

CHAPTER 18

TOXICITY OF EXPLOSIVES

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18 TOXICITY OF EXPLOSIVES

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18.1 AWARENESS SUMMARY

18.1.1 Introduction

Propellants, high explosives and pyrotechnics are chemicals and as such will exhibit toxic properties, that is, the ability to poison people. Those who use, manage or even more importantly test ammunition must be aware of the potential toxic hazards of the explosive content but also of the products of detonation and burning. This purpose of this chapter is to provide a basic understanding to assist in identifying toxic hazards when working with ammunition and explosive materials including a detailed toxicology of individual chemical components.

18.1.2 The Toxic Risk

18.1.2.1 Exposure to toxic chemicals

The toxic risk from any chemical will be a function, among other things, of the amount that enters the body by one of the three main routes:

1. **Ingestion** – passing into the stomach, usually in food or drink. This should be easily controlled; however, poor hygiene after handling explosive materials can inadvertently contaminate something that is handled while eating.
2. **Inhalation** – passing into the lungs either as gas/vapour, droplets or dusts. This route is the most difficult to protect as people breath in anything that is in the air unless respiratory protection is used.
3. **Skin Contact** – solids and more particularly liquids on the skin are often capable of being absorbed in to the body. This route is often ignored as a potential hazard.

Other routes of entry such as through cuts or grazes are less likely; however, contamination of the eyes by chemicals is a serious hazard. The risk of transfer to the eyes or to the mouth via contaminated hands or gloves must be remembered.

During much of the lifetime of a munition there is minimal potential for exposure to the explosive content and thus there is no toxic risk. However, there are times when explosive may exude during storage and then present a toxic hazard. The greatest risk of exposure is during manufacture when explosive materials are handled or filled in to the ammunition. There is also an increased risk of exposure during disposal or dismantling for surveillance or servicing. Products formed from explosion or burning of explosives are often highly toxic.

18.1.2.2 Exposure limits

The most likely entry route for chemicals in the workplace is by inhalation and the potential for a chemical in the air to enter the body will depend on two factors, the concentration present and how long it is breathed in. For example if a person is exposed to a certain concentration for, say, one hour and a second person is exposed to a concentration half of the first but for two hours they will have the same amount of chemical enter their systems. To provide safety guidelines for safe workplace concentrations of chemicals in the air the Health and Safety Executive publish Workplace Exposure Limits (WEL) as an acceptable level in the workplace.

A WEL can be for a Short Term Exposure Limit (STEL), usually for 15 minutes exposure, or a Long Term Exposure Limit (LTEL) of 8 hours. The STEL value is usually greater than the LTEL but may have additional controls relating to overall exposure during the working day. The LTEL means that a worker can be exposed to that concentration for 8 hours a day, 5 days a week during the working year. These limits are expressed in part per million (ppm) or mg m^{-3} . The LTEL may be quoted as a Threshold Limit Value (TLV) based on the American Conference of Governmental Industrial Hygienists. The workplace is anywhere people are working, in this instance, with explosives or ammunition and will include explosive storage, ranges, magazines, etc. as well as places where explosives and ammunition are manufactured. The HSE publish guidance in EH40⁽¹⁾ which lists workplace exposure limits of many chemical substances although the data for explosives is limited. The forward to the printed 2005 edition explains that many substances had been removed from earlier editions because the HSE had doubts that the limit was soundly-based. The absence of a substance from EH40 does not mean that it is safe.

18.1.2.3 Additional toxicity data

Anyone supplied with an article has the right to demand a Safety Data Sheet (SDS) for the chemicals contained therein. Safety Data Sheets contain doses or amounts required to produce acute lethal poisoning effects by all three main routes of entry. It may also cover skin and eye irritation, some chronic effects, warnings about potential cancer causing chemicals and other hazards such as danger to the unborn child or potential to cause reproductive defects. They can also repeat data from EH40 or the equivalents from other jurisdictions, usually the USA.

Acute toxicity is the adverse effects of a substance that result either from a single exposure or from multiple exposures in a short period of time, usually less than 24 hours. To be described as acute toxicity, the adverse effects should occur within 14 days of the administration of the substance. Chronic toxicity is the adverse health effects from repeated exposures, often at low levels, to a substance over a longer time period of months or years. The ability of some substances to cause cancer can also be considered as a chronic effect. Certain chemicals can cause cancer with a single exposure but are very rare.

There is a classification system for the toxicity of chemicals by what are called Hazard or H Statements, which are based on well-defined test methods and place the materials into bands of toxicity depending on the test results. There are also rules for defining the classification of mixtures. Annex A to this chapter contains the main H Statements and outlines the boundaries between them.

These H statements are based on LD₅₀ values and this represents the quantity of a chemical, usually expressed as mg of chemical per kg of body weight, that would cause lethality to 50 % of a large group of people exposed to that dose of the chemical. However, individuals may exhibit hypersensitivity to certain chemicals and therefore LD₅₀ values should be treated with extreme caution as a basis for a COSHH risk assessment when a large number of people are potentially exposed. Potentially an individual may die from exposure to well below the LD₅₀ dose. The Control of Substances Hazardous to Health (COSHH) Regulations⁽²⁾ require that a risk assessment be made of the potential harm that might come from any exposure to chemicals.

18.1.3 Explosive Chemicals with Significant Toxicity

Error! Reference source not found. provides a list of explosives and explosion products that have a significant toxic risk and where they are found. Explosive materials not included are still to be considered as toxic and included in any COSHH assessment.

Table 18-1 Explosives or Explosion Products with Significant Toxicity

Explosive	Route of entry	Where found	Major effect
Nitroglycerine	Inhalation (vapour) or skin contact	Gun propellants, double base rocket propellants, gelatine dynamites	Severe headache and lowers blood pressure
Ethyleneglycol dinitrate	Inhalation (vapour) or skin contact	Taggant in plastic explosives	Severe headache and lowers blood pressure
Propyleneglycol dinitrate	Inhalation (vapour) or skin	OTTO fuel	Severe headache and lowers blood pressure
Trinitrotoluene	Inhalation (dust) or skin contact	High explosive formulations	Cyanosis, liver and kidney damage
Tetryl	Inhalation (dust) or skin contact	Booster charges in warheads, only found in legacy ammunition	Severe dermatitis in some individuals, liver and kidney damage if inhaled
Lead	Inhalation (dust)	Indoor small arms ranges	Anaemia and central nervous system
Carbon monoxide	Inhalation (gas)	Detonation in confined areas	Headache, dizziness and then lack of oxygen in the blood

18.2 TOXICITY OF EXPLOSIVE SUBSTANCES AND EXPLOSION PRODUCTS

18.2.1 Classification of Toxic Hazards

Those who manage ammunition or undertake work with explosive materials must be aware of their obligations and rights under REACH (Registration, Evaluation, Authorisation and Restriction of Chemicals)⁽³⁾ and CLP (Classification, Labelling and Packaging)⁽⁴⁾. In June 2015 all previous Classification and Labelling requirements that fell under CHIP (Chemical Hazard Labelling and Packaging) were replaced by these two documents which implement the Globally Harmonised System (GHS) of Classification and Labelling of Substances within the European Union. Both REACH and CLP are enforced in UK law through Statutory Instruments (REACH in 2008 and CLP in 2015). The MOD can grant specific exemptions to REACH (May 2014) but state that their “internal standards will be at least as good as those in the regulations where reasonably practicable”.

