CHEMICAL WEAPONS AND THEIR EFFECTS ON THE ENVIRONMENT

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February 1991

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Summary

The Gulf war has brought with it the spectre of chemical weapons. The chemical weapons Iraq is thought to posess include nerve agents such as tabun and sarin, the blood agent hydrogen cyanide and the vesicant mustard gas. Iraq has used these weapons against Iran in the Iran-Iraq war and the Kurdish ethic minority in Kurdistan. The USA also has chemical weapons including the more toxic nerve gas VX. Under the 1925 Geneva Convention the USA reserved the right to retaliate in kind to a chemical weapons attack.

Nerve agents paralyse the nervous system in milligram amounts. If inhaled humans have difficulty breathing, salivate, vomit and have diarrhoea plus muscle twitching before death by respiratory failure. Domestic and wild animals will show a similar spectrum of signs since the nerve agent attacks a physiological system common to all species from insects to mammals. Antidotes are available but need care in their administartion or side effects are seen.

Hydrogen cyanide kills by rendering the cells of the body unable to utilise oxygen. Nausea vomiting, difficulty breathing, unconsciousness and death can occur within minutes if exposure is high. Again mammals and birds are also susecpible to hydrogen cyanide as they share similar basic cellular mechanisms. Treatment is possible but only with care.

Mustard gas disrupts a variety of cellular functions. Contact with skin causes blisters which may not appear for days or hours. Inhalation causes respiatory tract damage and long term lung damage and cancers are seen. Other animals will be similarly affected.

This report assesses the atmospheric spread of chemical weapons and concludes that considerable human and animal fatalities could occur following either the deliberate use of these weapons or by damage to installations containing them. For instance, at half a kilometer downwind from the instantaneous release of 18kgs of a nerve gas, sarin, 50% of the unprotected population would be killed. This does not allow for the long term effects of such agents. Leakage of less than 5kgs of sarin into a stream would produce concentrations over the threshold needed to severely affect the biota 80km downstrean for over 24hrs.

Iraq is thought to have between 20-40,000kgs of chemical weapon agents stockpiled. Between 0.7 and 615kgs of nerve agent can be delivered in one projectile or bomb.

Also at risk from chemical weapons are the wild and domestic animals who are unable to either use protective measures or avoid a contaminated area. The terrestrial ecosystem, like the marine ecosystem, of the area is highly stressed. The addition of another pressure could have serious effects on the indigenous wildlife. Because the agricultural system of the area depends on domestic species such as sheep, goats and camels this report also concludes that the livelihood of the local people could be threatened.

Most of these effects would be exerted over a short time span of hours to days. However, deposition of agents on vegetation and the persistance in the environment of mustard gas in particular could lead to prolonged contamination of land, food and water supplies.

Introduction

The spectre of chemical weapons use in the current conflict in the Gulf arises from the known possession of such agents by Iraq. Retaliatory action, reserved as a possibility by signatories to the 1925 Geneva Convention is possible as large stockpiles are held by the US (Carnes, 1989a). Chemical weapons are highly effective and are relatively easy and cheap to make. Add to this the overwhelming problems in policing and enforcing compliance of a global ban, and it becomes clear why chemical agents are favoured as a weapon by smaller countries with no nuclear capability (Hubbard, 1990). Iraq is known to have used these weapons both in the Iran-Iraq war and against the Kurdish ethnic minority in Iraq and possibly also in Turkey.

Chemical weapons have been in existence since the First World War where they were first deployed, and subsequently they have been refined and developed both in potency and spectrum of activity. There are now agents designed to act directly against adversaries which are lethal in microgram quantities or which are highly debilitating and disorientating at low concentration. Other compounds are designed to act indirectly by, for example, defoliation of covering vegetation to expose an enemy. In both cases, chemical weapons carry a particular stigma in that their action is insidious, they are difficult to detect and they are indiscriminate in their effects (Perry Robinson 1982).

Considerable debate has centred around the ways in which a workable Chemical Weapons Convention aimed at ending the production and use of these weapons worldwide (see: Stock & Sutherland 1990). In part this has been prompted by a recent resurgence of interest in the tactical uses of such material by the military in the light of scientific advances (Perry Robinson 1982). The development of binary weapons in the US has seriously increased the problems of chemical weapon control. These weapons contain two relatively innocuous low toxicity compounds which are mixed to produce the toxic chemical in the projectile or tank delivery system en route to the target.

Although there has generally been somewhat less concern shown for the potential effects of these compounds on the wider environment, some evaluation of these has been made as a result of the public concern surrounding the US plans to destroy obsolete chemical weapons held in stockpiles in the continental US and at bases in Germany (Hindman 1989).

