A Toxicological Review of the Products of Combustion

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ABSTRACT

The Chemical Hazards and Poisons Division (CHaPD) is frequently required to advise on the health effects arising from incidents due to fires. The purpose of this review is to consider the toxicity of combustion products. Following smoke inhalation, toxicity may result either from thermal injury, or from the toxic effects of substances present. This review considers only the latter, and not thermal injury, and aims to identify generalisations which may be made regarding the toxicity of common products present in fire smoke, with respect to the combustion conditions (temperature, oxygen availability, etc.), focusing largely on the adverse health effects to humans following acute exposure to these chemicals in smoke.

The prediction of toxic combustion products is a complex area and there is the potential for generation of a huge range of pyrolysis products depending on the nature of the fire and the conditions of burning. Although each fire will have individual characteristics and will ultimately need to be considered on a case by case basis there are commonalities, particularly with regard to the most important components relating to toxicity.

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EXECUTIVE SUMMARY

The Chemical Hazards and Poisons Division (CHaPD) is frequently required to advise on the health effects arising from incidents due to fires. The purpose of this review is to consider the toxicity of combustion products. Following smoke inhalation, toxicity may result either from thermal injury, or from the toxic effects of substances present. This review considers only the latter, and not thermal injury, and aims to identify generalisations which may be made regarding the toxicity of common products present in fire smoke, with respect to the combustion conditions (temperature, oxygen availability, etc.), focusing largely on the adverse health effects to humans following acute exposure to these chemicals in smoke.

The gaseous products formed during the combustion of most organic materials can be classified into two main categories on the basis of their toxicity either as asphyxiants or as irritants with a third category used to describe toxic products not falling within the two main categories.

The first category relates to fire smoke components which have asphyxiant properties and also oxygen depletion due to the fire itself. These asphyxiant gases can give rise to narcosis due to central nervous system depression. Exposure to these combustion products at sufficient concentration or duration of exposure can lead to unconsciousness and eventually death, due to tissue hypoxia. The principle asphyxiants produced during the combustion of organic materials are carbon monoxide, hydrogen cyanide and carbon dioxide together with low oxygen concentration which has similar effects as the asphyxiant gases. These asphyxiants can interact producing additive effects, resulting in higher toxicity.

The second category relates to the smoke components which cause irritation, either as sensory irritants affecting the eyes and upper respiratory tract (nose, mouth and throat), or as pulmonary irritants (affecting the lungs), although in many cases sensory and pulmonary irritation may be present simultaneously. The combustion of most commonly used materials, ranging from natural sources such as wood, to synthetic plastics and polymers will result in the generation of irritant gases. Therefore, irritant gases are present in most fire atmospheres, irrespective of whether the combustion is smouldering, flaming or ventilation-controlled. Irritant gases produced during combustion can commonly be divided into two main classes relating to their chemical composition, either as inorganic irritant gases or as organic irritants. The most common inorganic acid gases evolved during combustion include the halogen acids (HCl, HF, HBr) and oxides of sulphur, nitrogen and phosphorous. Other inorganic irritants present in combustion atmospheres include ammonia, chlorine and phosgene. The incomplete combustion of materials including; wood, fossil fuels, synthetic and natural polymers and foodstuffs will give rise to the formation of organic irritants, such as acrolein and formaldehyde. The injury following exposure to an irritant gas depends upon the chemical involved, its concentration, the exposure duration and its solubility. However, the initial effect of exposure to these irritant gases is likely to be sensory irritation.

A third category may often be used to describe products which may give rise to toxicity unlike that covered by the two main categories. In many cases, the combustion of

organic materials, particularly if it is incomplete, may also give rise to more complex molecules in the smoke plume which may typically include longer carbon chains and carbon-rings. Some of the compounds considered include polycyclic aromatic hydrocarbons (PAHs), dioxins, dibenzofurans, isocyanates, perfluoroisobutylene (PFIB) and particulate matter (PM). This third category may also include toxic chemicals which may be present at the site of the fire and do not undergo decomposition in the combustion atmosphere as may occur at a specialised chemicals factory.

