NERVE AGENTS, PESTICIDES, AND CHOLINESTERASE INHIBITION

by John S. Nordin, Ph.D.

You have heard it in the news media. Iraq is believed to possess chemical warfare agents, in particular, nerve agents such as the deadly VX and Sarin. There is no question that Iraq has used chemical warfare agents in the past, but samples of suspicious material collected by the U.S. military in Iraq have shown to be various pesticides. Are the chemical warfare agents hidden some place, or carried out of Iraq, or were (as the old Iraq regime claimed) the agents destroyed? And how secure are those stockpiles of nerve agents stored in the former Soviet Union?

The technologies and equipment used to manufacture pesticides are similar to those used to manufacture nerve gases. Some pesticides act by inhibiting the enzyme cholinesterase. Some chemical warfare agents (i.e. the nerve gases or agents) also act by inhibiting cholinesterase. Cholinesterase is an enzyme found in both humans, other animals, and insects. The enzyme is important in the functioning of the nervous system. There are differences between human and insect nerve transmission, and a goal in pesticide development is targeting the insect pests while leaving non-targeted species alone. Sarin was originally developed in Germany as a powerful pesticide for killing aphids. Pesticides on the market today are quite safe when used as directed, but workers who are in contact with certain pesticides should have their cholinesterase levels periodically checked. Deaths from pesticide poisoning still occur.

Let us take a look at the subject of cholinesterase inhibition and those chemicals that cause cholinesterase inhibition.

What is Cholinesterase Inhibition?

Let's take a look inside the human body. The human body, as well as other animals, contain electrical switching centers called 'synapses'. The body manufactures a chemical called 'acetylcholine' which turns on the switches and another enzyme called 'acetylcholinesterase' which breaks down the acetylcholine and turns off the switches. All this happens very fast. This is how the brain signals information throughout the body, to control respiration, muscle action, digestion, and other life functions.

Certain chemicals can throw this out of balance. A cholinesterase inhibiting chemical (nerve agents and some pesticides) interferes with the enzyme that breaks down the acetylcholine and excessive acetylcholine builds up at the synapses. There is nothing to switch off the synapses as acetylcholine builds up. Electrical impulses fire away continuously. Repeated and unchecked firing of electrical signals causes uncontrolled and rapid twitching of muscles, paralyzed breathing, convulsions, and in extreme cases, death.

Any chemical that can bind, or inhibit, cholinesterase (e.g. acetylcholinesterase) making it unable to breakdown acetylcholine is called a "cholinesterase inhibitor", or an "anticholinesterase agent". The nerve agents (chemical warfare agents) are the most

potent. Certain pesticides can also show some degree of cholinesterase inhibition. The pesticides that can result in cholinesterase inhibition fall into broad classifications of either (1) organophosphates or organophorphorous pesticides, (2) carbamate pesticides, or (3) pesticides based on chlorinated derivatives of nicotine. There are also many pesticides on the market that do not inhibit cholinesterase.

The offending chemical can be ingested, absorbed through the skin or eyes, or inhaled. The amount of chemical required to kill a human being can be as little as one drop of agent VX applied on the skin. On the other hand, some of the pesticides, which possess cholinesterase inhibition are of low enough toxicity that it would be difficult for a person to poison himself. .

Symptoms of Cholinesterase Inhibitation

1. Mild Poisoning: Tiredness, weakness, dizziness, nausea, and blurred vision. Symptoms appear usually within 4 to 24 hours of exposure in the case of pesticides, sooner in the case of toxic nerve agents.

2. Moderate Poisoning: Headache, sweating, tearing, drooling, vomiting, tunnel vision, and twitching. Symptoms appear usually within 4 to 24 hours of exposure in the case of pesticides, sooner in the case of nerve agents.

3. Severe Poisoning (a single large dose or repeated smaller dosages): Abdominal cramps, involuntary urination and/or defecation, muscular tremors, staggering gait, pinpoint pupils, hypotension (drop in blood pressure), slow heartbeat, difficulty breathing, possible convulsions, possible coma, and possible death. Symptoms can appear within seconds (by inhalation) or minutes (skin contact) in the case of lethal doses of nerve agents.

Some chemicals may also irritate the lining of the nose and respiratory tract, and in severe cases, result in pulmonary edema (filling of the lungs with fluid).

