iraqi soldiers to use chemical weapons to "retake the village" and that "each soldier must be instructed on how to respond during the chemical attack."

After the attack, some villagers returned for their belongings, but there was nothing left. They discovered that trees and plants had withered and yellowed. Furthermore, "the cats, the dogs, the birds and even the water snakes had died. But for some reason the victims had been removed by the troops. We saw no bodies."

In November 1993, a nine member U.N. inspection team arrived to take samples from the area of the alleged chemical attack. The results of the inspection were inconclusive.

It is also suspected that Iraq may have used biological agents (mycotoxins) during the 1984 attack on Majnoon Island, during the Iran-Iraq War, and in 1988 against the Kurds (cholera and typhus). However, no medical verification of Iraqi use of biological warfare agents yet exists.

The above documented instances of chemical weapons use (and suspected use) against Iranians, Kurds, and Shiites undermine Department of Defense assertions that Iraq may not have used these weapons against Coalition forces because they "feared contamination of their own men." Marine Brigadier General Richard Neil said that prisoner debriefings of Persian Gulf War EPWs had "yielded the impression that the Iraqis were not comfortable operating in a chemical environment...and...Iraqi soldiers had poor chemical protection equipment of their own." Lt. General Thomas Kelly stated in a press briefing that "the Iraqi Army was very uncomfortable, we are finding out from the POWs, about the use of chemical weapons because they are not familiar with it." However, as the preceding paragraphs make clear, the Iraqi Army had operational experience with the use of these weapons, unlike their American counterparts.

Gulf War Syndrome: The Case for Chemical/Biological Agent Exposure

As the preceding sections of this report make clear, the Government of Iraq possessed a large and sophisticated chemical and biological weapons production complex. Iraq's army, organized and equipped along Soviet lines, also appeared to employ Soviet chemical warfare doctrine, which advocated the use of mixed agent warfare. Iraq used these weapons against its own people in the 1980's, and possibly again in 1993. It should not be surprising that Baghdad would also use every weapon in its arsenal against the much more serious threat to its own survival posed by the massed Coalition forces. Additionally, the release of chemical and biological agents as a

result of Coalition bombing should have been expected by the Allied forces, based upon their own doctrine regarding the dispersal of chemical agents.

Several theories have been put forward to explain the cause(s) of Gulf War Syndrome. Most of them lack credibility because they do not explain transmission of similar symptoms across a broad and dissimilar population whose only commonality was the service of a family member in the Persian Gulf theater of operations or contact with materiel returned from that venue. Meanwhile, the passage of over three years since the appearance of the first symptoms, and the inability of the Departments of Defense and Veterans Affairs to find a cause, suggests that the illnesses may be caused by something that these institutions have not examined. Further, the absence of credible and verifiable published scientific research on the syndrome by the these agencies, providing specifics of the types of laboratory research that have been conducted, case histories, and methodologies used, leaves each interested scientist in the dark as to what diagnostic processes have been attempted and which have failed.

There is a growing body of evidence, outlined in detail below, which supports the claims of Gulf War veterans that exposure to chemical and/or biological warfare agents may be the cause of the complex of illnesses they currently suffer. There appear to be four primary sources of exposure:

1. As a result of direct attack, via missile, rocket, artillery, or aircraft munitions;
2. As a result of intermittent low-level exposure to fallout from Coalition bombings of Iraqi chemical and biological warfare plants and munitions bunkers;
3. As a result of administration of a nerve agent pre-treatment drug that acts in a manner similar to actual nerve agent;
4. As a result of continuing contact with the Iraqi enemy prisoners of war (EPWs).

In addition, there appear to be two secondary sources of exposure:

1. Exposure to occupational/environmental hazards in Southwest Asia and to contaminated material returned from the theater of operations
2. Transmission among family members. Exposure to endemic diseases and illnesses and diseases must also be thoroughly researched.

Hundreds of Gulf War veterans have been interviewed by the Committee staff. The events cited below are included because the veterans reporting them could remember approximately when they occurred, or because there were multiple independent confirming sources. A map showing the location of these events appears at the end of this section.

Chapter 2, Part 1.

Group I Exposures: Reported Direct Exposure Events

A number of direct exposure events are described below as reported by members of the U.S. Armed Forces who served in the Gulf War. Not every detail can be verified by multiple sources to date, but additional data from unofficial and unrelated sources continue to bolster initial accounts of events best explained as missile and rocket attacks or aerial explosions. Units located in areas where these events occurred are reporting high rates of illnesses. The areas in which these events occurred were key logistic and staging areas, as well as those areas which were breached during the liberations of Kuwait. Many veterans of these units have reported seeing large numbers of dead or dying animals in the area after the attacks; one veteran noted that "all the insects were dead too."

Department of Defense conclusions that no chemical or biological attacks occurred seem to be based on the assumption that there was no significant evidence of immediate chemical and biological casualties. However, since one of the primary goals of a biological attack is to debilitate your adversary's forces, while retaining a high degree of deniability, and since many of those interviewed describe both immediate physical reactions and long term debilitating effect, the issue of what these individuals may have been exposed to becomes highly critical.

Event 1:

January 17, 1991, early morning hours Cement City

Mr. Willie HIcks, then with the 644th Ordinance Company, was serving as the non-commissioned officer in charge of arms and ammunitions shipments. Staff Sergeant Hicks has testified before the Senate Committee on Armed Services' Subcommittee for Force Structure and Personnel that, at about 2:30a.m. on January 17, 1991, he heard a loud explosion, which was followed by a sounding of alarms. As Hicks was running to the bunker, his face began to burn. One member of the unit "just dropped." About ten minutes later, according to Hicks, the unit's first sergeant came by and told members of the unit to go to the highest level of alert. The unit remained at that level for 24 hours.

Two or three days later, Hicks began feeling ill and noticed blood in his urine. Several other members of the unit began experiencing "problems" with their rectums. Hicks testified that when members of the unit began to question what had happened, they were ordered by their

commanding officer not to discuss it. Of the unit's 100 soldiers, 85 now suffer from medical problems, and one, Staff Sergeant Bayle, who Hicks described as having been in good physical shape, has inexplicably died. HIcks described another member of the unit, Staff Sergeant Heal, as being seriously incapacitated.

Hicks, a former teacher and Vietnam veteran, carries a notebook with him everywhere. He claims to have a severe problem with memory loss. He quit his job because he kept passing out and getting lost on the way to work. Other symptoms being suffered by Mr. Hicks include headaches, blood in his urine, insomnia, joint and muscle pain, deteriorating vision, loss of mobility in his left arm, night sweats, and diarrhea (sometimes bloody). His illness has been classified by the Veterans Administration as post traumatic stress disorder.

Event2:

January 19, 1991, early morning hours. Camp 13, 6-7 miles west of Port of Jubayl, Kingdom of Saudi Arabia (Although some individuals reported this event as taking place on January 20, documentary evidence indicates that it took place on the 19th.)

Witness 01: Petty Officer Sterling Symms, then assigned to the Naval Reserve Construction Battalion 24, in an area south of the Kuwait border, testified before the Senate Armed Services Committee that between 2:00a.m. and 3:00a.m. on January 20, 1991, there was a "real bad explosion" overhead. The alarms went off and everybody started running towards their bunkers. Petty Officer Symms said there was a sharp odor of ammonia in the air. His eyes burned and his skin stung. HIs unit donned full chemical gear for nearly two hours until the "all clear" was given.

Later, according to Symms, members of the unit were advised that what they heard was a sonic boom. Petty Officer Symms said that he did not believe that it was a sonic boom because there was also a "fireball" associated with the explosion. Members of the unit were ordered not to discuss the incident. Petty Officer Symms says he has since experienced fatigue, sore joints, running nose, a chronic severe rash, and open sores which have been diagnosed as an "itching problem." He has also been treated for streptococcus infections. In his testimony, Symms stated that 4 or 5 other members of his unit and two of their wives have been treated for similar infections.

Witness 02: Mike Moore, assigned to the same unit as Symms, also reported that on January 20, 1991, at about 3a.m., he was awakened by a double explosion. As the sound of the explosion faded the alarms went off. The unit intercom announced "Go to MOPP level 4." Everyone in the tent put on their gas gear and went to the bunker. They stayed at MOPP level 4 until about 7a.m.. Later that day or the next, everyone's chemical suits and masks were collected and replaced. According to Mr. Moore, he was told the explosion was a sonic boom, to quit worrying about it, and to get back to work. Mr. Moore said that he later heard that what he heard was an incoming SCUD, but he also heard rumors that a Iraqi MIG was shot down in the area that night.

Mr. Moore said that he did not feel a spray or smell ammonia. He had no stinging or numb lips. Since returning home from the Gulf, he has suffered a severe thyroid problem, a heart attack, memory loss, tired and aching joints, rashes on his feet, nervousness, and muscle cramps, although he reported no bleeding. According to Mr. Moore, he has had about ten blood tests and two sets of x-rays performed at the Tuskeegee, Alabama, Veterans Affairs Medical Center. In past calls to the Tuskeegee, Alabama, VAMC, however, he had been told that there is no information in his record.

In February 1992, Mr. Moore's daughter began developing a thyroid problem and has been suffering from nervousness, headaches, and fatigue. Over the last year, his wife has begun to develop these symptoms as well. There is no history of thyroid problems in family.

Witness 03: Mr. William Larry Kay was an electrician assigned to Naval Mobile Construction Battalion 24. He was also assigned to Camp 13. On January 20, 1991, Mr. Kay heard two "booms", shaking the whole building. Sirens began going off. The camp intercom announced "confirmed mustard gas -- go to MOPP level 4." Mr. Kay was at the Recreation Center when the blasts occurred. He had fallen asleep. He went outside and put his gas mask on. It immediately filled with fumes. He recalls that it smelled like ammonia. Mr. Kay has been a member of a Hazmat (Hazardous Materials) team of the fire department in Columbus, Georgia; he said the strong smell of ammonia is unusual in an open area. There was an ammonia plant nearby, but he had never smelled such a strong odor of ammonia in the area. He reported to his assigned bunker. Each member of the unit had a duty during these attacks -- Mr. Kay was assigned to a decontamination team. There were other people assigned to test for chemical contamination. A radio call came in for these people to check for gas. Then, almost immediately, the intercom announced "all clear."

Mr. Kay said that after the incident, in response to questions from the unit as to what had occurrred, the unit Commanding Officer said "Have you ever heard of a sonic boom?" When members of the unit continued to question the unit commanders about what had occurred, they were ordered not to discuss the incident.

Witness 04: Mr. Terry Avery of Salem, Alabama worked on utilities for Naval Mobile Construction Battalion 24, and was also assigned to Camp 13. During the night of January 20, 1991, Mr. Avery said that he heard a double explosion. The alert siren went off. He put on his gas mask and went to the bunker. While in the bunker, his unit received the command to go to MOPP level 4 over the camp loudspeaker. He put on his chemical suit. Mr. Avery said he was almost completely dressed when they announced "all clear." He left the bunker and returned to his tent.

Mr. Avery was later told by his Master Chief that the noise he heard was just a sonic boom. A veteran of Vietnam who had heard sonic booms before, Mr. Avery felt that it was not a sonic boom, but he never got a good answer about the explosion. He reported that the rumor going around the camp was that an enemy plane had been shot down over the desert.

Late in the summer of 1991, Mr. Avery began feeling tired and having headaches. He saw a private doctor, who said he was probably working too hard in the sun. He says he does not think he is as ill as the rest of the men in his unit (NMCB24). He feels that he has leveled out, but he still has good days and bad days. He currently suffers from fatigue, headaches, weight gain, itching, muscle and joint pains, and memory loss (inability to concentrate).

His wife is also ill. Mr. Avery feels that she is more ill than he is. She has an enlarged spleen, an enlarged liver and abnormal liver functions, joint pains, night sweats, fatigue, stomach problems, itching, and rashes, but has not complained of memory loss. Two of his children are also complaining of headaches, joint pain, and abdominal pains. His 13 year old daughter was diagnosed as having mononucleosis. She also has sinus infections, and throat pains from the sinus drainage. His 11 year old son has had rashes, headaches, joint pain, itching, sinus and throat infections, and fevers.

Witness 05: The following are excerpts from one of two letters written by a U.S. serviceman present at Camp 13 during the January 19, 1991 incident. This individual has been interviewed by U.S. Senate professional staff. These original letters confirm the actual events of that morning. This individual has requested confidentiality. The original letters have been retained as evidence.

"8:00 pm
19 Jan 91

Dear Mom,

I just talked to you on the phone. I really didn't want to call you and tell you about the SCUD missile/gas attack so you wouldn't worry, but I really needed to hear a familiar voice...I'm trying like hell to keep my mind off the fact that it's night time again, and we could get hit again.

Mom, I can deal with getting shot at, because I can fight back and even if I got hit, I can be put back together, a missile, on the other hand, doesn't work like that, but I can even accept that. But gas scares the hell out of me. I know how to put on the protective suits and gear, but it's the thought. Once the missile hit (without warning!) we were so busy getting dressed in our chemical suits we never had time for it to sink in and be scared. I was proud of all of us because no one froze up - we all responded like we'd been trained to, but after we got suited up, we had to sit there and force ourselves to breathe slow and try and cool down - the suits are very hot. It's hard to slow your breathing when your heart's beating a million times a minute...[a] fire team [went] out and...patrolled the camp and checked all of the towers. The rest of the camp were in their bunkers except security and the chemical detection teams. I know they detected a cloud of dusty mustard gas because I was there with them, but today everyone denies it. I was there when they radioed the other camps north of us and warned them of the cloud...I talked to the look-outs that saw the air burst and cloud and had to stay with them for a few minutes to try and calm them down even though I was just as scared (probably more!). Jubail is South East of us, and that's where the Scud hit that was confirmed, but the air burst my guys saw was only 200+ yards west

of us. I don't know what that was, but that's where most of the gas came from I think. But the wind was almost blowing due North. I probably won't sleep much tonight, but at least I'll be able to respond faster..."

In the interview with Senate staff, the individual said that during patrols around Camp 13 in the days just after the incident he wrote about, he observed many animals that were either sick or dead. He also confirmed that after the attack, their chemical protective gear was replaced.

Witness 06: Mr. Mike Tidd was assigned to perform security duties with Naval Mobile Construction Battalion 24. He currently suffers from joint aches and pains, sinus infections, diarrhea, urinary urgency and frequency, rashes, small mosquito bite-like sores, heartburn, dizziness, occasional low temperatures, occasional night sweats, and chronic fatigue. Mr. Tidd kept a log while in Saudi Arabia.

According to his log, on January 19, a little past 0330hrs., Mr. Tidd was sitting on Tower 6 when all of a sudden, there was a double boom off to the northwest of the camp, accompanied by a bright flash of light. Within minutes, the general quarters alarm sounded. Mr. Tidd's unit first donned their gas masks and ponchos, and then minutes later, the call came to go to MOPP level 4. At about 0600hrs, the "all clear" was sounded.

While Mr. Tidd heard the bang and saw the flash, which he described as being fairly close, he does not recall seeing a cloud. He said that he did not experience any symptoms, but attributes that to having been in a covered guard tower about 20 feet off the ground with a 3' visibility area.

Event 3:

January 19, 1991, early morning hours (possibly January 20). King Abdul Aziz Naval Air Station (NMCB24-Air Det), 3 miles south of Port of Jubayl, Kingdom of Saudi Arabia

Witness 01: Mr. Larry Perry, of North Carolina, was a naval construction worker stationed near the port city of Al-Jubayl, at King Abdul Aziz Naval Air Station. He says the explosion on January 20, 1991 sent his entire unit running for the bomb shelter. When they emerged in their gas masks, they were enveloped by a mist.

Witness 02: Mr. Fred Willoughby of Columbus, Georgia was with Naval Mobile Construction Battalion 24 - Air Det. He currently suffers from headaches, diarrhea, aching joints, blood shot eyes, bloat, intestional problems, and chronic fatigue. He has had a polyp removed from his colon, and suffered from rectal bleeding in 1992.

Mr. Willoughby has reported that on January 20, 1991, at about 3-4a.m., he was "hanging out" outside his tent when he heard a long, loud explosion. Shortly thereafter, a siren sounded and he went inside the tent to get his gas mask. By the time he came out, people were yelling 'MOPP 4, MOPP 4, not a drill'. Immediately, his mouth, lips, and face became numb all over, a sensation

he likened to novocaine at the dentist's office. He was in the bunker for about an hour or an hour and a half. When he came out of the bunker, he and the others in the unit were told by the officers and chiefs that what they had heard was just a sonic boom. The next day, the unit was told not to talk about it. But the unit's MOPP gear was collected and replaced the next morning. Mr. Willoughby also heard that an enemy aircraft was shot down in the Gulf, not far from the base.

His wife has begun exhibiting similar symptoms, including fatigue, diarrhea, and aching joints.

Witness 03: Roy Morrow of Phenix City, Alabama was a builder with NMCB24 and was assigned to the Air Detachment at King Abdul Aziz Stadium. On January 20, 1991, he heard two explosions between 3:00-3:30a.m. He was awakened and went to the bunker. The unit went to MOPP level 2 for 25-30 minutes. The "all clear" was then given. When he exited the bunker, Mr. Morrow noticed the Marines running and screaming "MOPP level 4." The siren sounded again. He began to feel a burning sensation on his arms, legs, the back of his neck, and on his ears and face. His lips felt numb. His unit went to full MOPP level 4. Right before he went to the bunker the second time, Mr. Morrow saw a flash at the commercial port of Al-Jubayl. He had a radio in the bunker, and heard a call for the decontaminaton teams to respond.

BU2 Edwards was the head of the decontamination team in Mr. Morrow's unit. According to Mr. Morrow, BU2 Edwards said the next day that mustard gas and lewisite had been detected. When they began to discuss it, according to Mr. Morrow, the unit was told that the two explosions were a sonic boom, and they were ordered not to talk about it any more. The next day, all of their chemical gear was collected and replaced with new equipment.

The numbness experienced by Mr. Morrow remained for at least a week. Within two to three days after the incident, unit members began to suffer from rashes, diarrhea, and fatigue. The aching joints began a couple of weeks later. Mr. Morrow's symptoms have been getting progressively worse until the present time. He currently suffers from swollen lymph nodes, fatigue, diarrhea, night sweats, low grade temperature, weight loss, aching joints, muscle cramps, rashes (transient) blister, welts (2-3 times a month), a permanent hand rash, and short-term memory loss.

Witness 04: Mr. Harold Jerome Edwards, the chemical NCO in charge of the Nuclear/Biological/Chemical team for the Naval Mobile Construction Battalion 24 Air Detachment at King Abdul Aziz Naval Air Station was interviewed by U.S. Senate staff on January 13, 1994. During that interview Mr. Edwards said that he conducted three M-256 tests for chemical agents on the evening of this event. According to Edwards, two of the three tests he conducted were positive for chemical blister agent. He said that the negative test was conducted in an area in between a number of rows of tents. He also said that he reported this information to his unit commander. In addition, Mr. Edwards said that a member of the unit, Tom Muse, blistered in the area under his watch during this event. The "all clear" was given from a higher command. Mr. Edwards was called out to serve on a chemical decontamination team that day.

He said that the Mark 12 decontamination unit assigned to the team was inoperative and that he was assigned to take out a 500 gallon water truck and stand by to decontaminate incoming personnel. According to Mr. Edwards, no one was decontaminated by his team. He said that this was the only time he was called out on this type of mission throughout the entire war.

Other Information Regarding the Detection of Chemical Agents at Jubayl.

Ken Allison, then a Lance Corporal with the 174th Marine Wing Support Squadron, Group 37, was delivering supplies to Jubayl Airfield. During an interview with Senate staff, he reported that sometime during his deployment there, possibly in January 1991, he saw a sign posted on a guard shack at the airfield's southern gate. The sign warned: that the area had tested positive for chemicals; make sure your MOPP gear was ready; and that when the alarms go off it is for real. Although he did not recall the exact wording on the sign, he remembered the content clearly.

In addition, a number of British military personnel suffering from Gulf War Syndrome who were stationed near the Port of Jubayl have come forward and have described similar events.

Event 4:

Late February 1991 "Log Base Charlie", 7 miles from the Iraqi border near Rafha

Witness 01: Ms. Valerie Sweatman from Columbia, South Carolina, was serving as a telecommunications specialist with the U.S. Army, assigned to the 2nd MASH Hospital. Ms. Sweatman recalls that prior to moving to "Log Base Orange" in Iraq during the ground war, her unit packed up their equipment at "Log Base Charlie." "Log Base Charlie" was located about 7 miles from the Iraqi border, near Rafha. One night in late February 1991, she was awakened by a sergeant and was told there was a chemical alert and to go to MOPP level 4. She put on her MOPP suit and mask and began going outside while she was still putting on her gloves. Her unit stayed at MOPP level 4 for 1- 2 hours. That night, she heard that at least one soldier had come into the hospital showing symptoms of nerve agent exposure. She was told that there was a SCUD alert that night. She did not, however, hear any explosion. The morning after this incident, Ms. Sweatman's hands were itching from the wrists on down. She had developed little blisters which went away about a week later. She was treated with ointments and benadryl for a "skin condition."

Ms. Sweatman had heard the chemical alarms go off on other occasions prior to the incident reported above. She was the night telecommunications NCO for her unit, and heard alarms sounding during the first nights of the air war, when her unit was assigned to King Khalid Military City (KKMC). On one occasion during this period, she heard a blast and felt a mist in the same area. After this incident she experienced nausea, diarrhea, and bloody stools. Her unit began taking the nerve agent pre- treatment pills (NAPP), after these earlier alarms. Although the

alarms sounded, the NBC NCO claimed that they were sounding because the alarm equipment had bad batteries and not because of chemicals.

Ms. Sweatman currently suffers from headaches, exhaustion, fatigue, memory loss, nausea, muscle and joint pains, rectal and vaginal bleeding, and rashes. She has been diagnosed as having arthritis, headaches, and post traumatic stress disorder (PTSD).

Event 5:

Early February 1991 In the Desert Between Hafir Al Batin and King Khalid Military City, Northern Saudi Arabia

Witness 01: Ms. Michelle Hanlon of Killeen, Texas was assigned to the 1st Calvary Division as a communications specialist. On February 14, 1991, during lunch, she heard an explosion overhead. She thought at the time that it was a SCUD being intercepted by a Patriot missile and thought nothing more of it.

On another occasion, when her unit was assigned to a field base near Hafir Al Batin, she recalled that one night, the night air breeze made her eyes begin to water. She immediately put on her gas mask and thought nothing more of the incident. She also reported that on a number of days, she could actually smell sulfur from the Coalition bombings of Iraqi chemical plants during the air war.

She is currently suffering from intestinal problems, hemorrhoids, occasional fatigue, a rash on her finger (like little water blisters under the skin), cervical infections which coincide with intestinal problems, and some memory loss. She feels that she is becoming progressively more ill. Her rash has been diagnosed as exzema and has been treated with antibiotics. She is 23 years old. Her child, now 16 months old, has been getting fevers, yeast infections, rectal and penile disclorations.

Witness 02: Mr. Richard Voss was with the 207th Military Intelligence Brigade assigned to the 1st Infantry Division. Mr. Voss recalled witnessing what appeared to be a missile attack while stuck in slow-moving traffic heading west toward Hafir Al Batin on Tapline Road in early February 1991. Mr. Voss reports that sometime between noon and 4:00 p.m., he watched the missile, coming in from the north-northeast, impact to the east of Hafir Al Batin, about one mile away from his vehicle. He saw a large dark brown cloud rise up. Within two or three minutes, MPs came by giving the gas alert signal. He recalled that the wind was blowing from the north or northeast at the time of the incident. He continued to drive in traffic in MOPP gear for about 1-1/2 - 2 hours past Hafir Al Batin toward an assembly area. He got the "all clear" when he got near Log Base Echo.

Currently, Mr. Voss suffers from headaches, occasional fatigue, joint and muscle pain, memory loss/inability to concentrate, urinary urgency, dizziness, photosensitivity, shortness of breath,

rashes, recurring walking pneumonia, chest pains, numbness, and severe joint pains in both wrists and hands. His wife suffers from recurring yeast infections, menstrual irregularities, rashes, fatigue, muscle pain, and severe joint pain in her wrists.

Chapter 2, Part 2.

Group I Exposures: Reported Direct Exposure Events

Witness 03: Ms. Patricia Williams of Nolanville, Texas was assigned to the 1st Calvary Division, near Hafir Al Batin, as a civilian mechanic. One late afternoon in mid- February, she recalled an explosion somewhere in the desert. She described it as a very powerful explosion that she both heard and felt. To her knowledge, no chemical alarms had been set up. Coincidentially, her unit was told that they were going to have a chemical practice; they were told to put on their chemical gear. They were kept at MOPP level 4 for about twenty minutes, but told that this was just a practice. They were also told that the sound they had just heard was a sonic boom. Five civilians were so frightened that they departed that night. She reports that of the forty people originally in her unit, only half are left. She said the rest were so scared that they went home. Ms. Williams said that she did not get sick in the Persian Gulf until this incident. After this incident, she experienced headaches, diarrhea, and photosensitivity.

Ms. Williams currently suffers from headaches, fatigue, joint and muscle pain, memory loss, lumps on her arms and neck, night sweats, insomnia, urinary argency, diarrhea, photosensitivity, gastrointestinal problems, deteriorating vision, shortness of breath, coughing, thyroid problems, abnormal hair loss, swollen lymph nodes, sinusitis, and chest pains. She is forty-four years old.

Witness 04: A confidential source told Senate staff that, on February 14, he was in traffic between KKMC and Hafir Al Batin, near KKMC. Although he did not see or hear this event himself, Military Police with whom he spoke while in traffic told him that a SCUD had been shot down near Hafir Al Batin. He was told that it was nothing to worry about. No one around him went to MOPP.

Event 6: *February 22, 1991, late afternoon or early evening Near King Khalid Military City (KKMC), Kindgom of Saudi Arabia*

Witness 01: Charlene Harmon Davis was a medical secretary with the 34th Aeromedical Patient Staging Station at KKMC. She reported that, on February 22, she was getting ready for work (her shift began at 7:00 p.m.) when three of what she believed to be SCUD missiles were intercepted over KKMC by Patriot missiles. Ms. Davis recalls that the chemical alarms went off. After these explosions, her face, eyes, and throat began to burn, her nose began to run, and she began to feel nauseous. There was a funny taste in her mouth. These immediate symptoms lasted for about twenty minutes, but she has gotten progressively more ill since that incident. When she

sought medical attention after this event, the doctor told her that she might have had a contaminated gas mask, that the mask might have been contaminated by a previous user. Ms. Davis, however, said that she knew she was the first user of the mask because she broke the seal on it.

Ms. Davis currently suffers from migraine headaches, patellar syndrome, seborrheic dermatitis, hip pain, hair loss, insomnia, nightsweats, nightmares, numbness in toes, fatigue, joint and muscle pain, gastrointestinal problems, and dizziness. She also suffers recurring rashes which she says began after the first explosion, believed to be a SCUD missile attack, occurred near her location a few days after the beginning of the air war. Ms. Davis reports that these rashes continue to be a problem to this day. She has advised Senate staff that she is extremely concerned about her health as well as her prognosis. She is twenty-eight years old.

Witness 02: David Pena was a mechanic with the 63 Army Reserve Command (ARCOM), attached to the 3rd Armor Division. He was stationed at Camp Texas, near KKMC. He reports that on approximately February 22, 1991, he was leaving a meeting at about 5:30 p.m. when he heard an explosion, and saw a cloud. His unit went to MOPP level 4 for 1.5 - 2 hours. Mr. Pena recalls that he became nauseous and had blurry vision, lung disease, and skin problems.

Event 7:

Approximately January 20, 1991, early morning (pre-dawn hours). Vicinity of King Fahd International Airport

Mr. Rocky Gallegos was a Lance Corporal with Bravo Battery, 2nd Light Anti- aircraft Missile Battalion. He observed what he believed to be a SCUD missile shot out of the sky almost directly overhead by a Patriot missile while on the midnight-5:00 a.m. guard duty shift on approximately January 20. He reported that the explosion "blossomed like a flower." According to Mr. Gallegos, it exploded again when it hit the ground. Mr. Gallegos said that after the explosion he experienced a "very strong raunchy taste, like very bitter burnt toast" in his mouth. He also began experiencing headaches, nausea, diarrhea, and sensitivity to bright lights almost immediately after the attack. He did not hear the chemical alarms go off immediately. Approximately 10 minutes later, however, the alert alarms sounded and they were ordered to put on their masks.

Mr. Gallegos remained at his post until approximately 4:00 a.m., when he along with a lieutenant, a staff sergeant, and three other enlisted personnel, went on a patrol to investigate the incident. They drove in the general direction of the explosion, but were not able to find evidence of impact.

Mr. Gallegos remained outside until daylight, when he noticed that his hands were tingling and looked as though they were sunburned. During the events of the early morning, his hands had been the only exposed area; his face was covered by a hood, scarf, and glasses, but he removed his gloves to smoke a cigarette.

Later that morning, about a half hour after they returned from the patrol, Mr. Gallegos was assigned to drive the NBC NCO to check all of the chemical detection units. At the fourth or fifth unit, the NBC NCO came back with soimething written on a piece of paper. He shoved the paper in his pocket and told Mr. Gallegos: "get me back to camp -- Now!" Mr. Gallegos described him as "very excited about something," but when questioned the NBC NCO told Mr. Gallegos that it was none of his business.

Two days later, they again went out to patrol the area where the explosion occurred. According to Mr. Gallegos, they saw at least half a dozen dead sheep and a couple of camels that appeared to be very sick.

Unit officials would not tell Mr. Gallegos what had happened. He said that they told him that if it was of concern to him they would tell him. According to Mr. Gallegos, the wind was blowing from the northeast (southwesterly wind) at the time of the explosions.

Mr. Gallegos continued to suffer headaches, nausea, diarrhea, and photosensitivity during his tour of duty in the Saudi Arabia. He became more seriously ill about two weeks before leaving Saudi Arabia. He also suffers from sinus infections (bleeding), narcolepsy, blackouts, dizziness, rashes, hair loss, joint pains in his knees, elbows, and hands, dental problems, muscle pains and spasms, fatigue, night sweats, insomnia, nightmares, and blurred vision. Since his return from the Persian Gulf, his wife Laurie has had bladder surgery, mitral valve prolapse, disrupted menstruation, headaches, yeast infections, and a swollen thyroid. Her physician recently refused to continue treating her, according to Mr. Gallegos, telling her that she was so sick that he did not believe he could help her.

Event 8:

Early in the "Air War" -- Approximately January 20, 1991 Dhahran, Kingdom of Saudi Arabia

Witness 01: Ms. Patrecia Browning of New London, North Carolina, then a Staff Serveant assigned to the 227th Transportation Company, was at Khobar Towers in Dhahran when a Patriot missile intercepted what she believed to be a SCUD missile directly overhead. Her unit went to MOPP level 4 for 3 1/2 - 4 hours. Ms. Browning said that her eyes began to burn, and she smelled a strong odor that reminded her of ammonia. Shortly afterwards she broke out in a rash and began experiencing headaches, nausea, vomiting, and sensitivity to bright lights.

Ms. Browning also reports that she received the anthrax vaccine and the pyridostigmine bromide anti-nerve agent pretreatment pills. She reported that when the latter caused her to have episodes of bloody vomiting, she was told to cut the pills in half. The vomiting did not stop, however, until she stopped taking the pill.

Ms. Browning, who is thirty-seven years old, currently suffers from memory loss, severe recurring headaches, fatigue, joint and muscle pain, recurring rashes, night sweats, sleepiness,

diarrhea, gastrointestinal problems, dizziness, blurry vision and photosensitivity, coughing and shortness of breath, two duodenal ulcers, chest pains, heart arrhythmia, and erractic blood pressure. She said that many of these symptoms originated while she was still in Saudi Arabia.

Witness 02: Mr. Randall Vallee, a Sergeant with the 1113th Transportation Company, was at the "Expo," just north of Dhahran on January 20. He said that he remembers this incident well because it was the first time he came under attack. He heard two or three explosions and felt the concussion. He was outside at the time, with approximately fifteen others, getting ready to move to Tent City. It was nighttime, although he did not remember the exact time. They ran for cover in school buses parked nearby, but then officers began yelling at everyone to get back into the Expo center and go to MOPP level 4 immediately. While running back to the building, he recalled that the air raid sirens were not going off, but there were other alarms going off in the distance. He stated that he "did not think the alarms he heard were chemical alarms because he had been told that the chemical alarms didn't work; that they were just set up because it was standard operating procedure to have them." The air raid sirens went off after he got into the building. Once in the building, he put his chemical gear on and sat down. He recalled becoming nauseous, weak, dizzy, sweating profusely, his head throbbing, and becoming very, very thirsty, as though he were dehydrated. He stated that his vision became blurry, but at the time he thought it was either because of his mask or his sweating. The blurry vision didn't last long; the headache and nausea lasted about twenty minutes, and he continued to feel weak and dizzy for about forty-five minutes. When he went outside, after the all clear was given, he immediately noticed a "very suffocating smell, as though there wasn't enough air to breath," kind of like ammonia, but very strong." He recalled others commenting on the smell, which dissipated soon.