This document gives background information on chemical weapons, with emphasis on the types of chemical weapon which might be deployed in the Gulf conflict. These fall into four major categories: the aphyxiants, the blood gases, the vesicants and the nerve gases. The potential harmful effects upon exposed humans and upon the wider environment are examined following the deliberate use of these agents or their accidental release following an attack on production and storage facilities. In particular, attention is focussed on the environmental behaviour of the agents following release and upon the agricultural and natural ecosystems at risk.

The concerns are substantial and not merely idle speculation. In a recent UNEP report on Chemical Weapons and the Human Environment drawing on the expertise of a number of acknowledged experts in the field (Westing, 1990) it was concluded that:

"In the event of chemical warfare, the environment could be damaged, including both its floral and faunal components, again either inadvertently or by intent. This overall habitat (ecosystem) disruption could have catastrophic consequences, with ecological recovery taking years, decades or even centuries."

Background

1.Historical

Initial large scale deployment of chemical weapons took place in the First World War. The asphyxiants chlorine and phosgene (see below) were deployed at first, and although early results were somewhat haphazard (see Marshall 1987) the recognised tactical advantages led to the development of the mustard gases and by the end of the war, the potent arsenical vesicant "Lewisite". These agents were debilitating in effect rather than lethal. They were nonetheless responsible for a little over half a percent of the total US casualties in this conflict (Watson et al. 1989). Overall, between 1915 and 1918, over a million casualties were caused by chemicals and some ten percent of these were fatal (Murphy et al 1984). Despite the Geneva Protocol in 1925 (not in fact ratified by the US until 1975) banning the first use of chemical weapons, research into new agents and more sophisticated delivery systems was continued by Germany, the UK and the US (Murphy et al 1984).

The fledgling chemical industry of the early 1900s quickly found ways to adapt known compounds such as pesticides for weapons use and to develop progressively more toxic war gases. This research led in the late 1930s to the development of the organophosphate nerve agents. These originated from the same branch of organic chemistry as the familiar organophosphate pesticides such as Malathion (Gallo & Lawryk, 1991). Although in the Second World War, no official deployment of these weapons took place, there were accidents due to their transport (Spoehr, 1990). At the end of the conflict, large stockpiles were disposed of, generally by sea dumping. There were, however, several documented uses of other agents both before and after 1945.

In 1935-36 the Italians sprayed phosgene, tear and mustard gas over Ethiopia. During the Second World War, chemical weapons were used by the Japanese against the Chinese. Since 1945, allegations of chemical weapon use have been made against the USA in the Korean war and against Egyptian forces in the civil war in Yemen in 1967. In more recent years chemical weapons are alleged to have been used in Vietnam, Afghanistan, Kampuchea and Laos (Perry Robinson, 1982). Most recently, in the Middle East, allegations of use have been made against Iraq in the Iran-Iraq war and against the Kurdish people (Perry Robinson, 1986, Carus, 1989). Chemical weapons directed against trees and crops rather than people were used extensively by US forces in the Vietnam war.

2. Legislation

The Geneva Protocol, 1925 was designed to outlaw the use of chemical warfare. However it has several drawbacks which have resulted in its failure. For instance only first use of chemical agents is specifically prohibited and signatories can reserve the right to retaliate in kind. Approximately one third of all signatories have done this, including Iraq, the USA and the UK. This has resulted, unsurprisingly, in countries developing and stockpiling chemical weapons ostensibly for retaliatory use and using deterrence as a rationale (Perry Robinson, 1982). Since the development and stockpiling of chemical weapons is not specifically included in the Treaty new generation agents have been developed. In addition, the classes of agents covered by the Protocol have been argued not to include defoliants, anti-crop agents and anti-riot compounds by the USA with support from other countries including the UK (Murphy et al 1984).

Although the Geneva Protocol was followed by a Biological Weapons Convention in 1972, a Chemical Weapons Convention has yet to be agreed. Negotiations are dogged by problems of implementation and verification (see Working Party on Chemical and Biological Weapons,1988). Because many of the precursor chemicals are widely used in the

manufacture of other products and there is no free access to national production complexes, there are still many problems to be resolved (Timmerman, 1990).

3. Previous Middle East Use

The first reports of chemical weapons use in the Middle East occurred during the Iran-Iraq war. A UN team verified that mustard gas bombs and possibly tabun nerve gas bombs had been used in Iran-Iraq border areas during 1984 (Perry Robinson, 1986). More reports of the Iraqi use of chemical weapons came in 1985 with the same agents being used together with the blood gas hydrogen cyanide (Perry Robinson, 1986).

Between 1987 and 1989 it is thought that Iraq also used chemical weapons against the Kurdish people inside Iraq (e.g. in Halabja, Kurdistan) and possibly against refugees from this ethnic minority in Turkey (Ala'Aldeen et al, 1990). The chemicals used are thought to have included mustard gas, tabun and hydrogen cyanide. The Iranians claim that the Iraqis used phosgene against them over thirty times during their war (Carus, 1989). It is reported that the Iraqis have investigated the possibilities of producing VX (another nerve agent) but there is no evidence that they have ever produced it.