There is a derogation from labelling explosives, as referred to in section 2.1 of Annex I of the CLP (“placed on the market with a view to obtaining an explosive or pyrotechnic effect”) with anything other than their classification as explosive. There is no derogation from classifying the chemicals or having a classification for the chemicals. Anyone supplied with an article containing an explosive has the right to ask for a Safety Data Sheet (SDS) for the chemicals contained therein.

MOD should not have to classify the toxic hazards of an explosive unless the MOD actually produces them as novel chemicals. MOD should hold the SDS containing the classification for all explosives found in their ammunition stockpile. Prior to June 2015 some legacy MSDS (Material Safety Data Sheets) were acceptable but those who hold these will need to update them with SDS.

18.2.2 Toxicity of Explosive Chemicals Found in Propellants

18.2.2.1 Nitroglycerine (glycerol trinitrate)

Nitroglycerine (NG) is an extremely common constituent of many commercial explosives such as gelatine dynamites and of both certain gun (see 12.2.2) and rocket propellants (see 13.3.2). It is a liquid at most normal temperatures having a Melting Point of 13.2 °C⁽⁵⁾. NG is absorbed through the lungs and also through the skin. Nitroglycerine is a potent smooth-muscle relaxant and has been used as a vasodilator to widen blood vessels in the treatment of angina pectoris for over 100 years. Most of the SDS produced for NG are for its use as a medicine, where it can be found at the 1 to 2 % level diluted with other materials.

Because of this vasodilatory effect the early symptoms of exposure are a lowering of blood pressure leading to headache, dizziness, weakness and

nausea. The 'nitro headache' common in nitroglycerine and cordite production works⁽⁶⁾ is due to the cerebral vasodilation effect of NG. Because of this it is best to keep the sufferer in a standing position as this limits the blood supply to the brain⁽⁵⁾. At high doses NG causes methaemoglobinaemia, a condition described in section 18.3.1, where the blood has reduced capability to carry oxygen. NG has reasonable high volatility and sufficient vapour can exist to produce symptoms in enclosed spaces. For example, the distinctive smell will be noticeable in closed unvented propellant storehouses.

The US threshold limit value (TLV) for NG is 0.05 ppm (0.50 mg m⁻³) and exposure at this level can cause slight headaches. Although NG is not quoted in EH40, when it was included in 2002 it had a WEL (section 18.1.2.2) value of 0.2 ppm (2 mg m⁻³) and its short term exposure limit (STEL) was the same value. Exposure to this level has been shown to produce marked headaches in 50 % of those exposed within 3 minutes⁽⁵⁾. The saturated vapour pressure (SVP) of NG is such that a standing concentration of 0.65 ppm will occur in enclosed spaces at 25 °C.

The LD₅₀ (oral) for rats is 105 mg kg⁻¹⁽⁷⁾. A daily dose of 10 mg is safe and effective as a medicine⁽⁵⁾ but 9100 mg (taken with wine by mistake) has caused a fatality⁽⁵⁾. Alcohol is known to potentiate the symptoms. NG is classified as H300 (Acute Toxicity 2), H310 (Acute Toxicity 1) and H330 (Acute Toxicity 2)⁽⁷⁾.

The mechanism of toxicity of NG is complex. It is known that there is a marked build-up of tolerance to NG with increasing exposure⁽⁸⁾. For more information see section 18.3.1.3.

18.2.2.2 Nitrocellulose (NC)

Nitrocellulose is a large polymer which is both water and lipid insoluble which cannot be absorbed into the body. Animals fed on diets containing up to 30 % NC for long periods developed no symptoms of poisoning⁽⁹⁾. NC has an LD₅₀ greater than 5000 mg kg⁻¹.

18.2.2.3 Nitroguanidine (Picrite)

There is no evidence that nitroguanidine presents a toxic hazard to man under normal conditions. Animal studies confirm that this compound has exceptionally low toxicity both for acute and chronic poisoning. The LD₅₀ for rats (oral) is 4640 mg kg⁻¹ and mice fed nitroguanidine at 1000 mg g⁻¹ day⁻¹ for 90 days exhibited no ill effects⁽¹⁰⁾.

18.2.2.4 Propylene glycol dinitrate (PGDN)

Propylene glycol dinitrate is a major constituent (76 %) of OTTO Fuel II and is responsible for virtually all the toxic problems associated with exposure to this torpedo propellant used either as a monopropellant, or as a bi-propellant with hydroxyl ammonium perchlorate (HAP).

Animals exposed to PGDN vapours exhibit lethargy, prostration, anoxia and methaemoglobinaemia (section 18.3.1), with reduced response to external stimuli and at high doses, death. There is no reliable inhalation exposure data for PGDN; a rabbit continuously exposed to 35 ppm (265 mg m⁻³) died on the fourth day of exposure and one in nine monkeys exposed to the same level died after 9 days⁽¹¹⁾. Chronic exposure to high concentrations of PGDN vapour induced fatty changes in the livers of guinea pigs, dogs and monkeys. Monkeys exposed to concentrations of PGDN in the range of 0.3 to 4.2 ppm, 23 hours per day for 125 days, suffered no apparent physical effects; neither was there evidence from behavioural tests of any discernible effect that could be ascribed to PGDN exposure.

PGDN is a potential health hazard to man by inhalation, ingestion or by absorption through the skin. Most of the symptoms produced by exposure to this compound are typical of the nitrate esters. Ingestion of liquid OTTO Fuel can cause gastrointestinal disorders as well as the usual systemic effects. Both the liquid and the vapour are known to cause mild eye irritation. The vapour pressure of PGDN is small (0.07 torr at 22 °C⁽¹²⁾) compared to water (20 torr) at the same temperature, but not negligible and inhalation is the major health hazard posed by OTTO Fuel. The acute systemic effects of mild OTTO exposure are nasal congestion, headache, nausea, eye irritation, lowering of blood pressure, dizziness and laboured breathing. The most common acute symptom is the well-known 'nitro headache', which can be very severe and last for several hours after exposure. In common with NG, repeated exposure leads to a tolerance of the minor symptoms. Most of the effects of OTTO Fuel on individuals can be attributed to the cardiovascular action of PGDN. The National Institute of Occupational Safety and Health sets the TLV at 0.05 ppm⁽¹²⁾. Dizziness and a marked impairment in balance can be induced by concentration levels of 2 to 3 times the Occupational Exposure Limit. The saturated vapour pressure could create a concentration of 100 ppm in an enclosed unventilated space.

A comprehensive study of 87 workers chronically exposed to OTTO Fuel in their working environment, some for as long as 11 years, revealed a history of minor acute PGDN toxic symptoms but no serious chronic physical or neuro-physiological ill effects. No human deaths or other major toxic accidents due to PGDN have been reported. It seems likely that under normal working conditions, with good ventilation, peak PGDN concentrations are usually far below the Occupational Exposure Limit. The U.S. Department of Health and Human Services published a comprehensive study of the toxicological profile for OTTO Fuel II and its components in 1995⁽¹³⁾.

18.2.2.5 Triethylene glycol dinitrate (TEGDN)

TEGDN is an energetic plasticiser for explosives and propellants. Studies with animals indicate that TEGDN is 2-5 times less lethal than propylene glycol dinitrate (PGDN) when administered orally or subcutaneously. 'In vitro' studies have shown that TEGDN interfered with nerve/muscle communication, which

could explain why TEGDN dosed animals tremored violently and convulse while those exposed to PGDN were lethargic and did not exhibit convulsions⁽¹⁴⁾.