Some of the symptoms of cholinesterase inhibition can be confused with influenza (flu), gastroenteritis, pneumonia, heat prostration, alcohol intoxication, drug overdose, exhaustion, hypoglycemia (low blood sugar), asthma, or a brain hemorrhage, or even a heart attack.

First responder and law enforcement officers responding to the incident need to piece together quickly the circumstances. Is only one person affected or do many people display symptoms? Are dead animals or birds or insects present? Is there a chemical nearby that might indicate poisoning? Does the person carry medications or have a diabetic bracelet? Has the person been doing heavy labor? Does the person feel hot? This information needs to be conveyed to medical personnel.

Jane's Chem-Bio Handbook (published by Jane's Information Group, Alexandria Va, 1998) singles out three symptoms for first responders to look for in case of a mass nerve gas vapor exposure. These are (1) small pupils (miosis), (2) runny nose (rhinorrhea), and (3) shortness of breath. The pupils remain small even in dim light. Some casualties may have two of the effects, some will have only small pupils, some will have all three. A large concentration of vapor may also cause loss of consciousness and convulsions. If the vapor concentration is large enough, the symptoms may become apparent within a few seconds after inhalation.

If a nerve agent liquid contacts the skin, symptoms may appear within a few minutes after contact to about 18 hours depending upon the agent and amount. Only one drop (10 mg) of the agent VX on the skin can kill. Initial symptoms include sweating and muscular twitching (fasciculations) at the site of the drop. Later symptoms (after several hours) may include nausea and vomiting, especially if the amount is larger. A lethal drop will, within minutes of contract, cause loss of consciousness, convulsions, cessation of respiration, and paralysis.

Treatment

The person should be transported to the hospital, poison center, or treatment location at the first sign of poisoning.

Atropine (delivered by injection) is the antidote given when treating cholinesterase inhibition. A combination of Atropine and pralidoxime chloride (2-PAM or 2-PAMCl, Protopam) may be administered for organophosphate pesticide or nerve gas poisoning. Atropine blocks the effects of the neurotransmitter (the nerve gas or pesticide) that causes over stimulation. Pralidoxime chloride (2-PAMCl) is an oxime which removes the nerve agent or the organophosphate-based pesticide from the enzyme. Atropine is the only antidote for carbamate-based pesticide exposure. Fortunately, the breakdown of cholinesterase can be reversed by proper treatment.

The MARK I kit supplied to military personnel consists of two spring-driven injectors: (1) 2 mg of atropine in 0.7 ml of diluent and (2) 600 mg of 2-PAMCl in 2 ml of diluent.

Assisted breathing and supportive care will be required in cases of severe poisoning.

The Jane's Chem-Bio Handbook (published by Jane's Information Group, Alexandria, VA, in 1998) adds the following details (applicable to both nerve agents and certain pesticides):

Mild Poisoning: Usually no antidotes. However, if eye or head pain or nausea and vomiting (in absence of other symptoms) atropine/homatropine eye drops should be administered. Atropine only (2 mg) should be administered in the case of severe rhinorrhea (nasal discharge).

Moderate Poisoning: Vomiting and/or diarrhea, and/or shortness of breath: For nerve gases and organophosphate pesticide poisoning administer 2 mg of Atropine and 600 mg of 2-PAMCl (if injecting, 1000 mg of 2-PAMCl over 20 to 30 minutes). Follow with additional atropine (2 mg) at 5 to 10 minutes until breathing is improved. Assisted ventilation and oxygen is recommended in the case of a casualty with cardiac or pulmonary disease but probably unnecessary in most other situations.

Severe Poisoning: Convulsions, severe shortness of breath, unconscious, severe gastrointestinal effects, muscular twitching, or a combination of two or more of these symptoms. Administer 6 mg of Atropine by IM (not IV) and 1800 mg of 2-PAMCl (can be injected or alternatively by infusion of 1000 mg over 20 to 30 minutes). Administer diazepam (10 mg by IM or 5 to 10 mg slowly by IV). Diazepam is an antoconvulsant. Lorazepam may be administered instead of diazepam. Administer more atropine (2 mg at 5 to 10 mg slowly until improvement is noted. Administer more 2-PAMCl at hourly doses up to 3 doses.

Atropine does not reverse miosis (pupal size) so this cannot be used as an indicator of atropine effectiveness. Atropine should be administrated before attempting to insert an endotrachael tube to reduce bronchoconstriction in the situation of intense airway resistance.