The precise nature and magnitude of the Iraqi stockpile is in some doubt and the evidence is conflicting. According to a report in the UKSunday Times of Jan 27 1990, Iraq first tried to manufacture chemicals in 1975 with an approach to the Pfaudler Company of Rochester, New York, which was asked to supply a factory for making pesticides capable of manufacturing 1200 tons of material a year. Iraq unsuccessfully tried to procure similar equipment from Britain's Imperial Chemical Industries but was turned down. West Germany, Switzerland, the Netherlands, Belgium and Italy, however actually supplied equipment. It seems that the most important of these suppliers was West Germany where the industrial company Karl Kolb sold a pilot plant which with modifications was capable of making Sarin.

Iraq also bought technical assistance from Fritz Werner; heavy duty pumps and chemicals from Water Engineering Trading of Hamburg which sold $11m of chemicals including trichloride, a base used in the manufacture of nerve gas; and another company, Quast provided reactor vessels and centrifuges.

A series of setbacks in the war with Iran led the Iraqi leadership to consider that chemical weapons might provide a useful deterrent on the battlefield. Using the state-owned Enterprise for Pesticide Production as a front, the Iraqis began buying large quantities of chemicals in Europe and the United States. The most important was thiodiglycol which can be used in photographic developing and printing but is also a key component of mustard gas. In 1983, Phillips Petroleum in Tessenderlo, Belgium allowed a subsidiary to ship 500 tons of thiodiglycol to Iraq but became suspicious when a second order for 500 tons was placed a year later. By then Iraq was producing Mustard gas, which was used in the war with Iran and delivered by artillery, fighters and helicopters. Large numbers of western nations are implicated in the transfer of this dubious technology to Iraq (Timmerman, 1991).

Following the decision to start manufacture in the 1970s, mustard, Tabun & Sarin were in production by 1985 (Carus, 1989). At Salman Pak a research facility was constructed, while at Samarra, 62 miles north of Baghdad a 10 square mile facility was built to produce mustard gas, Sarin and Tabun and other complexes were built at Karbala and Fallujah. Estimated production figures are given below.

Agent (mode of action)	Production
Mustard gas (vesicant)	60 ton/month in 1986 (Carus, 1989)
Sarin (nerve agent)	4 ton/month in 1986 Possibly as high as 2,000 t/yr now (Carus, 1989)
Tabun (nerve agent)	4 ton/month in 1986 Possibly as high as 2,000t/yr now (Carus, 1989)

Total production capability is thought to have been around 700 tons/yr (in 1986) though unconfirmed estimates of between 3000 and 13200 tn/yr have been made. Iraq needs precursor for all these agents to be imported but is thought to be very close to independent mustard gas production (Carus 1989). A total stockpile of 2,000-4,000 tons of the three agents is thought to exist. Intelligence reports suggest that 90% of Iraqs chemical weapon arsenal consists of Mustard agent.

Properties of Chemical Weapons

Chemical weapons intended to act against personnel are usually classified as asphyxiants, blood gases, vesicants or nerve agents according to the effects they produce (see Murphy et al, 1984; Harris & Paxman, 1983). Other categories such as defoliants, other herbicides, riot control compounds and the psychotrpic agents, although important, are not considered here.

The medical effects of chemical agents in man have been summarised recently (Watson et al. 1989; Anon, 1991).

1.Asphyxiants

These were amongst the earliest chemical agents to be used on the battlefield (Marshall 1987).The best known asphyxiants are chlorine gas and phosgene. Chlorine gas was the first chemical weapon to be used in WWI. It is an intensely irritating greenish yellow gas which attacks the lungs leading to copious secretion of fluid and thickening of the blood. In the attack of 22nd April 1915 some 168 tonnes of chlorine were released in a light, steady wind along 7km of front - 20% of the 27,000 troops affected died. Phosgene was also extensively used in WWI. It is more toxic than chlorine and exerts its effects in a similar way except that there is frequently a latent period between exposure and onset of toxic symptoms. Death, as with chlorine, is caused by drowning in the fluid secreted by the blood into the lung.

SYMPTOMS OF POISONING

The common asphyxiant gases, chlorine and phosgene, as indicated above, exert their effect by irritation of the lung tissue, resulting in death by drowning. Sub-acute exposure can permanently impair lung function leading to an empyhsemic condition. Later infection may set in.

2. Blood gases

Blood gases are compounds which are absorbed through the lungs or skin and act systemically. Hydrogen cyanide, the compound possibly used by Iraq in the past (Perry Robinson, 1986), acts by impairing the ability of the body to utilise oxygen by interrupting the cellular respiratory chain. The oral LD_{50}[1] is 3.7mg/kg while the inhalation LC_{50}[2] is 120 ppm in air breathed for 60 minutes. It is relatively non-persistent (Marshall, 1987).