18.2.2.6 Ammonium Perchlorate

Ammonium perchlorate found in composite propellants is not classified as toxic under CLP. Ammonium perchlorate has a quoted LD₅₀ (rat) of 4200 mg kg⁻¹ but may cause serious eye irritation. It can, however, cause chronic thyroid problems if ingested in drinking water, from which it is very difficult to remove, as it interferes with iodide take up by the thyroid gland. The Environmental Protection Agency in the USA has set a limit of 15 mg L⁻¹ of drinking water at which there are not expected to be any adverse non-carcinogenic effects during a lifetime of exposure.

18.2.3 Toxicity of Explosive Chemicals Found in High Explosives

18.2.3.1 Trinitrotoluene (TNT) and related compounds

Typical symptoms of acute TNT exposure are paleness of face, grey lips, stomach ache, constipation, flatulence and possible jaundice. Changes in the blood, with a decrease in haemoglobin and the appearance of abnormal red blood cells are among the first signs to be present. Distinct poisoning has been observed in individuals exposed to an atmosphere containing TNT dust at a concentration of 3 mg m⁻³. TNT dust has been known to cause irritation of the digestive tract on ingestion and also skin rashes and dermatitis. EH40⁽¹⁾ contains a workplace exposure limit of 0.5 mg m⁻³ for TNT. This would be as the dust as the Saturated Vapour Pressure only produced 0.08 mg m⁻³ at 25° C.

Inhalation of TNT dust, ingestion or absorption through the skin can cause cyanosis, liver and kidney damage and even death. In the first 8 months of World War I 17,000 cases of TNT poisoning with 475 fatalities occurred in the United States of America⁽¹⁵⁾. In the United Kingdom some 106 deaths were attributed to the toxic effects of TNT during the whole of World War I. In contrast there were very few cases of poisoning reported in Germany during WW1. Many of these early casualties may have been caused by impurities in the TNT and by very poor industrial hygiene practices. There were only 22 fatalities in the United States of America during World War II caused by TNT intoxication.

There is a record of acute toxic jaundice caused by TNT and autopsy revealed atrophy of the liver⁽¹⁶⁾. Skin adsorption is supposed to be enhanced by the skin being oily or sweaty. Skin symptoms are a reddening of the skin⁽¹⁷⁾, it stains the skin of the hands a light yellow and discolours the hair to a reddish blond⁽¹⁸⁾.

Metabolism of TNT in the human produces many products including a dinitrohydroxylamine derivative that is a potent producer of methaemoglobin. After serious or prolonged exposure, cyanosis caused by methaemoglobinaemia (see 18.3.1), kidney disorders and liver damage causing severe jaundice will occur. TNT also destroys the bone marrow tissue causing aplastic anaemia. Death is usually due to severe jaundice, anaemia or toxic hepatitis. An LD₅₀ between 795 to 1320 mg kg⁻¹ has been quoted⁽¹⁹⁾ as have

similar values from other studies⁽⁵⁾. A manufacturer has classified TNT as H331, H311 and H301 but without references⁽²⁰⁾, which suggests that a more poisonous oral LD₅₀ may have been reported. It also gives a TLV from the US of 0.1 mg m⁻³.

Dinitrotoluene (DNT) is of interest as an impurity in TNT and as components of effluent from TNT production plants. While a mixture of isomers is produced, the main component is usually the 2,4-dinitro compound which is classified H301, H311 and H331. It is also H341 (suspected of causing genetic defects) and H350 (may cause cancer)⁽²¹⁾. The dinitrotoluenes are lipid-soluble and can be absorbed through the skin or by ingestion. Symptoms of poisoning include pallor, headache, giddiness, cyanosis and tremors. LD₅₀ of 2,4-dinitrotoluene for rats is 270 mg kg⁻¹ to 650 mg kg⁻¹⁽²²⁾. The other isomers have an LD₅₀ value in the same range. Chronic exposure of animals to DNT produced evidence of methaemoglobinaemia, central nervous system effects and liver damage. There is clear evidence from animal studies that the 2,4 isomer is carcinogenic. In the Ames test all DNT isomers are mutagenic. In general nitrotoluenes are reputed to be less toxic than nitrobenzenes, possibly because of the ease of metabolism within the body of the nitrobenzenes to the slightly toxic nitrobenzoic acids.

18.2.3.2 Nitrobenzenes

Nitrobenzene has been widely used as a semi-energetic fuel in improvised explosive mixtures. Both mono and the dinitrobenzenes can be produced during TNT manufacture particularly when using impure toluene.

Nitrobenzene can be readily absorbed into the body by inhalation of vapour or by absorption through the skin. As might be expected of an aromatic nitro compound, nitrobenzene will oxidise haemoglobin to methaemoglobin. It is also toxic to the liver and the nervous system. Exposure to vapour at a concentration greater than 5 mg m⁻³ is considered dangerous. Symptoms of nitrobenzene exposure include nausea, headache, dizziness, stomach upset and characteristic cyanosis. After severe acute exposure these symptoms will be intensified and heart palpitations, cramps, disturbance of vision and loss of consciousness may also occur. Chronic intoxication may lead to anaemia and liver damage.

It is likely that most of the casualties and fatalities caused during the early days of TNT manufacture were due to the presence of m-dinitrobenzene (DNB) as an impurity in the product. DNB is not very volatile but is readily absorbed percutaneously. An LD₅₀ for 1,3 dinitrobenzene of 59 mg kg⁻¹ has been quoted⁽²³⁾. During World War I, there were many cases of up to 50 % of exposed factory or munition workers becoming poisoned by nitrobenzene exhibiting cyanosis and headaches⁽²⁴⁾. Individuals exposed to DNB can develop methaemoglobinaemia, liver damage, convulsions and cerebral paralysis. After mild exposure, victims may completely recover in a few weeks but more serious cases may need 2 months or more to fully recover. In a few acute cases symptoms appear rapidly and death can occur in a few hours. Cases have been reported in which the onset of symptoms is much delayed, in some cases up to

several months after exposure has ceased. Dinitrobenzenes are classified as H300 (Acute Toxicity 2), H310 (Acute Toxicity 1) and H330 (Acute Toxicity 2).

18.2.3.3 Cyclotrimethylenetrinitramine (RDX)

RDX has a high melting point and at ambient temperatures has a low vapour pressure. Inhalation of RDX vapour therefore would not be expected to pose problems under normal circumstances. In spite of the low lipid solubility of RDX there have been some suggestions of dermal absorption. Inhalation of dust or ingestion will be the main routes of entry of RDX into the body.

The pharmacology of RDX is different to that of other previously mentioned organic nitro compounds and involves gastrointestinal, renal and central nervous system effects, the latter effect being the most prominent. The acute oral LD₅₀ in rats is about 100 mg kg⁻¹. One modern SDS records RDX as Acute Toxicity 3; H301 and Skin Irritant 2; H315⁽²⁵⁾. An older SDS records the chemical as “toxic” by all three routes of entry (R23/24/25). As there is evidence that inhaling the dust is dangerous H330 or H331 should always be considered when there is a chance of dust being produced.

Exposure to RDX dust can cause sudden loss of consciousness with convulsions, typically followed by a coma of several hours duration⁽²⁶⁾. When RDX is handled in a moist state and dusting suppressed, or when workroom ventilation is improved, RDX intoxication does not occur. Five cases of convulsions occurred among 26 workers engaged in pelletising RDX in 1962 in the United States of America⁽²⁷⁾. Typically the onset of symptoms occurs after a delay of several hours and may be preceded by preliminary signs of headache, nausea, vomiting or hyperirritability.