Afterwards, he took the nerve agent pre-treatment pill and boarded his bus for Tent City. Mr. Vallee recalled several attacks and the smell of ammonia several times while at Tent City. He said that the missiles were shot out of the sky so close to them that the fragments would land between the tents. Although his unit's chemical suits were used frequently, they were never replaced. He noticed as the days progressed that his chest "started getting tight," and he was getting flu symptoms." The nausea, fatigue headaches and respiratory problems continued off and on. Finally he became "so dizzy that he couldn't walk." He was diagnosed with an ear infection, and sent home on January 28.

Mr. Vallee currently suffers very severe recurring headaches, fatigue, respiratory problems, joint pain, memory loss, recurring rashes, depression and irritability, night sweats, insomnia, blood in his urine, constipation, nausea, dizziness, shortness of breath and coughing, thyroid problems, flu symptoms, sinus problems and sensitivity to smells. He always feels cold, and takes medication for pain. His wife suffers from fatigue, yeast infections and menstrual irregularities. Mr. Vallee is twenty-seven years old, his wife is twenty-six.

Event 9:

During ground war; Task Force Ripper

In September 1993, a copy of an excerpt from "NBC Survivability from a User's Perspective," by Brigadier General Carlton W. Fulford, Jr., USMC, Director, Training and Education Division, Marine Corps Combat Development Command, Quantico, Virginia was received by the Committee. It states: *"The most significant piece of detection equipment was the FOX NBC reconnaissance system. It demonstrated great detection and analysis capability and quickly moved. Its only disadvantage is that it looks like the Warsaw Pact BTR-60. To protect it from friendly fire, multiple U.S. markings were placed on the vehicle. The FOX was used primarily in mine field breaching operations. After the mine field was cleared, the FOX was sent through as the lead vehicle. Within minutes, the FOX could confirm or deny the presence of chemical contamination in the area. If a CAM alarm sounded while a unit was moving, the FOX was sent to that location to confirm or deny the CAM's reading. False CAM alarms were attributed to the massive numbers of burning oil wells. In the three-day offensive operation in Kuwait, the CAM alarm sounded four times. In three cases, the FOX confirmed a false alarm. In the fourth case, the FOX indicated a lewisite agent. In the opinion of the chemical experts, according to General Fulford, the lewisite reading was attributed to the burning oil wells."* (emphasis added)

Based on this report, research was done on the method with which the FOX vehicle detects chemicals. It uses some of the same techniques that field alarms might employ to detect chemical agents. In addition, however, it takes multiple air and ground samples and analyzes them using mass spectrometry.

Witness 01: On November 12, 1993, a Committee staff member interviewed CW03 Joseph Cottrel, the chemical detection supervisor assigned to this vehicle -- a U.S. Marine Corps NBC warrant officer. During the interview, Mr. Cottrel said that he detected chemical agents on three occasions during the Gulf War. According to a memorandum written by Mr. Cottrel, "The first detection occurred near N. 28 degrees, 32 minutes latitude, and E. 47 degrees, 52 minutes longitude. The FOX vehicle detected blister agents at levels below IMMEDIATE threat to personnel (levels below ICt50). It was determined at the time that the rapid movement through the breach sites would not pose a threat to continued combat operations or require decontamination. Exposure time for individuals was not tracked or limited."

"The next detection happened the evening of the first day of the ground attack." (Note: Since the ground war began at night, this would have been the second evening of the ground war.) "As Task Force Ripper held positions around the Ahmed Al Jaber Airbase (N. 28 degrees, 56 minutes latitude, and E. 47 degrees, 50 minutes longitude), the FOX vehicle detected Lewisite blister vapors. This report was produced by the vehicle operator and given to myself. I reported the findings to division headquarters and requested directions in regards to the chemical agent printout. I was told to forward the tape up the chain of command which I did. A report came back that the FOX had alerted on the oil smoke. That was checked against the FOX. The computer had separated the petroleum compound from the chemical agent. The computer tape has been lost."

The only other detection CW03 Cottrel was aware of occurred around a bunker complex in the vicinity of N. 29 degrees, 14 minutes latitude, and E. 47 degrees, 54 minutes longitude. The FOX crew was directed to check the area for chemical munitions. A report that some chemical vapors were found was reported. Shortly thereafter, Task Force Ripper was ordered back to the division support area and further detection operations were not carried out by the Task Force Ripper NBC Unit.

Witness 02: According to Sergeant Robert A. Maison, Task Force Ripper detected chemical agents on the second night of the ground war. Sergeant Maison reported that as a nuclear, biological, and chemical recon team member, "our team observed an artillery attack to our northwest, at a distance of approximately four kilometers. About five to six minutes later an alarm was sounded by our detection equipment (a mass spectrometer) which is used specifically for that purpose. Taking into account the wind speeds that we were encountering (approximately 40 to 50 knots steady) the reading was not expected to last for a long duration, as it did not (approx. three minutes). The specific agent detected was lewisite in a concentration considered to produce casualties but not death."

"A second [detection] occurred while performing an area recon of an orchard. The second agent type was benzyl-bromide. No liquid contamination was located but the vapor concentration was of casualty strength and documented by the specific ion concentration and identity being printed out by molecular weight on the spectrum analysis printout."

Witness 03: A source who requested confidentiality reported to Senate staff that, on the second night of the ground war, mustard gas was detected by three FOX vehicles at Ahmed Al Jaber Airfield.

He stated that, about 4:30 or 5:00 p.m. "gas, gas, gas" came in over the radio. His unit went to MOPP level 4 for two hours before they were given the "all clear." About a half hour later, they were told that three FOX vehicles had detected mustard agent. After that, he recalled, they were in and out of MOPP gear all night.

Chapter 2, Part 3.

Group I Exposures: Reported Direct Exposure Events

Event 10:

Riyadh, date unknown.

Mr. Michael Kingsbury was a driver/mechanic with the 601st Transportation Company during the Gulf War. He was interviewed by Committee staff for this report. Mr. Kingsbury was in Riyadh for six hours rest and relaxation when the first SCUD missile attack took place. Although he does not remember the date of the attack, he was certain that it was the first SCUD attack on Riyadh. Mr. Kingbury reported that three SCUDs came in, the alarms went off, and they went to MOPP level 4. He immediately began to experience nausea and a sore throat. His nose began to run and his eyes burned a little. He reported seeing a rainbow in the sky after the attack.

The symptoms that began with the attack never went away. In addition, he began to suffer skin irritation after the attack. He began having stomach problems when he returned from the Gulf and currently suffers from memory loss, rashes, aching joints, headaches, rectal bleeding, nausea, sensitivity to light, abnormal hair loss, high fevers, clammy skin, lumps, bloody oral/nasal mucous, night sweats, sore muscles, and fatigue.

Event 11:

January 18, 1991, around midnight (poss. very early on January 19) Log Base Alpha

Mr. William Brady was the Battalion Logistics NCO with the 217th Maintenance Battalion. Around midnight on Janury 18, or possibly very early on the 19th, Mr. Brady was awakened by what he believed to be a SCUD intercepted by a Patriot directly over his unit's position. He said there was a deafening sound, a flash of light, and everything shook. Chemical alarms were going off everywhere, and there was sheer panic. He remembered the chemical litmus paper turning red, and a positive reading from an M- 256 kit. Mr. Brady said that his nose began to run, and he smelled and tasted sulfur. He began coughing up blood a couple of days after the attack, and continued to do so "the whole time we were there after the attack." They remained at MOPP level 4 for five or six hours. They radioed the 16th Support Group, but did not get a response for a couple of hours. Eventually they were told to come to Group Headquartrers (Hq.) for a message that Hq. didn't want to radio over. The message said that what they heard was a sonic boom, and instructed them to perform another test. The second test, performed several hours after the initial test, was negative. Members of the unit were told that the M9 paper had turned red as a result of exposure to diesel fumes. The message also gave the "all clear" for people to come out of MOPP level 4, but, Mr. Brady recalled, everyone was afraid to unmask.

After they got out of MOPP level 4, Mr. Brady went with Lt. Bryant to deliver gas masks and nerve agent pre-treatment pills to the 344th Maintenance Company. When they arrived back at their unit, everyone was dressed in their full chemical suits at MOPP level 4. They were told that while they were out riding around (without a radio), there had been another attack.

Beginning on January 22, Mr. Brady began getting too sick to work. He had been taking the nerve agent pre-treatment pills since about January 17, and had been getting severe headaches from them. Approximately three days after the attack, his eyes began to burn, he developed a high fever, and "taking a breath of air made his lungs feel like they were burning up." He also had diarrhea, sores, nausea, and a runny nose. On January 24, he went to the 13th Evacuation Hospital, which had no beds available for him. He described the hospital as completely filled with people that seemed to have the same illness that he had. His January 26 diary entry said: "I'd rather die than feel like this."

Mr. Brady stated during the interview that he "is convinced that there was a chemical attack." He reported that "everyone started getting pneumonia or flu-like symptoms after the attack", ... that the nerve agent pre-treatment pills "were useless," ... and that he is convinced that the PB tabs gave people headaches, but that they also "got hit with a nerve agent."

Mr. Brady currently suffers from severe recurring headaches, chronic fatigue, joint and muscle pain, rashes, depression, night sweats, insomnia, urinary urgency, diarrhea, gastrointestinal problems, lightheadedness, photosensitivity, shortness of breath, coughing, abnormal hair loss, sensitivity in his teeth, burning and itching everywhere, arthritis, worsening leg cramps, "flu

symptoms all of the time," a tingling in his arms, and a "bulging disc" in his neck. He had a heart attack in May 1993. His wife is suffering from fatigue, yeast infections, a rash, sinus headaches, aching in her right arm and a loss of feeling in her thumb, and two ruptured discs in her neck. Mr. Brady is forty-seven years old, his wife is thirty-seven years old.

Event 12:

January 1991 (4-5 Days into the Air War) Near Ras Al Khafji

Mr. Norman Camp is a Staff Sergeant with the U.S. Marine Corps. He told Senate staff during an interview that he was near Ras Al Khafji several days into the air war when the chemical alarms went off, not only at their position, but also at their Division Supply Area, which was about 20 miles to their east. They went on 100% alert, but word was passed down from division not to go to MOPP. Sergeant Camp recalled that his whole platoon began falling ill the following night. He got headaches, nausea, and diarrhea for a day. Most others were sick for about a day and a half.

Sergeant Camp currently suffers from headaches, joint pain in knees and elbows, memory loss, night sweats, occasional insomnia, urinary urgency, dizziness, photosensivity, shortness of breath, coughing and heart problems. His wife suffers from fatigue, yeast infections, menstrual irregularities, joint and muscle pain, and chest pain. Sergeant Camp is thirty-six years old, his wife is thirty-two years old.

Event 13:

January 19 or 20, 1991, 3:30 a.m. 3-4 Kilometers West of Log Base Echo

Mr. Dale Glover was a Staff Sergeant with the 1165th Miliary Police Company. He recalled being awakened at 3:30 a.m. The Battalion NBC NCO was announcing that they were under chemical attack. An M-256 kit registered a positive reading for a chemical agent. They went to MOPP level 4 for four hours. Afterward, all of them had runny noses.

When asked if people were made sick from the attack, Mr. Glover responded that most people were already sick from the pyridostigmide bromide pills. He said that they had been taking them for two or three days before the attack and that "a lot of people got sick and three or four had to be medevaced out."

Mr. Glover currently suffers from headaches, fatigue, joint and muscle pain, an inability to concentrate, recurring rashes, irritability, night sweats, insomnia, diarrhea, gastrointestinal problems, dizziness, blackouts, excessive photosensitivity, sore gums, swollen lymph nodes, and a spot on his brain. His wife is suffering from fatigue, menstrual irregularities, yeast infections, joint pain, some memory loss, and hair loss. Mr. Glover is thirty years old, his wife is 28 years old.

Event 14:

February 25, 1991 In Iraq, near the Kuwait Border

Mr. John Jacob, a mechanic with the 1st Infantry Division, was on a road march with Task Force 216. He was sitting in the driver's seat in his humvee when he detected what he believed to be gas. He recalled "getting a whiff of" a sweet, almond-like taste and smell, accompanied by a sudden burning in his throat and lungs, watering eyes, blurry vision and photosensitivity, nausea, dizziness and diarrhea. He donned his mask and gloves, and sounded an alarm. He recalled that whatever it was seemed to come through the driver's side window, as though something was caught in the wind and just drifted into his face. Although no one else seemed to be affected - Mr. Jacob said the others looked at him as thought he were crazy - his symptoms never went away. Afterward, he began to get headaches as well. His coordination was "messed up" for a couple of days after this incident. Mr. Jacob said that he later heard that a couple of people in his convoy detected something, but does not have any additional information. He says his M9 did not register anything.

Mr. Jacob says that he has been sick ever since that incident, and in addition to those symptoms already described, currently suffers from fatigue, joint and muscle pain, memory loss, recurring rashes, lumps at joint areas, night sweats, depression and irritability, insomnia, urinary urgency, gastrointestinal problems, shortness of breath, coughing, abnormal hair loss, dental problems, swollen lymph nodes, and a foot fungus that will not go away. Mr. Jacob is thirty-two years old.

How these events occurred is a matter for legitimate debate. But given the absence of a credible explanation -- one which explains what occurred during these events, methodical and detailed testing and analysis of the causes of the symptoms these individuals are experiencing and how these symptoms are transmitted must be undertaken. This is not only a matter of providing medical care to veterans and their families, but also a matter of national security. Many of the servicemen and women interviewed believe the foregoing events occurred as a result of SCUD or FROG missile attacks. Since the first staff report was issued last September, however, it has been learned that there are other methods by which Iraqi chemical and biological materials might have been dispersed.

A number of troops who were assigned to perimeter security posts have described to Committee staff individuals, persons who appeared to be Bedouins, who would leave canisters of what they believed to be chemicals outside perimeter fences and would then speed off in their four wheel drive vehicles. In these cases unit NBC NCOs would be assigned to check the canisters. Others talk about indigenous peoples leaving dead animals laying on airstrips used by U.S. personnel or about their tossing dead animals over perimeter walls in protest of the U.S. presence in Saudi Arabia. Still others have told of snipers and other Iraqi special operations missions that occurred as far south as Dhahran during the war.

CHAPTER 2, Part 4.

Reports by Coalition Forces of Iraqi Chemical Mines Located During Breaching Operations

The following accounts provide additional evidence of exposure to chemical warfare agents.

Event 15:

February 24, 1991

The first encounter with chemical mines came at 6:31 a.m. on February 24, 1991, during the initial mine field breaching operation by the 2nd Marine Division. According to the Chicago Tribune, which interviewed officers and enlisted marines involved in the operation, a FOX vehicle confirmed positive readings for a nerve agent and for mustard gas. A second detecting device gave the same positive reading. General Keys, the 2nd Division commander, and Col. Livingston, commander of the 6th Marine Regiment, told reporters that they believe it is possible that a chemical mine was blown up or hit.

On April 20, 1994, Committee staff received the Battle Assessment Documentation of the 6th Marine Regiment, Operations Desert Storm.

According to that report:

```
24 Feb 1991                          G Day

0630    B Co., 1/6 [Regiment] blows line charge across first mine field
        in Lane
        Red 1.  C Co., 1/6 engages possible BMPs with M60A1 main tank
        gun.  Target missed due to poor visibility.

0631    B Co., 1/6 reports possible nerve agent in first minefield in
        Lane Red 1.

0635    B Co., 1/6 is at MOPP level 4.  Fox vehicle confirms positive
```

Sarin nerve agent and lewisite mustard gas, vic Lane Red 1.

0650 1/6 reports possible nerve agent/mustard agent between obstacle belts.

0730 Rgmt S-2 reports to the 2nd Marine Division that Lane Red 1 is considered contaminated for the first 300m only.

1210 Rgmt S-2 reports TACC reported large number of dead sheep near King Khalid, possible anthrax. MAG-13 reports enemy forces moving rapidly south along highway from Kuwait City. Unknown number of tanks.

26 Feb 1991 G+ 2

1537 3/23rd under NBC attack, in MOPP 4; remainder 8th Marin MOPP 2.

1640 B Co., 1/6 clears Army stores camp, finds large number of Weapons and ammo, to include 155 arty shells painted completely yellow. Fox vehicle reports negative findings.

During the war, General Schwarzkopf told reporters he considered the reports of chemical agent detection on 24 February 1991 "bogus."

Event 16:

During the Ground War

British troops discovered Iraqi chemical mines on the gulf battlefield, according to Gannett News Service. An official said that the incident was reported to Prime Minister John Major's war cabinet; no details were given.

Other Combat-Related Reports

Event 17:

January 21, 1991 Taif, Saudi Arabia

Sergeant Thomas House served with the 2953rd Combat Logistics Support Squadron (CLSS), attached to the 48th Tactical Fighter Wing in Taif. Sergeant House's duties included the decontamination of U.S. Air Force F-111s that returned from bombing raids against Iraqi chemical and biological warfare facilities. According to the unit's records, the unit aircraft bombed 32 chemical targets, 113 bunkers, 11 Scud Missile sites, and 4 mine entrances.

Sergeant House and several others in his unit assigned to perform decontamination duties had worn only MOPP suits and had used water to decontaminate the aircraft. Sergeant House, whole primary duties are as an aircraft mechanic, later learned that chemical decontamination solutions were supposed to be used and that special suits were supposed to be worn.

On the evening of January 21, 1991, after decontaminating several aircraft that had returned from a bombing raid, Sergeant House's face began to burn and swell. He also noted a pungent odor. The following day, Sergeant House went to a U.S. Air Force medical facility. His U.S. Air Force medical records confirm this report. Shortly after the incident he began to experience headaches, coughing, nausea, vomiting, and diarrhea.

Sergeant House is currently suffering from recurring headaches, fatigue, joint and muscle pain, memory loss, recurring rashes, lumps under the skin, depression, irritability, night sweats, insomnia, urinary urgency, diarrhea, gastrointestinal problems, dizziness, blurry vision, photosensitivity, shortness of breath, coughing, bleeding gums, swollen lymph nodes, seizures, shaking, vomiting, fevers, chest pains, sinus infections and sinus growths. He is 32 years old. His wife currently suffers from nearly all of the same symptoms.

Seven other members of this unit have reported to Committee staff that they experienced similar exposures. They also currently suffer from similar symptoms.

Conclusions

- Iraq intended to use weapons of mass destruction against coalition forces and had the means to deliver these weapons.
- Events did not occur until the air war began.
- There are multiple witnesses to what appear to be best explained as chemical or mixed agent attacks. Symptoms appeared simultaneously with alarms going off, Patriots intercepting Scuds, alert alarms going off, etc.
- Smells, tastes, burning, stinging, numbness are all consistent with chemical or mixed agent attacks.
- Removal and replacement of MOPP gear is consistent with SOP for contaminated equipment.
- Sonic booms are not explosions associated with fireballs and it is unlikely that a commander would order troops not to discuss sonic booms.
- Rates of illnesses are reportedly high in these units.
- Servicemen and women have not received credible explanations of the events from commanding officers.
- The Department of Defense has consistently denied that there is evidence of exposure to chemical and biological warfare agents by U.S. forces, altering its position on specific aspects of this issue only when challenged with evidence that is difficult to dispute.
- The fact that the "sonic boom" explanation was utilized in units subordinate to different services to describe suspicious events and was followed by orders not to discuss the event, suggests that

this explanation originated at least at the theater level. Visual observations reported by field forces suggest this explanation was unrelated to the actual nature of these events.

Chapter 3, Part 1.

Reports of Exposure of Coalition Forces Resulting from the Fallout of the Bombings of Iraqi Chemical, Biological, and Nuclear Facilities (Group II)

The Czechoslovak Chemical Defense Unit in the Persian Gulf and the Results of the Investigations of the Military Use of Poisonous Gases.

Other Related Information

U.S. Unofficial Reports of Downwind Exposure Due to Coalition Bombings of Iraqi Chemical and Biological Facilities

Weather Reports, Climatic Information, and Imagery Smoke Plume Data

---

Reports of Exposure of Coalition Forces Resulting from the Fallout of the Bombings of Iraqi Chemical, Biological, and Nuclear Facilities (Group II)

There were serious concerns expressed prior to the Persian Gulf War about the fallout that would be caused by the bombing of Iraqi chemical, biological, and nuclear weapons production facilities, storage depots, and bunkers. Certainly these bombings were a necessary part of the conflict, but the consequences as well as the necessity must be acknowledged.

U.S. military doctrine warns that, according to its calculations, the use of a nerve agent against a target area of no more than a dozen hectares (a hectare is about 2.47 acres) can, under certain weather conditions, create a hazard zone downwind of up to 100 kilometers in length. Within this downwind area, friendly military units would have to take protective measures.100 The amount of agent and materials targeted during the Coalition bombings in Iraq exceeded the amounts cited in the example above certainly by multiples and possibly by orders of magnitude.

The dispersal of the chemical agents and other hazardous substances is controlled by factors such as topography, wind velocity, direction, temperature, precipitation, vertical temperature gradient and atmospheric humidity. These factors will all contribute to the size and type of dispersal

pattern which will be observed.101 Unclassified U.S. satellite imagery confirms that debris from the Coalition bombings was upwardly dispersed, rather than downwardly dispersed as would occur in offensive use, causing chemical agents to be carried by upper atmospheric currents and distributing "trace amounts" of chemical fallout over "down weather" positions. Material distributed from the destruction of the ammunition bunkers and storage depots also travelled upward and outward as confirmed in videotaped records of the destruction of these bunkers obtained by Committee staff. These concerns relating to the fallout from the destruction of these materials were expressed by several credi! ble so uces as noted below:

1. As a result of these concerns prior to the war, several of the U.S. national laboratories were consulted and/or prepared reports for the U.S. Army, the U.S. Air Force, and the Department of Energy, advising of the hazards which were associated with bombing these facilities.102
2. Prior to the war, Soviet chemical weapons expert I. Yevstafyev publicly advocated withholding information from the Coalition forces on chemical weapons and military facilities supplied by Moscow to Iraq, on the grounds of national security. "Strikes on chemical and biological weapons facilities on Iraq's territory could rebound on us and cause damage to the population of our country."103
3. On February 4, 1991, media sources reported that General Raymond Germanos, a spokesperson for the French Ministry of Defense, confirmed that chemical fallout -- "probably neurotoxins" -- had been detected in small quantities, "a little bit everywhere," from allied air attacks of Iraqi chemical weapons facilities and the depots that stored them.104
4. In late July, 1993, the Czech Minister of Defense confirmed that a Czechoslovak Federative Republic military chemical decontamination unit assigned to an area near the Saudi-Iraqi border had detected the chemical nerve agent Sarin in the air during the early stages of the Gulf War. In this unit, 18 of 169 individuals are believed to be suffering from Gulf War illnesses.105 While the report goes to some length to refute any allegations of the detection being the result of a direct chemical attack, it does defend the ability of the Czech chemical detection equipment to irrefutably confirm traces of chemical warfare agents. Further, the U.S. Government, in the November 10, 1993, briefing only referenced the detection of the nerve agent Sarin (GB) by the Czech forces on January 19, 1991. The Czech document, however, states that both Sarin and Yperite (HD) were detected that day. The fact that multiple agents were detected in measurable airborne concentrations suggests ! that t he agents may have emanated from fallout from Coalition bombings of Iraqi chemical weapons plants or storage bunkers, or from a direct mixed agent attack.

The Czechoslovak Chemical Defense Unit in the Persian Gulf and the Results of the Investigations of the Military Use of Poisonous Gases.

This section contains the main body of the translated Czech government report, prepared by the Czech Ministry of Defense in response to requests from Members of the Congress of the United States. Following this translation of the report are related accounts from independent sources.

The unit of 169 Czechoslovak military specialists was dispatched into the Gulf on the basis of an agreement between the governments of the Czech and Slovak Federative Republic (CSFR) and the government of the Kingdom of Saudi Arabia (KSA) regarding their activities and the conditions of their stay in Saudi Arabia. This Agreement was signed in Prague on November 19, 1990 and amended in Riyadh on November 22, 1990. The Federal Assembly of the CFSR ratified this Agreement. Resolution 97 was modified by an amendment by the Federal Assembly, authorizing the government of the CFSR to accept a provision of the agreement to permit the crossing of the international borders between the Kingdom of Saudi Arabia and Kuwait. The Government gave its approval through Resolution 71, dated January 31, 1991.

By executive order of the Commander of the Northern Region of the Ministry of Defense of the Kingdom of Saudi Arabia, the unit was assigned on the 22nd of December, in accordance with Article IV of the Agreement, to the military configuration of that region. As of January 1, 1991, the two chemical defense platoons were assigned to the 4th and 20th Brigades of the Army of the Kingdom of Saudi Arabia. The remainder of the unit was assigned to the base camp and to the headquarters.

Dislocation and strategic command of the unit was completely within the power of the Army of Saudi Arabia. Colonel Jan Valo, commander of the unit, provided specific assignment orders. His duty was to assure that in the course of fulfilling their duties, no Czechoslovak law or basic standard of international law was violated.

Beginning on January 27, 1991, the two chemical defense platoons were assigned to the Kingdom of Saudi Arabia brigades, crossing the border into Kuwait. They participated actively in assuring the anti-chemical defenses of the allied units during their execution of the operational plan. On February 5, 1991, the unit was supplemented, bringing its total numbers to 198 people.

The Czechoslovak anti-chemical defense unit primarily performed the following tasks:

1. Anti-chemical defense of the headquarters of the northern region troops located in the area of King Khalid Military City;
2. Anti-chemical defense of the 4th and 20th Brigades of the Kingdom of Saudi Arabia;
3. In the case of chemical attack of personnel, provide on their behalf facilities for chemical treatment and decontamination.

A part of the anti-chemical defense provisions was continuous chemical intelligence and surveillance, with the objective to identify the use of poisonous substances, provide data for alerting forces, and assist commanders in their decision making.

During the period after the commencement of the war on January 17, 1991, borderline concentration of poisonous substances were identified in the air by our chemical surveillance. In the Commander's Report, covering the anti-chemical defense battalions' activities during the period from January 1, 1991 until February 28, 1991, it specifically stated.

"During this period borderline life threatening concentrations of the chemical agents yperite [HD] and sarin [GB] were identified several times in both areas of the brigades and in King Khalid Military City (i.e., in the military encampment where the unit was stationed) probably the result of the Allies' air attacks on the storage facilities of chemical ammunition in the territory of Iraq." This information had been published at the time in the Czechoslovak media."

This aforementioned fact was confirmed by members of the battalion, chemical defense specialists who evaluated and ordered measures for personal protection. (see Attachments - pages from the book of the Operations Unit of the General Command of the Czechoslovak Army in prague, record #56), and all means of anti-chemical defenses were employed. After about two hours the alert was called off when repeated confirmation tests provided negative results.

The concentrations found, "0.002 grams of yperite per cubic meter and 0.003 mg per liter of an unspecified poisonous substance," [later identified by DoD as Sarin] are at the border of the maximum permitted threshold concentration affecting human organisms. These, however, were only one-time positive results from chemical surveillance which were not confirmed by anyone from the other participating countries. This was supported by the report on January 31, 1991:

"Since January 19th, the Czechoslovak unit has not found any other chemical substances."

The Czechoslovak anti-chemical defense unit had at its disposal all modern chemical surveillance and control technology. These are able to identify borderline levels (levels that do not affect the functions of human organisms) of suspected toxic substances and they can differentiate the nerve agents, such as sarin, from "V" agents.

The assertion that the chemicals were of very low concentrations that do not even cause temporary or minute changes in human organisms can be supported by the following facts:

- The results of the aforementioned surveillance;
- No signs of exposure to toxic materials were traced to personnel on site (toxic nerve agents, like sarin, cause instant reactions; for example: myosis. In the case of yperite, the first clinical signs of poisoning usually appear within 4 to 6 hours exposure);
- None of the personnel present had any later effects (related to exposure).

All of the chemical specialists were professional soldiers (there were 56 of them assigned over the length of the conflict). They are all graduates of military colleges and middle schools with a chemical defense major, and according to the curriculum, worked with highly toxic substances both in the laboratories and in field training. The training of anti-chemical specialists with selected types of poisonous materials had been conducted practically since the beginning of the anti-chemical defense program in 1956 until February 1990, when such training was halted because of complaints of destruction of the environment from environmental protection movements and the mayors of communities.

The anti-chemical defense specialists who had undergone this training are professionals, and they are able to identify the presence of toxic materials in the terrain, on military equipment, and in the air within the sensitivity ranges of the instruments used. Therefore, there is a high probability that the identified presence of poisonous materials is an objective analysis. At the same time, concentrations that are used at chemical field exercises and in laboratories are several times higher than the concentrations that were measured in the Persian Gulf.

It has been proven that military use of chemical weapons by Iraq did not occur and any such fact would have already have been subjecfted to extensive investigation by agencies of world peace organizations. One can consider that the data measured could have had origins from industrial facilities or even storage facilities of chemical ammunition that were hit by allied bombardment. This is supported by a report of the unit's copmmander, by my statements, and by other direct participants. All members of the unit were equipped with the most modern means of protection against toxic substances. They were fully comparable with the current world standard. Any kind of exposure by these types of toxic substances would manifest itself immediately or in a very short time, and nothing of this kind has been reported. Latent damage, if it can even be considered in this group, would surely have been uncovered during exit examinations.

On the basis of the abovementioned facts, one can conclude that the event cannot in any way be connected with the use of chemical weapons or their use in battlefield activities, and harm to the Czechoslovak anti-chemical defense unit due to the military use of toxic substances could not have occurred.

These conclusions also are supported by health care specialists. Neither at the time of identification of the toxic substances, nor later, was any member of the unit put under medical care as a result of exposure at this event. All members of the unit were subjected to a complex examination in military hospitals after their return from the Persian Gulf -- primarily in the Central Military Hospital in Prague. Even there, no serious changes caused by demanding climatic conditions or by exposure to toxic substances were identified.

Many veterans of the Persian Gulf conflict later participated in, and still participate in, activities of the unit in the Czech Republic Army in Yugoslavia. Even at the time of their departure, no one mentioned any problems.

Despite this, as of 31 August 1993, military doctors had examined 18 Persian Gulf veterans who suffered certain health problems, and three of them remain under a doctor's supervision. So far, in their cases, nothing has been identified beyond 'routine' problems related to similar long-term stays abroad.

Other Related Information:

On October 8, 1993, U.S. Senate staff interviewed Joseph Boccardi, who initially came forward with information about the detection of chemical agents by the Czechoslovak chemical detection unit prior to the release of the Czech report quoted above. According to this witness, a former member of the U.S. Army assigned to the 1st Cavalry Division as an M1A1 tank crewmember (driver/loader/gunner), he was injured when he fell off a tank during his service in the Gulf War. He was sent to a medical holding area in northern Saudi Arabia. While there, he was befriended by a lieutenant assigned to the holding unit (Lt. Babika). The lieutenant came to him one day and told him to come along with him.

According to Mr. Boccardi, he and the lieutenant drove about 15-20 minutes to a facility that he was told had been used as a Saudi basic training camp. Mr. Boccardi described the facility as beautiful and palace-like (near King Khalid Military City). Once inside, the lieutenant began speaking a foreign language which Mr. Boccardi believed to be Russian to two soldiers armed with AK-47s standing at the top of a staircase. The soldiers answered. The lieutenant explained that he was speaking Czech and that these soldiers were also Czech.

Mr. Boccardi said that he and lieutenant went into a room where there were about nine soldiers, smoking, drinking vodka, and playing cards. He learned that they were a NBC (nuclear, biological, chemical) team. He asked someone there "if we were kicking their butts so bad, why didn't they hit us with chemicals?" At that, everyone in the room got quiet and the Czech colonel spoke in "broken English" for the first time. He said, according to Mr. Boccardi, "they did hit us with chemicals." According to the Czech colonel, a SCUD hit where they were staying. As soon as they learned that the Patriot had missed the SCUD, they put on their chemical gear and went out onto a balcony near the railing. The Czech colonel said they detected traces of Sarin and another gas which Mr. Boccardi believed began with the letter T.

According to Mr. Boccardi, the Czech colonel said that he called U.S. command officials about the result of their tests. He, the Czech colonel, said that he was told not to say anything about it. The colonel also said that he later heard that a number of the soldiers in the area developed skin rashes shortly after this incident.

After this part of the conversation, the individuals discussed in general terms why they were not supposed to discuss the incident. This Czech colonel was identified as the commanding officer of this unit.