SYMPTOMS OF POISONING

The effects of hydrogen cyanide are related directly to the amount inhaled and its effects on the cytochrome oxidase component of the respiratory chain. Nausea, vomiting, difficulty in breathing and unconsciousness followed by death are noted with increasing exposure. Death can be extremely rapid if the inhaled dose is high. A specific antidote is available, but this must be administered soon after exposure to be effecetive. Hydrogen cyanide affects other species in the same way.

3.Vesicants

Mustard gases and Lewisite are the best known examples of the vesicant or blister agents (Murphy et al. 1984). Mustard agents are either sulphur or nitrogen based compounds. The nitrogen mustards are more persistent and exert greater toxic effects on the immune system than sulphur mustards. They are between 10 and 500 times less toxic than the nerve gases with a skin LD_{50} of approximately 100mg/kg body weight. The incapacitating dose is considered to be between 12-70 mg-min/cubic metre for sulphur mustards. Exposure to 140 mg/cubic metre of sulphur mustard agent for ten minutes is fatal while 4 mg/kg body weight applied to the skin also causes death (Watson et al. 1989).

Mustard agents are oil-soluble liquids, which are colourless in the pure state and have a slight smell of mustard. They will be liquid at most daytime ambient temperatures experienced in the Gulf region, but at night may freeze, thus delaying dispersion for some time (Freezing points are between 1 &14 ^{o}C). Delivery systems range from artillery and mortar shells to spray tanks affixed to aircraft (Carnes 1989).

Cells exposed to mustard gas are destroyed by the reaction of the chemical with cellular proteins, enzymes and nucleic acid. Mustards can cause mutations in cells which is thought to account for the increased incidence of cancer in people exposed. Since the white cells of the body are particularly sensitive to mustard gas effects, the immune system of the body is severely compromised and life threatening infections can be a secondary consequence (Watson et al 1989).

Lewisite is another vesicant, but exerts its effect by fatally altering critical cellular enzyme systems. There is no latency period with lewisite, which causes immediate excruciating pain on contact with the skin and the eyes. It is also a systemic poison (liver and kidneys being the target organs) and can cause a slowly growing non-fatal form of skin cancer. The effective dose is estimated at 95 microgrammes per man. Severe systemic effects are estimated to be caused by 13 mg/kg body weight. Death can result from the inhalation of a concentration of 48

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[1] LD_{50} is the dose of a compound required to cause death in 50% of an exposed population

[2] LC_{50} is the lethal concentration required to kill 50% of a population

mg/cubic metre of air for just 30 minutes (Goldman & Dacre, 1989).

SYMPTOMS OF POISONING

a) Humans:

Mustard gas effects are related to its disruption of cellular functions (see above). These agents can act through contact with the skin or the respiratory tract. Following exposure to sulphur muistard agent there is usually a latency period of several hours before signs of toxicity begin to appear. Eye inflammation occurs at lower doses than any other effect. With increasing dose skin irritation occurs with the formation of blisters and irritation of the respiratory tract. Recovery from these toxic effects can take days or weeks. Special care is neccessary to prevent subsequent infection setting in.

Effects can progress to severe lung damage and impaired immune function following bone marrow suppression. Secondary pneumonia and septicaemia can be difficult to treat and require specialised medical and nursing care.

The mustard agents can also produce effects that appear years after exposure. Apparent healing of eye damage can be followed by permanent vision impairment. Respiratory tract damage can lead to chronic bronchitis. Other long term effects include increased incidence of tumours of the respiratory tract and of the skin.

As noted above, Lewisite does not show a latency period, results in severe pain on contact and can act as a systemic poison with the liver and kidneys being the target organs. No specific antidotes are available for the mustards where cell damage following exposure is inevitable. In the case of Lewisite, protection from effects can be gained up to an hour after exposure by intravenous or intramuscular injection of 2,3 dimercaptopropanol, known as British Anti-Lewisite.

b): Other Species

Other species are similarly susceptible to the effects of mustard gas. Although fur and plumage may offer some protection from skin contact, grooming behaviour will increase contact with the oral mucous membranes.

4. Nerve agents

Tabun was first developed in Germany in the 1930s. By the end of the Second World War, Sarin and Soman - both more toxic than Tabun - had been developed following research into organophosphorus insecticides (Murphy et al, 1984). The most toxic and persistent of the nerve agents, VX, was selected for military mass production from a group of compounds developed jointly by the US and the UK after 1945 (Murphy et al 1984).

The major agents likely to be used in the Gulf conflict, then, are Tabun, Sarin, Soman and VX. All are liquid at normal temperatures and can be delivered as the warhead of a weapons system or sprayed from tanks. Variants of the basic formulae of the agents also exist. Weapon payload of examples from the US stockpile ranges from around 0.7 kilos in the M360 105mm projectile to 157 kilos in the MK-116 Weteye bomb to 615 kilos in the TMU-28/B spray tank (Carnes, 1989b). It can be assumed that the Iraqi forces have similarly configured chemical weapons.