The likelihood of the presence of any of these toxic combustion products being formed during fires involving specific materials and under differing combustion conditions such as temperature, ventilation and stage of pyrolysis or combustion was considered.

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1 INTRODUCTION

A comprehensive review of the toxicology of combustion products was conducted to support the Health Protection Agency (HPA) strategic goals through Programme 2; 'To protect against the adverse health effects of acute and chronic exposure to chemicals, poisons and other environmental hazards'.

The Chemical Hazards and Poisons Division frequently has to advise on the health effects arising from incidents due to fires. The purpose of this review is to consider the toxicity of combustion products. Following smoke inhalation, toxicity may result either from thermal injury, or from the toxic effects of substances present. This review only considers the latter, and not thermal injury.

The pyrolysis and combustion of materials can result in the generation of many toxic smoke products which cause irritation, incapacitation, systemic toxicity, asphyxiation and may be lethal following acute exposures. Some of the common toxic chemicals which may be present in a fire effluent include asphyxiant gases, such as carbon monoxide (CO) and hydrogen cyanide (HCN), irritant gases such as hydrogen chloride (HCl) oxides of nitrogen (NO_x), acrolein and phosgene, and complex molecules such as polycyclic aromatic hydrocarbons (PAHs). The amounts of toxic products evolved during combustion vary with the type of combustion, the availability of oxygen, the temperature and the materials involved. Therefore, the conditions of combustion will affect the severity of the adverse health effects in those exposed to the products of combustion.

This is a complex area and there is the potential for generation of a huge range of pyrolysis products depending on the nature of the fire and the conditions of burning. Although each fire will have individual characteristics and need to be considered on a case by case basis there are commonalities, particularly with regard to the most important components relating to toxicity. This review aims to identify generalisations which may be made regarding the toxicity of common products present in fire smoke, with respect to the combustion conditions (temperature, oxygen availability, etc.) and materials involved, focusing largely on the adverse health effects to humans following acute exposure to these chemicals in smoke.

Consideration is given first to the general types of fire with respect to ventilation and temperature which are key factors governing the type of pyrolysis products formed. This is followed by a section giving consideration of the important toxic substances that may be produced, divided into general classes such as asphyxiant gases, irritant gases (inorganic and organic) and complex organic compounds and also includes consideration of specially sensitive groups in the population. This is followed by a section on the characteristics of fires from different combustible materials with regard to the type of toxic substances produced. Consideration is then given to how smoke and the pyrolysis products produced may be dispersed. Finally there is a conclusions section.

This document is intended as a scientific review of the toxicology of combustion products and does not cover the detailed precautions that may be appropriate in specific circumstances. However, most of the key chemicals involved, are covered in the

CHaPD Compendia of Chemical Hazards which contains a section on incident management. This section provides information that may be needed by health professionals during a chemical incident, including information on hazards and precautions that may be appropriate (eg, CHIP classification, risk phrases and safety phrases).

The Compendia of Chemical Hazards can be found at:
www.hpa.org.uk/HPA/Topics/ChemicalsAndPoisons/CompendiumOfChemicalHazards

2 TYPES OF FIRES

The thermal breakdown of materials can occur under a number of different conditions, which can affect how complete the degradation of the material may be. Thermal degradation can be classified into the main types described below, together with a few general comments on the principal products generated from the toxicological perspective.

2.1 Flaming combustion, well ventilated

Thermal breakdown and chemical conversion of materials in a “normal oxygen” environment in the presence of flaming can be described as well ventilated, flaming combustion. Flaming combustion is the highly efficient burning of materials above the auto-ignition temperature in the presence of sufficient oxygen [1]. Complete combustion occurs when an organic material is converted by oxidation into only carbon dioxide and water in a high oxygen environment. Flaming combustion of a material in a normal oxygen environment produces complete oxidation of that material. However, in most cases, carbon dioxide and water will not be the only products of combustion under these conditions, due to the presence of other elements in the atmosphere and in the material that is acting as fuel for the fire. For example, in a normal oxygen environment, nitrogen in the combustion atmosphere will also undergo oxidation to form nitrogen dioxide. Initially, under the conditions of well ventilated combustion, the production of smoke and toxic compounds tends to be low, with more toxic products formed as the fire develops [2].