On December 5, 1993, according to published press reports, Jean Paul Ferrand, a logistics officer with the French contingent, told Senator Richard Shelby that nerve agents and mustard agents were detected on January 24 or 25, 1991, in an area south of King Khalid Military City. According to an Agency France Press report on that date, Ferrand said that two chemical weapons alarms went off when a storm blew wind from Iraq. Ferrand was also attributed as having said that special badges worn on the troops protective suits also registered the presence of chemical weapons.106

On Monday, March 28, 1994, Committee staff were contacted by a member of the 371st Chemical Company, located in Greenwood, South Carolina. This individual said that during the Gulf War, he served with the 1st platoon of this unit in the vicinity of King Khalid Military City (KKMC). According to this individual and several other members of his platoon interviewed by Committee staff, two days after an Iraqi Scud missile warhead had exploded in the desert, his platoon was sent to a site in the desert a few miles south of KKMC to train with the Czech chemical detection team that had conducted tests. They also were trained on the Czech equipment. According to two additional members of the platoon who trained with the Czech team, and were interviewed by Committee staff on April 4, 1994 in the Army Reserve Center in Greenwood, South Carolina, the Czech colonel who commanded the unit had told them that his unit had detected measurable quantities of chemical nerve agent immediat! ely af ter the Scud attack. Unit members were not able to determine the exact date of the incident, but believe it was sometime in mid to late January 1991.

The members of the unit described the facility where the Czechoslovak team lived as the "glass palace." They believed that it had previously been used as a Saudi military engineering training facility. The members of the U.S. unit who trained with the Czechs, all NBC specialists, said that the Czech equipment appeared to be more reliable than their own.

The unit Executive officer and first sergeant, while not present during the training mission, confirmed that they too were aware of the training, the missile attack, and the reported detection of the chemicals. The unit first sergeant said that this information had been recorded in the units logs, but that he received a message to send the logs to Washington, D.C. for historical purposes shortly after they returned from the Persian Gulf.

When asked if their unit did biological agent testing after incoming missiles had detonated, members of the unit said that they had no biological agent testing capability. While there were several other NBC units in the area, they were unaware of any unit that was conducting biological agent tests.

Finally, the unit said that they had been deployed on several occasions to decontaminate the buses and other vehicles that were used to transport Iraqi enemy prisoners of war to detention facilities.

One member of the unit estimated that as many as 85% of the members of this unit are currently suffering from many of the symptoms associated with Gulf War Syndrome.

U.S. Unofficial Reports of Downwind Exposure Due to Coalition Bombings of Iraqi Chemical and Biological Facilities

1. During the early phases of the air war, there was extensive media coverage of the coalition bombing of Iraqi chemical, biological, and nuclear facilities. ABC News reported that on January 27, 1991, near the Saudi-Kuwaiti border, elements of the 82nd Airborne Division went through a chemical alert drill that was more than an exercise. According to ABC News coverage, their sensors actually registered traces of chemicals in the air, the result, it appeared, of allied bombing of chemical plants in Iraq. A U.S. medical corpsman told reporters, "When the Air Force bombers hit all the gas places there in Iraq, there's a lot of contamination in the air. Some may have filtered down and set these things off. They're very, very sensitive."107
2. Brian Martin, of Niles, Michigan, a Gulf War veteran of the 37th Airborne Combat Engineer Battalion, 20th Airborne Brigade, 18th Airborne Corps, arrived in Saudi Arabia on October 8, 1990. According to Martin, in late January 1991, while assigned to an area between Rafha and Naryian about six miles south of the Iraqi border, he recorded in his journal and on videotape that chemical "false alarms" were going off almost every day. At first, according to Martin, the alarms were explained as being caused by vapors coming off the sand. Later, since the alarms kept going off and the troops no longer believed that they were being caused by the vapors, Martin said he was informed by both his battalion commander and the battalion NBC NCO that the alarms were sounding because of "minute" quantities of nerve agent in the air, released by the coalition bombing of Iraqi chemical weapons facilities. The troops were assured that there was no danger.

   Mr. Martin believes that he witnessed a Patriot intercept of an incoming SCUD missile between Khafji and Wadi Al Batin during the air war period. He was also given the anti-chemical warfare medication pyridostigmine bromide, and suffered some adverse side effects. He says the drug made him jittery and made his vision "jiggle." Since returning from Saudi Arabia, Mr. Martin has experienced memory loss, swollen and burning feet, joint disorders, muscle weakness, heart palpitations, shortness of breath, rashes, fatigue, headaches, insomnia, bleeding from the rectum, chronic coughing, running nose, burning eyes, and uncontrollable shaking on his right-side extremeties.108

3. Mr. Troy Albuck, former anti-tank platoon leader with the 82nd Airborne Division, reported to Committee staff that his unit was told that the chemical alarms were going off

because of what was drifting down from the Coalition bombings. He explained that his understanding of the situation was that "it was a lot like the effect of gasoline funes," in that non-lethal exposure was not harmful and would be counteracted by fresh air.109

4. Another source who requested confidentiality reported that he was located approximately 40 miles due east of King Khalid Military City (KKMC), when at one position, every M-8 alarm went off -- over 30 at once. The date was between January 20th and February 1, 1991. The NBC NCO radioed in that a nerve agent plant had been bombed about 150 miles away. The source recalled that they were told to take no action and they did not.110

Weather Reports, Climatic Information, and Imagery Smoke Plume Data

Operation Desert Storm

Weather reports during this period were censored by the U.S. and Saudi governments. But environmental groups monitoring an oil spill in the Persian Gulf confirm that the winds were at times blowing from the northwest to the southeast. The chemical and biological warfare agent production plants heavily bombed by the coalition forces during this period are located in Iraq to the north and northwest of coalition troop deployments along the Saudi-Iraqi and Saudi-Kuwaiti border.111

As cited above, the dispersal of chemical agents and other hazardous substances is controlled by other factors in addition to wind direction and velocity, such as topography, temperature, precipitation, vertical temperature gradient, and atmospheric humidity. These factors all contribute to the size and type of dispersal that will be observed.

In March 1992, the U.S. Air Force Environmental Technical Application Center published a compendium of the weather during Operation Desert Shield and Operation Desert Storm. The following is a summary of relevant data for January 17, 1991 through March 2, 1991, excerpted from Gulf War Weather. The report documents the changing weather conditions, detailing the wind and rain patterns that could easily have delivered chemical and biological agents to Coalition troop emplacements. On many dates, this report notes the smoke and dust from the bombings and from the burning oil wells. The notation of visible smoke plumes is not intended to depict the actual fallout from the bombed chemical, biological, and nuclear facilities, but rather to generally reflect the direction of movement of debris from the bombings.

Chapter 3, Part 2

Gulf War Weather

17 January 1991

There were extensive early morning clouds over the entire area. These clouds gradually moved southward during the day as their bases raised to 8,000 feet. Broken to overcast high clouds from the approaching frontal system began to move in after 1200Z, quickly spreading over the entire area with ceilings from 20,000 to 25,000 feet. The high cloud base thickened and lowered to 10,000 to 15,000 feet by the end of the day.

Winds were from the south or southwest at 6-20 knots, becoming southeasterly after 1200Z and 3-12 after sunset.

Early morning visibilities were as low as 3,200 meters with patchy ground fog in west central Iraq. Blowing sand and suspended dust reduced visibiilty to 3,200 meters during the afternoon on the Saudi Arabia - Iraq border.

High temperatures were 13-22 degrees celsius; lows 6-10 degrees celsius.

18 January 1991

The entire area was covered by clouds with bases at 10,000-15,000 feet. These quickly lowered to 3,000 feet and, in some places, as to low as 1,500 feet. The western part of the area began clearing in the afternoon, but 200 foot ceilings formed throughout the area by the end of the day. Clouds were layered to 30,000 feet from central Iraq to southeastern Kuwait.

Light rain fell throughout the area, beginning as early as 0500Z and lasting past 1800Z in eastern Iraq.

Winds were east to southwesterly at 5-15 knots.

Visibilities were 5km in morning ground fog and 3,200 meters in rain. After the rain passed, visibilities were as low as 4,800 meters in haze and fog, falling to 1,600 meters by the end of the day.

High temperatures were 16-22 degrees C; lows, 8-13 degrees C.

19 January 1991

High pressure was centered in central Iraq and central Saudi Arabia. The stationary frontal system over the area became active when an upper-air disturbance crossed it. A low pressure cell developed and moved the front southward.

The entire area was covered with low clouds with bases at 100-200 feet. By 1200Z the northern part, including Baghdad had cleared. Ceilings in the southern section rose briefly at midday to 1,000-1,500 feet, then returned to 500-1,000 feet for the rest of the day. After sunset the clouds spread northward into the Baghdad area, where ceilings were 1,500 feet. Middle and high clouds, layered to 25,000 feet with bases at 10,000 feet, were also present over the southern area throughout the day.

Winds were east to southeasterly at 5-15 knots, becoming north to northeasterly at 10-20 knots in the afternoon and diminishing to 3-10 knots after sunset."

Morning visibilities were near zero in dense fog throughout the area. The northern section cleared by 1200Z. Visibilities in the south rose to 3,200 meters at midday, then returned to as low as 800 meters for the rest of the day. Visibilities in the northern section were as low as 1,600 meters after sunset.

Temperatures fell in response to northerly winds. Highs were 10-18 degrees C; lows, 0-10 degrees C.

The Czechoslovak chemical detection team detected the nerve agent Sarin (GB) in two separate locations during the morning hours. In addition, Yperite (HD) was also detected. As noted above, the frontal patterns during this period moved to the south-southeast.

20 January 1991

A weakening low-pressure area moved southeastward down the Persian Gulf to the Strait of Hormuz. Cool moist low-level air moved southwest and west over the northeastern half of Saudi Arabia and extreme southwestern Iraq. Mid-level disturbances across the subtropical jet stream resulted in extensive middle and high cloudiness over northern Saudi Arabia and southern Iraq. By day's end another low had crossed Syria toward western Iraq.

At 0000Z broken low clouds at 1,500-4,000 feet covered Baghdad and the Tigris-Euphrates river valley. These clouds slowly cleared from the northwest; by 2100Z, only broken middle and high clouds from 10,000 to 30,000 feet covered the southern half of the Valley. The northern half, including Baghdad saw only thin high clouds. Broken clouds were layered from 1,500 through 25,000 feet over the western slopes of the Zagros mountains. Over the southern Zagros, tops reached 30,000 feet. Isolated afternoon and evening thunderstorms reached 35,000 feet in the extreme southeast near the Zagros mountains. After 2100Z, patchy broken low clouds formed

again over the northern part of the Tigris Valley and the immediate Baghdad area; bases were 1,000 to 1,500 feet; tops, 3,000 feet.

Light rain or showers fell over the southern half of the Tigris-Euphrates river valley and southwestern Iraq. There were isolated afternoon and evening thundershowers over the extreme southeast. Intermittent drizzle fell in the cool air moving west away from the northern Persian Gulf.

Winds were northwesterly to northerly over the Baghdad area, becoming northeasterly over the southern Tigris-Euphrates river valley. Over Kuwait and extreme southern Iraq, winds were northeasterly to easterly. Speeds dimished from 10-15 knots in the morning to 5-10 knots by mid-evening.

Visibilities in southern and southwestern Iraq and in extreme northeastern Saudi Arabia were near zero in fog during the night, but as high as 2,000 meters in southern Iraq and Kuwait during mid-afternoon. After dark, they dropped rapidly to less than 500 meters. Visibilities in the Tigris-Euphrates river valley, northwest of the low clouds, improved to 10 km by late morning. Patchy dense river fog formed after 2100Z, dropping visibilities to less than 500 meters.

High temperatures were 7-10 degrees Centigrade in the north and 18 degrees Centigrade in the south. There were freezing temperatures in Iraq and north Saudi Arabia, and subfreezing temperatures above 6,000 feet in the mountains of southeastern Turkey and northeastern Iraq. Central Saudi Arabian lows were 5-12 degrees C.

21 January 1991

Mid-level disturbances continued to move east-northeastward along the subtropical jet stream, crossing northern Saudi Arabia and Kuwait into Iran.

Patchy dense fog and low clouds again plagued southwestern Iraq and extreme northeastern Saudi Arabia until they dissipated in late morning. Cloud bases were from zero to 1,000 feet; visibilities, from near zero to 500 meters. Layered middle and high clouds persisted from 10,000 to 32,000 feet over most of northern and central Saudi Arabia and the central Red Sea.

Extensive fog and low clouds also prevailed in this area. Cloud bases were from near zero to 500 meters, and tops reached 2,000 feet. The clouds and fog slowly dissipated by late morning over southwestern Iraq and northeastern Saudi Arabia as far east as Rafha and King Khalid Military City. Over northeastern Saudi Arabia, the low clouds and fog became broken with bases near 3,000 feet and tops at 6,000 feet by early afternoon. On the Saudi Arabian and Persian Gulf coast, early morning ceilings were also near zero, but by late morning, most clouds had become scattered. Patchy fog and low clouds reformed throughout all of northeastern Saudi Arabia and extreme southwestern Iraq shortly after dark. Ceilings dropped to 200-500 feet by 2100Z. Layered middle and high clouds from 10,000-32,000 feet moved slowly southeastward over

southern Iraq, northwestern Saudi Arabia, and Kuwait; by 2100Z, they were over central and northeastern Saudi Arabia just southeast of Kuwait. Inf! rared satellite imagery taken just before sunrise in Kuwait shows these layered decks.

Precipitation, outside of thunderstorms and showers, was limited to light drizzle in areas of dense fog and low clouds.

Winds were northwesterly at 5-10 knots in southeastern Iraq and northeastern Saudi Arabia; they slowly became southeasterly at 5-10 knots in southwestern Iraq and in north-central and northwestern Saudi Arabia.

Early morning visibilities in the fog and low cloud area ranged from zero to 500 meters, improving to 1,000-3,000 meters by late morning and to 5-5 km by late afternoon. Visibilities were as low as 100 meters in denser fog patches. Patchy dense fog again formed after dark. The thickset fog was found along the Persian Gulf coastline and in shallow depressions inland where sand was still moist or where showers had occurred earlier in the day. On the Persian Gulf coast, visibilities improved from near zero at dawn to 1,000-2,000 meters by 0900Z, but dropped below 500 meters in fog by 1900Z. Patchy dense fog over and near the Tigris and Euphrates Rivers northwest of Basrah dissipated by 0600Z, but reformed after 1900Z

22 January 1991

Easterly to east-northeasterly low-level winds continued to bring moisture to west-central and northwest Saudi Arabia. The subtropical jet stream slowly weakened, but it continued to bring middle and high clouds northeastward across the Arabian peninsula into Kuwait and southwestern Iran.

Multilayered broken middle and high clouds persisted over southwestern Iraq, the southern Persian Gulf, and central Saudi Arabia from 10,000 to 28,000 feet. Visibilities remained good except where mountains were obscured by cloud.

Fog and low clouds again persisted all night over northeastern Saudi Arabia, the northern Saudi Arabian Persian Gulf coast, and extreme southwestern Iraq. Ceilings were again from near zero to 500 feet. Low clouds slowly lifted and dissipated, moving to a small area southwest of Kuwait by late morning. Bases were now 3,000 feet, with tops to 7,000 feet. Isolated thunderstorms, with bases as low as 2,000 feet, formed in late morning and early afternoon in extreme northeastern Saudi Arabia, Kuwait, the northern Persian Gulf, and southwestern Iraq. Tops reached 40,000 feet. Layered broken middle and high clouds persisted from 10,000 to 27,000 feet throughout the day over central and northeastern Saudi Arabia, Kuwait, and extreme southwestern Iraq. Iraq northwest of Basrah was clear. A visual DMSP satellite image taken shortly after sunrise in Kuwait, shows layered clouds with embedded thunderstorms over northern Saudi Arabia, the Persian Gulf, and southwestern Iran.

By sunset, low clouds and fog began to reform along the Iraqi-Saudi Arabia border northwest as far as Rafha. By late evening, the fog had lifted into broken low clouds with bases from 1,000 to 2,000 feet and tops to 5,000 feet. These clouds had spread north and northeast as far as the central Tigris-Euphrates river valley by 2100Z.

Showers and thunderstorms fell over northeastern Saudi Arabia, Kuwait, the northern Persian Gulf, and southwestern Iran. Patchy nighttime drizzle fell in area of dense fog and low clouds.

Winds were northeasterly at 5 knots, becoming southeasterly at 5-10 knots after 0900Z.

Visibilities dropped to less than 100 meters in fog before dawn.

23 January 1991

By mid-afternoon, the frontal system had moved south of Baghdad. A weak high pressure center formed over Kuwait early in the day and moved slowly southeast in the northern Persian Gulf. The weak stationary frontal system in central Saudi Arabia weakened further. The southwest to northeast subtropical jet stream over central Saudi Arabia moved southeastward to Qatar by 2100Z.

Isolated blowing dust reduced visibilities to as low as 3,200 meters in western Iraq.

Extensive broken to overcast low clouds, with bases of 500-1,000 feet and tops to 1,500-2,000 feet, covered northeastern and central Saudi Arabia. By early afternoon, skies were scattered to broken and bases had lifted to 3,000 feet. This layer dissipated shortly before sunset over northeastern Saudi Arabia, but reformed by 2000Z. In early evening, broken low clouds from 3,000 to 5,000 feet moved north and northeastward over Kuwait and the southern Tigris-Euphrates river valley in advance of the southward moving cold front. By 1200Z, the leading edge of broken to overcast frontal cloud layers had moved south of Baghdad, with bases from 3,000 to 4,000 feet; tops were 12,000-15,000 feet with broken high clouds above. Isolated rainshowers along and just ahead of the front reached 20,000 feet. By 2000Z, the leading edge of the frontal clouds had moved to near An Najaf in the Tigris-Euphrates river valley--the trailing edge was 60 miles north of Baghdad. Figure 3-8, a vis! ual sa tellite image taken at 1037Z, shows these layered clouds well.

Isolated moderate to heavy rain showers fell in central Iraq along and within 100 miles either side of the southeastward moving cold front. Patch light drizzle fell in northeastern Saudi Arabia before 0500Z.

Winds were easterly at 5-7 knots before dawn, becoming southeasterly at 5-10 knots by late morning in extreme southern Iraq and northeastern Saudi Arabia. By 1700Z, wind had veered to southerly at 10-15 knots. In central Iraq, winds were light and variable until 1200Z, becoming southerly at 10-20 knots after 1500Z.

24 January 1991

At 1200Z, heavy rain fell in extreme northern Saudi Arabia near the western Iraq border. Fog formed during the night and through the morning in north-central Saudi Arabia, along the western Saudi Arabia-Iraq border, in northern Jordan, and in southern Syria.

Winds were westerly to northwesterly at 10 knots during the first 12 hours, becoming northerly to northeasterly at 10 knots later in the day.

Visibilities in fog were as low as 5 km from 0000 to 1000Z in Saudi Arabia./I>

25 January 1991

A low moved east-northeast across Syria and Iraq, producing light snow and rain showers, blowing dust, and extensive cloudiness. Conditions improved toward the end of the day as the system moved into Iran. By the end of the day, another low had developed along the eastern Mediterranean coast, increasing cloudiness in western Iraq.

The low produced light snow over western Iraq, northeastern Jordan, and Syria; light rainshowers fell in northwestern Saudi Arabia and Iraq. Winds to 20 knots in northern Saudi Arabia produced duststorms from 0900 to 1500Z as far south as 28 degrees north.

Cloud cover was extensive until evening, by which time the system had moved into Iran. Broken to overcast low and middle clouds, along with some high clouds, preceded the low and its cold front. Scattered to broken low clouds followed the front; ceilings were 2,000-3,000 feet, but as low as 500 feet in rainshowers.

Precipitation consisted of light rainshowers that developed with the frontal system. At Z, Baghdad skies were overcast with rainshowers.

From 0400 to 1100Z in Saudi Arabia, fog reduced visibility to as low as 3,600 meters. Fog redeveloped in the evening. Some dust may have been advected into the area from storms farther west.

26 January 1991

By 1800Z, a weak secondary low had formed along the front in Saudi Arabia near 27 degrees North, 44 degrees East, and drifted slowly east.

The low pressure system produced light snow over southern Syria and light rainshowers in northern Saudi Arabia and western Iraq. Blowing dust south of the rain in Saudi Arabia reduced visibilities to 5 km. Winds were 20 knots around the low, but reached 30 knots with rain in northwestern Saudi Arabia.

The low moving across southern Iraq produced extensive cloudiness, as well as thunderstorms with bases at 2,000 feet and tops to 35,000 feet. Ceilings were as low as 800 feet in rainshowers.

Precipitation fell as light rain and rainshowers in Saudi Arabia around the low. Rainshowers also fell in Iraq.

Winds were northwesterly at 5-15 knots most of the day, becoming northeasterly as the storm system approached and northwesterly again as it passed. Peak speeds were 23 knots, probably higher in Iraq.

Visibilities in eastern Iraq were 6 km in haze early in the day through 0600Z. Later in the day, visibilities on the south sides of showers and duststorms were reduced to 8 km.

27 January 1991

A low moving eastward from northeastern Saudi Arabia to the Persian Gulf coast produced extensive cloudiness over most of southeastern Iraq. The low gradually weakened throughout the day, leaving only some low clouds in the vicinity of the Gulf by late evening. A cold front extended west-southwest from the low across Saudi Arabia. A strong high moved into northwestern Saudi Arabia, driving the cold front into southern Saudi Arabia; strong winds behind the front produced duststorms.

The storm system produced significant weather over large parts of Saudi Arabia. Light rain and rainshowers moving east with the low persisted at some Gulf coastal stations until 1900Z. The low produced multilayered clouds the first half of the day, but only low cloud the second half. A low overcast with fog developed behind the front in northwestern Saudi Arabia; fog dropped visibilities to as low as 200 meters. Skies improved by mid-morning and cleared by afternoon. Strong winds behind the front produced duststorms. Visibilities in northwestern Saudi Arabia was near zero in early evening because of blowing dust in 35 knot winds.

Skies were initially overcast in the southeastern two-thirds of the area, but Baghdad and vicinity was clear. Middle and high clouds were only present the first half of the day; they dissipated and moved off to the east by 1200Z. The low clouds moved southeast during the day and were out of Iraq by 1500Z. After 1500Z, the low cloud remaining over Saudi Arabia and Kuwait was broken to overcast.

Precipitation fell from 0000 to 0600Z as light drizzle, rain, rainshowers.

Winds were initially southeasterly at 10-15 knots ahead of the low, by northerly to northwesterly across Iraq behind the front. Winds shifted across the area by 1200Z; northerly to northwesterly winds were 10-20 knots with peak gusts to 30 knots. Speeds dropped to less than 10 knots during the evening.

Visibilities dropped to 9 km under the cloud cover in rain, fog, and haze.

28 January 1991

High pressure began to dominate the weather over Iraq and northern Saudi Arabia, but parts of Saudi Arabia were still affected by weather left in the wake of the low pressure system that prevailed on the 27th.

Morning fog and low clouds north of Riyadh lowered ceilings and visibility to 2,000 feet and 8km. There were some scattered to broken low clouds in the western Persian Gulf and at coastal stations. There was broken fog and stratus, with blowing dust, in southern Saudi Arabia.

Skies were generally clear except for thin scattered high cloud at 22,000-28,000 feet over northern Iraq and heavy black smoke over southern Iraq--see Figure 3-14. Winds were northwesterly at 3-10 knots, becoming more northerly toward the end of the day. Visibilities were as low as 1,500 meters over southern Iraq in the heavy smoke.

Visible Smoke Plumes

NOAA visual imagery in Gulf War Weather shows smoke plumes visible originating in an area just south of the two large lakes west of Baghdad and extending to the southeast. Available NOAA thermal imagery details smoke plumes in eastern Iraq moving to the southeast.

29 January 1991

A high pressure area over Saudi Arabia weakened as it moved southeast toward Qatar. A mid to upper level disturbance moved across the northern part of the region, resulting in extensive cloudiness over northern Iraq and Turkey.

The disturbance produced light rain and snow in Syria and snow in Turkey. There was extensive black smoke along the Persian Gulf coast. Suspended dust still reduced visibility in southern Saudi Arabia.

Isolated evening thunderstorms from 3,000 to 35,000 feet developed over southeastern Iraq. Some formed southwest of Baghdad at 1800Z.

Winds were near calm during the night, becoming east-southeasterly at 5-10 knots in the morning and increasing 10-20 knots during the afternoon. On the Persian Gulf coast, however, winds were northwesterly at 5-10 knots for the first half of the day before switching to east-southeast.

Visibilities were 8 km in blowing dust in the afternoon as the winds picked up. Black smoke reduced visibilities along the Persian Gulf coast--one station reported 9 km.

30 January 1991

A low pressure system developed in the eastern Mediterranean and moved eastward across Syria, reaching western Iraq by the end of the day.

Low clouds moved into western Iraq during the day with ceilings around 3,000 feet. The subtropical jet stream produced high clouds across central Saudi Arabia.

Cloud cover from the previous day's disturbance remained over eastern Iraq and Kuwait; broken low clouds at 3,000-6,000 feet in the north around Baghdad dissipated by 1100Z. Over Kuwait, broken middle clouds from 8,000 to 14,000 feet moved off to the east by 0600Z. Broken to overcast low clouds with 3,000 foot ceilings and 6,000 foot tops entered the western part of the area in the evening.

With the storm system approaching, winds were southerly to southeasterly at 5-10 knots.

Visibilities were restricted, primarily by haze and smoke from burning oil. Morning fog reduced visibility to 5 km in some spots; most haze restrictions were reported at 8 km. Some dust was raised during the day with increasing winds from the approaching system.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows smoke plumes originating in an area west of the two large lakes west of Baghdad and extending to the southeast. The plume splits into two plumes, one extending to the east and the other to the SSE just south of the southernmost lake.

31 January 1991

A slow moving low in the eastern Mediterranean Sea spread stormy weather throughout the Middle East as an associated frontal system passed through Iraq. At 0600Z, a secondary low pressure cell was centered southwest of Baghdad. It rapidly moved northeast while the cold front moved south and weakened. In north-central Saudi Arabia, the strong subtropical jet stream spread extensive high clouds.

Broken to overcast low clouds extended over the area until about 1600Z, with ceilings over Iraq as low as 3,000 feet and tops to 6,000 feet. Baghdad was affected between 0200 and 0900Z. Skies became clear in central Iraq and Kuwait after 1600Z as the front moved southward.

Isolated thunderstorms with tops to 35,000 feet passed northeast of Baghdad near 1100Z. Rain fell in western Iraq when the low pressure cell moved through.

Winds were southwesterly at 5-10 knots before the front and westerly to northerly at 15-20 knots immediately behind it.

Visibilities were reduced to 4,000 meters by duststorms in Kuwait and southern Iraq as the front passed. Ground fog lowered visibilities to about 6 km in northeastern Saudi Arabia, Kuwait, and southern Iraq.

1 February 1991

High pressure was centered over northwestern Saudi Arabia, keeping central Iraq cloud-free. A weak cold front extended from a low centered in north-central Iran. The front spread middle clouds from Qatar southwestward across Saudi Arabia. A slow moving low-pressure system centered on the Turkey-Syria border caused rainshowers in western Iraq, Syria, and Jordan. By 1800Z, middle clouds from this low reached Baghdad. A weak low developed on the central Red Sea coast in response to an upper air distrubance.

Middle clouds covered the mountains to Iraq's west and north. Light rain fell in Syria and northern Iraq between 1800 and 2100Z. Extensive areas of mountain-wave turbulence developed in the west between 0300Z and 1500Z and reached as far east as 43 degrees east. Early-morning ground fog formed in low lying areas over most of the eastern Arabian Peninsula. Lowest visibilities were 2,000-4,000 meters.

Skies were clear to scattered before about 0900Z, except in the extreme northeast. Broken low clouds from the low in Turkey spread southward; by 1600Z, they had reached Baghdad, with 3,000 foot ceilings. Thin broken or scattered cirrus spread northeastward from the northern Red Sea, covering the area south of 31 degrees North by 1100Z.

Winds were northeasterly at 5-10 knots in the south, westerly to the north. Afternoon winds were light and variable over central Iraq. Visibilities were generally good, but morning ground fog reduced them to about 6 km in northeast Saudi Arabia, Kuwait, and southern Iraq.

2 February 1991

A trough of low pressure formed to the east of a high pressure cell centered over eastern Saudi Arabia, causing low clouds in southern Iraq. A frontal systrem with low centers in the southern Caspian Sea and in east-central Syria extended along the northern Iraq border. The strong subtropical jet stream spread high and middle clouds over central Saudi Arabia.

Low and middle clouds prevailed over northern and western Iraq. Early morning ground fog formed in low-lying areas over most of the eastern Arabian Peninsula. Extensive areas of mountain-wave turbulence developed near the Syria-Iraq border between 0300 and 1500Z and reached to 43 degrees East.

The subtropical jet stream caused layered middle and high clouds over the area south of 32 degrees North throughout the day. Ceilings were between 15,000 and 25,000 feet, with the lowest along the northern Persian Gulf. Frontal low clouds stretched along the Syria-Iraq

border. The broken low clouds southwest of Baghdad included 4,000 foot ceilings and 6,000 foot tops. These clouds gradually moved east; by 2000Z, they were on the Kuwait coast. Another layer of low clouds with ceilings of about 3,000 feet formed over the Tigris-Euphrates river valley north of 31 degrees North during the night.

Winds were easterly at 10-15 knots south of 30 degrees North, southerly in the central area, and westerly north of 33 degrees North. They were gusty in the southern areas.

Duststorms reduced visibilities in the northern Nafud Desert eastward to southern Kuwait between 1200 and 2000Z. Minimum visibility was about 2,400 meters. Dense smoke was reported in northwestern Kuwait before 0800Z--visibilities were probably below 2,000 meters.

High temperatures were 13-16 degrees Celsius; lows, 2-11 degrees Celsius.

3 February 1991

The frontal system in eastern Syria began to move slowly eastward and break up, resulting in lowered ceilings and gusty winds. The nearly dry front passed Baghdad at 2000Z. A weak front extended from central Iraq to near Riyadh, spreading low clouds to Iraq's eastern section. The subtropical jet stream became more westerly than northwesterly, leaving the northern Persian Gulf cloud-free but spreading scattered to broken middle and high clouds across Saudi Arabia.

Sustained winds to 25 knots were reported in extreme northwestern Saudi Arabia as the front passed. Extensive duststorms reduced visibilities to 1,000 meters near the front in the Syrian and Nafud Deserts. Duststorms were also reported at 1500Z between Riyadh and Kuwait.

Middle and high clouds from the subtropical jet stream had moved out of the area by 1000Z. In the morning, scattered to broken low clouds covered Iraq east of 43 degrees East and all of Kuwait, some locations reported 3,000 foot ceilings. By 1200Z, the clouds had moved eastward to the Iraq-Turkey border. A small area of low clouds with 3,000 foot ceilings formed about 100 miles west of Baghdad at 1600Z.

Winds were westerly at 10-25 knots west of 45 degrees East, but southeasterly at 10-15 knots to the east. There were gusts to 35 knots near the front.

Duststorms reduced visibilities to as low as 500 meters at about 1500Z in southern Kuwait and northeastern Saudi Arabia. Elsewhere, visibilities were above 6 km. High temperatures were 8-20 degrees Celsius; lows 3-16 degrees Celsius.

4 February 1991

High pressure centered in southeastern Egypt strengthened and built into northwestern Saudi Arabia. The low pressure system that had been affecting northern Iraq continued to move

eastward. By 0900Z, the trough had moved southeastward to the south of Qatar. The subtropical jet stream continued to spread high and middle clouds over the central Arabia Peninsula.

In the early morning, middle clouds produced 10,000 foot ceilings in a triangular area between 30 degrees North and a southwest-northeast line running from 60 miles south of Baghdad, then eastward to the Iraqi border. These clouds rapidly moved southeastward. By 0600Z, they affected only the coast of Kuwait. The area was almost cloud free by 1000Z.

Winds were northeasterly at 10-15 knots inland, but northerly at 15-20 knots on the Kuwait coast. Inland, winds became northerly at 5-10 knots aftrer 1800Z.

Visibilities were generally above 10 km, but scattered fires and smoke plumes reduced visibility in Kuwait to below 4 km. One smoke plume, originating in southern Kuwait, measured 35 miles long and 10-15 miles wide.

High temperatures were 3-13 degrees Celsius; lows, 2-8 degrees Celsius.

5 February 1991

High pressure centered in central Iraq kept skies clear or scattered most of the day, but clouds associated with the subtropical jet stream still spread high and middle clouds over the central Arabian Peninsula. These clouds were scattered in the morning, but denser clouds moved in from Egypt by 1000Z.