The nerve gases are extremely toxic. The human skin LD50 of VX agent is 0.04 mg/kg body weight. Effective inhalation doses range from 45 mg-min/cubic metre for VX to 100 mg-min/cubic metre for Sarin. In other words an ambient concentration in the air of 100mg per

cubic metre of Sarin, breathed for one minute can cause death (Watson et al. 1989).

The nerve agents act by causing an irreversible inhibition of the enzyme acetylcholinesterase. This enzyme is found at nerve endings where it breaks down the chemical (acetylcholine) released at those nerve terminals. Normally acetylcholine transmits messages between certain nerve cells or between nerves and muscles. When acetylcholine is not removed over stimulation of the nerve or muscle takes place until a response can no longer be sustained (Taylor, 1980). Acetylcholine is a neurotransmitter in the parasympathetic nervous system, at the motor end plate and at some central nervous system synapses. Therefore symptoms involve peripheral nervous tissues such as those serving the eye, intestine and the general muscular system and the central nervous system comprising the brain and spinal cord. In addition, other effects occur (non-cholinergic effects) which are not apparently related to interference with acetylcholinesterase are seen.

SYMPTOMS OF POISONING

The clinical effects in man and other animals parallel the inhibition of acetylcholinesterase and are related to the dose and route of contact. Nerve agents can cause poisoning following inhalation, ingestion or skin contact. Tabun is somewhat less effective in terms of incapacitating dose than Sarin by vapour inhalation but is equalyy toxic by skin absorbtion when applied as a liquid. VX is highly effective through skin penetration and is many times more toxic by this route than Sarin. Head and neck areas in man are very sensitive. The effective lethal dose by skin absorbtion decreases with increasing wind speed (Watson et al. 1989).

a) Humans

Miosis (pupillary constriction) and impaired vision may be the only signs of low dose through inhalation. However as the dose increases systemic signs are also seen. There will be difficulty breathing, vomiting and diarrhoea, salivation, muscle weakness and twitching. There may also be headache, anxiety, confusion and convulsions ending in coma. Death results from respiratory failure as bronchial secretions accumulate in the lungs, respiratory muscles become paralysed and the central respiratory centre is affected. If large amounts are inhaled loss of consciousness and death can occur within minutes.

Droplets of nerve agents contacting exposed skin may cause purely local signs with sweating and muscle twitching or, if one of the more potent agents is used or contact is high, the systemic signs given above become evident. Clinical signs may be delayed for several hours. Ingestion leads to signs associated the gastrointestinal tract initially and then signs progress as above if doses are high. The onset of signs is slower than for inhalation but quicker than skin exposure.

Self administered treatment with atropine and other oxime compounds is available but this is not without risks since proper dosage is somewhat empirical. Antidotes must be administered within minutes of exposure. Even with their use, artificial respiration and anticonvulsant drugs may be required together with specialised nursing. If these are used to protect from acute lethality, a delayed neuropathy syndrome may occur. Since atropine is a hallucinogen and may potentially be abused, it is not thought desirable to distribute it to the general public. Changes in brain wave (EEG) patterns may occur but the long term significance of these is unknown. Carcinogenic properties may exist but this is not certain. Other delayed effects of nerve agents have also been reported and are manifested as diverse neurological signs such as weakness, depression and poor memory. Delayed neurotoxicity is also a feature of poisoning with other, pesticidal, organophosphorus compounds (Barrett & Oehme, 1985) and the mechanism of toxicity may be similar.

b) Other Species.

Other species, including domestic and wild animals, are susceptible to the toxic effects of acetylcholinesterase inhibitors and the clinical effects of poisoning with organophosphate pesticides are well recognised in domestic species (Mayer, 1990). Since nerve agents are of the same class of chemical it is likley that the same effects will be seen. Delayed effects of organophosphorus compounds are also reported in domestic species (e.g.Jortner et al, 1983).

Chemical Agents in the Environment

Chemical weapons may enter the environment through their deliberate release or following an attack on chemical weapons factory or weapon store. The impact on the environment will depend on a variety of factors including the compound involved, the scale of the release, the area involved and the weather conditions at the time.

1. Dispersion in the environment

i) Atmospheric spread

Depending on the agent involved the initial release would result in a cloud of chemical vapour or aerosol which would be carried by the prevailing wind. The cloud would gradually increase in size and decrease in concentration in relation to the weather conditions (Westing, 1990; Miller & Kornegay 1989). Droplets would also tend to settle on ground, water and buildings and possibly be washed into the ground with any rain fall.

In the Gulf conditions where average temperatures in February in Kuwait are 11-18^{o}C and 6-18^{o} C in Iraq (Pearce & Smith, 1990) settled agent may evaporate again (revolatalise), particularly during the day. Where agent has settled revolatilisation will reduce the persistence of the agents but increase their acute toxicity. As average temperatures increase during the summer this effect would increase.