2.2 Flaming combustion, ventilation-controlled

Thermal breakdown and chemical conversion of materials in a low oxygen environment in the presence of flaming can be described as ventilation-controlled, flaming combustion. Ventilation-controlled fires occur when the air supply is restricted compared to the amount of fuel available for combustion. Most fires in confined spaces (such as in buildings) become ventilation-controlled fairly early on in the stages of combustion. Ventilation-controlled fires may be either small enclosed fires, or large post-flashover fires. Flashover is a term often used to describe the stage in a fire when all of the

combustible gases and materials in the vicinity ignite simultaneously at high temperature. The reduction in oxygen present in these types of fires leads to the production of high yields of carbon monoxide, carbon dioxide, hydrogen cyanide, other organic and inorganic gases and smoke [1, 2].

2.3 Oxidative pyrolysis (smouldering)

Oxidative pyrolysis, also known as smouldering, can be described as the thermal breakdown and chemical conversion of materials in a normal oxygen environment, but in the absence of flaming. Smouldering combustion progresses at a much slower rate than flaming combustion, most commonly involves a porous fuel material and is sustained by the heat given off during oxidation at the fuel surface [3]. Pyrolysis occurs at much lower temperatures than flaming combustion and is commonly defined as the thermal degradation of a material at a temperature below the auto-ignition temperature [1]. During pyrolysis, oxidation occurs at the surface of the solid material, in contrast to within the gas-phase around the material as occurs during flaming combustion. Smouldering combustion may occur in the initial stage of a fire and can provide a pathway to flaming combustion from a heat source which is insufficient to directly produce a flame [3]. Smouldering is a form of incomplete combustion due to the lower temperatures involved, and therefore may yield a much greater quantity of toxic products than flaming combustion [2, 3].

2.4 Anaerobic pyrolysis

Anaerobic pyrolysis can be described as thermal breakdown and chemical conversion of materials in a low oxygen environment and the absence of flaming [1]. This type of combustion may be initiated as oxidative pyrolysis, but can continue in a low oxygen environment, such as may occur following depletion of oxygen during pyrolysis in a closed compartment. In this situation, the higher the temperature of the combustion environment, the lower the amount of oxygen required. Anaerobic pyrolysis is similar to smouldering combustion in that the chemical conversion is incomplete and will therefore produce much greater quantities of toxic products than flaming combustion. However, due to the further limitation of oxygen in the case of anaerobic pyrolysis, this situation may therefore yield more toxic compounds than oxidative smouldering. There is no conclusive sign to distinguish the progression from oxidative pyrolysis to anaerobic pyrolysis, and therefore, both cases of pyrolysis are commonly described together as smouldering or non flaming fires [2, 3].

These types of fires, whilst having individual definitions, may occur simultaneously at different locations within the fire environment, or one of the types of fire may be most prevalent [1].

2.5 Composition of smokes

The composition of smoke in a combustion environment depends on a large number of variables and can be extremely complex due to the dynamic nature of a fire. Some of

the principal factors which can affect smoke composition include the nature of the fuels involved (chemical composition, structure and formulation), the stage of combustion (smouldering, flaming or post-flashover), the temperature of combustion and the availability of oxygen and ventilation in the fire environment [1, 4, 5].