Morning haze reduced visibilities in central Saudi Arabia; Riyadh reported 4,800 meters at 0500Z, improving to 8 km by 0800Z. Duststorms after 2000Z were the result of 20 knot winds over the Syrian and Nafud Desert; they lowered visibility to 5 km.

Clouds were limited to scattered cirrus until about 1100Z, but the subtropical jet stream moved scattered to broken middle and high clouds into southwestern Iraq later in the day. These clouds had moved over Kuwait by 1600Z, producing 10,000 foot ceilings during the night.

Winds were northeasterly at 10-15 knots along the coast, but light and variable in Iraq. As the front shifted farther north after 0900Z, winds in the south became stronger and more easterly. Visibilities were above 10 km except in Kuwait, where scattered fires and smoke plumes reduced visibility to below 4 km. High temperatures were 9-17 degrees Celsius; lows, 0-6 degrees Celsius.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows small visible smoke plumes over Kuwait extending to the south and southeast.

6 February 1991

High pressure was centered in northwestern Iraq, with a weak low pressure trough to the southeast between central Saudi Arabia--near Riyadh--and Israel. The subtropical jet stream remained over the northern Arabian Peninsula; associated high clouds became increasingly scattered after 0600Z.

At 0000Z, there were only scattered low, middle, and high clouds throughout the area. By 0300Z, the middle cloud deck had thickened; ceilings as low as 12,000 feet, with tops at about 18,000 feet, had formed over the southwestern half of Iraq. These clouds drifted eastward, and by 1900Z they were east of Baghdad and Kuwait. Middle and high clouds from the subtropical jet stream affected Kuwait and southern Iraq between 0400 and 1100Z; bases were at or above 10,000 feet, with tops to 32,000 feet.

Winds were light and variable in Iraq, but easterly at 5-10 knots in Kuwait. Morning fog, smoke, and dust reduced visibilities in Kuwait and southern Iraq to as low as 3,200 meters in spots. Afternoon visibilities in areas not affected by smoke were above 6 km.

Afternoon high temperatures were between 5 and 13 degrees Celsius. High pressure and almost clear skies drove morning low temperatures down to -2 degrees Celsius in the north and 7 degrees Celsius in the south.

Chapter 3, Part 3

Gulf War Weather (Continued)

7 February 1991

A low pressure system over northeastern Saudi Arabia resulted in afternoon and evening rainshowers and thunderstorms over Saudi Arabia and the Persian Gulf. The subtropical jet stream brought middle and upper cloudiness to central Saudi Arabia. Weak high pressure was centered over Iraq.

Scattered low clouds, with some middle and high clouds that were occasionally broken, extended across central Saudi Arabia and the Persian Gulf. Ceilings varied from 10,000 to 25,000 feet. Broken low clouds with bases at 2,000 feet were evident early in the morning over western Iraq. Light afternoon rainshowers fell over east-central Saudi Arabia. Isolated late evening thunderstorms were reported over the west-central part of the Persian Gulf. Tops were about 30,000-35,000 feet. Visibilities in northwestern Saudi Arabia were 7-9 km in haze and suspended dust. Suspended dust also reduced early morning visibilities in east-central Saudi Arabia to 4,800 meters.

Early morning skies were generally clear, but scattered middle clouds from the west moved into central Iraq and Kuwait by mid-morning. The middle clouds over central Iraq went scattered to broken at 10,000 feet by late morning. Skies became scattered by early afternoon. By early evening, cloud cover over Kuwait and southeast Iraq became scattered, variable to broken, at 10,000-18,000 feet. Isolated evening thunderstorms developed over extreme northern Kuwait; tops reached 30,000 feet.

Early morning winds were light and variable, becoming northwesterly to northerly at 10-15 knots by late morning. Haze and suspended dust reduced visibilities over central Iraq to 7-9 km. Smoke, haze, and suspended dust reduced visibilities in northern Kuwait to 5-7 km and to 1,600 meters in southern Kuwait.

High temperatures were 7-15 degrees Celsius; lows, 0-6 degrees Celsius.

Visible Smoke Plumes

NOAA visual imagery in Gulf War Weather shows visible smoke plumes extending in varying directions from the northeast to the south. Imagery captioned: "Smoke from Kuwait is being blown southward into northeast Saudi Arabia.112

8 February 1991

The low pressure system was now located over southeastern Saudi Arabia. The subtropical jet stream brought middle and high clouds across eastern Saudi Arabia. Weak high pressure was centered over Iraq.

Although skies were generally clear, broken middle clouds at 10,000 to 12,000 feet were observed over east-southeastern Saudi Arabia during early morning. Skies were broken to overcast at 4,000-5,000 feet between 0500 and 1300Z over northwestern and north-central Saudi Arabia. Scattered middle clouds were observed over western Iraq in the morning and afternoon, becoming broken at 10,000-12,000 feet during the evening. Blowing sand and dust lowered visibilities to 5-7 km in east-central Saudi Arabia.

Skies were generally clear, but scattered middle clouds were observed over southeastern Iraq during early morning. By mid-afternoon, there were scattered middle clouds over central Iraq. By late night, these became scattered to broken at 10,000-12,000 feet.

Winds were light and variable in the morning, becoming northwesterly to northerly at 10-15 knots. Visibilities in smoke over southern Kuwait and southeast Iraq was less than 1,600 meters. High temperatures were 7-15 degrees Celsius; lows, 0-6 degrees Celsius.

Visible Smoke Plumes

NOAA visual imagery in Gulf War Weather shows visible smoke plumes over southeast Iraq and southern Kuwait, extending to the south and southeast.

9 February 1991

High pressure dominated the region -- skies were generally clear.

Skies were clear except for scattered low clouds over northwestern Saudi Arabia and western Iraq. Winds increased to 15 knots with gusts of 25 knots across northern Saudi Arabia, where afternoon visibilities decreased to 4,800 meters in blowing sand and dust.

Early morning skies over central Iraq were broken at 10,000-12,000 feet. The clouds slowly moved into southeastern Iraq and dissipated during the day.

Winds were westerly to northwesterly at 10-20. Smoke and haze lowered visibilities in Kuwait to 5-7 km. High temperatures were 12-20 degrees Celdius; lows, 2-8 degrees Celsius.

10 February 1991

High pressure continued to dominate. Skies over Saudi Arabia and Iraq were clear during the day, but scattered to broken mid-level clouds with bases at 10,000-12,000 feet moved into western Iraq by late evening.

Skies were clear during the day, but by late evening, scattered to broken mid-level clouds with bases at 10,000-12,000 feet moved into central Iraq.

Winds were northwesterly at 5-10 knots. Thick smoke over east and southeast Iraq began to show in satellite photos by 0600Z and lasted until early evening. Smoke from southern Kuwait was also still visible; visibilities in the smoke were 800-1,600 meters. High temperatures were 10-15 degrees Celsius; lows, 0-5 degrees Celsius.

Visible Smoke Plumes

Imagery in Gulf War Weather shows smoke plumes visible over western, eastern, and southeastern Iraq. Direction: SSE; Smoke plumes are also visible over southern Kuwait. Direction: South.

11 February 1991

High pressure was centered over southeastern Saudi Arabia, but low pressure formed over western Saudi Arabia. The subtropical jet stream brought increased moisture in the mid and upper-levels to Iraq and nothern Saudi Arabia. A mid-level disturbance moving over Iraq and northern Saudi Arabia caused isolated thunderstorms and rainshowers.

Scattered high clouds over western Iraq became broken with bases at 20,000 feet from mid-morning through afternoon. There were scattered low clouds over northwestern and east-central Saudi Arabia during the day.

Scattered to broken upper clouds with bases at 22,000-25,000 feet were present during the morning and mid-afternoon over central Iraq. Morning skies were clear over Kuwait and southeastern Iraq, but smoke plumes were visible. Cloudiness decreased in central Iraq during the day, but increased in southeastern Iraq and Kuwait. Skies became scattered to broken, occasionally overcast, at 4,000-6,000 feet. By late afternoon, thunderstorms (tops 35,000 feet) and rainshowers had formed over Kuwait; they moved off to the east and dissipated by late evening.

Winds acompanying the thunderstorms in Kuwait reached 25-35 knots, but over the rest of the area, they were northwesterly at 10-15 knots.

Smoke and haze lowered morning visibilities to 4,800 meters in Kuwait and southeastern Iraq. Rain and rainshowers reduced evening visibilities to 1,600-3,200 meters. High temperatures were 10-15 degrees Celsius; lows were 0-5 degrees Celsius.

Visible Smoke Plumes

Imagery in Gulf War Weather shows smoke plumes clearly visible over Kuwait. Direction: South; Smoke plumes are also visible over southeastern Iraq. Direction: SSE.

12 February 1991

High pressure dominated, but a low-pressure system moved into the eastern Mediterranean by the end of the day, increasing cloudiness over western Iraq.

Broken middle clouds at 8,000-10,000 feet were present over northeast, east and central Saudi Arabia and the Persian Gulf during the morning. By late evening, there were scattered to broken high clouds at 20,000-25,000 feet over western Iraq.

Broken middle clouds at 8,000-10,000 feet remained over Kuwait until mid-morning. Scattered upper clouds moved into central Iraq by late evening.

Winds were northwesterly to northerly at 10-15 knots. High temperatures were 10-15 degrees Celsius; lows, 0-5 degrees Celsius. Visibilities in Kuwait were 5-7 km due to smoke and haze.

Visible Smoke Plumes

Imagery in Gulf War Weather shows smoke plumes clearly visible over eastern Iraq and Kuwait. direction: S-SSE.

13 February 1991

High pressure dominated the Saudi Arabian peninsula, but low pressure centered over the eastern Mediterranean sent moisture into Iraq and northern Saudi Arabia. The low moved to the northeast as high pressure intensified behind it.

Scattered skies became broken to overcast over western and northern Iraq. The 25,000 foot ceilings prevailing in the morning became 8,000-10,000 feet during the day. By the end of the day, skies were scattered again. Over northern Saudi Arabia, skies were scattered, but occasionally broken, at 20,000-25,000 feet.

Scattered skies became gradually broken over central, east-central, and southeast Iraq and Kuwait. Smoke plumes were visible over southern Kuwait and the northern Saudi Arabian Gulf Coast. (see below)

Winds were northwesterly at 10-15 knots. Visibilities in southeastern Iraq and Kuwait were 5-7 kilometers in smoke and haze. High temperatures were 13-16 degrees Celsius; lows, 1-4 degrees Celsius.

Visible Smoke Plumes

Imagery in Gulf War Weather shows smoke plumes clearly visible over southern Kuwait. Plume directions appear to be to the S-SSE.

14 February 1991

High pressure over Iran and Syria resulted in fair weather across most of the region.

Morning skies over northern Iraq were overcast with middle and high clouds; ceilings were as low as 10,000 feet. The clouds moved eastward and were over Iran by 1200Z. Broken low and middle clouds over central and southern Saudi Arabia produced 5,000 foot ceilings with scattered light rainshowers and 9 km visibilities. The clouds moved southeastward and became scattered after 1200Z. Visibilities in western and southern Iraq were as low as 6 km where 20 knot winds resulted in localized suspended and blowing dust.

Cloud cover consisted only of thin scattered high clouds over eastern Iraq and Kuwait: bases were 20,000 feet; tops 25,000 feet. The high clouds moved east into Iran by 1200Z. Winds were light and variable in the early morning, becoming northerly to easterly at 5-15 knots during the day.

Morning visibilities along the Persian Gulf coast near Kuwait were 8 km in fog. Widespread haze over northern Saudi Arabia produced visibilities of 8 km. High temperatures were 16-18 degrees Celsius; lows were 2-5 degrees Celsius.

Visible Smoke Plumes

Imagery in Gulf War Weather shows smoke plumes clearly visible over southern Iraq. Plume directions appear to be to the SE.

15 February 1991

High pressure over Iran and Turkey extended southward across most of the region. Broken high clouds passed through western Iraq to the east during the afternoon, followed in the evening by a large shield of high cloud entering from the west. Scattered to broken high clouds over parts of central and southern Saudi Arabia--with bases between 9,000 and 12,000 feet--dissipated partially during the day. Blowing dust in northern and western Saudi Arabia reduced visibilities to as low as 5 km.

Broken high clouds passed through the area between 1300Z and 2100Z with bases at 24,000 feet and tops to 32,000 feet. There were followed by scattered high clouds that moved into central Iraq from the west by the end of the day. Scattered bases were at 24,000 feet with tops to 32,000 feet.

Winds were northerly at 5-10 knots through the morning, gradually shifting to easterly at 5-15 knots in the afternoon and evening. Smoke that is clearly visible restricted visibility up to 14,000 feet. Evening ground fog developed along the Kuwait coast, dropping visibility to 8 km. High temperatures were near 20 degrees Celsius; lows varied from 2 degrees Celsius in the north to 8 degrees Celsius in the southeast.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows smoke plumes visible over Kuwait extending to the south over Saudi Arabia.

16 February 1991

High pressure over Iran weakened as a strong frontal system approached from the west. The polar jet stream dipped southward into the eastern Mediterranean as the subtropical jet stream crossed Egypt and brought in upper level moisture. A new low pressure center formed on the front over Syria by 1500Z and moved southeast. The low and its accompanying cold front reached western Iraq by 1800Z.

Multiple cloud layers covered the region southward to 25 degrees North with scattered to broken low clouds and broken to overcast middle and high clouds. Light rain and rainshowers lowered ceilings to 1,000 feet and visibilities to 1,100 meters. The blowing dust already present in northwestern Saudi Arabia at 0000Z spread to include much of northern Saudi Arabia, especially south of the rain. Winds up to 30 knots produced duststorms with visibilities as low as 200 meters in northern Saudi Arabia, Syria, and western Iraq.

Cloud cover increased and ceilings lowered during the day. Skies were initially scattered with high clouds from 27,000 to 30,000 feet, but became broken to overcast by morning, with multiple layers between 25,000 and 35,000 feet. Bases lowered to 20,000 feet by 0700Z. Broken middle clouds reached central Iraq at about 1100Z with 12,000 foot bases and 18,000 foot tops. Scattered low clouds moved in by early evening with 2,000 foot bases and 6,000 foot tops; middle-cloud ceilings were down to 8,000 feet by then. Low clouds increased in the evening. Light rain and rainshowers lowered ceilings to 1,000 feet.

Winds varied from easterly to southerly with the approaching frontal system. Initial 5 to 10 knot speeds increased during the day. The highest reported sustained speed was 30 knots.

Visibilities worsened throughout the day. Dense black smoke over the southern half of Kuwait reduced visibilities to 6 km--some pilots reported certain areas as "unworkable." Duststorms developed as wind speeds reached 20 knots around 0900Z; speeds to 30 knots dropped visibilities to as low as 200 meters later in the day. Local evening visibilities were as low as 1,100 meters. High temperatures increased to 20-25 degrees Celsius as the front brought warm air into the region; lows were 6-8 degrees Celsius.

17 February 1991

A low pressure area moved northeast from central Iraq across Iran as its cold front moved through most of Iraq. A weak secondary low formed along the front in south-central Iraq near the Saudi Arabian border. The cold front continued southward into central Saudi Arabia and weakened. High pressure intensified behind the front.

Rain fell along the front in northern Saudi Arabia early in the day, but moved into central Saudi Arabia by evening. Visibilities were 8 km, but dropped to 4,700 meters in a 1500Z thunderstorm in west-central Saudi Arabia. Blowing dust ahead of the front reduced visibilities to as low as 1,700 meters. Duststorms behind the front dropped visibility as low as 900 meters in western Iraq. Skies over central Saudi Arabia were scattered at 4,000 feet, and broken to overcast at 10,000 feet.

Cloud cover from 0000Z to 1100Z was broken to overcast with layered low and middle clouds; ceilings were 3,000 feet, tops to 15,000 feet. Skies over southern Iraq and Kuwait were overcast at between 20,000 and 35,000 feet. Skies in Iraq began to clear by 1100Z, leaving scattered low clouds from 3,000 to 6,000 feet that continued moving east and south; all of Iraq, except for its extreme northern border, was clear after 1600Z.

Rain and rainshowers fell over northeastern Saudi Arabia, southeastern Iraq, and Kuwait. The bases of late morning thunderstorms near the Saudi Arabia border were 3,000 feet, with tops to 35,000 feet. The rain moved eastward by evening.

Winds were southerly to southeasterly at 5-15 knots ahead of the low and cold front, and northerly to northwesterly at 5-20 knots behind it. Speeds diminished to 5-10 knots in the evening.

Visibility in rain was 4,700 meters. Blowing dust in some areas of northeastern Saudi Arabia that had remained dry lowered visibilities to 6 km. Evening fog formed locally where rain had fallen, lowering visibilities to 6 km.

Daytime temperatures were highest (20 degrees Celsius) in the west where skies cleared first, but highs in the east were as low as 14 degrees Celsius. Daily lows were in the evening after the cold front had passed. Low ranged from 6 degrees Celsius in the north to 12 degrees Celsius in the south.

18 February 1991

A high pressure cell moved over Iraq and dominated much of the region's weather. Morning fog developed over north-central and northwestern Saudi Arabia but dissipated by early afternoon. Clouds associated with yesterday's cold front were over central Saudi Arabia and the Persian Gulf, where they produced scattered light rain through the morning until moving into the

Arabian Sea in the afternoon. Skies were scattered from 3,000 to 6,000 feet, broken from 10,000 to 18.000 feet, and broken from 28,000 to 33,000 feet.

Thick morning ground fog lifted to form 1,000 foot ceilings that dissipated by about 1000Z. Broken middle clouds over southern Kuwait and northeastern Saudi Arabia moved off to the southeast during the first 6 hours of the day; ceilings were 7,000 feet with tops to 12,000 feet. Middle and high clouds moved into the region from the northwest during the second half of the day; scattered to broken middle clouds were from 8,000 to 18,000 feet, and thin broken high clouds were from 29,000 to 35,000 feet.

Winds were generally light and variable in the north, but northerly to northeasterly at 5-10 knots in the south. Visibilities ranged from near zero to 2,000 meters in thick and extensive morning fog across portions of Iraq and Saudi Arabia. The fog, which was concentrated over (and to the west of) the Tigris-Euphrates river valley in Iraq, didn't burn off until about 1000Z. Fog formed again in the evening over northern Saudi Arabia and Kuwait, dropping visibilities to 4,800 meters. High temperatures were 17-20 degrees Celsius; lows ranged from 5 degrees Celsius in clear areas to 11 dcgrees Celsius under the fog.

19 February 1991

A low pressure area developed over Syria and moved eastward into northwestern Iraq. A secondary low developed in northwestern Saudi Arabia and moved eastward into northern Saudi Arabia. Two lines of strong thunderstorms--one over northern Saudi Arabia, one over Iraq--developed between 1500 and 1800Z and continued well into the next day.

Scattered low clouds over western Iraq and northern Saudi Arabia early in the day were from 3,000 to 5,000 feet. Thin high clouds moved in that afternoon. Thunderstorm bases were at 3,000 feet, tops to 35,000 feet. Rain and rainshowers began after 1500Z. In central Saudi Arabia, scattered to broken low and middle clouds, with bases at 4,000 and 10,000 feet, produced scattered evening rainshowers.

Cloud cover in the first 6 hours was limited to southeastern Iraq and Kuwait, where skies were scattered at 3,000 and 5,000 feet and then broken from 20,000 to 25,000 feet. After these had cleared out in the afternoon, a new high thin broken layer at 22,000 to 25,000 feet moved in. Convective activity from the west entered the area at about 1800Z, producing bases that were generally 3,000 feet, but as low as 1,000 feet in thunderstorms; tops were to 35,000 feet. Convective cells consolidated to form a nearly solid, north-south line in central Iraq as another, similar line formed in northern Saudi Arabia. Middle-cloud ceilings outside showers were at 10,000 feet.

Thunderstorms produced localized moderate to heavy rain after 1800Z. Light rain and rainshowers fell outside the areas of strong convection.

Winds were east-southeasterly at 5-10 knots during the first half of the day, increasing to 15-20 knots by afternoon. Isolated gusts to 30 knots occurred with thunderstorms. Visibilities were less than 1,000 meters in rain associated with thunderstorms, but 7 and 9 km elsewhere in rainshowers, black smoke from Kuwait, fog, and/or blowing dust. High temperatures were 19-21 Celsius; lows, 7-11 degrees Celsius.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows smoke plumes visible over Kuwait and extreme northeastern Saudi Arabia.

20 February 1991

The low over Iraq moved eastward into Iran as the secondary low over northern Saudi Arabia moved southeast along the Persian Gulf coast. Thunderstorm activity that started the day before continued across eastern Iraq, Kuwait, and northeastern Saudi Arabia. Lines of thunderstorms moved graduallay eastward as new cells developed on their southwestern ends.

A cold front moved southeast across central and eastern Saudi Arabia, producing scattered rain showers and visibilities as low as 800 meters in blowing dust. Skies were scattered from 4,000 to 6,000 feet and broken from 10,000 to 15,000 feet. Thunderstorms that had been in northern Iraq earlier in the day moved into Iran, followed by broken to overcast low and middle clouds with ceilings at 3,500 feet.

Thunderstorms moved across the area from west to east. Bases were at 1,000 feet and tops reached 40,000 feet. Cloud cover outside thunderstorms was broken to overcast, and multilayered from 3,000 to 35,000 feet. Surface moisture helped produce low broken clouds west of the front in central Iraq; ceilings were 3,500 feet, with tops to 6,000 feet. There were also broken middle clouds from 10,000 to 15,000 feet. Parts of south-central Iraq and north-central Saudi Arabia cleared as thunderstorms moved east.

Precipitation was moderate to heavy in thunderstorms, but light away from the strong cells.

Winds were east-southeasterly at 10-20 knots, becoming west-northwesterly at 10-25 knots as the storm moved through. Isolated thunderstorms were above 30 knots.

Visibilities were 9 km outside thunderstorms, but less than 1,000 meters in heavy thundershowers. Blowing dust in areas along the front that had not received much rain lowered visibilities to 7 km. Evening fog formed along the Persian Gulf coast, lowering visibilities to 1,500 meters by 2300Z.

High temperatures ranged from 24 degrees Celsius in the southeast ahead of the cold front to as low as 15 degrees Celsius in the northwest behind it. Lows were 9 degrees Celsius in the north and 14 degrees Celsius in the southeast.

21 February 1991

A low pressure system moving south along the Persian Gulf neared Dhahran by 0300Z; by 1500Z, it was on the United Arab Emirates coast near 53 degrees East. Its cloudless cold front extended southwest across the Arabian Peninsula. By 0900Z, an area of high pressure had formed in northwestern Saudi Arabia near the Iraqi border.

The low pressure system spread a wide area of clouds, rain, and isolated thunderstorms over the Persian Gulf and along the coast as it passed. Ceilings were generally 10,000 feet in rainshowers, but ceilings in thunderstorms were reported at 3,000 feet. Inland, the front caused duststorms as it passed, reducing visibilities in some places to 800 meters. Fog blanketed northern Saudi Arabia in the wake of the low pressure system, but dissipated by 0800Z at most locations; visibilities were as low as 2,800 meters along the coast, but much lower in protected wadis. Along the eastern Saudi Arabian coast, visibilities were 4,800 meters in dense haze. Between 0500 and 1300Z, sporadic duststorms reduced visibilities to 6 km in the Syrian Desert.

An overcast layer of low clouds resulted in 500 foot ceilings over Kuwait and Iraq south of Baghdad. Clouds tops were about 1,200 feet. The clouds lifted to 1,000-3,000 feet by 0500Z and dissipated by 0700Z. South of 29 degrees North, broken middle clouds with 10,000 foot bases persisted until about 0700Z. Skies were clear after 0900Z.

Winds were northeasterly or northerly at 10 knots in the south, easterly at 10-15 knots in the north. Central Iraq's winds were light and variable. Highest speeds--20 knots along the northeastern Saudi Arabian border--were reported at 1500Z.

Fog and visibilities of 500 meters were common. The fog dissipated in the northwest first, but lingered until 0800Z in Kuwait and Iraq south of 32 degrees North. Dense smoke reduced visibilities in southern Kuwait and northern Saudi Arabia. The afternoon high temperature was 15 degrees Celsius. Morning lows were 6-11 degrees Celsius, but by evening, temperatures in the north had fallen to about 3 degrees Celsius.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows smoke plumes in southern Kuwait extending southward in Saudi Arabia.

22 February 1991

High pressure was centered over east-central Saudi Arabia. A cold front extending from a low in the eastern Mediterranean spread scattered to broken high clouds across Syria and northwestern Iraq. The system had moved into eastern Syria by 1600Z. A low pressure system near the Strait of Hormuz brough low cloudiness and rain to the southeastern Arabian Peninsula.

A dense band of smoke aloft extended from the northern Persian Gulf along the Saudi Arabian coast into the Rub al Khali. Bases were about 10,000 feet, tops to 18,000 feet. Skies were clear to scattered, but scattered to broken middle and high clouds moved over the extreme northeast by 1800Z. Ceilings, where present, were 10,000 feet with tops to 15,000 feet. The middle and high clouds were nearing Baghdad by 2300Z.

Visibilities were unrestricted except for areas affected by smoke, where they were generally about 6 km. Pilots reported smoke tops to about 15,000 feet and inflight visibilities as low as 1,000 feet. Dense smoke over and south of Kuwait.

Winds were light and variable before 1500Z, becoming southeasterly to easterly at 5-10 knots to the east of the front after 1500Z. Elsewhere, winds remained light. After sunset, winds were nearly calm. High temperatures were 13-18 degrees Celsius; lows, 1-8 degrees Celsius. The lowest temperatures were in the eastern Nafud Desert.

Visible Smoke Plumes

NOAA visual imagery in Gulf War Weather shows smoke plumes visible over Kuwait moving southward over coastal Saudi Arabia.

23 February 1991

High pressure centered over the eastern Arabian peninsula moved southeastward into the Rub al Khali by 2000Z. Even though the frontal system dissipated as it moved across northwest Saudi Arabia, it still caused isolated light showers and duststorms. The subtropical jet stream brought middle and high clouds eastward over the area after 0900Z. Low pressure formed over the Red Sea.

Fog reduced visibilities along the central Persian Gulf to about 1,000 meters between 0100 and 0400Z and reformed after 2000Z. Scattered to broken low and middle clouds with light isolated rainshowers reduced visibilities to 10km along the weak low pressure system in the west. Duststorms caused 4,000 meter visibilities in the Syrian and Nafud Deserts between 0900 and 1700Z. Middle and high clouds produced 10,000 foot ceilings over northwestern Saudi Arabia after 0900Z. Smoke from the Kuwaiti oilfields had reached Qatar; although concentrated at 10,000-12,000 feet, the smoke mixed with haze at lower levels to produce 6 km visibilities.

In the west, the low pressure system caused scattered to broken clouds at 10,000 feet until about 0600Z, when they became scattered. By 1200Z, middle and high clouds began to move into the

area south of 31 degrees North, causing broken to overcast ceilings at 10,000 to 12,000 feet. These clouds were east of 45 degrees East by 1900Z. Between 0400 and 1600Z, another band of middle and high clouds formed along the Iran-Iraq border north of 32 degrees North. Ceilings were about 8,000 feet, with tops to 32,000 feet. Isolated thunderstorms formed over Kuwait by 2000Z, with 2,500 foot bases and tops to 35,000 feet.

Winds were northerly to northwesterly at 10-15 knots east of 45 degrees East. Elsewhere, winds were easterly at 5-10 knots. Duststorms reduced visibilities to 8 km along the Iraq-Saudi Arabia border between 0800 and 1500Z. Dense smoke covered Eastern Kuwait and reduced visibilities generally to less than 8 km, with isolated cases as low as 1,000 meters. High temperatures were 13-16 degrees Celsius; lows, 7-13 degrees Celsius.

Chapter 3, Part 4

Gulf War Weather (Continued)

24 February 1991

A low pressure system moved slowly eastward along the Iraq-Saudi Arabia border. High pressure was still centered over the southeastern Arabian Peninsula. The subtropical jet stream's middle and high clouds moved eastward over the Persian Gulf. They were out of the area by about 1900Z, but another upper level disturbance brought more high and middle clouds eastward. At 1500Z, these clouds were in north-central Saudi Arabia.

Morning fog again blanketed the central Persian Gulf coast between Dhahran and the Strait of Hormuz. Visibility was poorest (2,000 meters) south of Qatar. Gust winds and blowing dust accompanied the low near the northern Saudi Arabian border where 15 to 20 knot winds raised dust that reduced visibilities to 6 km. Broken to overcast middle clouds produced 8,000 foot ceilings over the northern Persian Gulf, but embedded low clouds resulted in isolated ceilings at 3,000 feet. Isolated rainshowers fell near the low, reducing visibilities to 8 km. Isolated thunderstorms, with bases at 2,500 feet and tops to 30,000 feet, developed southwest of Riyadh between 1500 and 2200Z. Smoke reduced visibilities and obscured skies along the Persian Gulf as far south as 23 degrees North.

In Kuwait and southern Iraq, skies were broken to overcast with 8,000 foot ceilings until about 0500Z. Tops of these multiplayered clouds reached 35,000 feet. There were isolated 2,500 foot ceilings. By 0500Z, the higher clouds had moved east, leaving scattered to broken low clouds over Kuwait. In the evening, more middle and high clouds began to move into the southern half from the west. They reached western Kuwait by 2000Z, bringing 9,000 foot ceilings and tops to 30,000 feet.

Isolated rainshowers and thunderstorms affected Kuwait and southeastern Iraq unti 0600Z. Rain, heavy at time, reduced visibility to 5 km. Winds in Saudi Arabia and western Iraq were southwesterly to westerly at 10-15 knots, increasing to 15-25 knots by 0900Z south of 32 degrees North, with gusts to 30 knots. By 2100Z, speeds had diminished to 10-15 knots. Winds in the Tigris-Euphrates river valley were southeasterly at 10-20 knots, but dropped to 3-5 knots after sunset.

Duststorms reduced visibilities to as low as 1,000 meters in Kuwait and southern Iraq between 0900 and 2100Z. Dense smoke from the Kuwaiti oil fires moved northwestward. Visibilities just south of Baghdad were less than 3,000 meters. Fog formed after 2100Z in Kuwait and southern

Iraq, reducing visibilities to less than 4,000 meters. Afternoon high temperatures were 10-21 degrees Celsius; morning lows ranged from 1 degree Celsius in the northeast to 15 degrees Celsius in the southeast.

Visible Smoke Plumes

DMSP visual imagery in Gulf War Weather shows smoke plumes from Kuwait moving westward into Iraq.

(As the ground war began, Iraqi forces set fire to Kuwaiti oil wells, resulting in extremely heavy smoke concentrations over the entire region.)

25 February 1991

An upper air disturbance moving northeastward spread stormy weather over the northern Arabian Peninsula; by 0900Z, most of the region was covered with clouds. A low pressure system lingered over northwestern Saudi Arabia as the high pressure cell in the southeast moved eastward. Low pressure moving east from the northeastern Mediterranean spread clouds southeastward over northern Iraq.

Fog reduced visibilities to as low as 1,500 meters from northeastern Saudi Arabia to the United Arab Emirates coast (and to as low as 500 meters in the Tigris-Euphrates river valley) before 0400Z and again after 2000Z. Broken middle and high multilayered clouds with tops to 30,000 feet spread 9,000 foot ceilings from the northern Red Sea to the northern Persian Gulf and along the Iran-Iraq border. Isolated thunderstorms and rainshowers formed over northwestern Saudi Arabia throughout the day. They were most intense and widespread at about 1600Z northwest of Riyadh, along the southern Iraq-Iran border, and in extreme western Iraq near the Jordan border. Some of these storms were dry, creating intense, localized duststorms that reduced visibilities to well below 1,000 meters. Widespread duststorms were reported in the northern Arabian Peninsula and the Syrian Desert between 0900 and 2000Z with visibilities as low as 4,000 meters. Prevailing winds were as high as 30 kno! ts in northeastern Saudi Arabia.

At 0300Z, broken high clouds with 24,000 foot ceilings prevailed over Iraq and Kuwait; but as denser clouds continued to move in, a solid overcast from 7,000 to 33,000 feet formed throughout southern Iraq and Kuwait. After 1300Z, isolated thunderstorms with tops to 35,000 feet developed in the area's southern half; skies in the heaviest storms wree obscured. Conditions over southern Iraq and Kuwait improved after 1800Z. In southwestern Iraq between the Tigris River and the Iranian border, skies were scattered with isolated low clouds from 2,000 to 20,000 feet. South of 30 degrees North, skies wre broken to overcast with 20,000 foot ceilings; there were also isolated low clouds from 10,000 to 35,000 feet. Elsewhere, skies remained overcast between 8,000 and 35,000 feet.