Of the nerve agents that may beused in the Gulf, VX is the least volatile so would be expected to settle but to revolatilise at a slower rate than tabun or sarin. Mustard gas is also one of the most persistent agents and can remain potent in the field for several weeks (Westing, 1990). Hydrogen cyanide, however, is likely to rapidly disperse and not to pose a long term threat of ground or water contamination.

Table 1 shows the human fatalities at given distances downwind of a release of the nerve gases, sarin or VX, and mustard gas which may be either instantaneus (as in deliberate use or major accidentat an installation), semicontinuously (from installation damage or continuous deliberate use), and from spill evaporation (following installation damage and weapon leakage) (Miller & Kornegay, 1989). These have been calculated from air dispersion models and share the shortcomings of these. For instance, they do not account for variable topography, changes in wind direction and other spatial changes in atmospheric conditions. It assumes the agent would be dispersed as an elliptical plume . Therefore these figures must be treated with some caution, although such models are considered to be underestimates of the true situation. The modelling results do indicate that if a weapons manufacturing plant were destroyed without a fire taking hold, then the effect on the local human and animal population would depend upon agent density (VX is much denser, and also more toxic, than Sarin), the amount released and whether release is instantaneous, semicontinuous or evaporative.

What is clear, however, is that relatively small amounts of these nerve agents have the capacity to cause death on a widespread scale in an unprotected population and will

inevitably also affect animal life. It should also be noted that this is only a representation of lethal effects, sub-acute or chronic effects will be superimposed on this. No effect distances (assuming a no effect concentration of $10^{-9}mg/m^3$ for both VX andSarin) are some seven times greater than the no death distances.

To put the quantities of gas in context Perry Robinson (1982) has estimated, using hypothetical Soviet chemical weapons, that a 120mm Mortar could deliver a maximum of 200Kgs of nerve gas, 350kgs of mustard gas or 125kgs of hydrogen cyanide in one minute. Carus (1989) has outlined the possible scale of the chemical weapons arsenal in Iraq (See above) and suggests that up to 4,000 tons (approximately 40,000kgs) of chemical weapon agents have been produced and stockpiled at various sites in Iraq.

TABLE 1 The amount of VX , Sarin or Mustard gas (in kgs) which will result in 50%, 1% and 0% human fatalities at distances of 0.5, 2.0 and 10.0km downwind of a release (from Miller & Kornegay, 1989).

	Downwind distance (km)	**AMOUNT RELEASED (kg) to cause**		
		50%	**1%**	**0%**
			fatalities	
SARIN				
i) Instant release	0.5	18	3	2
	2.0	320	45	27
	10.0	9,000	1,600	900
ii) Semicontinuous	0.5	90	14	7
release - 60 min	2.0	1,100	180	90
	10.0	36,400	7,000	3,000
iii) Spill evaporation	0.5	45	4	2.7
- 60 min	2.0	650	70	36
	10.0	2,300	400	230
VX				
Semicontinuous	0.5	4	0.7	0.4
release	2.0	39	7.0	3.6
	10.0	700	90	70
MUSTARD GAS				
Spill evaporation	0.5	13,600	1,400	900
- 60 min.	2.0	180,000	18,000	13,600
	10.0	-	-	270,000

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ii) Water impacts.

Chemical weapons agents may enter the water supply through direct settlement of agent on water, through runoff from contaminated land or from direct introduction into the watercourse. In all three cases the effects of the weapons may then be exerted in areas well away from the immediate impact zone predicted by simple atmospheric modelling. (Tolbert and Breck 1989). The polluted water may then become a source of danger to those animals and humans using it for drinking or other purposes. The potential impacts may be considerable. It has been estimated that aquatic resources in streams up to a medium size

could be severely affected by spills of around 5kg of nerve agent, over most of their length. Overall, the impact wll depend upon the behaviour of the agent in the wider environment.

2. Degradation in the environment

Chemical weapon agents may be broken down by chemical reactions or by bacterial decomposition or by photodecomposition. Use of decontaminating agents may also produce a spectrum of reaction products of environmental significance (Watson et al. 1989). While not all possible products of these diverse reactions have been fully evaluated, none of the known breakdown products are as acutely toxic as the parent compounds.