3 COMMON TOXIC COMBUSTION PRODUCTS

The gaseous products formed during the combustion of most organic materials can be classified into two main categories on the basis of their toxicity. The first category relates to fire smoke components which have asphyxiant properties and also oxygen depletion due to the fire itself. The second category relates to the smoke components which cause irritation, either as sensory irritants affecting the eyes and upper respiratory tract (nose, mouth and throat), or as pulmonary irritants (affecting the lungs), although in many cases sensory and pulmonary irritation may be present simultaneously. A third category may occasionally be used to describe products which may give rise to toxicity unlike that covered by the two main categories. There are, however, few documented cases of specific toxic combustion products which fall into this third category [6]. Also considered in this third category are toxic chemicals which may be present at the site of the fire and do not undergo decomposition in the combustion atmosphere. This may be an important consideration in scenarios such as a fire at a specialised chemicals factory.

3.1 Asphyxiant gases

Asphyxiant gases produced during combustion can give rise to narcosis due to depression of the central nervous system. Exposure to these combustion products at sufficient concentration or duration of exposure can lead to unconsciousness and eventually death, due to tissue hypoxia [6, 7]. The principal asphyxiants produced during the combustion of organic materials are carbon monoxide, hydrogen cyanide and carbon dioxide together with low oxygen concentration [1, 2, 6, 7]. The effect of these can interact producing additive effects resulting in higher toxicity. Table 1 at the end of this section gives occupational exposure limits and acute emergency guideline levels for the substances considered.

Chemical asphyxiants prevent the normal uptake of oxygen by tissues by interfering with specific elements in oxygen delivery and metabolic processes. Thus carbon monoxide and hydrogen cyanide are chemical asphyxiants. Simple asphyxiants are physiologically inert gases that, if inhaled, displace oxygen from the alveoli and lead to hypoxia. Carbon dioxide, nitrogen and methane are considered to be simple asphyxiants. Breathing a reduced concentration of oxygen also has this effect, but is not considered as a simple asphyxiant. The reduced concentration of oxygen is due to the presence of other gas assuming the barometric pressure is unchanged.

3.1.1 Carbon monoxide (CO)

Carbon monoxide (CO) is the most common asphyxiant product in most fire environments and is formed during both smouldering and flaming combustion of all organic materials [6, 8]. The production of CO in a fire is dependent upon the availability of oxygen in the combustion environment, with an increase in CO formation with decreasing availability of O_2 [6]. The production of CO is therefore, greater in cases of ventilation-controlled combustion, than with well ventilated combustion. At the point of flashover, the production of CO increases significantly, due to the combustion becoming ventilation-controlled and the rapid increase in the mass burning rate [6].

The toxicity of CO is due to a reduction in the oxygen-carrying capacity of the blood, known as hypoxia [1, 6, 9]. Hypoxia following exposure to CO results from the conversion of haemoglobin to carboxyhaemoglobin (COHb) due to competition between O_2 and CO for the haem-binding sites [6]. The main factor for the toxicity of CO is widely regarded to be that the affinity of haemoglobin for CO is around 200 – 250 times greater than the affinity for O_2 [1, 6, 10]. As such, once COHb is formed it will not be readily displaced by O_2. Therefore, only partial conversion of haemoglobin to COHb significantly reduces the oxygen carrying capacity of the blood [1, 6]. The formation of COHb also leads to a left shift in the dissociation curve of oxyhaemoglobin: the oxygen that is bound will have a greater affinity for the haemoglobin and will be released less readily at the tissues [6, 10]. This alteration in oxygen dissociation from haemoglobin resulting from COHb formation further increases the likelihood of hypoxia. The concentration of COHb in the blood will be elevated in most cases in individuals exposed to a combustion atmosphere, with the concentration of COHb being dependent upon the duration of exposure and the concentration of CO in the fire environment. The presence of COHb in low concentrations in the blood of individuals exposed to a fire atmosphere may be difficult to interpret, since environmental factors (eg, an urban environment or tobacco smoking) may be associated with raised levels of COHb [1]. Most fatalities following inhalation of CO, have reported the concentration of COHb in the blood to be greater than 50% [1, 11, 12]. A post-mortem COHb concentration of greater than or equal to 70% following acute exposure to CO can be associated with fatality caused directly by CO poisoning alone. A COHb concentration in the range of 30 – 70%, is likely to be associated with cause of death being due to a combination of both CO poisoning and other factors, such as the presence of additional toxic combustion products. A fatality in which there is a COHb concentration of less than 30%, the main cause of death is likely to be due to effects other than CO poisoning [13].