Cases have been reported of troops chewing plastic explosive containing RDX, the object being to produce a 'high' similar to the effects of alcohol and suffering severe RDX intoxication. Some military victims of RDX poisoning had used a similar RDX containing plastic explosive as a fuel for a cooking fire and either inhaled the toxic fumes or introduced particles of the material into their food⁽⁵⁾.

The symptoms of RDX intoxication are quite severe, however, few deaths have occurred and survivors appear to recover completely over a period which can vary from days to several months in the case of long exposures. The US Department of Health and Human Services published a comprehensive review of toxicological studies on RDX in 2012⁽²⁸⁾.

18.2.3.4 Cyclotetramethylenetetranitramine (HMX)

The toxic symptoms of HMX are reported as being similar to those induced by RDX but HMX is regarded as being somewhat less toxic. The US Department of Health and Human Services published a comprehensive review of toxicological studies on HMX in 1997 in which they noted very few studies relating to effects on humans⁽²⁹⁾. Some animal studies were reported in rats suggesting that acute LD₅₀ for HMX is around 6000 mg kg⁻¹ in rats for oral ingestion and about half this value in mice. This seems to be related to the low solubility of HMX

compared to RDX. At the Human and Environmental Toxicology of Explosives and related Compounds Meeting 2012 (Amsterdam), LD₅₀ (rat) values were reported for RDX and HMX as 100 mg kg⁻¹ and 6000 mg kg⁻¹ respectively⁽¹⁹⁾. Their water solubilities were 50 mg L⁻¹ for RDX and only 6 mg L⁻¹. HMX has been classified as Acute Toxicity 4; H302 and Acute Toxicity 3; H311, an 8 hr Time Weighted Average (LTEL) of 5mg m⁻³ and an oral LD₅₀ of 1670mg kg⁻¹⁽³⁰⁾.

18.2.3.5 Pentaerythritol tetranitrate (PETN)

The toxic action of PETN is the same as other nitrate esters such as nitroglycerine. It has been used in medicine as a long lasting vasodilator in the treatment of angina. However the vapour pressure of PETN is negligible being 1.2×10^{-8} torr at 25 °C ⁽³¹⁾ leading to a saturated vapour concentration of 5×10^{-4} mg m⁻³ at 25 °C so inhalation of vapour will not occur at ambient temperatures. It also has a low solubility and even breathing small amounts of PETN dust has been reported as causing little effect⁽³²⁾, 0.25 g being required to produce irritation of the lungs. PETN is absorbed only slowly from the gastrointestinal tract, with a 64 mg oral dose in man produced no ill effects⁽⁵⁾. There is little, if any, absorption through the skin. The toxicity of PETN is certainly much less than that of NG. Some cases of dermatitis have been attributed to contact with PETN in ordnance facilities, but short term tests on a limited number of volunteers did not support this⁽⁵⁾.

18.2.3.6 Trinitrophenylmethyl nitramine (Tetryl)

The hazard from tetryl is due to the dust being inhaled, or coming into contact with the skin. The main symptom of tetryl is the development of a skin irritation after repeated handling but there is also a hazard from dust which will turn skin yellow and cause sensitisation dermatitis. In one processing plant where over 5000 people worked in a series of buildings 23 % suffered from the symptoms of tetryl illness of which 75 % developed definite tetryl dermatitis. The illness was not confined to those in direct contact with tetryl, 20 % of the operatives working in the same building but who did not handle tetryl developed the illness⁽⁵⁾.

At low concentrations the dust irritates the upper respiratory tract. Breathing an atmosphere containing 1.5 mg m⁻³ of tetryl will cause symptoms to appear 2-3 weeks after exposure. Other symptoms of tetryl poisoning are insomnia, loss of appetite and giddiness. Chronic exposure can result in nitro induced anaemia and liver or kidney damage.

18.2.3.7 Hexanitrostilbene (HNS)

HNS is classified as R20/22 under the old toxicity classification system, harmful by inhalation and if swallowed. The studies on which this classification is based have not been found and the noted ingestion dose of >4000 mg kg⁻¹ would not lead to it being classified under either the old or new system. One SDS notes that it has not been recorded as a carcinogen but may be a weak mutagen. Another SDS notes that pregnant women should not work with HNS.

18.2.3.8 Triaminotrinitrobenzene (TATB)

TATB is classified as R20/21/22 under the old toxicity classification system. LD₅₀ for oral ingestion is quoted in one SDS as >5000 mg kg⁻¹ which would not lead to this classification but there is evidence to irritation of the respiratory tract at the 5 mg m⁻³ level.

18.2.3.9 Ethylene glycol dinitrate (EGDN)

Ethylene glycol dinitrate is usually used in Dynamites mixed with nitroglycerine (NG) to lower the freezing point of the latter and thus avoid the dangers associated with low temperature applications. It is also used as a taggant in some explosives as it is more volatile than NG and thus is easier to detect as a vapour.

Ethylene glycol dinitrate produces the symptoms and tolerances typical of organic nitrates. It can be absorbed by inhalation or percutaneously. Headaches occur in non-habituated humans within 3 minutes at 2 mg m⁻³. Chronic exposure can cause very low blood pressure and slowing of the heart rate known as bradycardia. Protracted inhalation of large quantities of vapour can give rise to fatal acute circulatory or cardiac failure after a prolonged latency period of 24-48 hours. Between 1927 and 1952 49 fatalities were recorded with a fatal heart attack occurring during the weekend or on a Monday morning⁽⁵⁾. Consumption of alcohol makes humans particularly susceptible to EGDN⁽³³⁾. Since EGDN is 160 times as volatile as nitroglycerine⁽³¹⁾, most vapour exposure casualties are caused by the EGDN when used together with NG. Good exhaust ventilation at the workplace with fresh air devices at head height are reported as being adequate to prevent problems due to inhalation of EGDN vapours.

18.2.3.10 New explosives

There are a number of new explosives that are being considered for next generation explosive formulations both propellants and high explosives. There is limited information on their toxic properties.

DNAN, 2,4-dinitroanisole, is classified as H302 harmful if swallowed and H341 suspected of causing genetic defects. However, the SDS from one manufacturer is careful to say that the acute and chronic toxicity of this substance is not fully known.

FOX12, guanylureadinitamide (GUDN), does not have any classification for toxicity and the SDS notes that LD₅₀ (rat) is >2000 mg kg⁻¹ for both oral and skin absorption.

HNF, hydrazonium nitroformate, is classified as toxic by ingestion with a quoted LD₅₀ (rat) of 128 mg kg⁻¹. As a hydrazine salt it has, by analogy, a suspected carcinogenicity effect.

FOX7, 1,1-Diamino-2,2 Dinitroethene, has a SDS that quotes a very wide LD₅₀ (rat) of between 200 and 2000 mg kg⁻¹ without specifying a route of entry.

18.2.4 Toxicity of Chemicals Found in Initiating Substances

Many of the primary explosives used as initiators in the munitions industry are salts (e.g. styphnates or azides) of silver, lead or mercury. Most heavy metals are highly cumulative poisons and toxic doses can be acquired over a period of several years of exposure. There is much published information on these compounds including some concise toxicology⁽³⁴⁾.

18.2.4.1 Mercury

Mercury fulminate, the first common detonating primary explosive, is highly toxic as are most mercury salts. Cases of mercury poisoning are well documented amongst workers engaged in the first stages of production of this compound. Mercury metal itself is sufficiently volatile to produce a vapour hazard even though the vapour pressure at room temperature is low. Absorption can be through the skin, resulting from careless handling of the liquid metal or by breathing in vapour. Serious acute poisoning by elemental mercury or its salts, particularly the water soluble mercury salts which are much more toxic than the insoluble compounds, is not very common. Chronic human mercurialism is however very well documented. Toxic doses can be gradually built up in the body over a long period of exposure to the vapour.