i) Degradation in water

Most chemical weapon agents are subject to hydrolysis which eventually results in the production of less toxic compounds. Some intermediary compounds may also be toxic although by orders of magnitude less than the parent compound (Westing, 1990). The rate of hydrolysis will vary for the chemical involved (Trapp,1985). With Sarin the rate of hydrolysis is related to the pH and temperature in a predictable way, being more rapid at higher temperature and under more alkaline conditions. The half life of sarin in water of pH 7.0 at 25°C is 75 hours which is reduced by a factor of approximately 10 in salinewater (Trapp, 1985). VX agent is much more stable and only poorly soluble in water. Therefore half life data are much more difficult to interpret with any confidence. The solubilty and degradation of VX that may have adsorbed to sediments is difficult to calculate. However, under laboratory conditions the half life of VX in water of pH 7.0 at 25°C will be in the order of months (Trapp, 1985). Tabun is the least persistent of the nerve agents and will rapidly hydrolyse in water. The hydrolytic half-lives can be misleading when considering impacts on aquatic resources. Modelling, using the known properties of Sarin spilt into a 6th order stream with a flow rate of 51 metres^3 per second suggests that 4.54 kgs will result in a downstream concentration of 0.1mg/l 80 km downstream 27.1 hours after the event. This would severely affect the stream biota (Breck 1990).

The sulphur mustards hydrolyse more rapidly than nitrogen mustards. A half life of 8.5 minutes in distilled water (pH 7.0) at 25°C has been given for sulphur mustards but this must be treated with extreme caution. Mustard agents hydrolyse rapidly in water if they are dissolved but do not in fact dissolve readily and will tend to form a surface slick. The persistence of this will then depend upon wind and wave action. VX agent and some mustards are more dense than water and could form globules which sink to the bottom or are transported downstream. If mustard was spilled into cold water, it would solidify and sink to the bottom. These globules have the potential to persist for between several months and several years and there is little information on toxic action involving contact with slicks or globules (Tolbert & Breck ,1989). It is also worth bearing in mind that leaking containers of mustard gas dumped in the Baltic Sea in 1945 are still dangerous (Trapp, 1985).

ii) Degradation in soils.

Again it is difficult to give accurate figures for breakdown rates as these will depend on the physical charateristics of the soil itself, the weather and the agent involved. Trapp (1985) quotes figures for the duration of hazard on bare soil with low vegetation of 3-4 days for Mustard gas and 32 days for V/G agent (similar to VX). However it must be remembered that the fauna of the area will be unable to avoid the area during such a hazard period. Physico-chemical process will also affect the behaviour of the agent. For instance, rainfall will wash out the chemical but this may then enter the water course or agents may be bound to soil particles which will render them inert in the short term but act as a long term reservoir.

iii) Microorganisms in soil or water.

Microorganisms may also be responsible for the breakdown of some of the chemical agent. This component has not been quantified in any detail (Trapp, 1985; Westing, 1990)

iv) Decontamination

Human decontamination procedures for compounds such as the mustards using chlorinated limes ("supertropical bleaches" have beendeveloped for military use) may be as toxic to the environment and the user as the original compound (Trapp, 1985). In addition their use may result in the formation of compounds such as chloroform, which are of environmental significance.

3. Combustion products.

Following a fire in a chemical munitions factory or store some of the chemical agents themselves will be released to the environment togther with the precursor chemicals and toxic products of combustion. These may be dispersed over considerable distances. If fire took hold of an attacked facility, several toxic gases could be generated. These include hydrogen cyanide and nitrogen dioxide from Tabun, hydrogen fluoride from sarin, sulphur dioxide from VX, hydrogen chloride from sulphur mustard and chlorine from Lewisite (Watson et al. 1989). These gases would be of immediate local concern while others such as the polycyclic aromatic hydrocarbons (PAHs) and polychlorinated dibenzodioxins (PCDDs)/ polychlorinated dibenzofurans (PCDFs) would be of concern in the longer term.

Ecosystem Impacts - the resources at risk

Public perception of the countries of Iraq, Kuwait and Saudi Arabia is often of non-productive areas of desert. However, as McKinnon (1990) so graphically demonstrates in his book "Arabia, Sand, Sea,Sky" this is very far from the truth. As a meeting point between Asia and Africa the region has a diverse flora and fauna with elements from each, some of which have subsequently evolved to become unique to that area.

WILDLIFE

Mammals found in the region include species as diverse as the Arabian sand gazelle (*Gazella subgutturosa marica*), the long eared hedgehog (*Hemiechinus auritus*) in Kuwait, the Euphrates jerboa (*Allactaga euphratica*), and the Indian gerbil or antelope rat (*Tatera indica*) to name but a few.

In the spring many of birds of prey pass over Kuwait during their migration including the steppe eagle (*Aquila rapax*), booted eagles (*Hierae tuspennatus*), Bonelli's' eagle (*H. sciatus*), spotted eagle (*Aqui laclanga*) and Imperial eagles (*Aquila helica*). There are many other birds which live in the region and depend upon it for more than just a safe migration route. These include the Houbara bustard (*Chlamydotis undulata*), Crowned sandgrouse (*Pterocles coronatus*) and Marsh Harrier (*Cirus aeruginosus*) (Heinzel et al, 1979).

As well as birds and mammals there are large numbers of insects and reptiles which all play their part in the finely tuned ecosystem of the Gulf. The organophosphate nerve agents, originally developed for their insecticidal properties, will inevitably affect this group of animals.