Acute health effects resulting from CO induced hypoxia at concentrations below that causing lethality can include neurological effects such as, headache, dizziness, confusion, disorientation, loss of coordination, memory loss, fainting, cerebral oedema and coma [1, 14]. These neurological effects of CO exposure in a fire environment may hinder the ability to perform tasks, recognise danger and escape from a hazardous fire situation [1]. Neurological symptoms following severe acute toxicity may appear 2-40 days post exposure, including lethargy, irritability and lack of concentration, and possible severe effects including dementia and pshychosis, which may not all be related to CO induced hypoxia [12]. Inhalation of CO is also likely to give rise to metabolic acidosis [1, 14]. The heart is particularly sensitive to the effects of CO, and acute exposure may give

rise to cardiovascular effects including reduced myocardial function, hypotension, vasodilation, cyanosis, cardiac arrhythmias, shock, circulatory failure and cardiac arrest [15, 16]. Pregnant women, the foetus *in utero* and newborn infants are at an increased risk from CO exposure. CO readily crosses the placenta and binds to foetal haemoglobin with a higher affinity than for maternal haemoglobin. CO is also cleared from foetal blood much slower than from maternal blood, resulting in 10-15% increase in COHb formation in the foetus relative to the mother [12].

3.1.2 Hydrogen cyanide (HCN)

Any organic material containing carbon and nitrogen will produce hydrogen cyanide (HCN) during combustion under most conditions. Polymers such as nylons, polyurethanes and polyacrylonitrile are all known to be prominent sources of HCN on thermal decomposition or combustion [1, 17, 18]. The yield of HCN from the combustion of materials containing nitrogen is dependent upon the temperature and availability of oxygen in the fire environment. In an oxygen-limited environment, the generation of HCN is initiated at high temperatures, with the yield increasing with further increases in temperature. In air, the evolution of HCN begins at much lower temperatures than in an anaerobic environment, with the yield increasing with temperature, up to around 625°C. However, at temperatures in the range of approximately 625 - 925°C the yield of HCN decreases, followed by a secondary increase as the temperature rises above this range [1, 19]. The generation of HCN has been shown to be proportional to the nitrogen content of the polymer involved in the combustion, in one study [20]. However, there is also evidence to suggest that the yield of HCN may not be proportional to the nitrogen content of the burning material since the yield of HCN from polyacrylonitrile (1500 ppm) has been shown to be around 7.5 times greater than from wool (200 ppm), although the nitrogen content of the two materials is similar (19% and 14.3%, respectively) [18].

Following exposure and systemic uptake of HCN, it undergoes dissociation in the blood, to form the cyanide ion. The cyanide ion is readily distributed within the body and is responsible for the toxicity of HCN by reducing the cellular utilisation of oxygen (cellular respiration). The cyanide ion binds to cytochrome oxidase, which is a principal enzyme involved in the utilisation of oxygen in most cells throughout the body, and inhibits it by forming a cytochrome oxidase-cyanide complex. The inhibition of cytochrome oxidase results in a rapid onset of cytotoxic hypoxia and loss of cellular function. The cardiac and cerebral tissues are particularly susceptible to the effects of cyanide on cellular respiration. The most common cause of death from HCN intoxication is due to depression of the respiratory system resulting from the cytotoxic hypoxic effect of the cyanide ion on the central nervous system, but effects on the cardiovascular system may also be a cause of death [6]. Some features of acute exposure to hydrogen cyanide at below fatal concentrations may include headache, nausea, dizziness, confusion, muscle weakness, loss of coordination, hyperventilation, cardiac arrhythmia, bradycardia, rapid loss of consciousness and coma [1, 6, 21]. These acute effects of HCN inhalation may impede the escape from a fire environment.