Mercury vapour primarily attacks the brain, however, the kidney is the critical organ of injury following exposure to mercury salts. Characteristic symptoms of mercury poisoning include excitability, irascibility, fatigue, depression and tremor. In the environment, metallic or elemental mercury is converted to organomercury compounds which can be concentrated in the natural food chains and are potent and highly cumulative nervous system and kidney poisons. Methyl mercury produces teratogenic effects. In the notorious Minamata (Japan) poisoning episode inorganic mercury from an industrial process was discharged into seawater where micro-organisms converted it into methyl mercury. This was concentrated in plankton which in turn were eaten by shellfish and small fish which were eaten by large fish and eventually by humans leading to 765 deaths and over 2000 cases of non-fatal mercury poisoning⁽³⁴⁾.

18.2.4.2 Lead

Apart from specialist applications virtually all initiators for detonating explosive trains contain lead azide. Also, the majority of initiators for igniferous systems will contain lead styphnate, for example the percussion caps in small arms ammunition. In an era of improving environmental control the presence of lead in initiators is of increasing concern.

Lead is a highly cumulative poison and its chronic toxicity effects in man are well known⁽³⁵⁾. Dust and fumes of the metal as well as soluble lead salts are

poisonous. Lead poisoning produces many complex symptoms. Red blood cells are attacked producing anaemia. The nervous system is particularly sensitive to accumulation of lead; convulsions, delirium and other brain symptoms can be observed. The effects among industrial workers of chronic lead exposure leading to kidney and renal failure have been well documented. High concentrations of atmospheric lead dust have been found in indoor firing ranges and several cases of mild lead poisoning among shooting instructors have been reported; the lead dust originated both from the bullet cartridge primer and also from partial vaporisation of the bullet itself, both on firing and on fragmentation when impacting a hard target.

18.2.4.3 Silver

Silver azide finds use in specialised detonators. Chronic severe industrial exposure to silver fumes or dusts results in a condition known as argyria, which is a permanent light blue/grey pigmentation of the skin and eyes. The lethal acute dose for man is very high. However, silver nitrate solutions and dusts are both toxic and, even at very low concentrations are irritant to the skin and eyes. Taken orally silver nitrate (AgNO_3) can cause violent abdominal pain, vomiting and even death; the oral LD_{50} in the mouse is estimated to be 50 mg kg^{-1} . This difference between the toxicity of the metallic element and its salts has been recognised by the American Conference of Governmental Industrial Hygienists (ACGIH) which has set the Threshold Limit Value (TLV) at 0.1 mg m^{-3} for metal dust and fumes, and a factor of 10 lower at 0.01 mg m^{-3} for soluble airborne silver compounds.

18.2.4.4 Azides

Exposure to azide is only likely in the manufacturing process of lead or silver azide through contact with the precursor sodium azide. On initiation the azide will convert to nitrogen gas. Most toxicological data in the literature about azides relates to hydrazoic acid or its sodium salt. Inorganic azides are well known poisons. Sodium azide (NaN_3) is about as toxic as sodium cyanide and, like cyanide, is a strong inhibitor of the vital metabolic enzyme cytochrome oxidase; it also binds strongly to haemoglobin and blocks oxygen transport from the blood. Hydrazoic acid, which is formed from aqueous solutions of azide salts by hydrolysis, is a volatile, highly toxic gas with an intolerable pungent odour. Sodium azide is classified as H300 and H310⁽³⁶⁾. Fatal doses can occur at 10 mg kg^{-1} body weight.

In man the acute effects of exposure to hydrazoic acid vapour are eye irritation, headache, a fall in blood pressure, dizziness and collapse. The effects are due to the conversion of iron to iron oxide so that it can no longer fulfil its functions of the transport of oxygen as haemoglobin. No deaths have been reported for workers engaged in the manufacture of lead azide, but mild acute symptoms of hydrazoic acid poisoning are common. Long term exposure of animals to azide vapours has been reported to cause permanent damage to the nervous system but no such chronic effects have been reported in humans.

18.2.5 Toxicity of Chemicals Found in or Associated With Pyrotechnics

18.2.5.1 Screening Smoke

18.2.5.1.1 White phosphorus

White Phosphorus (WP) was commonly used as the main filling in screening smoke munitions but now has limited UK service use. It is found in legacy and foreign ammunition. WP is one of the most highly toxic inorganic substances known. It can be absorbed through the skin, the respiratory system and also by ingestion. Severe symptoms have been reported in man following a single dose of 15 mg. The human LD₅₀ may be as low as 1 mg kg⁻¹. Skin contact with white phosphorus results in very painful burns which can penetrate deep into flesh. The US Department of Health and Human Services produced a Toxicological Profile for white phosphorus and the smoke that it emits in 1997⁽³⁷⁾.

The chronic effects of phosphorus poisoning among industrial workers is well known. Often the symptoms take several years to appear. The classic effect is on bone and the 'phossy jaw', a jaw bone necrosis, was an occupational disease among those involved in the match and firework industries until the use of white phosphorus was abandoned. The substance is classified as H300 Cat 1 (oral).

18.2.5.1.2 Red Phosphorus

Red phosphorus is much less toxic than the white variety. It is formed from white phosphorus by careful anaerobic heating and is a polymeric chain form rather than P₄ pyramids. This changes "toxicokinetics" so that the red form is relatively non-toxic but beware of contamination by white phosphorous. It is also chemically less reactive. White Phosphorus is H300 Cat 1 (Oral) whereas Red Phosphorus is not classified as toxic.

18.2.5.1.3 Phosphine

Phosphine gas, PH₃, can be liberated from both red and white phosphorus in storage if moisture contamination occurs. It has a STEL is 0.3 ppm and causes kidney and usual phosphorous skeletal effects. As an inhalable vapour its absorption by the body can be rapid and it has been used in suicides by ingesting a phosphide rat poison which forms phosphine in the stomach. The LC₅₀ (rat) for 1 hr exposure is 22 ppm. LC₅₀ is the concentration of a chemical in air, usually expressed as ppm or mg m⁻³, that would cause lethality to 50 % of a large group of people exposed to that dose of the chemical. It is important to note that an LC value must relate to a given time of exposure. Emsley⁽³⁸⁾ has provided further information on phosphorus and phosphine toxicity and the toxicology is presented in section 18.3.3.

18.2.5.1.4 Hexachloroethane

Hexachloroethane (HCE) is found in HCE/zinc oxide/aluminium smoke compositions. It is a toxic chemical with a distinctive odour and most countries apply a TWA (8 hr) of 1 ppm. There is still limited use of HCE compositions in UK Service but will certainly be found in legacy munitions.

18.2.5.1.5 Products of Combustion

When phosphorus burns in air phosphorus pentoxide is produced, which in the presence of atmospheric moisture hydrolyses to phosphoric acid (H_3PO_4) or poly- phosphoric acids such as pyrophosphoric acid ($\text{H}_4\text{P}_2\text{O}_7$) and triphosphoric acid ($\text{H}_5\text{P}_3\text{O}_{10}$)⁽³⁹⁾. The American Office of the Army Surgeon General of the USA published recommendations regarding permissible levels to which soldiers should be exposed. The emergency 15 minute concentration was 40 mg m^{-3} with longer exposures having lower limits. They also published data for hexachloroethane smoke, where the main toxic product is zinc chloride, as 10 mg m^{-3} of ZnCl_2 .