AGRICULTURE

Although large parts of the Middle East are not used for agricultural purposes there is still a basic dependence on settled farming systems and animal herding/pastoralism (Beaumont et al. 1988). Since defoliants are unlikely to be used in the Gulf war and none of the chemicals to be used have direct effects on vegetation little direct impact on crops might be expected. However, following the deposition of chemical agent on vegetation, this may then be ingested by humans or animals and cause poisoning (Sigal & Suter, 1989). Toxic effects might also be expected to result from agent uptake from surface and soil contamination. Long term effects on the ecosystem overall might be expected to result from decimation of the particularly sensitive insect component.

Guidelines issued issued by US regulatory authorities specify that all forage crops, grains and garden produce be quarantined until tested, and the public provided with alterantive sources of water and food. In practice in the Middle East, facilities for such testing and provision are unlikely to be widely available. Crops and arable areas would be unusable for a year or more depending upon the agent involved. GB is volatile and disperses relatively readily while VX is less volatile and much more persistent. The mustards can remain active in the environment for years and can adsorb onto soils and vegetation. This is why they are recommended by military strategists as "terrain denial" material.

The agricultural systems most at risk are those which depend on animals, these include pastoral systems, animal herding on a less extensive scale and confined animal systems. The domestic animals kept in these systems include the camel (Camelus dromedarius) sheep, goats and cattle. Table 2 shows the numbers of these species kept in Iraq.

TABLE 2. Estimated numbers of animals kept in Iraq in 1985 (from Beaumont et al, 1988)

('000s of animals)

Sheep	Goats	Camels	Buffaloes	Donkeys	Horses
8,500	2,350	55	155	450	50

All these species of animals are at risk from the chemical weapons agents that may be released in the Gulf conflict. They may inhale aerosol, ingest contaminated foliage, ingest chemical during grooming of contaminated coat, or may absorb material through hairless parts of their body. Modelling predicts (Sigal & Suter, 1989) that animal deaths would be expected outside a no effect zone for humans due to the contamination of forage crops.

On the basis of ingestion toxicity, cattle and sheep are most sensitive to VX, rats and rabbits the least sensitive. There is evidence that chemical agents and their breakdown products are accumulated by plants and deaths in grazing animals could occur for a considerable time after the event. Although it is considered that the consumption of meat and dairy produce from animals exposed but not killed by the nerve agents should be safe, this is not certain. Animals killed by nerve agents would be unfit for consumption, those acutely affected would have to be destroyed. Precautions to prevent residual contamination problems would need to be carried out for all animals slaughtered for consumption from an area affected by a release. It would be necessary to quarantine all forage and grains until proved safe by testing. Birds and insects may be particularly sensitive to the effects of chemical weapons (Sigal & Suter, 1989)

If losses were on a large scale the communities which depend on such agricultural systems for their food and livelihood will be put at increased risk in both the short and the long term.

Finally, another factor needing consideration is the decontamination of buildings and personal effects. Depending on the extent of the attack and the agent used, the inability to prevent public access to contaminated areas will result in civilian populations being inadequately prrotected. Many of the decontamination methods may prove destructive to many common building materials since they involve heating to temperatures of 1000 ◦ C. Hence buildings contaminated with the persistent mustards or VX agent may have to be subject to indefinite access restrictions. In practice these effects are likely to be exacerbated by the unavailability of decontaminating agents to the civilian population. The input of resources would need to be considerable to even begin to partially address the problem (Trapp, 1985).

THE OVERALL EFFECT

It is clear that the risk of chemical weapons to the wider environment cannot be directly quantified. What is certain, however, is that as highly potent toxins designed to kill and incapacitate, some considerable environmental damage is likely to result from their deployment or accidental release. However, it is possible to predict where some of the dangers to the environment lie. Although the nerve agents, sarin and tabun, and the blood gas hydrogen cyanide would rapidly disperse in the hot climate of the Gulf, their short term lethal effects would be exerted on wild animals, domestic animals and humans alike. In the case of natural systems severely impacted, it is possible that the original ecological balance would never be restored in its original form.

Mustard gas, because of its chemical properties is likely to persist in the environment for much longer periods. It may pose particular threats if water courses become contaminated. Like the other agents both short and long term effects would be exerted upon all animal life.

The animal population of the Gulf states has an added disadvantage, not only does it have no protection against such agents,it cannot choose to avoid contaminated areas. The evolution of the ecosystems in the area has resulted in delicate ecological structures which are already under threat from human interference (McKinnon,1990).

Domestic animals are also threatened which may affect the livelihoodand diet of the local people from weapons which are highly indiscriminate in their lethal effects The environmental effects of the chemical weapons used during the Viet Nam war have been profound for flora, fauna and the human population (Westing,1977; 1982) particularly in terms of the highly persistent residues resulting from the use of Agent Orange.

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