At 0500Z, a line of rainshowers spread from west-central Saudi Arabia northeastward to the Saudi Arabia-Iraq border and eastward into southern Kuwait. The line expanded and intensified to cover most of Kuwait, southern Iraq, and north-central Saudi Arabia by 1600Z. Intermittent precipitation fell the rest of the day.

Winds were southeasterly at 5-10 knots until 0900Z. Afternoon winds were stronger at 15-20 knots, with gusts to 40. Fog and smoke reduced visibilities to below 2,000 meters in southern Iraq and Kuwait. Visibilities improved to 8 km by 0600Z, but sporadic duststorms in the afternoon reduced them to 4,000 meters. High temperatures were 14-21 degrees Celsius; lows, 3-16 degrees Celsius.

26 February 1991

As the upper air disturbance moved northeast, it continued to produce heavy rainshowers and duststorms over the area. A surface trough formed between the low pressure area in central Saudi Arabia and another moving through southern Turkey. By 2100Z, the trough stretched through Iraq along 43 degrees East. An area of high pressure was located in centrtal Iran and the extreme southeastern Arabian Peninsula.

Several lines of rainshowers and thunderstorms moved through the northern Arabian Peninsula throughout the day. Between 0000 and 0300Z, an area of thunderstorms spread from the Red Sea near 25 degrees North to the Iraq-Saudi Arabia border near 45 degrees East. Another formed in northeastern Iraq near the Iranian border. By 0900Z, a third area had formed over northeastern Saudi Arabia at 28 Degrees North, 47 degrees East. Bases were at 2,500 feet and tops reached 35,000 feet. Thunderstorms were embedded in scattered to broken middle clouds west of 45 degrees East. Multilayered clouds were broken to overcast from 8,000 to 33,000 feet north of 25 degrees North. By 1100Z, the northern area had spread southwestward and the southern areas had moved southeastward. Storm intensity and coverage increased throughout the day until 1600Z, when a line of isolated thunderstorms extended from the northern Persian Gulf to southwest of Riyadh. Areas west of 43 degrees East had clea! red. Clouds, rainshowers, and thunderstorms spread southeastward again in the evening, reaching as far southwest as 20 degrees North, 44 degrees East, by 1900Z.

Broken to overcast clouds between 8,000 and 20,000 feet covered the entire area before 0300Z. The lower cloud deck gradually dissipated in the northwest, leaving scattered skies over most of Iraq, and high clouds with tops to 32,000 feet over southern Iraq and Kuwait. Scattered to broken clouds between 4,000 and 6,000 feet formed over central Iraq between 0500 and 1600Z. At 1100Z, there were isolated thunderstorms or rainshowers embedded in these clouds in a line from 35 degrees North, 45 minutes East, to 31 degrees North, 41 minutes East. Thunderstorms also formed over eastern Kuwait and extreme southwestern Iraq after 1500Z. Cloud bases were 3,000 feet and tops reach 35,000 feet.

Intermittent rainshowers and thunderstorms fell southeast of a line extending from 34 degrees North, 46 minutes East, to 31 degrees North, 42 minutes East throughout the day.

Winds were southeasterly at 10-15 knots, but by 1200Z, speeds in the east reached 20-30 knots. Winds in the west shifted to northwesterly at 10-15 knots as the trough moved eastward. Fog and smoke reduced visibilities to below 2,000 meters from the central Tigris-Euphrates river valley to Kuwait. Visibilities improved to 8 km by 0600Z, but sporadic duststorms in the afternoon reduced visibilities to 4,000 meters. Visibilities in heavy rainshowers may have dropped to as low as 1,000 meters. High temperatures were 17-21 degrees Celsius; lows, 7-16 degrees Celsius.

27 February 1991

A low pressure cell that had been centered in southwestern Iraq at 1000Z slid southeastward into the Persian Gulf throughout the day. A weak frontal system in the eastern Mediterranean Sea moved onshore and was in central Iraq by the end of the day.

In western Iraq and northwestern Saudi Arabia, skies were scattered with bases at 10,000 feet throughout the morning. Clouds from the front approaching from the Mediterranean began moving in by 1100Z, forming ceilings rapidly. Rain began lowering visibilities to 5 km by 1300Z. Thunderstorms developed in the afternoon as the clouds moved eastward. By the end of the day, clouds and rain were confined to the western Saudi Arabia-Iraq border. In north-central and northeastern Saudi Arabia, broken clouds, multilayered from 3,000 to 25,000 feet with rainshowers and thunderstorms, prevailed. Visibilities were 4,800 meters in ground fog, rain and haze, but near zero in blowing dust from thunderstorms. Clouds moved slowly southeast to east-central Saudi Arabia by day's end.

Broken multilayered clouds from 3,000 to 25,000 feet covered the southern half of the area, but cleared from the northwest by noon, leaving scattered clouds at 3,000 feet and broken clouds at 6,000-8,000 feet over southeast Iraq and Kuwait. These also cleared by 1900Z. A smoke layer at 2,500 feet covered most of central and southern Kuwait throughout the day. A line of broken 4,000 foot clouds associated with the from the Mediterranean invaded the western part of the area by noon. The line was past Baghdad and into north-central Saudi Arabia by the end of the day. Rainshowers and thunderstorms were widespread in the southern half of the area through the morning. Light rain fell in the western half as the front passed.

Winds were northwesterly to northeasterly at 5-15 knots (but up to 25 knots in thunderstorms) in the southern half of the area. Winds became southwesterly at 5-15 knots as the front approached, and northwesterly at 8-20 knots behind it. Visibilities were near zero in dense fog along the Tigris-Euphrates river basin. There were also near zero in the southern half of the area, where thunderstorms produced blowing dust. Elsewhere, morning visibilities were 5 km in ground fog, rain, and haze. Rain lowered visibilities to 4,800 meters in the western half of the area as the front passed. Smoke limited visibility aloft to 1,600 meters over Kuwait. High temperatures were 13-20 degrees Celsius; lows, 7-16 degrees Celsius.

28 February 1991

Low pressure was centered over the southeastern part of the Saudi Arabian peninsula while high pressure intensified in the rest of the region. Remnants of a weak frontal system remained in northern Saudi Arabia and southern Iraq.

Skies were broken to overcast over north-central, northeastern, east, and east-central Saudi Arabia. Ceiling heights were 3,000-4,000 feet. By evening, skies were mostly clear to scattered. Light rain and drizzle fell over north-central and eastern Saudi Arabia. Thunderstorms with tops to 35,000 feet were observed over east-central Saudi Arabia and the northern part of the Persian Gulf during early morning. Winds were northwesterly at 10-20 knots with gusts to 25 knots. Fog lowered morning visibilities to 4,800 meters in east-central Saudi Arabia. Fog reduced early afternoon visibilities to 3,200 meters in northeastern Saudi Arabia, which improved to 6-8 km in smoke and haze by late afternoon. A sandstorm in east-central Saudi Arabia, with winds speeds of 30-40 knots, reduced late afternoon and early evening visibilities to 1,600-4,000 meters, with isolated reports of 100 meters.

Skies wre broken to overcast over southern and southeastern Iraq and Kuwait--ceilings were 3,000 to 4,000 feet, but 800 feet in showers. By early evening, skies were cleared to scattered. Isolated afternoon thunderstorms (tops to 35,000 feet) and rainshowers were present over southeastern Iraq and Kuwait.

Winds were northwesterly to northerly at 10-20 knots, with gusts to 35 knots near thunderstorms. In Kuwait, visibilities were less than 3,200 meters in smoke and 2,000 meters in thunderstorms. High temperatures were 13-18 degrees Celsius; lows, 5-10 degrees Celsius.

1 March 1991

High pressure dominated, but a low pressure system developed over the eastern Mediterranean by the end of the day, sending moisture into western areas. Skies were clear to scattered over most of the area, but scattered to broken at 1,000-2,000 feet over northeast and eastern Saudi Arabia due to smoke. Scattered to broken low clouds at 3,000 feet, with occasionally broken middle and high ceilings, moved into western Iraq and northwestern Saudi Arabia by mid-afternoon. Winds were northwesterly to northerly at 10-15 knots. Visibilities in northeastern Saudi Arabia were 6-8 km in smoke and haze. Blowing sand and dust reduced early morning visibilities to 3,200 meters in east-central and eastern Saudi Arabia. Scattered, occasionally broken, middle clouds at 8,000-10,000 feet moved into central Iraq by mid-afternoon.

Winds were northwesterly at 10-15 knots. Smoke and haze reduced visibilities in Kuwait to 5-7 km, occasionally to 3,200 meters. High temperatures were 15-20 degrees Celsius; lows, 5-10 degrees Celsius.

2 March 1991

High pressure dominated as a low pressure system moved northeast and brought moisture across Iraq and northern Saudi Arabia. Skies were broken to overcast at 8,000-10,000 feet, but early morning ceilings were 3,000-5,000 feet in showers over western and northern Iraq. Smoke formed a broken layer at 2,000-3,000 feet over northeast and east-central Saudi Arabia during the morning. Skies over the rest of the area were clear to scattered. Isolated rainshowers and thunderstorms with tops to 35,000 feet developed during early morning in Northern Iraq. Visibilities were 6-8 km in precipitation. Haze reduced morning visibilities to 4,800 meters in east-central Saudi Arabia. Smoke reduced morning visibilities in eastern Saudi Arabia to 4,800 meters; in the afternoon and evening, to 4,000 meters.

Over central Iraq, skies were broken with middle clouds at 8,000-10,000 feet in the early morning, becoming scattered in early afternoon. Winds were northwesterly at 10-15 knots. Smoke reduced visibilities in Kuwait to 5-7 km, with isolated areas of less than 1,600 meters. High temperatures were 15-20 degrees Celsius; lows, 5-10 degrees Celsius.

Chapter 3, Part 5

Conclusions

The following facts provide significant evidence that coalition forces were exposed to mixed chemical agents as a result of coalition bombings of Iraqi nuclear, chemical, and biological facilities and that the fallout from these bombings may be contributing to the health problems currently being suffered by Gulf War veterans.

- Iraqi nuclear, chemical, and biological weapons plants and storage sites were priority targets for U.S. and Coalition forces and were repeatedly bombed.
- Chemical alarms began sounding and the servicemen were put on chemical alert simultaneous with the beginning of the air war.

  The nature of diesel, oil, etc., did not alter during the air war, suddenly causing the alarms to sound. These substances were present before the initiation of the air war, and did not set off the chemical alarms. (The automatic alarms have no sensitivity control.)

  These chemical alarms are battlefield instruments. Battlefields are full of fumes, propellants, explosives, and so forth. It is difficult to believe that they would have been procurred if they were ineffective in this environment.

- U.S. military personnel, and the Czech and French governments have confirmed that the chemical alarms were sounding as the result of nerve agent detection.
- The combination of prevailing wind directions, the open terrain, the lack of structural impediments, and other factors listed above, indicate that chemical and possibly nuclear and biological agents from allied bombings became airborne and were being blown and carried across coalition forces emplacements along the Saudi-Iraqi and Saudi-Kuwaiti border.
- Chemical nerve agents, such as Sarin and others, are known to have a cumulative effect, i.e., they have a slow rate of detoxification. Little is known about the long-term effects of continuous low levels of exposure. Many of the veterans claiming to be suffering from Gulf War Syndrome are exhibiting symptoms of neurophysical disorders.

Chapter 4, Part 1

Other Identifiable Exposures

Chemical/Biological Warfare Pre-Treatment Drug Reaction113
Other Identifiable Exposures
Transmission

Those exposures covered in Chapter 2 and Chapter 3 have received much greater attention in this report than those which are identified in Chapter 4. Since the actual exposures to the materials identified below have been confirmed or are unchallenged, the development of evidence to confirm the exposures is not required.

Chemical/Biological Warfare Pre-Treatment Drug Reaction113

Another area in which further research appears to be warranted is the relationship between some Gulf War Syndrome cases and the administration of the nerve agent pretreatment drug given to US troops to protect them against Iraqi gas attacks. It appears possible that the use of this nerve agent pre-treatment drug may have permanently damaged some veterans' health. In addition to research on the effects of the drug itself, the possibility that these drugs might have a synergistic effect either with other drugs, or with chemical agents or other, environmental exposure, should also be thoroughly researched.

Maj. Gen. Ronald Blanck, commander of the Walter Reed Army Medical Center, has said that, "Military intelligence reports indicated there was a real possibility that Iraqi forces would employ biological and chemical weapons; in response to that threat, anthrax vaccine and botulinum vaccine were administered." The Army also gave soldiers a course of pyridostigmine bromide pills, normally used for neuro-muscular disorders. A public interest group, the Public Citizen, had filed a suit to stop experimental drugs being used on soldiers without their consent, but in the patriotic fervor immediately before the war, the suit was dismissed.114

Anthrax and Botulinum Toxoid Vaccines

Maj. General Blanck has advised Committee staff that the anthrax vaccine was administered to 150,000 soldiers and the botulinum vaccine to 8,000 soldiers.115 Both the anthrax vaccine and the botulinum toxoid vaccine were manufactured by the Michigan Department of Public Health. All of the anthrax vaccine is believed to be the same type of vaccine that has been administered

to veterinarians and agricultural workers in the United States since the late 1950s and approved by the Food and Drug Administration in 1971. The botulinum toxoid, manufactured using techniques similar to those employed in the production of the tetanus toxoid, has been administered to medical and laboratory workers since the early 1970s. It is still listed as investigatonal drug (IND). These vaccines are designed to raise the body's level of antibodies should the individual come into contact with the bacteria or toxin.116

While immediate local and some systemic reactions are reportedly experienced with the administration of these drugs, no information was developed by Committee staff that suggests that these vaciinations have widespread long term risks. Nevertheless, the effectiveness of these drugs, their possible long term effects, and the efficacy of manner in which they were administered does merit further study. No other biological warfare defense program immunizations, other than those commonly administered to travellers, have been identified by Committee staff.

Committee staff has received reports of recurring rumors that experimental recombinant DNA (rDNA) biological defense vaccines were used by the military during the Persian Gulf War. No evidence of any rDNA vaccine immunization program has been uncovered thus far. In addition, there has been some concern raised about the fact that soldiers were told that the immunizations they received were "secret." The issue of the secrecy of the vaccines is one that relates to the need to deny the enemy knowledge of those materials against which your forces have been protected, rather than to the vaccines themselves.117

Pyridostigmine Bromide (Group III)

During House of Representative hearings in 1993, Carol Picou, assigned to a combat support hospital during the Gulf War, recalled that when the ground war began, "we were ordered to take the drug pyridostigmine to protect us against chemical attack. Within one hour of taking the drug, I began to experience serious side-effects, such as uncontrollable twitching eyes, runny nose, excessive frothing from the mouth, neck and shoulder pain."118 Dr. Sidney Wolfe, director of the Public Citizen's health research group, who filed a suit against use of the drug, said it was administered so sloppily that nobody knew who took it. Maj. Gen. Blanck said that there was a risk of minor side effects, but that these were worthwhile to be "prepared for exposure to deadly biological and chemical warfare agents."119

As reported above under Group II disorders, Brian Martin also claimed to have had side effects from the drug pyridostigmine. According to Martin, the drug made him jittery and made his vision "jiggle."

Steve Hudspeth, assigned to the 1454th Transportation Company, also reported getting very sick from the nerve agent pre-treatment pills. He reported severe nausea and diarrhea that did not abate until he stopped taking the pills after two days. He recalled thinking that "if I'm going to feel like this I might as well be dead." Mr. Hudspeth currently suffers from memory loss, fatigue,

sore muscles and joints, insomnia, cough, some night sweats, diverticulitis, diarrhea, kidney stones, bloody stools, urinary urgency, growth on his eye, rashes, tingling and itching sensations, and depression and irritability.120

Chemically related to pesticides, nerve agents such as Sarin, Soman, Tabun, and VX kill by interfering with the metabolic processes, and cause a buildup of a chemical messenger in the human metabolic process called acetylcholine, which operates in the gap between the nerve and the muscle cells. A buildup of acetylcholine may cause drooling, excessive sweating, cramping, vomiting, confusion, irregular heart beat, convulsions, loss of consciousness and coma.121 Little, however, is known about the consequences of non-lethal exposure to these toxins.

Nerve gas pre-treatment drugs such as pyridostigmine bromide, paradoxically, also meddle with these metabolic processes by creating carbamate-inhibited acetylcholinesterase, which interferes with the actions of nerve gas -- theorectically permitting the process to be partially reversed.122

An article concerning a retrospective study conducted by the military on the effects of administration of pyridostigmine bromide appeared in the August 1991 Journal of the American Medical Association. According to the article 30mg of oral pyridostigmine bromide was administered to 41,650 members of the XVIII Airborne Corps, every eight hours for 1-7 days while under threat of nerve agent attack during Operation Desert Storm. The study observed that "about half of the population that received the drug noted physiologic changes that were not incapacitating, such as increased flatus, abdominal cramps, soft stools, and urinary urgency." "Approximately 1% of the soldiers believed they had effects that warranted medical attention, but fewer than 0.1% had effects sufficient to discontinue the drug. Non-incapacitating symptoms often occurred; however, the military mission was not impaired." Other symptoms noted in the article are headaches, rhinorrhea (running nose), diaphore! sis (p erspiration), nausea, and tingling of the extremities.

The results of this study and the coincidence of symptoms with many of those now being experienced by the veterans of the Gulf War suggests that the raw data and case histories which formed the basis for this study should be made available to researchers. This data can provide valuable information to conduct a second study of the possible long term effects of the administration of this drug on otherwise healthy individuals. Further, another independent study of the additive, synergistic, or even possible potentiating effects of pyridostigmine bromide combined with organophosphate pesticides, insect repellents such as DEET, and/or trace amounts of nerve agents on key neurotransmission regulators and secondary regulators must be considered.123

Other Identifiable Exposures

Reported Contact with Iraqi Enemy Prisoners of War

On April 4, 1994, several members of the 371st Chemical Company, Army Reserve Center, Greenwood, South Carolina, reported to Committee staff that elements of the unit were deployed on several occasions to decontaminate buses used to transport Iraqi enemy prisoners of war (EPWs) to detention camps inside Saudi Arabia. They were never advised of the reason these decontamination missions were necessary.124

A number of military police and other units who guarded the EPWs had close and continuing contact with them. Many individuals in these units are now reporting very high rates of illnesses in their units to Committee staff. The symptoms these Gulf War veterans describe are consistent with those commonly associated with Gulf War Syndrome. Committee staff has also been informed that the 300th Medical Brigade was responsible for the EPW health care during and after the war. While the Committee has received anecdotal information regarding the health status of the Iraqis and their symptoms, certainly medical records were established and retained regarding their care while in Coalition custody.

Given the reported high rate of illnesses among these military police units, and the possible relationship between the illnesses being suffered by these veterans and those which were reported by the EPWs, the raw data from the medical units which treated the EPWs should be immediately released to aid independent research into the causes of these illnesses.

Chemical Agent Resistant Coating (CARC)

CARC coatings need to be resistant to chemicals that are required to decontaminate military equipment that has been exposed to chemical and/or biological warfare agents. After cleaning with these decontamination chemicals, vehicles treated with CARC can be placed back into service immediately, without stripping and repainting. There have been several generations of CARC coatings. According to published sources, the military specifications for these coatings vary with the type of equipment to be used.125

The first generation of CARC contained lead and hexavalent chrome. Later these items were removed and the CARC was made VOC (volatile organic compound compliant). Prior to the Gulf War, the CARC specification was for a "high solids coating without exempt solvent."126

Committee staff has received calls from members of several National Guard and Army Reserve Units in Florida and Michigan who were detailed to apply these coatings to U.S. military vehicles during their service in the Gulf War. According to these veterans, many members of the units are suffering from the symtoms similar to those of other affected veterans. There have also been a number of anecdotal reports received by the Committee suggesting that unprotected exposures to CARC can have neurotoxological effects similar to exposures to other neurotoxins.

According to a published report in 1993, Dr. William Johnson, formerly of the Eisenhower Army Medical Center at Fort Gordon, Georgia, noted in a report prepared for Congress, that many of

these soldiers worked 12 hour days in poorly ventilated enclosures -- initially with no respirators. This report is consistent with information received by Committee staff.

While these duties were certainly necessary to perform, the failure to provide appropriate safety equipment to these individuals should be investigated further, not only for its impact on the health of the individuals but also for its impact on the ultimate readiness of these units to perform their mission. The chemical nature and the hazards associated with exposure to the various CARCs should be easily identified. Further, a study into the rates and types of illnesses being experienced by these units could be easily undertaken since the units would be readily identifiable.

Committee staff has developed no information to date that suggests these coatings represent a hazard once they are applied and cured.

Depleted Uranium Ammunition

Several different armor-penetrating munitions used during Operation Desert Storm were tipped with depleted uranium (DU) and encased in aluminum. The Persian Gulf War marked the first time such shells were used in combat. The penetrators are made of uranium rods from nuclear power plants and according to James Mathews, in an article that appeared in the Journal of the National Cancer Institute in July 1993, the uranium is depleted of the more volatile material, including the potent isotopes U-235 and U-238.127

According to Mathews, "When depleted-uranium penetrators strike their target, the aluminum covering is torn away and a large protion of the kinetic energy is dissipated as heat. The heat of the impact causes the depleted uranium to oxidize or burn momentarily."128

When uranium particles enter the body, they become lodged in bones or major organs, affecting the bone marrow and producing DNA damage. In previous congressional testimony, Mathews reports, Maj. Gen. Ronald R. Blanck, commander of Walter Reed Army Medical Center, Washington, D.C., stated that "careful analysis of [servicemen exposed to] depleted uranium suggests there will be no significant increase in risk to health, either in the short or long-term." Medical evaluations have been conducted by the Boston VA Medical Center on a number of soldiers identified as having the greatest potential for inhaling or ingesting depleted uranium dusts, mainly soldiers that prepared damaged battlefield vehicles for shipment back to the United States.

"The results of those examinations have shown no effects of uranium toxicity, and no uranium residues or byproducts were detected," said Blanck.129

The U.S. Armed Forces Radiological Reserach Institute is conducting a five year study into the hazards associated with U.S. military equipment and munitions that use depleted uranium. The preliminary results of that research should be made available to researchers and physicians to

provide a basis for determining if exposure to unexpended or expended depleted uranium munitions is a serious health hazard.

Finally, individual dosimeters were reportedly issued to many of the soldiers who fought in the Persian Gulf War to measure radiological hazards. It has been reported to Committee staff that at least some of these dosimeters were collected from the soldiers who participated in the conflict prior to their leaving the Gulf.130 In order to facilitate the research currently underway, and to provide information to researchers as to the level of exposure by location, the Department of Defense should release information regarding the readings from these dosimeters. If this information is not readily available, the National Institute of Standards and Technology could easily assess the level of emission related to these materials, if necessary, to provide information to both scientific and medical researchers attempting to find a cause for the illnesses being experienced to those individuals who may have been exposed to these materials. This information is also vital to ensure t! hat if a danger exists, appropriate safeguards will be taken, whenever possible, in future conflicts.

Environmental Exposures

With the initiation of the "ground war" on February 24, 1991, Iraqi forces set fire to over 600 oil wells located inside Kuwait. The contamination from these fires had a dramatic impact on the environment and the smoke was so thick that often there was darkness. In the areas where the fires were burning, Coalition soldiers were covered with an inhaled oil and soot. Even when they were able to shower, often they had no clean uniforms to replace the oil soaked ones. Other environmental hazards that have been previously considered include heater fuel fumes, pesticides, insect repellent, petrochemicals, and electromagnetic radiation from radars and communications equipment. While many researchers have discounted these exposures as causing Gulf War Syndrome since these exposures are not unique to the Gulf War environment, nevertheless, the results of the research that has been conducted in these areas by the Department of Defense and the Department of Veteran Affairs, incl! uding available data sets, case histories and diagnostic breakdown informati on, must be made available to assist medical researchers in furthering their research, and physicians in treating their patients.

Decontamination of Equipment Returned from the Persian Gulf Theater of Operations

Beginning in November 1993, Committee staff began receiving reports that a number of Department of Defense civilian personnel at the Anniston, Alabama and Sharpsite, California Army Depots were beginning to experience symptoms consistent with those of the Gulf War veterans. These individuals were assigned to clean, repair, and upgrade military vehicles and other equipment returning from the Southwest Asia theater of operations.131

No further information has been developed regarding these reports. Further investigation, however, appears warranted into what, if any, hazardous substances may have been transported

on equipment that would cause these symptoms. This type of information may assist in narrowing the scope of the research in determining the causes of Gulf War Syndrome.

Transmission

Over the past seven months, Committee staff has interviewed in varying detail over 1,000 Gulf War veterans who claim to be suffering from many of the symptoms commonly associated with Gulf War Syndrome. As a result of these interviews, it has been learned that most of the spouses and many of the children of Gulf War veterans are suffering from many of the same symptoms. Several published reports have recently appeared that suggest that Gulf War Syndrome may be transmittable, that it may be causing miscarriages, and that it may be causing birth deficiencies and some birth defects in newborns.132

During a February 21, 1994 Gulf War veterans round table meeting held in Lansing, Michigan, Chairman Donald W. Riegle, Jr. met with 9 Gulf War veterans and their wives to discuss their health problems. Of the 9 Gulf War veterans present, 20 additional individuals in their immediate families who were also suffering from many of the same symptoms were also identified.

In an effort to determine the scope and nature of symptom transmission, a survey of those individuals who have contacted the Committee is currently underway. The purpose of the survey is to determine the symptoms currently being suffered by the Gulf War veterans, those being transmitted to family members, and the number and rate of birth deficiencies being experienced within this population. The final results of this survey will not be available until later this year.

The issue of the possible transmission of Gulf War Syndrome is one that recrafts the issue of national security. Surely there are some aspects of the Department of Defense's chemical and biological warfare defense programs that merit secrecy. However, when the secrecy of those programs interfere with the safety of the citizenry, then one must understand that the notion of national security rests primarily in the security of the people and not in the secrecy of vulnerability.

Chapter 4, Part 2: Conclusions

Chemical/Biological Warfare Agent Exposure: Why Wasn't Everyone Affected?
Chemical/Biological Warfare Agent Exposure: Did the Military Know or Suspect that Individuals were Exposed to these Hazardous Substances?
The Need for Immediate Primary Scientific Research and Advanced Medical Research
Conclusions

Chemical/Biological Warfare Agent Exposure: Why Wasn't Everyone Affected?

The ability of someone to resist an illness, disease, or the adverse effects of a medication varies with each individual. Not everyone who received nerve agent pre-treatment drugs exhibited adverse effects. According to the Centers for Disease Control not everyone who is exposed to nerve gas will cross a toxic threshold at the same time. Certainly, there is a threshold beyond which such exposure will surely be lethal. This is what has come to be accepted as the effect of nerve gas exposure.

The results of this investigation suggest that there is, in fact, a relationship between dosage and harmful effects. A number of units who believe they suffered a direct chemical weapons attack report illness rates over 50%. The Czech chemical decontamination unit, which suffered only indirect exposure and might be expected to be well prepared against chemical exposure, reports an illness rate of 10%. The extent of exposure in the larger population in the Gulf at the time, and the rate of illnesses, is unknown. The number of Gulf War veterans who have signed up for the Persian Gulf Registry examination is now over 17,000.

Nerve agents like Sarin kill by disrupting the metabolic processes, causing a buildup of a chemical messenger (acetylcholine) by inhibiting the production of acetylcholinesterase, a key regulator of neurotransmission. Nerve agent pre-treatment drugs (NAPP) administered to U.S. servicemen and women, such as pyridostigmine bromide, also disrupt these metabolic processes by creating a carbamate-inhibited acetylcholinesterase, which preempts the action of the nerve agent. Several veterans suffering from Gulf War illnesses have testified before House and Senate Veterans Affairs Committee and believe that these illnesses are related to the permanent adverse side effects from this drug. Further, the efficacy of the biological warfare defense inoculations merits further research.

Chemical/Biological Warfare Agent Exposure: Did the Military Know or Suspect that Individuals were Exposed to these Hazardous Substances?

The evidence cited above and the statements of the witnesses will have to be evaluated on their own merits in this regard. During the course of this investigation, a medical questionnaire was received from one of the veterans currently suffering from Gulf War illnesses. This questionnaire like the other evidence and statements must be weighed on its own merits. The following information is solicited on this document, an overprint to SF600:

1. What diseases or injuries did you have in the Southwest Asia region?
2. Are you receiving any medicine, or other treatment, at the present time?
3. Do you have fever, fatigue, weight loss, or yellow jaundice?
4. Do you have any swelling of lymph nodes, stomach, or other body parts?
5. Do you have any rash, skin infection, or sores?
6. Do you have a cough or sinus infection?
7. Do you have stomach or belly pain, nausea, diarrhea, or bloody bowel movements?
8. Do you have urinary problems such as blood or stones in urine or pain and burning with urination?
9. Have you had nightmares or trouble sleeping?
10. Have you had recurring thoughts about your experiences during Desert Shield/Desert Storm?
11. Do you have any reason to believe that you, or any members of your unit, were exposed to chemical warfare or germ warfare?

Forms such as this suggest that the military expected, for whatever reason, to see symptomologies such as those that are currently being experienced. This information, as well as the information maintained in the medical records of U.S. forces and the Iraqi EPWs may provide information that will assist medical researchers in determining causal links. This issue should be further investigated.

The Need for Immediate Primary Scientific Research and Advanced Medical Research

Thousands of veterans of the Gulf War are reporting symptoms of memory loss, muscle and joint pain, intestinal and heart problems, fatigue, rashes, sores, and running noses. A number of veterans who have exhibited these symptoms since returning from the Gulf War have subsequently died. Physicians have been unable to diagnose the cause of the disorders.

The following symptoms have been identified as those most commonly reported by veterans:

- recurring severe headaches
- fatigue

- joint and muscle pain (particularly in knees, ankles, shoulders, and back)
- memory loss (often described as an inability to concentrate)
- recurring rashes (sometimes severe and often causing skin discoloration or described as mosquito bite-like or small with water pustules)
- lumps at joint areas
- lumps under skin

Symptoms most commonly reported by veterans (cont.):

- depression, irritability
- night sweats
- insomnia
- urinary urgency and frequency
- diarrhea (sometimes bloody) or constipation
- gastrointestinal problems (mausea, swollen stomach, gas)
- dizziness or blackouts
- blurry vision
- photosensitivity (excessive sensitivity to bright lights)
- shortness of breath
- coughing
- abnormal hair loss
- bleeding gums (or other serious dental problems)
- swollen lymph nodes
- sinus infections
- chest pains

female veterans only:

- chronic or recurring vaginal yeast infections
- menstrual irregularities
- excessive bleeding and severe cramping

Little is known about the long-term consequences of exposure to low levels of nerve gas, and even less about complications which might arise from using combined agent weapons. Further, little is known about other difficulties associated with interfering with the neurotransmission process. Non-lethal exposure to pesticides, however, has manifested itself in memory loss. Nearly every bodily process requires a properly functioning nervous system to operate.

The following is a summary, not offered as diagnostic evidence, suggesting how some of the symptoms noted could be rooted in neurotransmission related disorders:

- Memory Loss: Although neuroscience is a long way from explaining the memory functions of the human brain, considerable strides have been made towards understanding how neurons are modified by experience and how those modifications are maintained for extended periods of

time. The ability to remember is regulated, however, by neural processing.133 On August 25, 1993, Dr. Howard Hu, a researcher with Physicians for Human Rights who participated in the investigation of the use of nerve gas by the Iraqi government against the Kurds, suggested that the effects of non-lethal exposures to nerve agents could be similar to those involving non-lethal exposures to pesticides. Dr. Hu said these disorders are generally neuropsychological and include memory loss.134

- Muscle Pains: Myasthenia Gravis is a disease causing progressive muscle weakness. It has been shown that the disease is an autoimmune reaction to the acetylcholine-gated channels in the neuromuscular junction. According to Lloyd D. Partridge and L. Donald Partridge, many drugs and toxins, including pesticides and nerve gas, are known to exhibit their effects through specific actions at the neuromuscular junctions, blocking the action of acetylcholinesterase.135
- Joint Pains: When the force generated by a muscle acts on a load, there is a requisite exchange of energy between the muscle and the load. A failure of the nervous system to send impulses to effector muscles can result in the failure of effector muscles to provide the resistance necessary to protect joints from excessive torque. This failure, and the resultant joint pain, is consistent with the action of any agent or medicaton which functions by disrupting the communication process operating in the gap between the nerve and the muscle cells.136
- Gastrointestinal Disorders: As a combined neural operation, the neural signals that control digestive functions, such as in the complex nervous system of the gut, are largely, but not entirely, independent of the central nervous system (CNS). Many of the control functions are conducted by local nerve networks and the endocrine systems. These digestive functions, however, depend on the ability of the CNS and local nerve networks to function properly.137
- Heart problems, running noses and virtually every other problem lumped under the heading of Gulf War Illnesses can be explained by neurophysical and neuropsychiatric disorders. Some of the non-chemical warfare related diseases involving a disruption in the acetylcholine-gated channels in the neuromuscular messenger junctions, such as myasthenia gravis, while treatable, are irreversible. Neurotransmission disorders resulting from disrupted physiological processes, such as those regulating acetylcholines (including toxin acetylcholine and acetylcholinesterase) may be contributing to the symptomologies observed. Detection of these types of disorders may only be possible using highly sophisticated, computer-read electroencephalograms (EEG). Further, given the possibility that some of these individuals were exposed to biotoxins and other biological agents, scientists and physicians will need to use sophisticated procedures including DNA plasmid screening, bacteriological! screening, mycological screening, viral screening, and toxicological screening.