Signalling smoke, both white and coloured, consists of solid particles in air. These have to be of a certain size to remain suspended for useful periods of time, a size that puts them in the class of respirable dusts. EH40⁽¹⁾ offers an almost universal upper limit of 10 mg m^{-3} for the 8 hr exposure limit to inhalable dust and 4 mg m^{-3} for respirable dust unless the limit is lower on other grounds. Signalling smokes will be denser than this to be effective.

The smoke itself is formed by evaporating a dye with a cool burning pyrotechnic. However it is inevitable that a small proportion of the organic materials in the smoke will be pyrolysed into unpredictable by-products. The compositions may also contain calcium oxalate which is harmful. In the past some of the dyes used were highly carcinogenic but modern compositions contain much safer materials. Most are anthraquinone based dyes and have an LD_{50} of about 5000 mg kg^{-1} ⁽⁴⁰⁾. Cinnamic acid, frequently used to give a white smoke, is classified as a skin and respiratory irritant⁽⁴¹⁾. The riot control agent CS (2-chlorobenzalmalononitrile) in pellet form is relatively non-toxic. Like signalling smokes it is pyrotechnically evaporated from a canister forming a particulate cloud known as 'tear gas' causing intense irritation of eyes, nose, mouth and throat mucous membranes causing profuse coughing, disorientation, and difficulty breathing, partially incapacitating the subject. It has an LC_{50} estimated as over $50,000 \text{ mg m}^{-3}$ for a one minute exposure but it is intensely irritating at 1 mg m^{-3} .

18.2.5.2 Other pyrotechnics

All pyrotechnics except the phosphorus smokes are fuel-oxidiser mixtures with a wide range of potential ingredients. The toxicity of each ingredient must be considered separately and then all effects combined for the full toxicity classification. The additivity rules for classification are such that it is unlikely that any part of a pyrotechnic mixture will be present in such a small amount that it can be disregarded. It is not the intention to produce a comprehensive guide to the toxicity of such a large number of compounds.

In reality, the toxic hazards associated with pyrotechnic compositions are largely the concern of manufacturers that handle the chemicals. Once filled in to a munition the toxic hazard is no longer present unless the contents leak from the munition body. Nearly all of the ingredients are either crystalline solids or metal powders and so, even if the composition is exposed, there will not be a vapour hazard and then only skin contact must be avoided.

18.2.5.3 Pyrotechnic oxidisers

Inorganic nitrates, such as those of barium, potassium, sodium and strontium are used as oxidisers in pyrotechnic compositions. Inorganic nitrates do not produce methaemoglobinaemia symptoms in adults. The barium, sodium and strontium salts are classified as H302, harmful if swallowed. Potassium nitrate and ammonium nitrate are not classified as toxic, though the latter may cause skin and eye irritation and is found in a number of improvised explosive mixtures (see 16.3).

Other oxidisers include chlorates and perchlorates found in both pyrotechnic and improvised explosive mixtures. Potassium perchlorate and potassium chlorate are both H302 with the chlorate also being H332. In some formulations, particularly delays, uncommon oxidisers such as chromates or dichromates can be found which are H330 (Category 2 by inhalation), H301 (Category 3 if swallowed) and H312 (Category 4 in contact with skin). Permanganates may be found in some compositions and has an LD₅₀ oral of 750 mg kg⁻¹ for potassium permanganate and classified as H302, harmful if swallowed.

18.2.5.4 Pyrotechnic fuels and other ingredients.

Fuels can include non-toxic organic materials such as lactose sugar; metals including aluminium, magnesium, and zirconium; non-metals and metaloids including charcoal, boron and silicon and other materials such as antimony sulphide. Burn rate moderators such as calcium oxalate may be present and other processing aids could also be found. The major toxic hazard from these chemicals is accidental ingestion.

18.2.5.5 Pyrotechnic combustion products

Pyrotechnic compositions are often complex mixtures and when they burn can reach temperatures well in excess of 2000 °C. In most cases standard chemistry will predict the majority of the products, however, due to the complexity of the mixtures there may be a large number of minor products that are difficult to predict. Attempts have been made to quantify the products from some pyrotechnics, however, it is certain that many have not been studied. On functioning all pyrotechnics will create airborne products and so care should be taken to avoid inhaling them.

18.2.6 Combustion Products of High Explosives and Propellants

Any combustion or explosion in a confined space may reduce the oxygen content of the atmosphere to a level too low to support respiration. The oxygen

content in the workplace atmosphere should never be allowed to fall below 18 % by volume at atmospheric pressure, where the normal level is 20.8 %, though this level would be survivable.

Apart from the danger of asphyxiation, the burning of propellants or detonation of high explosives will always result in the production of toxic gaseous combustion products. The most important of these are the oxides of nitrogen (NO and NO₂) and of carbon (CO and CO₂). Some nitrate esters will also produce small amounts of hydrogen cyanide if an excess of oxygen is not available during combustion. Adequate ventilation is thus essential in order to maintain an environmentally acceptable atmosphere.

18.2.6.1 Carbon Dioxide

Carbon dioxide is not an asphyxiant at low levels but it is mildly narcotic at a concentration level of 3 % and according to some reports exposure to 7 %-10 % of CO₂ renders individuals unconscious within minutes even though the atmosphere contains enough oxygen to support life.

18.2.6.2 Carbon Monoxide

Carbon monoxide, a colourless, odourless gas, is universally recognised as being highly toxic. CO is rapidly absorbed through the lungs where it binds tightly to haemoglobin thus reducing ability of the blood to provide oxygen to the cells. There is evidence to support the view that exposure to concentrations of CO in the air much above 100 ppm for several hours will begin to have physiological effects on most individuals. The accepted TLV or WEL (section 18.1.2.2) has been set at half of this value. Carbon monoxide toxicology is covered in greater detail in section 18.3.2.

18.2.6.3 Hydrogen Cyanide

Hydrogen cyanide (HCN) may be produced when burning some of the nitrate esters such as propylene glycol dinitrate, an OTTO Fuel II component, as a monopropellant at up to 0.4 % in exhaust gases. HCN is a highly toxic substance and is a potent inhibitor of the vital respiratory enzyme cytochrome oxidase, thus stopping the utilisation of oxygen by the tissues of the body. At low doses the earliest symptoms are quickening of breathing, headache, confusion and sometimes vomiting and nausea. Hydrogen cyanide is absorbed extremely rapidly through the lungs and high concentrations (several hundred ppm) can prove fatal almost instantaneously.

18.2.6.4 Oxides of Nitrogen

Perhaps the most insidious of combustion products in explosion products are the oxides of nitrogen. These are effective at very low concentrations of producing delayed symptoms of lung damage in a manner similar to that of the chemical warfare agent phosgene. Although thermodynamics predicts that nitrogen gas is the most favourable product of explosion, studies of reaction products have found the presence of nitric oxide and nitrogen dioxide.

Nitric oxide is a colourless odourless gas. It can be formed by combination of nitrogen and oxygen of the air at elevated temperatures. The products of combustion of most fires and the gases produced during firing of high explosives or propellant charges contain varying amounts of nitric oxide. It is converted spontaneously, if slowly, in air to nitrogen dioxide. The WEL of nitric oxide is 25 ppm (30 mg m^{-3}). The chief effect of nitric oxide has been ascribed to the formation of methaemoglobin and subsequent action on the central nervous system. Animal experimentation suggests that nitric oxide is about one fifth as toxic as nitrogen dioxide.