Atropine at 6 mg dose should not be given by IV to a hypoxic patient because of the possibility of ventricular fibrillation. The initial dose must be given by IM. 2-PAMCl if injected too rapidly (less than 20 minutes) can result in hypertension. Phentolamine (5 mg, by IV) will reverse the hypertension.

In a normal adult without nerve gas or pesticide poisoning, 2 mg of atropine will cause an increase in heart rate of about 35 beats per minute which can be tolerated by someone without heart disease. Atropine given to an adult without nerve gas or pesticide poisoning also may result in blurred vision for 24 hours and drying of secretions (including sweat inhibition).

The recommended starting dose for children between 2 and 10 years is 1 mg of atropine. For infants under 2 years, administer 0.5 mg.

Is There a Test for Cholinesterase Inhibition?

Workers who are routinely exposed to cholinesterase-inhibiting pesticides (organophosphate and/or carbamate pesticides) should have their blood checked for cholinesterase activity. This is required by law by many states. This includes workers who service equipment. Federal regulations also require applicators to be tested when using pesticides in the "highly toxic" or "moderately toxic" category (EPA lists categories for pesticides). A baseline sample should be taken before employment (or taken during a time when the worker has not been exposed to organophosphate or carbamate pesticides for 30 days). At least two baseline samples should be taken. If later tests show a 20% decrease in cholinesterase level, he/she should be retested. If tests show a 30% or greater decrease, the worker must be removed from all exposure to organophosphate and carbamate pesticides.

Humans have three types of cholinesterase: (1) red blood cell cholinesterase, (2) plasma cholinesterase, and (3) brain cholinesterase. When a blood sample is withdrawn, the patient's red blood cell cholinesterase and plasma cholinesterase can be measured. Brain cholinesterase cannot be directly measured in a practical situation, but the same red blood cell cholinesterase enzyme is also in the nervous system Plasma cholinesterase, which is manufactured in the liver, is different from red blood cell cholinesterase. Plasma cholinesterase and red blood cell cholinesterase numbers have different meanings. Plasma cholinesterase numbers give an acute or early warning number while red blood cell cholinesterase numbers are useful in evaluating chronic, long-term exposure.

Toxicity of Nerve Agents

Nerve agents can be absorbed into the body by inhalation, through the skin(including the eyes), or by ingestion. In a terrorist attack, the routes of entry will probably be through the skin or by inhalation. We will look at the toxicity of four nerve agents: (1) Sarin, also called GB; (2) Soman, also called GD; (3) Tabun, also called GA; and (4) O-ethyl-S-(2-diisopropylaminoethyl) methylphosphonothiolate , better known as VX. All are organophosphate compounds. These are not the only nerve agents in existence but the ones that are the most widely known. There are also chemical warfare agents that are not cholinesterase inhibitors; these include blister agents (e.g. mustard gas, lewisite, phosgene oxime, etc.), pulmonary agents (e.g. phosgene, chlorine), or otherwise very toxic chemicals (e.g. hydrogen cyanide, cyanogen chloride). The nerve agents and some of the blistering agents are banned by international treaty, but other toxic chemicals have legitimate uses in industry.

Information on toxicity is obtained from running tests using animals, usually on rats or mice. The lethal dose required to kill 50% of the test animal (LD_{50}) is expressed in units of milligrams per kilogram of body weight. The liquid nerve agent is placed under a patch on the animal's skin. The assumption is made that the test results can be extrapolated to a 70 kg man. For the inhalation tests, the animals are placed in an enclosure with a certain concentration (milligrams per cubic meter) of the agent and allowed to breathe the agent for specified time (usually 1 or 4 hours, or other time). The lethal concentration required to kill 50% of the test animal (LC_{50}). The dose (LCt_{50}) has units of mg-min/m^3 . [mg = milligrams; m^3= cubic meter]. When adjusting the data to humans, the assumption is made that the human is at rest and breathing at the rate of 20 liters/minute If a person is breathing heavily, he/she could receive a higher dose.

Table 1 lists LD_{50} and LCt_{50} values extrapolated to a 70 kg man obtained from animal studies, as cited in Lane's Chem-Bio Handbook:

Table 1. Lethal Dose of Nerve Agents for a 70 kg Person

Nerve Agent	Dermal LD_{50} , mg liquid on skin	Inhalation LCt_{50} , mg-min/m^3
Tabun (GA)	1000	400
Sarin (GB)	1700	100
Soman (GD)	350	50
VX	10	10

The inhalation dose of tabun, sarin, or soman required to produce miosis (small eye pupils) is about 2 or 3 mg-min/m^3.