Conclusions

Thousands of American servicemen and women are reportedly suffering from memory loss, muscle and joint pain, intestinal and heart problems, fatigue, rashes, sores, and running noses as a result of their service in the Gulf War. A number of veterans who have exhibited these symptoms since returning from the Gulf War reportedly have died. Members of their immediate families are now beginning to contract some of the illnesses. Physicians have been unable to diagnose or treat the cause of the disorders.

Despite the Department of Defense's position that no evidence exists for exposure to chemical warfare agents during the Gulf War, this investigation is establishing that there is substantial evidence supporting claims that U.S. servicemen and women were exposed to low level chemical warfare agents and possibly biological agents and toxins from a variety of sources. This exposure may account for many of the Gulf War Illness symptoms. Little is known about the long term consequences of exposure to low levels of nerve gas, although most are known to have cumulative toxic effects.

Even less is known about complications which might arise from exposure to combined agents and combined agent weapons. The combined agent strategy is intended to frustrate efforts at diagnosing these illnesses. Non-lethal exposure to pesticides can result in memory loss, and nerve agents are chemically related to pesticides. Many of the veterans complaining of Gulf War Syndrome illnesses suffer from, among other disorders, memory loss. Many of the identified chemical and biological agents interfere with the body's neurotransmission processes, effecting the regulation of acetylcholine, neurotoxin acetylcholine, and other necessary enzymes required by nearly every bodily process. In order to detect irregularities such as those which might be caused by exposure to nerve gas, computer read electroencephalograms are needed; a physician probably would not be able to recognize the abnormalities during a visual EEG interpretation.

If biotoxins or biological agents were used or released in the Gulf War, detection requires that physicians and scientists have some idea of what they are looking for. Further, if mycotoxins or viruses were used or released, they would be difficult to detect without the aid or advanced laboratory screen methods.

Non-lethal exposure to chemical warfare agents, some biological agents, mixed chemical/biotoxin agents and/or the administration of nerve agent pre-treatment drugs could explain many of the symptoms of the Gulf War illness, as well as the inability to diagnose the disorders. Other possible causes for Gulf War syndrome have been suggested, such as exposure to pesticides, petrochemicals, burning landfills and oil wells, depleted uranium from anti-tank munitions, or exposure to other environmental hazards. Many of these possibilities already have been investigated and discounted. Additionally, these types of exposures are not specific to the Middle East or to the Gulf War and the evidence for these hazards causing the large number of unexplained illnesses is less than compelling. Each of these possible causes of unexplained illnesses, however, should be systematically researched.

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - Tabun (GA)
  - Sarin (GB)
  - Soman (GD)
  - VX
- Blister Agents
  - Sulfur Mustard (HD, THD)
  - Sulfur Mustard (HT)

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - Tabun (GA)

Material Safety Data Sheet -- Lethal Nerve Agent Tabun (GA)

Section I: General Information
Section II: Composition
Section III: Physical Data
Section IV: Fire and Explosion Data
Section V: Health Hazard Data
Section VI: Reactivity Data
Section VII: Spill, Leak and Disposal Procedures

Section I: General Information

MANUFACTURER'S NAME: Department of the Army

MANUFACTURER'S ADDRESS:

U.S. Army Armament, Munitions and Chemical Command
Chemical Research, Development and Engineering Center
ATTN: SMCCR-CMS-E
Aberdeen Proving Ground, MD 21010-5423

CAS REGISTRY NUMBER: None

CHEMICAL NAME: Ethyl N,N-dimethylphosphoramidocyanidate

TRADE NAME AND SYNONYMS:

- Ethyl dimethylplosphoramidocyanidate
- Dimethylaminoethoxy-cyanophosphine oxide
- Dimethylamidoethoxyphosphoryl cyanide
- Ethyldimethylaminocyanophosphonate
- Ethyl ester of dimethylphosphoroamidocyanidic acid
- Ethyl phosphorodimethylamidocyanidate
- GA
- EA1205
- Tabun

CHEMICAL FAMILY: Organophosphorous compound

FORMULA: C5 H11 N2 02 P

NFPA 704 SIGNAL:

- Health - 4
- Flammability - 2
- Reactivity - 1

Section II: Composition

INGREDIENTS NAME	FORMULA	PERCENTAGE BY WEIGHT	AIRBORNE EXPOSURE LIMIT (AEL)
GA	C5 H11 N2 02 P	100	0.0001 mg/m3

Section III: Physical Data

BOILING POINT DEG F (DEG C): 247.5 DEG C

VAPOR PRESSURE (mm hg): 0.07 @ 24 DEG C

VAPOR DENSITY (AIR=1): 5.6

SOLUBILITY IN WATER (g/100 g): 9.8 @ 25 DEG C/ 7.2 @ 20 DEG C

SPECIFIC GRAVITY (H20=1): Not available

FREEZING (MELTING) POINT: -50 DEG C

AUTOIGNITION TEMPERATURE DEG F (DEG C): Not available

VISCOSITY (CENTISTOKES): 2.18 @ 25 DEG C

PERCENTAGE VOLATILE BY VOLUME: 610 mg/m3 @ 25 DEG C

EVAPORATION RATE: Not available

APPEARANCE AND ODOR: Colorless to brown liquid. Faintly fruity; none when pure

---

Section IV: Fire and Explosion Data

FLASHPOINT: 78 DEG C

FLAMMABILITY LIMITS (% by volume): Not available

EXTINGUISING MEDIA: Water, fog, foam, CO2 - Avoid using extinguishing methods that will cause splashing or spreading of the GA.

UNUSUAL FIRE & EXPLOSION HAZARDS: Fires involving this chemical may result in the formation of hydrogen cyanide.

SPECIAL FIRE FIGHTING PROCEDURES:

All persons not engaged in extinguishing the fire should be immediately evacuated from the area. Fires involving GA should be contained to prevent contamination to uncontrolled areas. When responding to a fire alarm in buildings or areas containing agents, firefighting personnel should wear full firefighter protective clothing (without TAP clothing) during chemical agent firefighting and fire rescue operations.

Respiratory protection is required. Positive pressure, full facepiece, NIOSH-approved self-contained breathing apparatus (SCBA) will be worn where there is danger of oxygen deficiency and when directed by the fire chief or chemical accident/incident (CAI) operations officer. The M9 or M17 series mask may be worn in lieu of SCBA when there is no danger of oxygen deficiency. In cases where firefighters are responding to a chemical accident/incident for

rescue/reconnaissance purposes vice firefighting, they will wear appropriate levels of protective clothing (see Section 8).

Section V: Health Hazard Data

AIRBORNE EXPOSURE LIMIT (AEL): The suggested permissible airborne exposure concentration for GA for an 8-hour workday or a 40 hour work week is an 8 hour time weight average (TWA) of 0.0001 mg/m3 (2 x 10-5 ppm). This value is based on the TWA of GA as proposed in the USaEHA Technical Guide 169, "Occupational Health Guildelines for the Evaluation and Control of Occupational Exposure to Nerve Agents, GA, GB, GD, and VX." To date, however, the Occupational Safety and Health Administration (OSHA) has not promulgated a permissible exposure concentration for GA.

EFFECTS OF OVEREXPOSURE:

GA is an anticholinesterase agent similar in action to GB. Although only about half as toxic as GB by inhalation, GA in low concentrations is more irritating to the eyes than GB.

The number and severity of symptoms which appear are dependent on the quantity, and rate of entry of the nerve agent which is introduced into the body. (Very small skin dosages sometimes cause local sweating and tremors with few other effects.)

Individuals poisoned by GA display apaproximately the same sequence of symptoms regardless of the route by which the poison enters the body (whether by inhalation, absorption, or ingestion). These symptoms, in normal order of appearance, runny nose; tightness of chest; dimness of vision and pin pointing of the eye pupils; difficulty in breathing; drooling and excessive sweating; nausea; vomiting, cramps, and involuntary defecation and urination; twitching, jerking, and staggering; and headaches, confusion, drowsiness, coma, and convulsion. These symptoms are followed by cessation of breathing and death.

Onset Time of Symptoms: Symptoms appear much more slowly from skin dosage than from respiratory dosage. Although skin absorption great enough to cause death may occur in 1 to 2 minutes, death may be delayed for 1 to 2 hours. Respiratory lethal dosages kill in 1 to 10 minutes, and liquid in the eye kills almost as rapidly.

Median Lethal Dosage, Animals: LD50 (monkey, percutaneous) = 9.3 mg/kg (shaved skin); LCt50 (monkey, inhalation) = 187 mg-min/m3 (t = 10)

Median Lethal Dosage, Man: LCt50 (man, inhalation) = 135 mg-min/m3 (t = 0.5-2 min) at RMV (Respiratory Minute Volume) of 15 1/min; 200 mg-min/m3 at RMV* of 10 1/min

GA is not listed by the International Agency for Research on Cancer (IARC), American Conference of Governmental Industrial Hygienists (ACGIH). Occupational Safety and Health Administration (OSHA), or National Toxicology Program (NTP) as a carcinogen.

EMERGENCY AND FIRST AID PROCEDURES:

- INHALATION: Hold breath until respiratory protective mask is donned. If severe signs of agent exposure appear (chest tightens, pupil construction, incoordination, etc.), immediately administer, in rapid succession, all three Nerve Agent Antidote Kit(s), Mark I injectors (or atropine if directed by the local physician). Injections using the Mark I kit injectors may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will be maintained of all injections given. If breathing has stopped, give artificial respiration. Mouth-to-mouth resuscitation should be used when approved mask-bag or oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists. If breathing is difficult, administer oxygen. Seek medical attention IMMEDIATELY.
- EYE CONTACT: IMMEDIATELY flush eyes with water for 10-15 minutes then don respiratory protective mask. Although miosis (pinpointing of the pupils) may be an early sign of agent exposure, an injection will not be administered when miosis is the only sign present. Instead, the individual will be taken immediately to the medical treatment facility for observation.
- SKIN CONTACT: Don respiratory protection mask and remove contaminated clothing. Immediately wash contaminated skin with copious amounts of soap and water, 10% sodium carbonate solution, or 5% liquid household bleach. Rinse well with water to remove decontaminate. Administer an intramuscular injection with the MARK I kit injectors only if local sweating and muscular twitching symptoms are observed. Seek medical attention IMMEDIATELY.
- INGESTION: Do not induce vomiting. First symptoms are likely to be gastrointestinal. IMMEDIATELY administer 2 mg. intramuscular injection of the MARK I kit auto-injectors. Seek medical attention IMMEDIATELY.

Section VI: Reactivity Data

STABILITY: Stable

INCOMPATIBILITY: Not available

HAZARDOUS DECOMPOSITION: Decomposes within 6 months at 60 DEG C. Complete decomposition in 3-1/4 hours at 150 DEG C. May produce HCN. Oxides of nitrogen, oxides of phosphorus, carbon monoxide, and hydogen cyanide.

HAZARDOUS POLYMERIZATON: Not available

Section VII: Spill, Leak and Disposal Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED: If leaks or spills occur, only personnel in full protective clothing (see section 8) will remain in area. In case of personnel contamination see section V "Emergency and First Aid Instructions."

RECOMMENDED FIELD PROCEDURES:

Spills must be contained by covering with vermiculite, diatomaceious earth, clay, fine sand, sponges, and paper or cloth towels. This containment is followed by treatment with copious amounts of aqueous Sodium Hydroxide solution (a minimum 10 wt percent). Scoop up all material and place in a fully removable head drum with a high density polyethylene liner. The decontamination solution must be treated with excess bleach to destroy the CN formed during hydrolysis. Cover the contents with additional bleach before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations.

All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Sections 2 and 8).

If 10 wt percent Sodium Hydroxide is not available then the following decontaminants may be used instead and are listed in order of preference: Decontamination Solution No. 2 (DS2), Sodium Carbonate and Supertropical Bleach Slurry (STB).

RECOMMENDED LABORATORY PROCEDURES:

A minimum of 56 grams of decon solution is required for each gram of GA. The decontamination solution is agitated while GA is added and the agitation is maintained for at least one hour. The resulting solution is allowed to react for 24 hours. At the end of 24 hours, the solution must be tritrated to a pH between 10 and 12. After completion of the 24 hour period, the decontamination solution must be treated with excess bleach (2.5 mole OC1-/mole GA) to destroy the CN formed during hydrolysis.

Scoop up all material and place in a fully removable head drum with a high density polyethylene liner. Cover the contents with additional bleach before affixing the drum head. All contaminated

clothing will be placed in a fully removable head drum with a high density polyethylene liner. Cover the contents of the drum with decontaminating solution as above before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW state, EPA and DOT regulations.

All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW State, EPA and DOT regulations. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Sections 2 and 8).

WASTE DISPOSAL METHOD: Open pit burning or burying of GA or items containing GA.

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - Sarin (GB)

Material Safety Data Sheet -- Lethal Nerve Agent Sarin (GB)

Section I: General Information
Section II: Composition
Section III: Physical Data
Section IV: Fire and Explosion Data
Section V: Health Hazard Data
Section VI: Reactivity Data
Section VII: Spill, Leak and Disposal Procedures
Section VIII: Special Protection Information
Section IX: Special Precautions
Section X: Transportation Data

Section I: General Information

MANUFACTURER'S NAME: Department of the Army

MANUFACTURER'S ADDRESS:

U.S. Army Chemical and Biological Defense Agency
Edgewood Research, Development and Engineering Center
ATTN: SCBRD-ODR-S
Aberdeen Proving Ground, MD 21010-5423

CAS REGISTRY NUMBER: 107-44-8 or 50642-23-4

CHEMICAL NAME AND SYMONYMS:

- Phosphonofluoridic acid, methyl-, isopropyl ester
- Phosphonofluoridic acid, methyl-, 1- methylethyl ester

ALTERNATE CHEMICAL NAMES:

- Isopropyl methylphosphonofluoridate
- Isopropyl ester of methylphosphonofluoridic acid
- Methylisoproposfluorophosphine oxide
- Isopropyl Methylfluorophosphonate
- O-Isopropyl Methylisopropoxfluorophosphine oxide
- O-Isopropyl Methylphosphonofluoridate
- Methylfluorophosphonic acid, isopropyl ester
- Isoproposymethylphosphonyl flouride

TRADE NAME AND SYNONYMS:

- GB
- Sarin
- Zarin

CHEMICAL FAMILY: Fluorinated organophosphorous compound

FORMULA: C4 H10 F02 P

NFPA 704 SIGNAL:

- Health - 4
- Flammability - 1

- Reactivity - 1

Section II: Composition

INGREDIENTS NAME: GB

FORMULA: C4 H10 FO2

PERCENTAGE: 100

AIRBORNE EXPOSURE LIMIT (AEL): 0.0001 mg/m3

Section III: Physical Data

BOILING POINT DEG F (DEG C): 316 (158)

VAPOR PRESSURE (mm hg): 2.9 @ 25 DEG C

VAPOR DENSITY (AIR=1): 4.86

SOLUBILITY IN WATER: Complete

SPECIFIC GRAVITY (H20=1): 1.0887 @ 25 DEG C

FREEZING/MELTING POINT: -56 DEG C

LIQUID DENSITY (g/cc): 1.0887 @ 25 DEG C/1.102 @ 20 DEG C

PERCENTAGE VOLATILE BY VOLUME: 22,000 m/m3 @ 25 DEG C/ 16,090 m/m3 @ 20 DEG C

APPEARANCE AND ODOR: Colorless liquid. Odorless in pure form.

Section IV: Fire and Explosion Data

FLASH POINT (METHOD USED): Did not flash to 280 DEG F

FLAMMABLE LIMIT: Not applicable

LOWER EMPLOSIVE LIMIT: Not available

UPPER EXPLOSIVE LIMIT: Not available

EXTINGUISING MEDIA: Water mist, fog, foam, CO2 - Avoid using extinguishing methods that will cause splashing or spreading of the GB

SPECIAL FIRE FIGHTING PROCEDURES:

GB will react with steam or water to produce toxic & corrosive vapors. All persons not engaged in extinguishing the fire should be evacuated. Fires involving GB should be contained to prevent contamination to uncontrolled areas. When responding to a fire alarm in buildings or areas containing agents, firefighting personnel clothing (without TAP clothing) during chemical agent firefighting and fire rescue operations.

Respiratory protection is required. Positive pressure, full facepiece, NIOSH-approved self-contained breathing apparatus (SCBA) will be worn where there is danger of oxygen deficiency and when directed by the fire chief or chemical accident/incident (CAI) operations officer. In cases where firefighters are responding to a chemical accident/incident for rescue/reconnaissance purposes vice firefighting, they will wear appropriate levels of protective clothing (see Section 8).

UNUSUAL FIRE AND EXPLOSION HAZARDS: Hydrogen may be present

Section V: Health Hazard Data

AIRBORNE EXPOSURE LIMIT (AEL): The permissible airborne exposure concentration for GB for an 6 hour workday or a 40 hour work week is an 8 hour time weight average (TWA) of 0.0001 mg/m3. This value is based on the TWA or GB which can be found in "AR 40-8, Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX." To date, however, the Occupational Safety and Health Administration (OSHA) has not promulgated a permissible exposure concentration for GB.

EFFECTS OF OVEREXPOSURE: It is a lethal anticholinergic agent. Doses which are potentially life threatening may be only slightly larger than those producing minimal effects.

Route	Form	Effect	Type	Dosage
ocular	vapor	miosis	ECt50	less than 2 mg-min/m3
inhalation	vapor	runny nose	ECt50	less than 2 mg-min/m3
inhalation		severe incapacitation	ICt50	35 mg-min/m3
inhalation	vapor	death	LCt50	70 mg-min/m3
percutaneous	liquid	death	LD50	1700 mg/70 kg man

Effective dosages for vapor are estimated for exposure durations of 2-10 minutes.

Symptoms of overexposure may occur within minutes or hours--depending upon dose. They include: miosis (constriction of pupils) and visual effects, headache and pressure sensation, runny nose and nasal congestion, salivation, tightness in the chest, nausea, vomiting, giddiness, anxiety, difficulty in thinking, difficulty sleeping, nightmares, muscle twitches, tremors, weakness, abdominal cramps, diarrhea, involuntary urination and defecation.

With severe exposure symptoms progress to convulsions and respiratory failure. GB is not listed by the International Agency for Research on Cancer (IARC), American Conference of Governmental Industrial Hygienists (ACGIH), Occupational Safety and Health Administration (OSHA), or National Toxicology Program (NTP) as a carcinogen.

EMERGENCY AND FIRST AID PROCEDURES:

- INHALATION: Hold breath until respiratory protective mask is donned. If severe signs of agent exposure appear (chest tightens, pupil constriction, incoordination, etc.), immediately administer, in rapid succession, all three Nerve Agent Antidote Kit(s), Mark I injectors (or atropine if directed by the local physician). Injections using the Mark I kit injectors may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will be maintained of all injections given. If breathing has stopped, give artificial respiration. Mouth-to-mouth resuscitation should be used when approved mask-bag or oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists. If breathing is difficult, administer oxygen. Seek medical attention IMMEDIATELY.
- EYE CONTACT: Immediately flush eyes with water for 10-15 minutes, then don respiratory protective mask. Although miosis (pinpointing of the pupils) may be an early sign of agent exposure, an injection will not be administered when miosis is the only sign present. Instead, the individual will be taken IMMEDIATELY to the medical treatment facility for observation.
- SKIN CONTACT: Don respiratory protective mask and remove contaminated clothing. Immediately wash contaminated skin with copious amounts of soap and water, 10% sodium carbonate solution, or 5% liquid household bleach. Rinse well with water to remove decontaminant. Administer an intramuscular injection with the MARK I Kit injectors only if local

sweating and muscular twitching symptoms are observed. SEEK MEDICAL ATTENTION IMMEDIATELY.

- INGESTION: Do not induce vomiting. First symptoms are likely to be gastrointestinal. Immediately administer an intramuscular injection of the MARK I kit auto-injectors. SEEK MEDICAL ATTENTION IMMEDIATELY.

Section VI: Reactivity Data

STABILITY: Stable when pure.

INCOMPATIBILITY: Attacks tin, magnesium, cadmium plated steel, some aluminums. Slight attack on copper, brass, lead, practically no attack on 1020 steel, Inconel & K-monel.

Hydrolyzes to form HF under acid conditions and isopropyl alcohol & polymers under basic conditions.

Section VII: Spill, Leak and Disposal Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED: If leak or spills occur, only personnel in full protective clothing (see section 8) will remain in area. In case of personnel contamination see section V "Emergency and First Aid Instructions."

RECOMMENDED FIELD PROCEDURES:

Spills must be contained by covering with vermiculite, diatomaceous earth clay, fine sand, sponges, and paper or cloth towels. Decontaminate with copious amounts of aqueous Sodium Hydroxide solution (a minimum 10 wt percent). Scoop up all material and place in a fully removable head drum with a high density polyethylene liner. Cover the contents of the drum with decontaminating solution as above before affixing the drum head.

After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Dispose of material used to decontaminate exterior of drum IAW Federal, state and local regulations. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Sections 2 and 8).

If 10 wt. percent aqueous Sodium Hydroxide solution is not available then the following decontaminants may be used instead and are listed in the order of preference: Decontamination Solution No. 2 (DS2), Sodium Carbonate, and Supertropical Bleach Slurry (STB).

RECOMMENDED LABORATORY PROCEDURES:

A minimum of 56 grams of decon solution is required for each gram of GB. Decontaminant/agent solution is allowed to agitate for a minimum of one hour. Agitation is not necessary following the first hour. At the end of the one hour, the resulting solution should be adjusted to a pH greater than 11.5. If the pH is below 11.5, NaOH should be added until a pH above 11.5 can be maintained for 60 minutes.

An alternate solution for the decontamination of GB is 10 wt percent Sodium Carbonate in place of the 10 percent Sodium Hydroxide solution above. Continue with 56 grams of decon to 1 gram of agent. Agitate for one hour but allow three (3) hours for the reaction. The final pH should be adjusted to above 10. It is also permitted to substitute 5.25% Sodium Hypochlorite or 25 wt percent Monoethylamine (MEA) for the 10% Sodium Hydroxide solution above. MEA must be completely dissolved in water prior to addition of the agent. Continue with 56 grams of decon for each gram of GB and provide agitation for one hour. Continue with same ratios and time stipulations.

Scoop up all material and place in a fully removable head drum with a high density polyethylene liner. Cover the contents of the drum with decontaminating solution as above before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Dispose of material used to decontaminate exterior of drum IAW Federal, state and local regulations. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Sections 2 and 8).

WASTE DISPOSAL METHOD: Open pit burning or burying of GB or items containing or contaminated with GB in any quantity is prohibited. The detoxified GB using procedures above can be thermally destroyed by incineration in an EPA approved incinerator in accordance with appropriate provisions of Federal, state and local RCRA regulations.

Section VIII: Special Protection Information

RESPIRATORY PROTECTION:

Concentration	Respiratory Protective Equipment
less than 0.0001 mg/m3	A full face piece, chemical canister, air purifying protective mask will be onhand for escape. (The M9-, or M40-series masks are acceptable for this purpose).
0.0001 to 0.2 mg/m3	A NIOSH/MSHA approved pressure demand full facepiece SCBA or supplied air respirator with escape air cylinder may be used. Alternatively, a full facepiece, chemical canister air purifying protective mask is acceptable for this purpose(for example, M9-, M17-, or M40-series mask or other mask certified as equivalent) is acceptable. (See DA PAM 385-61 for determination of appropriate level)
greater than 0.2 mg/m3 or unknown	NIOSH/MSHA approved pressure demand full facepiece SCBA suitable for use in high agent concentrations With protective ensemble (see DA PAM 385-61 for examples).

VENTILATION: Local exhaust: Mandatory must be filtered or scrubbed to limit exit concentration to less than 0.0001 mg/m3 averaged over 8 hr/day indefinitely. Air emissions shall meet local, state and federal regulations.

- SPECIAL: Chemical laboratory hoods shall have an average inward face velocity of 100 linear feet per minute (1fpm) plus or minus 10% with the velocity at any point not deviating from the average face velocity by more than 20%. Existing laboratory hoods shall have an inward face velocity of 150 1fpm plus or minus 20 percent. Laboratory hoods shall be located such that cross drafts do not exceed 20 percent of the inward face velocity. A visual performance test utilizing smoke producing devices shall be performed in the assessment of the hood's ability to contain agent GB. Emergency backup power necessary. Hoods should be tested semi-annually or after modification or maintenance operations. Operations should be performed 20 cm inside hood face.
- OTHER: Recirculation of exhaust air from agent areas is prohibited. No connection is allowed between agent areas and other areas through ventilation system.

PROTECTIVE GLOVES: Butyl Glove M3 and M4, Norton, Chemical Protective Glove Set

EYE PROTECTION: Chemical goggles. For splash hazards use goggles and faceshield.

OTHER PROTECTIVE EQUIPMENT: For general lab work, gloves and lab coat shall be worn with M9, M17 or M40 mask readily available.

MONITORING:

Available monitoring equipment for agent GB is the M8/M9 Detector paper, detector ticket, blue band tube, M256/M256A1 kits, bubbler, Depot Area Air Monitoring System (DAAMS), Automatic Continuous Air Monitoring System (ACAMS), real time monitoring (RTM), Demilitarization Chemical Agent Concentrator (DCAC), M8/M43, M8A1/M43A2, Hydrogen Flame Photometric Emission Detector (HYPED), CAM-M1, Miniature Chemical Agent Monitor (MINICAM) and the Real Time Analytical Platform (RTAP).

Real-time, low-level monitors (with alarm) are required for GB operations. In their absence, an IDLH atmosphere must be presumed. Laboratory operations conducted in appropriately maintained and alarmed engineering controls require only periodic low-level monitoring.

Section IX: Special Precautions

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING: In handling, the buddy system will be incorporated. No smoking, eating and drinking in areas containing agent is permitted. Containers should be periodically inspected for leaks (either visually or by a detector kit). Stringent control over all personnel practices must be exercised. Decontamination equip shall be conveniently located. Exits must be designed to permit rapid evacuation. Chemical showers, eye-wash stations, and personal cleanliness facilities must be provided. Wash hands before meals and each worker will shower thoroughly with special attention given to hair, face, neck, and hands, using plenty of soap before leaving at the end of the work day.

OTHER PRECAUTIONS: Agents must be double contained in liquid and vapor tight containers when in storage or when outside of ventilation hood.

For additional information see "AR 385-61, The Army Toxic Chemical Agent Safety Program'" "DA PAM 385-61, Toxic Chemical Agent Safety Standards," and "AR 40-8, Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX."

Section X: Transportation Data

PROPER SHIPPING NAME: Poisonous liquids, n.o.s.

DOT HAZARD CLASSIFICATION: 6.1 Packing Group I Hazard Zone A

DOT LABEL: Poison

DOT MARKING: Poisonous liquid, n.o.s. (Isopropyl methylphosphonofluoridate) UN2810

DOT PLACARD: Poison

PRECAUTIONS TO BE TAKEN IN TRANSPORTATION: Motor vehicles will be placarded regardless of quantity. Driver shall be given full and complete information regarding shipment and conditions in case of emergency.

AR 50-6 deals specifically with the shipment of chemical agents. Shipments of agent will be escorted in accordance with AR 740-32. EMERGENCY ACCIDENT PRECAUTIONS AND PROCEDURES: See sections IV, VII, and VIII.

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - Soman (GD)

Material Safety Data Sheet -- Lethal Nerve Agents Somain (GD and Thickened GD)

Section I: General Information
Section II: Hazardous Ingredients
Section III: Physical Data
Section IV: Fire and Explosion Data
Section V: Health Hazard Data
Section VI: Reactivity Data
Section VII: Spill, Leak and Disposal Procedures
Section VIII: Special Protection Information
Section IX: Special Precautions
Section X: Transportation Data
Addendum A: Physiological Effects
Addendum B: First Aid Procedures
Addendum C: Additional Information for Thickened GD

Section I: General Information

MANUFACTURER'S NAME: Department of the Army

MANUFACTURER'S ADDRESS:

U.S. Army Armament, Munitions and Chemical Command
Chemical Research, Development and Engineering Center
ATTN: SMCCR-CMS-E
Aberdeen Proving Ground, MD 21010-5423

CAS REGISTRY NUMBER: 96-64-0 or 50642-24-5

CHEMICAL NAME: Phosphonofluoridic acid, methyl-, 1, 2, 2-trimethylpropyl ester

ALTERNATE CHEMICAL NAMES:

- Pinacolyl methylphosphonofluoridate
- 1,2,2-Trimethylpropyl methylphosphonofluoridate
- Methylpinacolyloxyfluorophosphine oxide
- Pinacolyloxymethylphosphonyl flouride
- Pinacolyl methanefluorophosphonate
- Methylfluoropinacolylphosphonate
- Fluoromethylpinacolyloxyphosphine Oxide
- Methylpinacolyloxyphosphonyl flouride
- Pinacolyl methylfluorophosphonate
- 1,2,2,-Trimethylpropoxyfluoromethylphosphine oxide

TRADE NAME AND SYNONYMS:

- GD
- EA 1210
- Soman, Zoman
- PFMP

CHEMICAL FAMILY: Fluorinated organophosphorus compound

FORMULA: C7 H16 F02 P

NFPA 704 SIGNAL:

- Health - 4
- Flammability - 1
- Reactivity - 1

Section II: Hazardous Ingredients

INGREDIENTS	FORMULA	PERCENTAGE BY WEIGHT	AIRBORNE EXPOSURE LIMIT
GD	C7 H16 FOP	100	0.00003 mg/m3

Section III: Physical Data

BOILING POINT DEG F (DEG C): (198 DEC C) 388 DEG F

VAPOR PRESSURE: 0.40 mm Hg @ 25 DEG C

VAPOR DENSITY (AIR=1): 6.3

SOLUBILITY IN WATER: Moderate

SPECIFIC GRAVITY (H20=1): 1.022 @ 25 DEG C

VOLATILITY: 3900 mg/m3 @ 25 DEC C

MELTING POINT: -42 DEG C

APPEARANCE AND ODOR: When pure, colorless liquid with fruity odor. With impurities, amber or dark brown, with oil of camphor odor

---

Section IV: Fire and Explosion Data

FLASHPOINT: 121 DEG C (Open Cup)

FLAMMABILITY LIMITS: Unknown

LOWER EXPLOSIVE LIMIT: Not applicable

UPPER EXPLOSIVE LIMIT: Not applicable

EXTINGUISING MEDIA: Water, fog, foam, CO2 - Avoid using extinguishing methods that will cause splashing or spreading of the GD.

SPECIAL FIRE FIGHTING PROCEDURES:

Fires involving GD should be contained to prevent contamination of uncontrolled areas. All persons not engaged in extinguishing the fire should be evacuated immediately. Contact with GD or its vapors can be fatal. When responding to a fire alarm in buildings or areas containing agents, firefighting personnel should wear full firefighter protective clothing (without TAP clothing) during chemical agent firefighting and fire rescue operations.

Respiratory protection is required. Positive pressure, full facepiece, NIOSH approved self contained breathing apparatus (SCBA) will be worn where there is danger of oxygen deficiency

and when directed by the fire chief or chemical accident/incident (CAI) operations officer. The M9 or M17 series mask may be worn in lieu of SCBA when there is no danger of oxygen deficiency. In cases where firefighter are responding to a chemical accident/incident for rescue/reconnaissance purposes vice firefighting, they will wear appropriate levels of protective clothing (see Section 8).

UNUSUAL FIRE AND EXPLOSION HAZARDS: Hydrogen produced by the corrosive vapors reacting with metals, concrete, etc., may be present.

Section V: Health Hazard Data

AIRBORNE EXPOSURE LIMIT (AEL): The suggested permissible airborne exposure concentration of GD for an 8 hour workday or a 40 hour work week is an 8 hour time weighted average (TWA) of 0.00003 mg/m3 (2 x 10-5 ppm). This value is based on the TWA of GB as proposed in the USaEHA Technical Guide No. 169, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX." To date, however, the Occupational Safety and Health Administration (OSHA) has not promulgated permissible exposure concentration for GD.