Nitrogen dioxide (NO_2) is a reddish brown gas and in high concentrations is partly associated to the dimer dinitrogen tetroxide (N_2O_4) which is colourless. Varying amounts of nitrogen dioxide result from the detonation of nitro or nitrate explosives. Many deaths from pulmonary oedema, induced by inhalation of high concentrations of nitrogen dioxide, have been reported.

Brief exposure to 250 ppm causes coughing and increased difficulty in breathing; within 2 hours severe pulmonary oedema may develop and death may soon follow. In other cases there may be a latent period of 8 hours or more where the victim feels quite well, but after this interval the onset of pulmonary oedema is rapid. Latent periods of up to several weeks after exposure and then sudden symptoms and death are not unknown. The incidence of chronic effects, from long exposures at low doses, is less well defined: The WEL of nitrogen dioxide is 3 ppm (6 mg m^{-3}).

18.2.6.5 Hydrogen chloride/hydrochloric acid

When composite propellants containing ammonium perchlorate burn the efflux contains hydrogen chloride, which when mixed with moisture in the atmosphere is converted to a hydrochloric acid mist with a STEL (section 18.1.2.2) of 5 ppm.

18.3 TOXICOLOGY

This section is not comprehensive but provides an insight into the more prominent toxicological effects caused by explosives and their explosion products.

18.3.1 Methaemoglobinaemia

The ability of haemoglobin to carry oxygen depends on the iron it contains being in the ferrous state. Methaemoglobin is a changed form of haemoglobin in which the haem iron has been oxidised to the ferric state, which alters the oxygen binding curve so that it will no longer bond to the oxygen. In a normal individual the proportion of methaemoglobin does not exceed about 1 % - 2 % unless the individual smokes cigarettes when this can rise to between 5 % and 10 %.. Compounds containing inorganic and organic nitrogen can oxidise haemoglobin to methaemoglobin. The ferric pigment causes little ill effect in the victim up to a concentration of about 1 g per 100 ml (15 %) but beyond this level cyanosis sets in. First the extremities and then the whole body assumes a bluish hue and the patient complains of fatigue and difficulty in breathing. Symptoms of anoxia, headache and dizziness occur when the methaemoglobin level is 30 % - 40 %; at greater than 40 % vomiting usually occurs; more than 60 % - 70 % is a life endangering situation and death can occur from a breakdown of the respiratory function. Acute methaemoglobinaemia normally reverts at a rate of about 12 % per hour. In chronic cases of exposure to certain compounds the red blood cells can contain inclusions of precipitated methaemoglobin termed 'Heinz bodies'.

18.3.1.1 Organic amines

Aromatic amines such as aniline and its derivatives can be metabolised to hydroxylamines and nitroso compounds⁽⁴²⁾ and produce methaemoglobin with the formation of Heinz bodies. Many of these amino compounds can be absorbed through the skin. Acute exposure symptoms include cyanosis, giddiness, headache, nausea, vomiting and respiratory distress together with neurotoxic effects such as coma and convulsions leading to death. Chronic exposure victims can exhibit weakness, giddiness, loss of appetite, anaemia with occasional Heinz bodies and mild to severe cyanosis. Some amines e.g. acrylamide cause severe neurotoxic lesions, disturbance in gait, hallucinations and muscular atrophy. Exposure to aromatic nitro compounds and amines can also be responsible for liver damage. Among aromatic amines the phenylhydroxylamines are probably the most potent producers of methaemoglobinaemia induced cyanosis.

18.3.1.2 Organic nitro compounds

All organic nitro compounds act very similarly producing, at low concentrations, methaemoglobin, with cyanosis and the appearance of Heinz bodies. In addition, central nervous system and possible liver damage are quite common.

The aromatic nitro compounds oxidise haem by an indirect mechanism and intolerance to alcohol often occurs. Nitrobenzenes can be absorbed through the skin. Typical nitro poisoning symptoms include headache, cyanosis, vomiting, gastric colic, convulsions and coma. The lethal dose taken orally is usually a few millilitres and there is often a delay of several hours before symptoms occur. Nitrophenols inhibit oxidative phosphorylation leading to weight loss and for this reason they were once used in the treatment of obesity but have since been abandoned as being too toxic with damage to liver and kidneys being recorded.

18.3.1.3 Inorganic and organic nitrite and nitrate compounds

Nitrites relax smooth muscle. Sodium nitrite has been used orally, and amyl and ethyl nitrites by inhalation, as vasodilators in relieving angina pains.

Nitrites are very effective in oxidising the ferrous iron of haemoglobin to the ferric state to produce methaemoglobin in a direct reversible process, the mechanism of which is not very well understood. One mole of nitrite will oxidise 1 mole of haem. In contrast to the action of nitro compounds where no Heinz bodies are formed.

Inorganic nitrate is normally pharmacologically ineffective since, at physiological pH, the nitrate ion is a relatively poor oxidising agent and cannot convert haem to the +3 state. However, nitrates can be converted to nitrites by the intestinal flora of infants and cases of methaemoglobinaemia due to very low nitrate concentrations are not uncommon among newborn babies often called the blue baby syndrome.

Organic nitrate esters are very potent vasodilators⁽⁴³⁾, and can be absorbed orally, through the skin or by inhalation. Typical symptoms are:

- a. Fall in blood pressure
- b. increase in heart rate
- c. headache ('nitro-head') and at higher doses
- d. dizziness, nausea, production of methaemoglobin, cyanosis
- e. even sudden death

Alcohol often potentiates the symptoms. Organic nitrates are rapidly broken down in the liver, and act pharmacologically as free nitrite ions produced by reduction by tissue sulphhydryl groups. The typical tolerance that is built up by those regularly exposed to organic nitrates is due to depletion of sulphhydryl groups with a consequent inability to metabolise the nitrate to the active nitrite form. Relatively short respites from exposure to nitrate esters allow the regeneration of free tissue sulphhydryl groups and loss of 'nitro tolerance', which explains the habit of munitions workers of keeping a small piece of propellant in their clothing and handling it during weekends and holidays. Those working with EGDN pursued a similar practice with cloth impregnated with the substance. This tolerance effect probably explains the fact, noted above, that 105 mg kg⁻¹

is the LD₅₀ for NG taken as one dose yet 1058 mg kg⁻¹ day⁻¹ can be tolerated by the test animal for two years with no obvious ill effects.

18.3.2 Carbon Monoxide

Carbon monoxide (CO) is colourless, odourless, tasteless and non-irritating. It is produced during the incomplete combustion of carbon and carbon containing compounds and oxygen deficient explosives. Carbon Monoxide binds to a haemoglobin molecule at the same sites as an oxygen molecule. Bour⁽⁴⁴⁾ states that CO has a binding affinity that is approximately 200-300 times greater than oxygen. If there is CO present it will bind more readily to haemoglobin regardless of the amount of oxygen available. Figure 18-1 shows the dissociation curves for both carbon monoxide-haemoglobin (carboxyhaemoglobin) and oxygen-haemoglobin (oxyhaemoglobin). A much smaller amount of CO is required to reach 100% haemoglobin saturation than oxygen. A normal partial pressure of oxygen is about 160 mm Hg. The curve demonstrates why low oxygen levels can be tolerated in the absence of carbon monoxide as the affinity of haemoglobin (Hb) for oxygen only starts to decline significantly below about 10% oxygen (80 mm Hg partial pressure).