The concentration of HCN which is fatal to humans following inhalation is dependent upon the duration of exposure. It has been widely reported that a concentration of 130

ppm for 30 minutes is likely to be fatal, a concentration of 180 ppm HCN is likely to be fatal after just 10 minutes, and a HCN concentration of 270 ppm is considered to be immediately fatal [21, 22]. A blood cyanide concentration of greater than 1μg/ ml in blood samples taken post mortem from fire fatalities is considered to suggest significant toxicity of HCN. Blood cyanide levels of 3μg/ml or greater are considered to be lethal levels of cyanide [6]. However, measurements of blood cyanide levels can be problematic and analysis should be cautious with respect to factors including the time between sample removal and analysis, and the storage method [23].

The majority of fire fatalities cannot be attributed to inhalation of HCN alone, since in a combustion atmosphere where HCN is being generated, many additional toxic products are also likely to be evolved. CO is produced in all fires involving organic materials and there is likely to be an interaction between CO and HCN, causing hypoxia by two separate mechanisms. In a combustion environment where both CO and HCN are present at sublethal doses, the combination of the two vapours have an additive effect which can prove to be fatal [6, 24, 25]. Hyperventilation resulting from acute inhalation of HCN could also give rise to increased toxicity of CO, reducing the time to death by increasing the amount of CO respired [1]. Therefore, in many cases of fire fatalities resulting from smoke inhalation it is difficult to isolate a cause of death to one chemical in particular from COHb and blood cyanide measurements, when it is likely to be due to a combined effect from both chemicals.

3.1.3 Carbon dioxide (CO_2)

Carbon dioxide (CO_2) is generated in all fires involving organic materials, although its rate of production is largely dependent upon the availability of oxygen in the fire environment. As the level of oxygen present in a fire environment diminishes, there is a shift from production of CO_2 to CO. In this respect, the highest rate of generation of CO_2 occurs when combustion is complete due to sufficient ventilation [5].

An increase in the amount of CO_2 inhaled at concentrations such as those likely to be generated in a fire, is not considered to cause significant toxicity on its own. However, the inhalation of CO_2 in a fire atmosphere will give rise to physiological effects which enhance the toxicity of other combustion products. An increase in CO_2 concentration will stimulate the rate and depth of respiration, increasing the respiratory minute volume [6]. This leads to an increase in the amount of any toxicants present which are inspired over a given period. A concentration of 2% CO_2 has been shown to increase the respiratory minute volume by 50%, whilst 10% CO_2 may increase the volume of air respired in one minute by as much as 10-fold [6]. Human volunteers exposed to 7.5% CO_2 for 15 minutes have also reported difficulty breathing, headache, sweating, increased heart rate, restlessness, disorientation and visual distortion [26]. Prolonged exposures over a few hours to similar concentrations of CO_2 (7 to 10%) can cause the onset of severe distress, nausea and may lead to a loss of consciousness [2].

The presence of CO_2 has been shown to potentiate the toxicity of CO, by increasing the formation of COHb due to an increase in the volume of CO inspired [1, 27]. Additionally, exposure to elevated levels of CO_2 will give rise to respiratory acidosis, which in

conjunction with metabolic acidosis caused by CO will result in severe acidosis, with a prolonged recovery period following cessation of exposure [27].

3.1.4 Low oxygen concentration

In a combustion atmosphere, as oxygen is consumed by the fire, the level of oxygen is depleted, particularly if the fire is in a closed environment. The depletion of oxygen below normal levels (21%) in a fire environment can give rise to adverse health effects. Therefore, a reduction in oxygen levels should also be considered to be a toxic product of combustion [6]. A reduction in the concentration of oxygen of only 4% (17% O_2) can lead to an impairment of motor co-ordination. Further reduction of the level of oxygen in the combustion environment to around 14 to 10%, can lead to fatigue and an increased likelihood of making faulty judgements. These effects of low oxygen concentration could seriously hinder the escape from a hazardous fire situation. If the level of oxygen falls below approximately 10%, it is likely that an exposed individual will become unconscious and to prevent fatality will require immediate removal to fresh air or treatment with oxygen [6, 28].