The U.S. Department of Energy has published Temporary Emergency Exposure Limits for inhalation of Sarin or VX (table 2):

Table 2. DOE-recommended Temporary Emergency Exposure Limits for Inhalation of Nerve Agents

Nerve Agent	TEEL-1, mg/m^3	TEEL-2, mg/m^3	TEEL-3, mg/m^3
Sarin (GB)	0.0075	0.05	0.6
VX	0.00035	0.002	0.015

TEEL is an acronym for Temporary Emergency Exposure Limit. TEEL numbers are developed by the Subcommittee on Consequence Assessment and Protective Actions (SCAPA), under the U.S. Department of Energy (DOE). They are considered temporary values to be used until peer-review numbers are established as Emergency Response Planning Guidelines (ERPG), which is another list published by the American Industrial Hygiene Association. Their definitions are as follows:

TEEL-1: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed without experiencing other than mild transient adverse health effects or perceiving a clearly defined, objectionable odor.

TEEL-2: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed without experiencing or developing irreversible or other serious health effects or symptoms which could impair an individual's ability to take protective action.

TEEL-3: The maximum airborne concentration below which it is believed that nearly all individuals could be exposed without experiencing or developing life-threatening health effects.

The exposure time is not stated in the DOE definition but one hour is implied.

Toxicity of Pesticides

We will limit the discussion to pesticides that inhibit cholinesterase. The two major classifications are (1) organophosphate pesticides and (2) carbamate pesticides. Pralidoxime chloride (2-PAMCl) may be administered along with atropine in the case of organophosphate pesticide poisoning, but pralidoxime chloride (2-PAMCl) should not be administered in the case of carbamate pesticide poisoning (it may be ineffective and possibly do more harm than good). Table 3 and 4 present toxicity data based on rat studies, by ingestion, skin absorption, and inhalation. The source of the information contained in tables 3 and 4 is the EXTOXNET website at http://ace.ace.orst.edu/info/extoxnet/pips/ghindex.html .

Table 3. Lethal Dose of Organophosphate Pesticides to Rats (unless otherwise specified)

Pesticide (typical brand name)	Oral LD_{50} mg/kg	Dermal LD_{50} mg/kg	Inhalation LC_{50} mg/m^3 in 1 or 4 hours
Azinphos-methyl (Guthion)	4.4 to 16	88 to 220	400 (1 hour)
Chlorpyrifos (Dursban, Lorsban)	95 to 270	> 2000	> 200 (4 hours)

Coumaphos (Co-Ral)	13 to 41	860	340 (1 hour)
Dichlorvos (DDBP, Vapona)	25 to 80	70.4 to 250	>200 (1 hour)
Diazinon (Spectracide)	300 to 400	3600 (rabbit)	350 (4 hours)
Dimethoate (Cygon, De-Fend)	180 to 330	100 to 600	>200 (4 hours)
Disulfoton (Di-Syston)	1.9 to 12.5	3.6 to 15.9	300 (1 hour)
Ethion (Acithion, Ethion)	21 to 191	62	864 (4 hours)
Fenamiphos (Nemacur)	2 to 19	72 to 154	110 to 170 (*)
Fonofos (Dyfonate)	3.2 to 18.5	147	900 (4 hours)
Isofenphos (Oftanol, Amaze)	28 to 38	188	130 to 144 (4 hours)
Malathion (Cythion)	1000 to 10000+	>4000	-
Methidathion (Supracide)	25 to 54	85 to 94	360 (4 hours)
Methyl Parathion (Baldwin M, Metapos)	6 to 50	67	240 (1 hour)
Mevinphos (Phosdrin)	3 to 12	4.2	125 (1 hour)
Naled (Dibrom)	91 to 430	800	> 150 (6 hours, mice)
Parathion (Niran, Phoskil)	2 to 30	6.8 to 50	84 (4 hours)
Phorate (Thimet)	1.1 to 3.7	2.5 to 6.2	60 (4 hours)
Phosmet (Irnidan, Prolate)	113 to 160	3160 to 4640	276 (1 hour)
Propetamphos (Blotic, Safrotin)	75 to 119	2300 to 3100	204 (4 hours, rabbit)
Temephos (Abate)	1226 to 13000	-	-
Terbefos (Counter)	1.3 to 1.74	1.1 (rabbit)	-
Trichlorfon (Dylox, Neguvon)	450 to 650	2000 to 5000	>500 (4 hours)