EFFECTS OF OVEREXPOSURE: GD is a lethal anticholinesterase agent with the median lethal dose in man being: LCt50 (inhalation) = 70 mg min/m3 (t = 10 min); LD50 (PC, bare skin) = 0.35 g/man (70 kg).

1. One to several minutes after overexposure to airborne GD the following acute symptoms appear:
    1. . LOCAL EFFECTS (lasting 1-15 days, increase with dose)
        1. On eyes: Miosis (constriction of pupils); redness, pressure sensation on eyes.
        2. By inhalation: Rhinorrhea (runny nose), nasal congestion, tightness in chest, wheezing, salivation, nausea, vomiting
    2. SYSTEMIC EFFECTS (increases with dose): When inhaled GD will cause excessive secretion causing coughing/breathing difficulty: salivation and sweating: vomiting, diarrhea; stomach cramps; involuntary urination/defecation; generalized muscle twitching/muscle cramps; CNS depression including anxiety, restlessness, giddiness, insomnia, excessive dreaming and nightmares. With more severe exposure, also headache, tremor, drowsiness, concentration difficulty, memory impairment, confusion, unsteadiness on standing or walking, and progressing to death.
2. After exposure to liquid GD, the following acute symptoms appear:
    1. LOCAL EFFECTS:
        1. On eyes: Miosis (constriction of pupils); redness, pressure sensation on eyes.

2. By ingestion: salivation, anorexia, nausea, vomiting, abdominal cramps, diarrhea, involuntary defecation, heartburn.
3. On skin: Sweating, muscle twitching

2. Chronic exposure to GD causes forgetfulness, thinking difficulty, vision disturbances, muscular aches/pains. Although certain organophosphate pesticides have been shown to be teratogenic in animals, these effects have not been documented in carefully controlled toxicological evaluations for GD.

GD presently is not listed by the International Agency for Research on Cancer (IARC), National Toxicology Program (NTP), Occupational Safety and Health Administration (OSHA), or American Conference of Governmental Industrial Hygienists (ACGIH) as a carcinogen.

EMERGENCY AND FIRST AID PROCEDURES:

- INHALATION: Hold breath until respiratory protective mask is donned. If severe signs of agent exposure appear (chest tightens, pupil constriction, incoordination, etc.), immediately administer, in rapid succession, all three Nerve Agent Antidote Kit(s), Mark I injectors (or atropine if directed by the local physician). Injections using the Mark I kit injectors may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will be maintained of all injections given. If breathing has stopped, give artificial respiration. Mouth-to-mouth resuscitation should be used when approved mask-bag of oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists. If breathing is difficult, administer oxygen. Seek medical attention IMMEDIATELY.
- EYE CONTACT: IMMEDIATELY flush eyes with water for 10-15 minutes, then don respiratory protective mask. Although miosis (pinpointing of the pupils) may be an early sign of agent exposure, an injection will not be administered when miosis is the only sign present. Instead, the individual will be taken IMMEDIATELY to the medical treatment facility for observation.
- SKIN CONTACT: Don respsiratory protective mask and remove contaminated clothing. Immediately wash contaminated skin with copious amounts of soap and water, 10% sodium carbonate solution, or 5% liquid household bleach. Rinse well with water to remove decontaminant. Administer nerve agent antidote kit, Mark I, only if local sweating and muscular twitching symptoms are present. Seek medical attention IMMEDIATELY.
- INGESTION: Do not induce vomiting. First symptoms are likely to be gastronintestinal. IMMEDIATELY administer Nerve Agent Antidote kit, Mark I. Seek medical attention immediately.

Section VI: Reactivity Data

STABILITY: Stable after storage in steel for 3 months at 65 Deg. C. GD corrodes steel at the rate of 1 x 10-5 inch/month.

GD will hydrolyze to form HF--H-H-O-CH3 and (CH3) 3-C-C-O-P-OH

HAZARDOUS POLYMERIZATION: Will not occur.

Section VII: Spill, Leak and Disposal Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED: If leak or spills occur, only personnel in full protective clothing (see Section 8) will remain in area. In case of personnel contamination, see Section V "Emergency and First Aid Procedures."

RECOMMENDED FIELD PROCEDURES:

Spills must be contained by covering with vermiculite, diatomaceous earth, clay, fine sand, sponges and paper or cloth towels. This containment is followed by treatment with copious amounts of aqueous Sodium Hydroxide solution (a minimum of 10 percent). Scoop up all material and place in a fully removable head drum with a high density polyethylene liner. Cover the contents of the drum with decontaminating solution as above before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations.

All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Dispose of material used to decontaminate exterior of drum IAW Federal, state and local regulations. Contaminated clothing will be placed in a fully removable head drum with a high density polyethylene liner and the contents shall be covered with decontaminating solution as above before affixing the drum head. Conduct general area monitoring to confirm that the atmospheric concentrations do not exceed the exposure limits (see Section 8).

If 10 wt percent aqueous Sodium Hydroxide solution is not available then the following decontaminants may be used instead and are listed in the order of preference: Decontaminating Solution No. 2 (DS2), Sodium Carbonate, and Supertropical Tropical Bleach Slurry (STB).

RECOMMENDED LABORATORY PROCEDURES:

A minimum of 55 grams of decon solution is required per gram of GD. Decontaminant/agent solution is allowed to agitate for a minimum of one hour. Agitation is not necessary following the first hour provided a single phase is obtained. At the end of the first hour the pH should be checked and adjusted up to 11.5 with additional NaOH as required.

An alternate solution for the decontamination of GD is 10 percent Sodium Carbonate in place of the 10 percent Sodium Hydroxide solution above. Continue with 55 grams of decon per gram of GD. Agitate for one hour and allow to react for 3 hours. At the end of the third hour adjust the pH to above 10. It is also permitted to substitute 5.25% Sodium Hypochlorite for the 10% Sodium Hydroxide solution above. Continue with 55 grams of decon per gram of GD. Agitate for one hour and allow to react for 3 hours then adjust the pH to above 10.

Scoop up alll material and place in a fully removable head and a high density polyethylene liner. Cover the contents with additional decontaminating solution before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All contaminated clothing will be placed in a fully removable head drum with a high density polyethylene liner.

Cover the contents of the drum with decontaminating solution as above before affixing the drum head. After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Conduct general area monitoring to confirm that the atmospheric concentrations do not exceed the exposure limits (see Section 8).

WASTE DISPOSAL METHOD: Open pit burning or burying of GD or items containing or contaminated with GD in any quantity is prohibited. The detoxified GD (using procedures above) can be thermally destroyed by incineration in an EPA approved incinerator in accordance with appropriate provisions of Federal, state and local RCRA regulations.

NOTE: Some states define decontaminated surety material as a RCRA Hazardous Waste.

Section VIII: Special Protection Informaton

RESPIRATORY PROTECTION:

```
GD Concentration Respiratory Protective Equipment

Less than 0.00003 mg/m3  M9, M17, or M40 series mask shall be available for
escape as necessary 0.00003 mg/m3 to 0.06 mg/m3   M9, or M40 series mask with
Level A or Level B ensemble (see AMCR 385-131 for determination of
appropriate level).

                          Demilitarization Protective Emsemble (DPE), or
                          Toxicological Agent Protective Ensemble Self
                          Contained(TAPES), used with prior approval from AMC
                          Field Safety Activity.
```

Greater than 0.06 mg/m3 or DPE or TAPES used with prior approval from AMC
Field unknown Field Safety Activity

NOTE: When DPE or TAPES is not available the M9 or M40 series mask with Level A protective ensemble can be used. However, use time shall be restricted to the extent operationally feasible, and may not exceed one hour. As an additional precaution, the cuffs of the sleeves and the legs of the M3 suit shall be taped to the gloves and boots respectively to reduce aspiration.

- Local Exhaust: Mandatory. Must be filtered or scrubbed to limit exit conc. to <.00001 mg/m3 (averaged over 8 hr/day, indefinitely).
- Special: Chemical laboratory hoods shall have an average inward face velocity of 100 linear feet per minute (lfpm) + 10 percent with the velocity at any point not deviating from the average face velocity by more than 20 percent. Laboratory hoods shall be located such that cross-drafts do not exceed 20 percent of the inward face velocity. A visual performance test utilizing smoke-producing devices shall be performed in assessing the ability of the hood to contain agent GD.
- Emergency back-up power necessary: Hoods should be tested semi-annually or after modification or maintenance operations. Operations should be performed 20 cm inside hood face.
- Other: Recirculation of exhaust air from agent areas is prohibited. No connection between agent areas and other areas through ventilation system is permitted.

PROTECTIVE GLOVES: Butyl Glove M3 and M4; Northon, Chemical Protective Glove Set

EYE PROTECTION: Chemical Goggles. For splash hazards use goggles and faceshield.

OTHER PROTECTIVE EQUIPMENT: Full protective clothing will consist of M9 mask and hood, butyl rubber suit (M3), M2A1 butyl boots, M3 and M4 gloves, unimpregnated underwear, or demilitarization protective ensemble (DPE). For laboratory operations, wear lab coats and have a protective mask readily available.

MONITORING: Available monitoring equipment for agent GD is the Automatic Chemical Agent Detector Alarm (ACADA), bubblers (GC method), and Chemical Agent Monitor (CAM).

Section IX: Special Precautions

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING: In handling GD, the buddy system will be incorporated. No smoking, eating or drinking is permitted in areas containing agent GD. Containers should be periodically inspected for leaks (either visually or by a detector kit) and prior to transferring

the containers from storage to work areas. Stringent control over all personnel practices must be exercised. Decontamination equipment shall be conveniently located. Exits must be designed to permit rapid evacuation. Chemical showers, eyewash stations, and personal cleanliness facilities shall be provided. Wash hands before meals and each worker will shower thoroughly with special attention given to hair, face, neck, and hands, using plenty of soap before leaving at the end of the workday.

OTHER PRECAUTIONS: Agent must be double-contained in liquid and vapor-tight containers when in storage or when outside of the ventilation hood.

For additional information, see AMC-R 385-131, "Safety Regulations for Chemical Agents H, HD, GB, and VX" and USaEHA Technical Guide No. 169, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX."

Section X: Transportation Data

PROPER SHIPPING NAME: Poisonous liquid, n.o.s.

DOT HAZARD CLASSIFICATION: Poison A

DOT LABEL: Poison gas

DOT MARKING: Poisonous liquid, n.o.s. (Pinacolyl methylphosphonofluoridate) NA 1955

DOT PLACARD: POISON GA

EMERGENCY ACCIDENT PRECAUTIONS AND PROCEDURES: See Section IV, VII and VIII.

PRECAUTIONS TO BE TAKEN IN TRANSPORTATION:

Motor vehicles will be placarded regardless of quantity. Driver shall be given full and complete information regarding shipment and conditions in case of emergency.

AR 50-6 deals specifically with the shipment of chemical agents. Shipments of agent will be escorted in accordance with AR 740-32.

While the Chemical Research Development and Engineering Center, Department of the Army believes that the data contained herein are factual and the opinions expressed are those of

qualified experts regarding the results of the tests conducted, the data are not to be taken as a warranty or representation for which the Department of the Army or Chemical Research Development Engineering Center assumes legal responsibility. They are offered solely for your consideration, investigation, and verification. Any use of these data and information must be determined by the user to be in accordance with applicable Federal, State, and local laws and regulations.

Addendum A: Physiological Effects

ACUTE PHYSIOLOGICAL EFFECTS:

Site of Action	Signs and Symptoms
Muscarine-like-Pupils	Miosis, marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting.
Conjunctivae	Hyperemia
Nasal mucous membranes	Rhinorrhea, hyperemia
Bronchial tree	Tighness in chest, sometimes with prolonged wheezing, expiration suggestive of broncho-constriction or increased secretion, cough.
	Following Systemic Absorption
Bronchial tree	Tightness in chest, with prolonged wheezing, expiration suggestive of broncho-constriction or increased secretion, dyspnea, slight pain in chest, increased, bronchial secretion, cough, pulmonary edema, cyanosis.
Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm)with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation.
Sweat glands	Increased sweating
Salivary glands	Increased salivation
Lacrimal glands	Increased lacrimation
Heart	Slight bradycardia

	Pupils	Slight miosis, occasionally unequal, later maximal miosis (pinpoint).
	Ciliary body	Blurring of vision
	Bladder	Frequent, involuntary micturition
Nicotine-like		
Striated muscle		Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis.

Sympathetic ganglia Pallor, occasional elevation of blood pressure.

Central Nervous system Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG, especially on over-ventilation, drowsiness, difficult concentration, slowness on recall, confusion, slurred speech, ataxia, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respirations, convulsions, depression of respiratory and circulatory centers, with dyspnea, cyanosis, and fall in blood pressure.

CHRONIC PHYSIOLOGICAL EFFECTS:

- Acute Exposure: If recovery from nerve agent poisoning occurs, it will be complete unless anoxia or convulsions have gone unchecked so long that irreversible central nervous system changes due to anoxemia have occurred.
- Chronic Exposure
    - The inhibition of cholinesterase enzymes throughout the body by nerve agents is more or less irreversible so that their effects are prolonged. Until the tissue cholinesterase enzymes are restored to normal activity, probably by very slow regeneration over a period of weeks or 2 to 3 months if damage is severe there is a period of increased susceptibility to the effects of another exposure to any nerve agent. During this period the effects of repeated exposures are cumulative; after a single exposure, daily exposure to concentrations of a nerve agent insufficient to produce symptoms may result in the onset of symptoms after several days. Continued daily exposure may be followed by increasingly severe effects. After symptoms subside, increased susceptibility persists for one to several days. The degree of exposure required to produce recurrence of symptoms, and the severity of these symptoms, depend on duration of exposure and time intervals between exposures. Increased susceptibility is not limited to the particular nerve agent initially absorbed.
    - Estimates have been made for the times as which 50% of exposed subjects would be affected (Et50's) at median incapacitating doses. These are presented below:

Et50	Degree of Effectiveness	ICt50	Exposure Time
min		mg min/m3	min
1.5	Moderate	27	0.5
3.0	Incap.	27	2.0
6.0		40	10.0
1.0	Severe	37	0.5
3.8	Incap.	37	2.0
7.8		56	10.0
2.0	Very	47	0.5
4.5	Severe	47	2.0
9.5	Incap.	72	10.0
6.5	Death	70	5.0
9.0		70	2.0
13.5		103	10.0

- Exposure to high concentrations of nerve agent may bring on incoordination, mental confusion and collapse so rapidly that the casualty cannot perform self-aid. If this happens, the man nearest to him will give first aid.
- Onset Time of Symptoms

Types of Effects	Route Absorption	Description	When Effects Appear After of Effects Exposure
Vapor	Lungs	Rhinorrhea, nasal	Hyperemia One to several minutes
Local		tightness in chest, wheezing	
Vapor	Eyes	Miosis, conjectival	hyperemia One to several minutes
Local		eye pain, frontal headache	
Vapor	Lungs or	Muscarine-like, nicotine-like	Less than 1 min. to a few min.
Systemic	eyes and central nervous system after moderate or marked effects.		

(See 2a above) exposure: about 30 min. after mild exposure

Liquid Local	Eyes	Same as vapor effects	Instantly	
Liquid	Ingestion	Gastrointestinal. (See 2a above).		About 30 min. after ingestion
Local				
Liquid Local	Skin	Local sweating and muscular twitching		3 min to 2 hours
Liquid Sytemic	Lungs	See 2a above	Several minutes	
Liquid Systemic	Eyes	Same as for vapor	Several minutes	
Liquid Systemic	Skin	Generalized sweating	15 minutes to 2 hours	
Liquid Systemic	Ingestion	Gastrointestinal (see 2a above)		15 minutes to 2 hours

Duration of Effects After

Types of Effects	Route of Absorption	Mild Exposure	Severe Exposure
Vapor Local	Lungs	A few hours	1 to 2 days
Vapor Local	Eyes	Miosis - 24 hours	3 to 14 days 2 to 5 days
Vapor Systemic	Lungs or eyes	Several hours	8 days
Liquid Local	Eyes	Similar to effects of vapor	
Liquid Local	Ingestion	3 days	5 days
Liquid Local	Skin	3 days	5 days

Liquid Systemic	Lungs	1 to 5 days
Liquid Systemic	Eyes	2 to 4 days
Liquid Systemic	Skin	2 to 5 days
Liquid Systemic	Ingestion	3 to 5 days

Addendum B: First Aid Procedures

1. Exposed personnel will be removed immediately to an uncontaminated atmosphere. Personnel handling casualty cases will give consideration to their own safety and will take precautions and employ the prerequisite protective equipment to avoid becoming exposed themselves.

   CAUTION: Due to the rapid effects of nerve agents, it is extremely important that decontamination of personnel not be delayed by attempting to blot off excessive agent prior to decontamination with sodium hypochlorite.

2. The causalty will then be decontaminated by washing the contaminated areas with commercial liquid household bleach (nominal 5% solution hyprchlorite or 10 percent sodium carbonate solution) and flushing with clean water. Mask will be left on the victim until decontamination has been completed unless it has been determined that areas of the face were contaminated and the mask must be removed to facilitate decontamination. After decontamination, the contaminated clothing will be removed and skin contamination washed away. If possible, decontamination will be completed before the casualty is taken to the aid station of medical facility.

   CAUTION: Care must be taken when decontaminating facial areas to avoid getting the hypochlorite into the eye or mouth. Only clean water shall be used when flushing the eyes or mouth. Skin surfaces decontaminated with bleach should be thoroughly flushed with water to prevent skin irritation from the bleach.

3. If there is no apparent breathing, artifical resuscitation will be started immediately (mouth-to-mouth, or with mechanical resuscitator). The situation will dictate method of choice, e.g., contaminated face. Do not use mouth-to-mouth resuscitation when facial contamination exists. When appropriate and trained personnel are available, cardiopulmonary resuscitation (CPR) may be necessary.

4. An individual who has received a known agent exposure or who exhibits definite signs or symptoms of agent exposure shall be given an intramuscular injection immediately with the MARK I kit auto-injectors.
    1. Some of the early symptoms of a vapor exposure may be rhinorrhea (runny nose) and/or tightness in the chest with shortness of breath (bronchial constriction).
    2. Some of the early symptoms of a percutaneous exposure may be local muscular twitching or sweating at the area of exposure followed by nausea or vomiting.
    3. Although myosis (pin-pointing of the pupils) may be an early sign of agent exposure, an injection shall not be administered when myosis is the only sign present. Instead, the individual shall be taken immediately to the medical facility for observation.
    4. Injections using the MARK I kit injectors (or atropine only if directed by the local physician) may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will maintained of all injections given.
    5. Administer, in rapid succession, all three MARK I kit injectors (or atropine if directed by the local physician) in the case of SEVERE signs of agent exposure.
5. If indicated, CPR should be started immediately. Mouth-to-mouth resuscitation should be used when approved mask-bag or oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists.

    CAUTION: Atropine does not act as a prophylactic and shall not be administered until an agent exposure has been ascertained.

Addendum C: Additional Information for Thickened GD

TRADE NAME AND SYNONYMS: Thickened GD, TGD

HAZARDOUS INGREDIENTS: K125 (acryloid copolymer, 5%) is used to thicken the GD. K125 is not known to be a hazardous material except in a finely divided, powder form.

PHYSICAL DATA: Essential the same as GD except for viscosity. The viscosity of TGD is approximately 1180 centistokes.

FIRE AND EXPLOSION DATA: Same as GD

HEALTH HAZARD DATA: Same as GD except for skin contact. For skin contact, don respiratory protective mask and remove contaminated clothing. Immediately scrape the TGD from the skin surface, then wash the contaminated surface with acetone. Administer Nerve Agent Antidote Kit, MARK I, only if local sweating and muscular twitching symptoms are observed. Seek medical attention IMMEDIATELY.

SPILL, LEAK AND DISPOSAL PROCEDURES: If spills or leaks of TGD occur, follow the same procedure as those for GD, but add the following step: Since TGD is not water soluble, dissolve the TGD in acetone prior to introducing any decontaminating solution. Containment of TGD is generally not necessary. Spilled TGD can be carefully scraped off the contaminated surface and placed in a drum with a fully removable head and a high density, polyethylene lining. The TGD can then be decontaminated after it has been dissolved in acetone, using the same procedures as for GD. Contaminated surfaces should be treated with acetone, then decontaminated using the same procedures as for GD.

SPECIAL PROTECTION INFORMATION: Same as GD.

SPECIAL PRECAUTIONS: Same as GD with the following addition. Handling the TGD requires careful observation of the "stringers" (elastic, thread-like attachments) formed when the agents are transferred or dispensed. These stringers must be broken cleanly before moving the contaminating device or dispensing device to another location, or unwanted contamination of a working surface will result.

TRANSPORTATION DATA: Same as GD.

Appendix - Material Safety Data Sheets

- Chemical Nerve Agents
  - VX

Material Safety Data Sheet -- Lethal Nerve Agent VX

Section I: General Information
Section II: Composition
Section III: Physical Data
Section IV: Fire and Explosion Data
Section V: Health Hazard Data
Section VI: Reactivity Data
Section VII: Spill, Leak and Disposal Procedures
Section VIII: Special Protection Information
Section IX: Special Precautions
Section X: Transportation Data
Addendum A: Physiological Effects
Addendum B: First Aid Procedures

Section I: General Information

MANUFACTURER'S NAME: Department of the Army

MANUFACTURER'S ADDRESS:

U.S. Army Armament, Munitions and Chemical Command
Chemical Research, Development and Engineering Center
ATTN: SMCCR-CMS-E
Aberdeen Proving Ground, MD 21010-5423

CAS REGISTRY NUMBER: 50782-69-9, 51848-47-6, 53800-40-1, 70938-84-0

CHEMICAL NAME: Phosphonothioic acid, methyl-, S-(2bis(1-methylethylamino)ethyl) 0-ethyl ester

ALTERNATE CHEMICAL NAMES:

- O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothioate
- S-2-Diisopropylaminoethyl O-ethyl methylphosphonothioate
- S-2 (2-Diisopropylamino)ethyl) O-ethyl methylphosphonothiolate
- O-ethyl S-(2-diisoproplyaminoethy) methylphosphonothioate
- O-ethyl S-(2-diisoproplyaminoethy) methylthiolphosphonoate

TRADE NAME AND SYNONYMS:

- VS
- EA 1701
- TX60

CHEMICAL FAMILY: Sulfinated organophosphorus compound

FORMULA: C11 H26 N 02 P S

NFPA 704 SIGNAL:

- Health - 4
- Flammability - 1
- Reactivity - 1

Section II: Composition

INGREDIENTS NAME	FORMULA	PERCENTAGE BY WEIGHT	AIRBORNE EXPOSURE LIMIT
VX	C11H26N02PS	100	.00001 mg/m3

Section III: Physical Data

BOILING POINT DEG F (DEG C): 568 (298)

VAPOR PRESSURE (mm Hg): 0.0007 @ 25 DEG C

VAPOR DENSITY (AIR=1): 9.2

SOLUBILITY IN WATER: Moderate

APPEARANCE AND ODOR: Colorless to straw colored liquid & odorless, similar in appearance to motor oil.

Section IV: Fire and Explosion Data

FLASHPOINT: 159 DEG C (McCutchan-Young)

FLAMMABILITY LIMITS: (% by volume): Not available

LOWER EXPLOSIVE LIMIT: Not applicable

UPPER EXPLOSIVE LIMIT: Not applicable

EXTINGUISING MEDIA: Water mist, fog, foam, CO2 - Avoid using extinguishing methods that will cause splashing or spreading of the VX.

SPECIAL FIRE FIGHTING PROCEDURES:

All persons not engaged in extinguishing the fire should be immediately evacuated from the area. Fires involving VX should be contained to prevent contamination to uncontrolled areas. When

responding to a fire alarm in buildings or areas containing agents, firefighting personnel should wear full firefighter protective clothing (without TAP clothing) during chemical agent firefighting and fire rescue operations.

Respiratory protection is required. Positive pressure, full facepiece, NIOSH-approved self contained breathing apparatus (SCBA) will be worn where there is danger of oxygen deficiency and when directed by the fire chief of chemical accident/incident (CAI) operations officer. The M9 or M17 series mask may be worn in lieu of SCBA when there is no danger of oxygen deficiency. In cases where firefighters are responding to a chemical accident/incident for rescue/reconnaissance purposes vice firefighting, they will wear appropriate levels of protective clothing (see Section 8).

Do not breathe fumes. Skin contact with V-agents must be avoided at all times. Although the fire may destroy most of the agent, care must still be taken to assure the agent or contaminated liquids do not further contaminate other areas or sewers. Contact with VX or VX vapors can be fatal.

UNUSUAL FIRE AND EXPLOSION HAZARDS: None known.

Section V: Health Hazard Data

RECOMMENDED EXPOSURE LIMIT (REL):

The suggested permissible airborne exposure concentration of VX for an 8 hour workday or a 40 hour work week is an 8 hour time weighted average (TWA) of 0.00001 mg/m3 (9 x 10-7 ppm). This value is based on the TWA of VX as proposed in the USAEHA Technical Guide No. 169, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX." To date, however, the Occupational Safety and Health Administration (OSHA) has not promulgated permissible exposure concentration for VX.

VX is not listed by the International Agency for Research on Cancer (IARC), American Conference of Governmental Industrial Hygienists (ACGIH), Occupational Safety and Health Administration (OSHA), or National Toxicology Program (NTP) as a carcinogen.

EFFECTS OF OVEREXPOSURE:

VX is a lethal anticholinergic agent with the median dose in man being: LC50 (skin) = 0.135 mg/kg; ID50 (Skin) - 0.07 - 0.71 mg/kg; LCt50 (inhalation) = 30 mg min/m3; LCt50 (inhalation) - 30 mg min/m3; LCt50 (inhalation) - 24 mg min/m3.

1. One to several minutes after overexposure to airborne VX the following acute symptoms appear:
    1. LOCAL EFFECTS (lasting 1-15 days, increases with dose)
        1. On eyes: Miosis (constriction of pupils); redness, pressure sensation on eyes.
        2. By inhalation: Rhinorrhea (runny nose), nasal congestion, tightness in chest, wheezing, salivation, nausea, vomiting
    2. SYSTEMIC EFFECTS (increases with dose): By inhalation - excessive secretion causing coughing/breathing difficulty; salivation and sweating; vomiting, diarrhea; stomach cramps; involuntary urination/defecation; generalized muscle twitching/muscle cramps; CNS depression including anxiety, restlessness, giddiness, insomnia, excessive dreaming and nightmares. With more severe exposure, also headache, tremor, drowsiness, concentration difficulty, memory impairment, confusion, unsteadiness on standing or walking, and progressing to death.
2. After exposure to liquid VX, the following acute symptoms appear:
    1. Local Effects:
        1. On eyes: Miosis, redness, pressure sensation on eyes.
        2. By ingestion: salivation, anorexia, nausea, vomiting, abdominal cramps, diarrhea, involuntary defecation, heartburn.
        3. On skin: Sweating, muscle twitching
    2. Systemic Effects: Similar to generalized effects from exposure to airborne VX.
3. Chronic overexposure to VX causes forgetfulness, thinking difficulty, vision disturbances, muscular aches/pains. Although cer-organophosphate pesticides have been shown to be teratogenic in animals, these effects have not been documented in carefully controlled toxicological evaluations for VX.

EMERGENCY AND FIRST AID PROCEDURES:

- INHALATION: Hold breath until respiratory protective mask is donned. If severe signs of agent exposure appear (chest tightens, pupil constriction, incoordination, etc.), immediately administer, in rapid succession, all three Nerve Agent Antidote Kit(s), Mark I injectors (or atropine if directed by the local physician). Injections using the Mark I kit injectors may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will be maintained of all injections given. If breathing has stopped, give artificial respiration. Mouth-to-mouth resuscitation should be used when approved mask-bag or oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists. If breathing is difficult, administer oxygen. Seek medical attention IMMEDIATELY.
- EYE CONTACT: IMMEDIATELY flush eyes with water for 10-15 minutes, then don respiratory protective mask. Although miosis (pinpointing of the pupils) may be an early sign of agent exposure, an injection will not be administered when miosis is the only sign present. Instead, the individual will be taken IMMEDIATELY to the medical treatment facility for observation.
- SKIN CONTACT: Don respsiratory protective mask and remove contaminated clothing. Immediately wash contaminated skin with a solution of 5% household bleach. Rinse well with water to remove excess bleach followed by copious soap and water wash. Administer nerve

agent antidote kit, Mark I, only if local sweating and muscular twitching symptoms are present. Seek medical attention IMMEDIATELY.

- INGESTION: Do not induce vomiting. First symptoms are likely to be gastronintestinal. IMMEDIATELY administer Nerve Agent Antidote kit, Mark I. Seek medical attention immediately.

Section VI: Reactivity Data

STABILITY: Relatively stable at room temperature. Unstabilized VX of 95% purity decomposed at a rate of 5% a month at 71 deg. C.

HAZARDOUS DECOMPOSITION PRODUCTS:

During basic hydrolysis of VX up to about 10% of the agent is converted to EA2192 (diisopropylaminoethyl methylphosphonothioic acid). Based on the concentration of EA2192 expected to be formed during hydrolysis and its toxicity (1.4 mg/kg dermal in rabbit at 24 hours in a 10/90 wt% ethanol/water solution), a Class B poison would result.

The large scale decon procedure, which uses both HTH and NaOH, destroys VX by oxidation and hydrolysis. Typically the large scale product contains 0.2 - 0.4 wt% EA2192 at 24 hours. At pH 12, the EA2192 in the large scale product has a half-life of about 14 days. Thus the 90 day holding period at pH 12 results in about a 64-fold reduction of EA2192 (six half-lives). This holding period has been shown to be sufficient to reduce the toxicity of the product below that of a Class B poison.

Other less toxic products are ethyl methylphosphonic acid, methylphosphinic acid, diisopropylaminoethyl mercaptan, diethyl methylphosphonate, and ethanol.

The small scale decontamination procedure uses sufficient HTH to oxidize all VX thus no EA2192 is formed.

HAZARDOUS POLYMERIZATION: Will not occur.

Section VII: Spill, Leak and Disposal Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED: If leak or spills occur, only personnel in full protective clothing (see Section 8) will remain in area. In case of personnel contamination, see Section V "Emergency and First Aid Instructions." Spills must be contained by covering with vermiculite, diatomaceous earth, clay or fine sand. This containment is followed by the following treatment.

RECOMMENDED LABORATORY PROCEDURES (For Quantities less than 50 grams):

If the active chlorine of the Calcium Hypochlorite (HTH) is at least 55 percent, then 80 grams of a 10 percent slurry is required for each gram of VX. Proportionally more HTH is required if the chlorine activity of the HTH is lower than 55 percent. The mixture is agitated as the VX is added and the agitation is maintained for a minimum of one hour. If phasing of the VX/decon solution continues after 5 minutes, an amount of denatured ethanol equal to a 10 wt percent of the total agent/decon shall be added to assist miscibility. NOTE: Ethanol should be minimized to prevent the formation of a hazardous waste.

Upon completion of the one hour agitation the decon mixture shall be adjusted to a pH between 10 and 11. Conduct general area monitoring to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Secions 2 and 8).

RECOMMENDED FIELD PROCEDURES (For quantities greater than 50 grams):

NOTE: These procedures can only be used with the approval of the CRDEC Safety Officer.

An alcoholic HTH mixture is prepared by adding 100 milliliters of denatured ethanol to a 900 milliliter slurry of 10 percent HTH in water. This mixture should be made just prior to use since the HTH can react with the ethanol. Fourteen grams of alcoholic HTH solution is used for each gram of VX. Agitate the contaminaton mixture as the VX is added. Continue the agitation for a minimum of one hour. This reaction is reasonable exothermic and evolves substantial off gassing. The evolved reaction gases should be routed through a decontaminate filled scrubber prior to release through filtration systems.

After completion of the one hour minimum agitation, 10 percent Sodium Hydroxide is added in a quantity equal to that necessary to assure that a pH of 12.5 is maintained for a period not less than 24 hours. Hold the material at a pH between 10 ad 12 for a period not less than 90 days to ensure that a hazardous intermediate material is not formed.

After sealing the head, the exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Conduct general area monitoring to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Section 2 and 8).

If the alcoholic Calcium Hypochlorite (HTH) mixture is not available then the following decontaminants may be used instead and are listed in the order of preference: Decontamination Solution No. 2 (DS2), Supertropical Bleach Slurry (STB), and Sodium Hypochlorite.

WASTE DISPOSAL METHOD: Open pit burning or burying of VX or items containing or contaminated with VX in any quantity is prohibited. The detoxified VX (using procedures above) can be thermally destroyed by incineration in an EPA approved incinerator in accordance with appropriate provisions of Federal, state and local RCRA regulations. NOTE: Some states define decontaminated surety material as a RCRA Hazardous Waste.