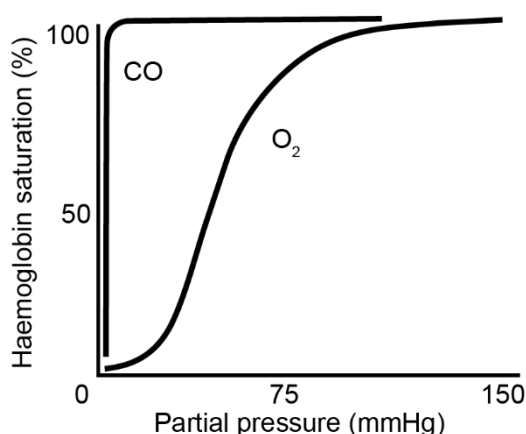


Figure 18-1 Carboxyhaemoglobin and Oxyhaemoglobin Dissociation Curves⁽⁴⁵⁾.

Oxygen usually binds 'imperfectly' to Hb. If it bound strongly it would not leave the haemoglobin when it was required to diffuse into the tissues it supplies⁽⁴⁶⁾. Once one CO molecule is bound to a Hb molecule, it then gives Hb a higher affinity for O₂⁽⁴⁷⁾. The oxyhaemoglobin curve shifts to the left⁽⁴⁵⁾ and the affinity is great enough that the O₂ will not leave the Hb to enter the tissues and supply them with the O₂ they need to survive. So a combination of fewer O₂ molecules bound to the Hb and a higher affinity of haemoglobin for oxygen is what causes the hypoxia, the deprivation of tissues of oxygen⁽⁴⁷⁾. Once one molecule of CO is bound to a Hb molecule, Hb's affinity to CO will drop by two orders of magnitude⁽⁴⁶⁾. This protects the body against poisoning by CO naturally produced in the body as this would take up a lot of the binding spaces if the affinity remained as high for all four sites per Hb molecule.

J. S. Haldane was the first person to confirm that the binding affinity of Hb is far greater for CO than oxygen. Haldane studied carbon monoxide and its effects on the body, usually his own body or those of his children, in great detail in the 1890s. He would subject himself to varying levels of carbon monoxide and note down the effects it had on him. He concluded “the effects to be similar to those produced by the lack of O₂”⁽⁴⁸⁾. In partnership with Lorrain-Smith, Haldane developed what is now known as the Haldane Principle shown in Equation (18-1)⁽⁴⁹⁾.

$$\frac{[\text{HbCO}]}{[\text{HbO}_2]} = \frac{M \cdot P_{\text{CO}}}{P_{\text{O}_2}} \quad (18-1)$$

This equation, taken from McGrath⁽⁴⁸⁾, represents the relationship between haemoglobin, oxygen and carbon monoxide in blood. [HbCO] and [HbO₂] represent the concentration in blood of CO and O₂ respectively, P_{CO} and P_{O₂} represent the partial pressures of CO and O₂ and M is the Haldane constant, which is the ratio of the affinity of blood for CO to that for O₂, and is approximately 250.

18.3.3 Phosphorus and Phosphine.

White phosphorus is extremely toxic whereas red and other forms such as black phosphorus are not classed as being toxic. White phosphorus is a small pyramidal molecule made of 4 phosphorus atoms whereas the other forms are polymeric chains. This makes them not susceptible to spontaneous ignition in air and not toxic. Both white and red phosphorus will produce phosphine when damp. Phosphine is a gas and thus has an inhalational route of entry.

It is important to separate the acute and chronic effects of phosphorus and phosphine. Unless it is ingested most exposure to phosphorus will be low level and chronic. Acute exposure to phosphine is possible and can lead to heart and lung damage as phosphine interferes with enzymes and protein synthesis. The US Agency for Toxic Substances and Disease Registry (ATSDR), in its leaflet about phosphine states⁽⁵⁰⁾ “Most deaths occur within the first 12 to 24 hours after exposure and are cardiovascular in origin. If the patient survives the first 24 hours the ECG typically returns to normal indicating that heart damage is irreversible. Deaths after 24 hours are usually due to liver failure.”

Phosphine is also a central nervous system depressant. The Health Protection Agency (HPA) note that pure phosphine is odourless and that it is impurities that lead to the characteristic odour somewhere between garlic and decaying fish⁽⁵¹⁾. This means that the quoted odour threshold of 0.1 to 0.2 ppm (0.14 to 0.28 mg m⁻³) is an unreliable indicator that phosphine is present and is not detected until just below the UK STEL (see 18.1.2.2) (15 minutes) of 0.3 ppm. The HPA say that 400 to 600 ppm of phosphine will be fatal after an exposure of 30 minutes to an hour⁽⁵¹⁾. They note that post-mortem evidence from 26 deaths found that lung oedema was the most common cause of death.

Chronic exposure to low levels of phosphine can cause bronchitis, gastrointestinal problems, visual and speech problems and the skeletal defects in the jaw and teeth noted from white phosphorus.

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Appendix A GHS Classification

By 2015 all classification on Safety Data Sheets (SDS) should be as H statements. However, there are still many Material Safety Data Sheets (MSDS) in use that include risk (R) phrases and some safety documents include both R and H Statements together. The boundaries of the H statements are given below.

Acute Toxicity, Oral, LD₅₀ (mg/kg)

1. H300 Category 1, 0 to less than or equal to 5
2. H300 Category 2, 5 < to less than or equal to 50
3. H301 Category 3, 50 < to less than or equal to 300
4. H302 Category 4, 300 < to less than or equal to 2000

Acute Toxicity, Dermal, LD₅₀ (mg/kg)

1. H310 Category 1, 0 to less than or equal to 50
2. H310 Category 2, 50 < to less than or equal to 200
3. H311 Category 3, 200 < less than or equal to 1000
4. H312 Category 4, 1000 < less than or equal to 2000

Acute Toxicity, Gases, (ppm volume).

These are materials which are above or very close to their boiling point at the temperature of exposure (see below). This is not the same as the definition of a gas as a substance that is above its critical temperature.

- 1 H330 Category 1, 0 to less than or equal to 100 (0 to 0.01 %)
- 2 H330 Category 2, 100 < to less than or equal to 500 (0.01 % to 0.05 %)
- 3 H331 Category 3, 500 < to less than or equal to 2500 (0.05 % to 0.25 %)
- 4 H332 Category 4, 2500 < to less than or equal to 20000 (0.25 % to 2 %)

n.b. For many flammable gases the high end of category 4 will be in the explosive range.

Acute Toxicity, Vapours, (mg litre⁻¹).

A vapour is the gaseous form of a substance released from its liquid or solid state with the provision that if the liquid is so close to its boiling point that it cannot be held as a liquid in the open it should be considered as a gas.

1. H330 Category 1, 0 to less than or equal to 0.5 (0 to 500 mg m⁻³)
2. H330 Category 2, 0.5 < to less than or equal to 2 (500 to 2000 mg m⁻³)
3. H331 Category 3, 2 < to less than or equal to 10 (2000 to 5000 mg m⁻³)
4. H332 Category 4, 10 < to less than or equal to 20 (5000 to 20000 mg m⁻³)

Acute Toxicity, Aerosols, Dusts and mists (as mg/l).

A dust is a suspension of solid particles in air; a mist is a suspension of droplets in air. Both may have vapour associated with them but the concentration of the vapour from a mist will be generally higher than from a dust.

- 1 H330 Category 1, 0 to less than or equal to 0.05 (0 to 50 mg m⁻³)
- 2 H330 Category 2, 0.05 < to less than or equal to 0.5 (50 to 500 mg m⁻³)
- 3 H331 Category 3, 0.5 < to less than or equal to 1 (500 to 1000 mg m⁻³)
- 4 H332 Category 4, 1 < to less than or equal to 5 (1000 to 5000 mg m⁻³)