(*): time not specified but probably 4 hours

Table 4. Lethal Dose of Carbamate Pesticides to Rats (unless otherwise specified)

Pesticide (typical brand name)	Oral LD_{50} mg/kg	Dermal LD_{50} mg/kg	Inhalation LC_{50} mg/m^3 in 1 or 4 hours
Aldicarb (Temik); not used in U.S.	0.5 to 1.5	-	-
Bendiocarb (Ficam)	34 to 156	566	550 (4 hours)
Carbaryl (Sevin)	250 to 850	> 2000 (rabbit)	>200 (4 hours)
Carbofuran (Furadan)	5 to 13	> 1000 (rabbit)	43 to 53 (4 hours, guinea pig)
Methomyl (Lannate, Nudrin)	17 to 24	5880 (rabbit)	120 to 170 (4 hours)
Oxamyl (Vydate	514	2960 (rabbit)	120 to 170 (4 hours)

Propoxur (Baygon)	100	1000 to 2400+	1440 (1 hour)

The information from which tables 3 and 4 were constructed came from many researchers. The researchers came up with different results. Sometimes different animals were used instead of rats. Some researchers used a 4-hour inhalation test, some used one hour. Some used all male rats, some used female rats (female rats usually more susceptible to the chemical). A range of LD_{50} or LC_{50} values are presented for the different results.

Tables 3 and 4 obviously have different units than table 1. The assumption is made that the rat (or other test animal) translates to a 70 kg man, and that the oral LD_{50} and dermal LD_{50} animal values in units of mg of chemical per kg of body weight can be multiplied by 70 to estimate the dose for a 70 kg man. For the inhalation LC_{50} comparison, the calculations get somewhat murky. The accumulated dose up to l hour in time is assumed to be linear, e.g. Dose = (concentration)x(time), x denotes multiplication. After one hour, the accumulative dose drops off and tends to be proportional to the square root of the exposure time. This means that to convert the 4-hour test to a 1-hour equivalent test, the 4-hour concentrations (mg/m^3) should be multiplied by 2. To get units of $mg\text{-}min/m^3$, the 1-hour test equivalent should be multiplied by 60. For example, phorate is one of the most toxic organophosphate pesticides listed with a 4-hour LC_{50} for rats of 60 mg/m^3. The 1-hour equivalent LC_{50} calculates out to be 120 mg/m^3. This number is multiplied by 60 to get 72,000 $mg\text{-}min/m^3$ (or 72 $grams\text{-}min/m^3$). The major conclusion is that the lethal dose required to kill is several orders of magnitude higher for pesticides than for any of the nerve agents.

The more toxic of the pesticides are classified as "Restricted Use Pesticides" in the United States, meaning, that they can only be purchased and used by certified applicators and applied according to directions. Other criteria such as possible contamination of groundwater and toxicity to wildlife are considered. In this list, Restricted Use Pesticides are (1) azinphos-methyl, (2) dichlorvos, (3) diazinon, (4) disulfoton, (5) fenamiphos, (6) fonofos, (7) isofenphos, (8) most formulations of methidathion, (9) methyl parathion, (10) mevinphos, (11) parathion, (12) phorate, (13) some formulations of propetamphos, (14) terbefos, (15) some formulations of bendiocarb, (16) carbofuran, (17) methomyl, (18) oxamyl, and (19) some formulations of propoxur. The very toxic aldicarb is no longer used. The less toxic pesticides are classified as "General Use Pesticides", meaning, that formulations containing these pesticides can be purchased and used by the general public.

The U.S. Department of Energy has published TEELs for inhaling many of the organophosphate and carbamate pesticides (tables 5 and 6).