Section VIII: Special Protection Informaton

RESPIRATORY PROTECTION:

VX Concentration	Respiratory Protective Equipment
Less than 0.00001 mg/m3	M9, M17, or M40 series mask shall be available for escape as necessary
0.00001 mg/m3 to 0.02 mg/m3	M9, or M40 series mask with Level A or Level B protective ensemble (see AMCR 385-131 for determination of appropirate level).
	Demilitarization Protective Emsemble (DPE), or Toxicological Agent Protective Ensemble Self-Contained (TAPES), used with prior approval from AMC Field Safety Activity.
Greater than 0.02 mg/m3 or DPE or TAPES used with prior approval from AMC Field unknown Field Safety Activity	
	NOTE: When DPE or TAPES is not available the M9 or M40 series mask with Level A protective ensemble can be used. However, use time shall be restricted to the extent operationally feasible, and may not exceed one hour.
	As an additional precaution, the cuffs of the sleeves and the legs of the M3 suit shall be taped to the gloves and boots respectively to reduce aspiration.

VENTILATION: Local Exhaust: Must be filtered or scrubbed to limit exit conc.to <.00001 mg/m3.

Special: Chemical laboratory hoods shall have an average inward face velocity of 100 linear feet per minute (lfpm) + 10 percent with the velocity at any point not deviating from the average face velocity by more than 20 percent. Laboratory hoods shall be located such that cross-drafts do not exceed 20 percent of the inward face velocity. A visual performance test utilizing smoke-producing devices shall be performed in assessing the ability of the hood to contain agent VX.

Emergency back-up power necessary. Hoods should be tested semi-annually or after modification or maintenance operations. Operations should be performed 20 cm inside hood face.

Other: Recirculation of exhaust air from agent areas is prohibited. No connection between agent areas and other areas through ventilation system is permitted.

PROTECTIVE GLOVES: Butyl Glove M3 and M4; Northon, Chemical Protective Glove Set

EYE PROTECTION: Chemical Goggles. For splash hazards use goggles and faceshield.

OTHER PROTECTIVE EQUIPMENT:

Full protective clothing will consist of M9 mask and hood, m3 butyl rubber suit (M3), M2A1 butyl boots, M3 and M4 gloves, unimpregnated underwear, or demilitarization protective ensemble (DPE). For laboratory operations, wear lab coats and have a protective mask readily available.

In addition, daily clean smock, foot covers, and head covers will be required when handling contaminated lab animals.

MONITORING: Available monitoring equipment for agent HD is the M8/M9 Detector paper, ACADA), detector ticket, M256/M256A1 kits, bubbler. Depot Area Air Monitoring System (DAMMS), Automated Continuous Air Monitoring System (ACMS), Real-Time Monitor (RTM), Demilitarization Chemical Agent Concentrator (DCAC), M8/M43, M8A1/M43A1, CAM-M1, Hydrogen Flame Photometric Emission Detector (HYFED), and the Minature Chemical Agent Monitor (MINICAM).

Section IX: Special Precautions

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING: In handling, the buddy system will be incorporated. No smoking, eating or drinking in areas containing agent is permitted. Containers should be periodically inspected for leaks (either visually or by a detector kit). Stringent control over all personnel practices must be exercised. Decontamination equipment shall be conveniently located. Exits must be designed to permit rapid evacuation. Chemical showers, eyewash stations, and personal cleanliness facilities shall be provided. Wash hands before meals and each worker will shower thoroughly with special attention given to hair, face, neck, and hands, using plenty of soap before leaving at the end of the workday.

OTHER PRECAUTIONS: Agent must be double-contained in liquid and vapor-tight containers when in storage or when outside of the ventilation hood.

For additional information, see AMC-R 385-131, "Safety Regulations for Chemical Agents H, HD, HT, GB, and VX" and USAEHA Technical Guide No. 169, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Nerve Agents GA, GB, GD, and VX."

Section X: Transportation Data

PROPER SHIPPING NAME: Poisonous liquid, n.o.s.

DOT HAZARD CLASSIFICATION: Poison A

DOT LABEL: Poison gas

DOT MARKING: Poisonous liquid, n.o.s. (O-ethyl S-(2-diisopropylaminoothyl) methyl phosphonothioate) NA 1955

DOT PLACARD: POISON GAS

EMERGENCY ACCIDENT PRECAUTIONS AND PROCEDURES: See Section IV, VII and VIII.

PRECAUTIONS TO BE TAKEN IN TRANSPORTATION:

Motor vehicles will be placarded regardless of quantity. Driver shall be given full and complete information regarding shipment and conditions in case of emergency. AR 50-6 deals specifically with the shipment of chemical agents. Shipments of agent will be escorted in accordance with AR 740-32.

While the Chemical Research Development and Engineering Center, Department of the Army believes that the data contained herein are factual and the opinions expressed are those of qualified experts regarding the results of the tests conducted, the data are not to be taken as a warranty or representation for which the Department of the Army or Chemical Research Development Engineering Center assumes legal responsibility. They are offered solely for your consideration, investigation, and verification. Any use of these data and information must be determined by the user to be in accordance with applicable Federal, State, and local laws and regulations.

Addendum A: Physiological Effects

ACUTE PHYSIOLOGICAL EFFECTS:

Site of Action	Signs and Symptoms
	Following Local Exposure Muscarine-like-
Pupils	Miosis, marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting.
Conjunctivae	Hyperemia
Nasal mucous membranes	Rhinorrhea, hyperemia
Bronchial tree	Tightness in chest, sometimes with prolonged wheezing expiration suggestive of broncho-constriction or increased secretion, cough..
	Following Systemic Absorption
Bronchial tree	Tightness in chest, with prolonged wheezing, expiration suggestive of broncho-constricion or increased secretion, dyspnea, slight pain in chest, increased bronchial secretion, cough, ulmonary edema, cyanosis.
Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation.
Sweat glands	Increased sweating
Salivary glands	Increased salivation
Lacrimal glands	Increased lacrimation
Heart	Slight bradycardia
Pupils	Slight miosis, occasionally unequal, later maximal miosis (pinpoint).
Ciliary body	Blurring of vision
Bladder	Frequent, involuntary micturition

Nicotine-like

Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis.
Sympathetic ganglia	Pallor, occasional elevation of blood pressure.
Central Nervous system	Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG, especially on over-ventilation, drowsiness, difficult concentration, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respirations, convulsions, depression of respiratory and circulatory enters, with dyspnea, cyanosis, and fall in blood pressure.

CHRONIC PHYSIOLOGICAL EFFECTS:

Acute Exposure

- If recovery from nerve agent poisoning occurs, it will be complete unless anoxia or convulsions have gone unchecked so long that irreversible central nervous system changes due to anoxemia have occurred.

Chronic Exposure

- The inhibition of cholinesterase enzymes throughout the body by nerve agents is more or less irreversible so that their effects are prolonged. Until the tissue cholinesterase enzymes are restored to normal activity, probably by very slow regeneration over a period of weeks or 2 to 3 months if damage is severe there is a period of increased susceptibility to the effects of another exposure to any nerve agent. During this period the effects of repeated exposures are cumulative; after a single exposure, daily exposure to concentrations of a nerve agent insufficient to product symptoms may result in the onset of symptoms after several days.
- Continued daily exposure may be followed by increasingly severe effects. After symptoms subside, increased susceptibility persists for one to several days. The degree of exposure required to produce recurrence of symptoms, and the severity of these symptoms, depend on duration of exposure and time intervals between exposures. Increased susceptibility is not limited to the particular nerve agent initially absorbed.

- Estimates have been made for the times as which 50% of exposed subjects would be affected (Et50's) at median incapacitating doses. These are presented below:

Et50	Degree of Effectiveness	ICt50	Exposure Time
min		mg min/m3	min
1.5	Moderate	27	0.5
3.0	Incap.	27	2.0
6.0		40	10.0
1.0	Severe	37	0.5
3.8	Incap.	37	2.0
7.8		56	10.0
2.0	Very	47	0.5
4.5	Severe	47	2.0
9.5	Incap.	72	10.0
6.5	Death	70	5.0
9.0		70	2.0
13.5		103	10.0

Exposure to high concentrations of nerve agent may bring on incoordination, mental confusion
and collapse so rapidly that the casualty cannot perform self-aid. If this happens, the man
nearest to him will give first aid.

Onset Time of Symptoms

Types of Effects	Route of Absorption	Description	When Effects Appear After of Effects	Exposure
Vapor Local	Lungs	Rhinorrhea, nasal Hyperemia tightness in chest, wheezing		One to several minutes
Vapor Local	Eyes	Miosis, conjectival hyperemia eye pain, frontal headache		One to several minutes
Vapor	Lungs or	Muscarine-like, nicotine-like		Less than 1 min. to a few min.

Systemic	eyes	and central nervous system effects. (See 2a above)		after moderate or marked exposure: about 30 min. after mild exposure
Liquid Local	Eyes	Same as vapor effects	Instantly	
Liquid Local	Ingestion	Gastrointestinal. (See 2a above).		About 30 min. after ingestion
Liquid Local	Skin	Local sweating and muscular twitching		3 min to 2 hours
Liquid Sytemic	Lungs	See 2a above	Several minutes	
Liquid Systemic	Eyes	Same as for vapor	Several minutes	
Liquid Systemic	Skin	Generalized sweating	15 minutes to 2 hours	
Liquid Systemic	Ingestion	Gastrointestinal (see 2a above)		15 minutes to 2 hours

Onset Time of Symptoms. (cont'd)

Duration of Effects After

Types of Effects	Route of Absorption	Mild Exposure	Severe Exposure
Vapor Local	Lungs	A few hours	1 to 2 days
Vapor Local	Eyes	Miosis - 24 hours	3 to 14 days 2 to 5 days
Vapor Systemic	Lungs or eyes	Several hours	8 days
Liquid Local	Eyes	Similar to effects of vapor	
Liquid Local	Ingestion	3 days	5 days

Liquid Local	Skin	3 days	5 days
Liquid Systemic	Lungs		1 to 5 days
Liquid Systemic	Eyes		2 to 4 days
Liquid Systemic	Skin		2 to 5 days
Liquid Systemic	Ingestion		3 to 5 days

Addendum B: First Aid Procedures

1. Exposed personnel will be removed immediately to an uncontaminated atmosphere. Personnel handling casualty cases will give consideration to their own safety and will take precautions and employ the prerequisite protective equipment to avoid becoming exposed themselves.

   CAUTION: Due to the rapid effects of nerve agents, it is extremely important that decontamination of personnel not be delayed by attempting to blot off excessive agent prior to decontamination with sodium hypochlorite.

2. The causalty will then be decontaminated by immediately removing any contaminated clothing and washing the contaminated areas with copious amounts of soap and water, 5% sodium hypochlorite solution, or liquid household bleach (nominal 5% solution sodium hypochlorite) and flushing with clean water. Mask will be left on the victim until decontamination has been completed unless it has been determined that areas of the face were contaminated and the mask must be removed to facilitate decontamination. After decontamination, the contaminated clothing will be removed and skin contamination washed away. If possible, decontamination will be completed before the casualty is taken to the aid station of medical facility.

   CAUTION: Care must be taken when decontaminating facial areas to avoid getting the hypochlorite into the eye or mouth. Only clean water shall be used when flushing the eyes or mouth. Skin surfaces decontaminated with bleach should be thoroughly flushed with water to prevent skin irritation from the bleach.

3. If there is no apparent breathing, artifical resuscitation will be started immediately (mouth-to-mouth, or with mechanical resuscitator). The situation will dictate method of choice, e.g.,

contaminated face. Do not use mouth-to-mouth resuscitation when facial contamination exists. When appropriate and trained personnel are available, cardiopulmonary resuscitation (CPR) may be necessary.

4. An individual who has received a known agent exposure or who exhibits definite signs or symptoms of agent exposure shall be injected immediately with the Nerve Agent Antidote Kit, MARK I.
    1. Some of the early symptoms of a vapor exposure may be rhinorrhea (runny nose) and/or tightness in the chest with shortness of breath (bronchial construction).
    2. Some of the early symptoms of a percutaneous exposure may be local muscular twitching or sweating at the area of exposure followed by nausea or vomiting.
    3. Although myosis (pin-pointing of the pupils) may be an early sign of agent exposure, a Mark I Kit shall not be administered when myosis is the only sign present. Instead, the individual shall be taken immediately to the medical facility for observation.
    4. Injections using the MARK I kit injectors (or atropine only if directed by the local physician) may be repeated at 5 to 20 minute intervals if signs and symptoms are progressing until three series of injections have been administered. No more injections will be given unless directed by medical personnel. In addition, a record will maintained of all injections given.
    5. Administer, in rapid succession, all three MARK I kit injectors (or atropine if directed by the local physician) in the case of SEVERE signs of agent exposure.

    CAUTION: The nerve Agent Antidote Kit, MARK I does not act as a prophylactic and shall not be administered until an agent exposure has been ascertained.

5. If indicated, CPR should be started immediately. Mouth-to-mouth resuscitation should be used when approved mask-bag or oxygen delivery systems are not available. Do not use mouth-to-mouth resuscitation when facial contamination exists.

Appendix - Material Safety Data Sheets

- Blister Agents
  - Sulfur Mustard (HD, THD)
  - Sulfur Mustard (HT) (No Data Found)

Material Safety Data Sheet -- Lethal Nerve Agents Sulfur Mustards (HD and THD)

Section I: General Information
Section II: Composition
Section III: Physical Data
Section IV: Fire and Explosion Data
Section V: Health Hazard Data
Section VI: Reactivity Data
Section VII: Spill, Leak and Disposal Procedures
Section VIII: Special Protection Information
Section IX: Special Precautions
Section X: Transportation Data
Addendum A: Additional Information for Thickened HD

Section I: General Information

MANUFACTURER'S NAME: Department of the Army

MANUFACTURER'S ADDRESS:

U.S. Army Armament, Munitions and Chemical Command
Chemical Research, Development and Engineering Center
ATTN: SMCCR-CMS-E
Aberdeen Proving Ground, MD 21010-5423

CAS REGISTRY NUMBER: 505-60-2, 39472-40-7, 68157-62-0

CHEMICAL NAME AND SYNONYMS:

- Sulfide, bis (20chloroethyl)
- Bis (beta-chloroethyl) sulfide
- Bis (2-chloroethyl) sulfide
- (beta-chloroethylthio) ethane
- beta, beta'-dichlorodiethyl sulfide
- 2,2' dichlorodiethyl sulfide
- Di-2-chloroethyl sulfide
- beta, beta'-dichloroethyl sulfide
- 2,2'-dichloroethyl sulfide

TRADE NAME AND SYNONYMS:

- HD
- Senfgas
- H
- Sulfur mustard
- S-lost
- HS
- Iprit
- Suphur mustard gas
- Kampstoff "Lost"
- S-yperite
- Lost
- Yellow Cross Liquid
- Mustard Gas
- Yperite

CHEMICAL FAMILY: chlorinated sulfur compound

FORMULA: C4(H8)C12(S)

NFPA 704 SIGNAL:

- Health - 4
- Flammability - 1
- Reactivity - 1

Section II: Composition

INGREDIENTS NAME	FORMULA	PERCENTAGE BY WEIGHT	AIRBORNE EXPOSURE LIMIT (AEL)
Sulfur Mustard	C4(H8)C12(S)	100	0.003 mg/m3 (8 hr-TWA)

Section III: Physical Data

BOILING POINT DEG F (DEG C): 422 DEG F. (217 DEG C)

VAPOR PRESSURE (mm Hg): 0.072 mm Hg @ 20 DEG C (0.11 mm Hg @ 25 DEG C)

VAPOR DENSITY (AIR=1): 5.5

SOLUBILITY IN WATER: Negligible. Soluble in acetone, CH3(C1), tetrachloroethane, ethylbenzoate, and ether.

SPECIFIC GRAVITY (H20=1): 1.27 @ 20 DEG C

VOLATILITY: 610 mg/m3 @ 20 DEG C; 920 mg/m3 @ 25 DEG C

APPEARANCE AND ODOR: Water clear if pure. Normally pale yellow to black. Slight garlic type odor. The odor threshold for HD is 0.0006 mg/m3

Section IV: Fire and Explosion Data

FLASHPOINT (METHOD USED): 105 DEG C (ignited by large explosive charges)

FLAMMABILITY LIMITS (% by volume): Unknown

EXTINGUISING MEDIA: Water, fog, foam, CO2. Avoid using extinguishing methods that will splash or spread mustard.

SPECIAL FIRE FIGHTING PROCEDURES:

All persons not engaged in extinguishing the fire should be immediately evacuated from the area. Fires involving HD should be contained to prevent contamination to uncontrolled areas. When responding to a fire alarm in buildings or areas containing agents, firefighting personnel should wear full firefighter protective clothing (without TAP clothing) during chemical agent firefighting and fire rescue operations.

Respiratory protection is required. Positive pressure, full facepiece, NIOSH-approved self contained breathing apparatus (SCBA) will be worn where there is danger of oxygen deficiency and when directed by the fire chief of chemical accident/incident (CAI) operations officer. The M9 or M17 series mask may be worn in lieu of SCBA when there is no danger of oxygen deficiency. In cases where firefighters are responding to a chemical accident/incident for rescue/reconnaissance purposes vice firefighting, they will wear appropriate levels of protective clothing (see Section 8).

Section V: Health Hazard Data

AIRBORNE EXPOSURE LIMIT (REL): The AEL for HD is 0.003 mg/m3 as proposed in the USAEHA Technical Guide No. 173, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Mustard Agents H, HD and HT." No individual should be intentionally exposed to any direct skin or eye contact.

EFFECTS OF OVEREXPOSURE: HD is a vesicant (causing blisters) and alkylating agent producing cytotoxic action on the hematopoietic (blood-forming) tissues which are especially sensitive. The rate of detoxification of HD in the body is very slow and repeated exposures produce a cumulative effect. HD has been found to be a human carcinogen by the International Agency for Research on Cancer (IARC).

Median doses of HD in man are:

- LD50 (skin) = 100 mg/kg
- ICt50 (skin) = 2000 mg-min/m3 at 70-80 DEG F (humid environment); = 1000 mg-min/m3 at 90 DEG F (dry environment)
- ICt50 (eyes) = 200 mg-min/m3
- ICt50 (inhalation)=1500 mg-min/m3 (Ct unchanged with time)
- LD50 (oral) = 0.7 mg/kg

Maimum safe Ct for skin and eyes are 5 and 2 mg-min/m3, respectively.

ACUTE PHYSIOLOGICAL ACTION OF HD IS CLASSIFIED AS LOCAL AND SYSTEMIC:

- LOCALLY, HD affects both the eyes and the skin. SKIN damage occurs after percutaneous resorption. Being lipid soluble, HD can be resorbed into all organs. Skin penetration is rapid without skin irritation. Swelling (blisters) and reddening (erythema) of the skin occurs after a latency period of 4-24 hours following the exposure depending on degree of exposure and individual sensitivity. The skin healing process is very slow. Tender skin, mucous membrane and perspiration covered skin are more sensitive to the effects of HD. HD's effect on the skin, however, is less than on the eyes. Local action on the eyes produces severe necrotic damage and loss of eyesight. Exposure of eyes to HD vapor or aerosol produces lacrimation, photophobia, and inflammation of the conjunctiva and cornea.
- SYSTEMIC ACTIONS occur primarily through inhalation and ingestion. The HD vapor or aerosol is less toxic to the skin or eyes than the liquid form. When inhaled, the upper respiratory tract (nose, throat, trachea) is inflamed after a few hours latency period, accompanied by sneezing, coughing, and bronchitis, loss of appetite, diarrhea, fever, and apathy. Exposure to nearly lethal dose of HD can produce injury to bone marrow, lymph nodes, and spleen as indicated by a drop in WBC count and, therefore, results in increased susceptibilty to local and systemic infectons. Ingestion of HD will produce severe stomach pains, vomiting, and bloody stools after a 15-20 minute latency period.
- CHRONIC EXPOSURE to HD can cause sensitization, chronic lung impairment, (cough, shortness of breath, chest pain), and cancer of the mouth, throat, respiratory tract, skin, and leukemia. It may also cause birth defects.

EMERGENCY AND FIRST AID PROCEDURES:

- INHALATION: Remove from the source IMMEDIATELY. If breathing has stopped, give artificial respiration. If breathing is difficult, administer oxygen. Seek medical attention IMMEDIATELY.
- EYE CONTACT: Speed in decontaminating the eyes is absolutely essential. Remove person from the liquid source, flush the eyes immediately with water by tilting the head to the side, pulling the eyelids apart with the fingers and pouring water slowly into the eyes. Do not cover eyes with bandages but, if necessary, protect eyes by means of dark or opaque goggles. Transfer the patient to a medical facility IMMEDIATELY.
- SKIN CONTACT: Don respsiratory protective mask and gloves; remove victim from agent source immediately. Flush skin and clothes with 5 percent solution of sodium hypochlorite or liquid house hold bleach within one minute. Cut and remove contaminated clothing, flush contaminated skin area again with 5 percent sodium hypochlorite solution, then wash contaminated skin area with soap and water. If shower facilities are available, wash thoroughly and transfer to medical facility. If the skin becomes contaminated with a thickened agent, blot/wipe the material off immediately with an absorbent pad/paper towel prior to using decontaminating solution.
- INGESTION: Do not induce vomiting. Give victim milk to drink. Seek medical attention immediately.

Section VI: Reactivity Data

STABILITY: Stable at ambient temperatures. Decomposition temperature is 149 DEG C to 177 DEG C. Mustard is a persistent agent depending on pH and moisture, and has been known to remain active for up to three years in soil.

INCOMPATIBILITY: Conditions to avoid. Rapidly corrosive to brass @ 65 DEG C. Will corrode steel at a rate of .0001 in. of steel per month @ 65 DEG C.

HAZARDOUS DECOMPOSITION PRODUCTS: Mustard will hydrolize to form HCI and thiodiglycol.

HAZARDOUS POLYMERIZATION: Will not occur.

Section VII: Spill, Leak and Disposal Procedures

STEPS TO BE TAKEN IN CASE MATERIAL IS RELEASED OR SPILLED: Only personnel in full protective clothing (see Section 8) will remain in area where mustard is spilled.

RECOMMENDED FIELD PROCEDURES:

The mustard should be contained using vermiculite, diatomaceious earth, clay or fine sand and neutralized as soon as possible using copious amounts of 5.25 percent Sodium Hypochlorite solution.

Scoop all material and place in an approved DOT container. Cover the contents of the drum with decontaminating solution as above. The exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Dispose of the material used to decontaminate exterior of drum IAW Federal, state and local regulations. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limit (see Sections 2 and 8).

If 5.25 percent Sodium Hypochlorite solution is not available then the following decontaminants may be used instead and are listed in the order of preference: Calcium Hypochlorite

Decontamination Solution No. 2 (DS2), and Super Tropical Bleach Slurry (STB). WARNING: Pure, undiluted Calcium Hypochlorite (HTH) will burn on contact with liquid blister agent.

RECOMMENDED LABORATORY PROCEDURES:

A minimum of 65 grams of decon per gram of HD is allowed to agitate for a minimum of one hour. Agitation is not necessary following the first hour if a single phase is obtained. At the end of 24 hours, the resulting solution shall be adjusted to a pH between 10 and 11. Test for presence of active chlorine by use of acidic potassium iodide solution to give free iodine color. Place 3 ml of the decontaminate in a test tube. Add several crystals of Potassium Iodine and swirl to dissolve. Add 3 ml of 50 wt percent Sulfuric Acid water and swirl. IMMEDIATE Iodine color indicates the presence of active chlorine. If negative, add additional 5.25 percent Sodium Hypochlorite solution to the decontamination solution, wait two hours, then test again for active chlorine. Continue procedure until positive chlorine is given by solution.

A 10 wt percent Calcium hypochlorite (HTH) mixture may be substituted for Sodium Hypochlorite. Use 65 grams of decon per gram of HD and continue the test as described for Sodium Hypochlorite.

Scoop up all material and place in approved DOT containers. Cover the contents of the drum with decontaminating solution as above. The exterior of the drum shall be decontaminated and then labeled IAW EPA and DOT regulations. All leaking containers shall be overpacked with vermiculite placed between the interior and exterior containers. Decontaminate and label IAW EPA and DOT regulations. Dispose of the material IAW waste disposal methods provided below. Dispose of the material used to decontaminate exterior of drum IAW federal, state and local regulations. Conduct general area monitoring with an approved monitor (see Section 8) to confirm that the atmospheric concentrations do not exceed the airborne exposure limits (see Section 8).

NOTE: Surfaces contaminated with HD and then rinse decontaminated may evolve sufficient mustard vapor to produce a physiological response.

WASTE DISPOSAL METHOD:

All decontaminated material should be collected, contained and chemically decontaminated or thermally decomposed in an EPA approved incinerator, which will filter or scrub toxic by-products from effluent air before discharge to the atmosphere. Any contaminated protective clothing should be decontaminated using HTH or bleach and analyzed to assure it is free of detectable contamination (3X) level. The clothing should then be sealed in plastic bags inside properly labeled drums and held for shipment back to the DA issue point. Decontamination of waste or excess material shall be accomplished in accordance with the procedures outlined above with the following exception:

HD on laboratory glassware may be oxidized by its vigorous reaction with concentrated nitric acid.

Open pit burning or burying of HD or items containing or contaminated with HD in any quantity is prohibited.

NOTE: Some states define decontaminated surety material as a RCRA Hazardous Waste.

Section VIII: Special Protection Informaton

RESPIRATORY PROTECTION:

Concentration mg/m3	Respiratory Protection/Emsemble Required
Less than or equal to 0.003 as an 8-hr TWA	Protective mask not required provided that: (a) Continuous real-time monitoring (with alarm capability is conducted in the work area at the 0.003 mg/m3 level of detection. (b) M9, M17 or M40 mask is available and donned if ceiling concentrations exceed 0.003 mg/m3. (c) Exposure has been limited to the extent practicable by engineering controls (remote operations, ventilation, and process isolation) or work practices.
	If these conditions are not met then the following applies:
	Full facepiece, chemical canister, air purifying respirators. (The M9, M17 or M40 series or other certified equivalent masks are acceptable for this purpose in conjunction with the M3 toxicological agent protective (TAP) suit for dermal protection.)
Greater than 0.003 as an mil, 8 hr TWA may be used with prior approval from the AMC Field Safety Activity. Use time for the 30 mil DPE must be restricted to two hours or less.	The Demilitarization Protective Ensemble (DPE), 30

NOTE: When 30 mil DPE is not available the M9 or M40 series mask with Level A protective ensemble including impregnated innerwear can be used. However, use time shall be restricted to the extent operationally feasible, and may not exceed one hour.

As an additional precaution, the cuffs of the sleeves and the legs of the M3 suit shall be taped to the gloves and boots respectively to reduce aspiration.

VENTILATION:

Local Exhaust: Mandatory. Must be filtered or scrubbed.

Special: Chemical laboratory hoods shall have an average inward face velocity of 100 linear feet per minute (lfpm) plus or minus 10% with the velocity at any point not deviating from the average face velocity by more than 20%. Laboratory hoods shall be located such that cross-drafts do not exceed 20% of the inward face velocity. A visual performance test utilizing smoke-producing devices shall be performed in assessing the ability of the hood to contain agent HD.

Other: Recirculation of exhaust air from agent areas is prohibited. No connection between agent areas and other areas through ventilation system is permitted. Emergency backup power is necessary. Hoods should be tested semi-annually or after modificaton or maintenance operations. Operations should be performed 20 cm inside hoods.

PROTECTIVE GLOVES: MANDATORY. Butyl toxicological agent protective gloves (M3, M4, gloveset).

EYE PROTECTION: As a minimum, chemical goggles will be worn. For splash hazards use goggles and faceshield.

OTHER PROTECTIVE EQUIPMENT:

Full protective clothing will consist of the m3 butyl rubber suit with hood, M2A1 butyl boots, M3 gloves, impregnated underwear, M9 series mask and coveralls (if desired), or the Demilitarization Protective Ensemble (DPE). For general lab work. gloves and lab coat shall be worn with M9 or M17 mask readily available.

In addition, when handling contaminated lab animals, a daily clean smock, foot covers, and head covers are required.

MONITORING: Available monitoring equipment for agent HD is the M8/M9 Detector paper, blue bank tube, M256/M256A1 kits, bubbler. Depot Area Air Monitoring System (DAMMS), Automated Continuous Air Monitoring System (ACMS), CAM-M1, Hydrogen Flame

Photometric Emission Detector (HYFED), and the Minature Chemical Agent Monitor (MINICAM).

Section IX: Special Precautions

PRECAUTIONS TO BE TAKEN IN HANDLING AND STORING:

During handling, the "buddy" (two-man) system will be used. Containers should be periodically inspected for leaks either visually or using a detector kit, and prior to transfering the containers from storage to work areas. Stringent control over all personnel handling HD must be exercised. Chemical showers, eyewash stations, and personal cleanliness facilities must be provided. Each worker will wash their hands before meals and shower thoroughly with special attention given to hair, face, neck, and hands, using plenty of soap before leaving at the end of the workday. No smoking, eating, or drinking is permitted at the work site.

Decontaminating equipment shall be conveniently located. Exits must be designed to permit rapid evacuation. HD should be stored in containers made of glass for Research, Development, Test and Evaluation (RDTE) quantities or one-ton steel containers for large quantities. Agent shall be double-contained in liquid-tight containers when in storage.

OTHER PRECAUTIONS: For additional information, see AMC-R 385-131, "Safety Regulations for Chemical Agents H, HD, HT, GB, and VX" and USAEHA Technical Guide No. 173, "Occupational Health Guidelines for the Evaluation and Control of Occupational Exposure to Mustard Agents H, HD, and HT."

Section X: Transportation Data

PROPER SHIPPING NAME: Poisonous liquid, n.o.s.

DOT HAZARD CLASSIFICATION: Poison A

DOT LABEL: Poison gas

DOT MARKING: Poisonous liquid, n.o.s. (Sulfide, bis 2-chloroethyl) NA 1955

DOT PLACARD: POISON GAS

EMERGENCY ACCIDENT PRECAUTIONS AND PROCEDURES: See Section IV, and VIII.

PRECAUTIONS TO BE TAKEN IN TRANSPORTATION:

Motor vehicles will be placarded regardless of quantity. Driver shall be given full and complete information regarding shipment and conditions in case of emergency. AR 50-6 deals specifically with the shipment of chemical agents. Shipments of agent will be escorted in accordance with AR 740-32.

While the Chemical Research Development and Engineering Center, Department of the Army believes that the data contained herein are factual and the opinions expressed are those of qualified experts regarding the results of the tests conducted, the data are not to be taken as a warranty or representation for which the Department of the Army or Chemical Research Development Engineering Center assumes legal responsibility. They are offered solely for your consideration, investigation, and verification. Any use of these data and information must be determined by the user to be in accordance with applicable Federal, State, and local laws and regulations.

Addendum A: Additional Information for Thickened HD

TRADE NAME AND SYNONYMS: Thickened HD, THD

HAZARDOUS INGREDIENTS: K125 (acryloid copolymer, 5%) is used to thicken HD, K125 is not known to be hazardous except in a finely-divided, powder form.

PHYSICAL DATA: Essentially the same as HD except for viscosity. The viscosity of HV is between 1000 and 1200 centistokes @ 25 DEG C.

FIRE AND EXPLOSION DATA: Same as HD.

HEALTH HAZARD DATA: Same as HD except for skin contact. For skin contact, don respiratory protective mask and remove contaminated clothing IMMEDIATELY. IMMEDIATELY scrape the HV from the skin surface, then wash the contaminated surface with acetone. Seek medical attention IMMEDIATELY.

SPILL, LEAK, AND DISPOSAL PROCEDURES:

If spills or leaks of HV occur, follow the same procedures as those for HD, but dissolve the THD in acetone prior to introducing any decontaminating solution. Containment of THD is generally not necessary. Spilled THD can be carefuly scraped off the contamininated surface and placed in a fully removable head drum with a high density, polyethylene lining. The THD can then be decontaminated, after it has been dissolved in acetone, using the same procedures used for HD.

Contaminated surfaces should be treated with acetone, then decontaminated using the same procedures as those used for HD.

NOTE: Surfaces contaminated with THD or HD and then rinse-decontaminated may evolve sufficient mustard vapor to produce a physiological response.

SPECIAL PROTECTION INFORMATION: Same as HD.

SPECIAL PRECAUTIONS: Same as HD with the following addition. Handling the THD requires careful observation of the "stringers" (elastic, thread-like attachments) formed when the agents are transferred or dispensed. These stringers must be broken cleanly before moving the contaminating device or dispensing device to another location, or unwanted contamination of a working surface will result.

TRANSPORTATION DATA: Same as HD.