Table 5. DOE-recommended Temporary Emergency Exposure Limits for Inhalation of Organophosphate Pesticides

Pesticide (typical brand name)	TEEL-1 , mg/m^3	TEEL-2, mg/m^3	TEEL-3, mg/m^3
Azinphos-methyl (Guthion)	0.6	0.7	10
Chlorpyrifos (Dursban, Lorsban)	4	7	35
Coumaphos (Co-Ral)	4	30	125
Dichlorvos (DDBP, Vapona)	2.5	20	750
Dimethoate (Cygon, De-Fend)	15	30	30
Disulfoton (Di-Syston)	0.3	2	75

Ethion (Acithion, Ethion)	1.2	13	350
Fenamiphos (Nemacur)	0.3	0.9	40
Fonofos (Dyfonate)	0.3	1.3	200
Malathion (Cythion)	30	250	250
Methidathion (Supracide)	3	20	400
Methyl Parathion (Baldwin M, Metapos)	0.34	0.35	15
Mevinphos (Phosdrin)	0.27	4	4
Parathion (Niran, Phoskil)	0.3	2	10
Phorate (Thimet)	0.1	0.6	0.6
Phosmet (Irnidan, Prolate)	0.075	0.54	40
Terbefos (Counter)	0.6	1	1

Table 7. DOE-recommended Temporary Emergency Exposure Limits for Inhalation of Carbamate Pesticides

Pesticide (typical brand name)	TEEL-1, mg/m^3	TEEL-2, mg/m^3	TEEL-3, mg/m^3
Aldicarb (Temik)	0.21	0.3	0.3
Carbaryl (Sevin)	15	25	100
Carbofuran (Furadan)<	0.3	0.43	0.5
Methomyl (Lannate, Nudrin)	7.5	10	200
Oxamyl (Vydate)	1	1.7	15
Propoxur (Baygon)	1.5	20	20

The TEEL concentrations for pesticides are almost always several orders of magnitude greater than for nerve agents. However the TEEL-3 values for a few of the most toxic pesticides do indeed approach the TEEL-3 value for Sarin.

Do People Exposed to These Chemicals Fully Recover?

Deaths have occurred due to nerve agents and pesticide poisoning. There can be complications administrating the antidote atropine to patients with heart disease or are hypoxic. Patients exposed to these chemicals also recover and their cholinesterase levels return to normal.

Additional details on pesticide exposure can be found at the EXTOXNET website at http://ace.ace.orst.edu/info/extoxnet/pips/ghindex.html . Even though cholinesterase levels recover after exposure, some patients experience neurological problems after chronic exposure to some pesticides for very long time and may never fully recover. The subject is a controversial one.

Animal tests in most cases show that the organophosphate or carbamate chemical is metabolized or excreted directly and does not accumulate within the body. Long term animal feeding studies sometimes show liver and kidney and blood changes. Available information seems to indicate that most of the chemicals are not carcinogenic. For some of the chemicals the animal test cancer data are inconclusive or can result in cancer at relatively high doses.

Can a Terrorist Disperse Pesticides To Get the Same Effects as Nerve Agents?

Fortunately, the available pesticides which inhibit cholinesterase have very low vapor pressures and are much less toxic than the nerve agents. Also the formulations that are commercially available are not easily dispersed in large quantities into the air. Other toxic chemicals (e.g. ammonia, chlorine, etc.) may be more attractive to a terrorist. This is not to say that pesticides would not be used as a terrorist weapon.

Delivery

The four nerve agents (Tabun, Sarin, Soman, and VX) are liquids. Sarin is somewhat volatile and can disperse in a gaseous (vapor) form. VX is an oily liquid which does not readily volitilize as a vapor. But VX droplets stick to surfaces, and a person can be poisoned if his/her clothing or skin contacts these surfaces.

The pesticides listed in tables 3 and 4 are mostly crystalline solids. Some are liquids. Most have a very low vapor pressure, some have higher vapor pressures. The product formulations for any of these pesticides may be a liquid, granules, emulsions, flowable powder, or even (for some) dispersed as an aerosol using a gas to propel the chemical into the air. These product formulations have been developed to maximize the effect against the target insects (or other pests) while minimizing exposure to the applicant. May of the solid pesticides are soluble in water, alcohol, or other solvent. Oily or otherwise hard-to-handle chemicals can be emulsified into some other liquid using surfactants, or clays or other materials mixed allowing dispersal as a fine powder.

The same technologies used in the pesticide industry in developing product formulations to ease dispersal can potentially be used by a terrorist to disperse a toxic chemical into crowd. The terrorist wants to disperse a highly toxic material as a gas, a vapor, or as very fine aerosol or powder into the air, or by contact.