The effects of a reduction of the oxygen concentration in a combustion environment are due to hypoxic hypoxia caused by a decrease in the partial pressure of oxygen (PO_2) in the arterial blood [1]. Depletion of oxygen in a fire environment could potentially enhance the toxicity of the asphyxiant gases produced during combustion. As CO and HCN also give rise to hypoxia, a reduction in the level of atmospheric oxygen will be likely to reduce the concentration at which significant toxicity may be observed, compared to a normal oxygen environment, due to a further reduction in the availability of oxygen for cellular respiration. Low oxygen concentration in addition to raised carbon dioxide levels has a marked effect on breathing. Physical exertion such as may be required to escape a hazardous fire environment will increase the individuals demand for oxygen, which may also accelerate the onset of hypoxia [6].

Table 1: Worker Exposure Limits (WEL) and Acute Emergency Guideline Levels (AEGL) for asphyxiant gas combustion products.

	WEL [29](ppm)		AEGL – 2 / AEGL – 3 [30](ppm)				
	LTEL	STEL	10 min	30 min	60 min	4 hr	8hr
CO†	30	200	420 / 1700	150 / 600	83 / 330	33 / 150	27 / 130
HCN	-	10	17 / 27	10 / 21	7.1 / 15	3.5 / 8.6	2.5 / 6.6
CO_2	5000	15000	Data not available				

WEL = Workplace Exposure Limits, LTEL = Long Term Exposure Limit (8 hour Time Weighted Average), STEL = Short Term Exposure Limit (15 minute reference period), AEGL – 2 = Acute Exposure Guideline Level – 2 (The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape), AEGL – 3 = Acute Exposure Guideline Level – 3 (The level of the chemical in air at or above which the general population could experience life-threatening health effects or death), † = Interim AEGL.

3.2 Irritant gases

The combustion of most commonly used materials, ranging from natural sources such as wood, to synthetic plastics and polymers will result in the generation of irritant gases. Therefore, irritant gases are present in most fire atmospheres, irrespective of whether the combustion is smouldering, flaming or ventilation-controlled [6, 10]. The irritant gases evolved and the rate of generation may however, depend upon the mode of combustion, relative to temperature and ventilation. Irritant gases produced during combustion can be divided into two main classes relating to their chemical composition, either as inorganic acid gases or as organic irritants. The injury following exposure to an irritant gas depends upon the chemical involved, its concentration, the exposure duration and its solubility. However, the initial effect of exposure to these irritant gases is likely to be sensory irritation [6, 10]. Irritation of the eyes will cause pain and stinging of the eyes, initiation of a blinking reflex and lachrimation [6]. The severity of sensory irritation is dependent upon the concentration of the irritant present, and is independent of the exposure duration. An individual exposed to irritant gases in a combustion atmosphere with the effect of stinging or burning of the eyes and throat may shut their eyes and hold their breath to alleviate the irritation, hindering their ability to escape from the hazard [6]. An additional characteristic sign of exposure to irritant gases is a burning sensation of the mucous membranes of the upper respiratory tract, including the nose, mouth and throat [2, 6, 10]. Pulmonary irritation will commonly occur following sensory irritation, due to inhalation of the irritant gas into the lungs. This irritation of the lungs gives rise to bronchoconstriction, coughing and breathing difficulties. Unlike sensory irritation, the severity of pulmonary irritation is dependent upon both the concentration and the duration of exposure to the irritant gas. Exposure to high concentrations of irritant gases can cause inflammation of the lung tissues, pulmonary oedema and could potentially be fatal in a period of between 6 and 48 hours after removal from the exposure [6].

3.2.1 Inorganic acid gases

The most common inorganic acid gases evolved during combustion include the halogen acids (HCl, HF, HBr) and oxides of sulphur, nitrogen and phosphorous [2]. The degree of generation of these gases during combustion will, however, be dependent upon the chemical composition of the materials involved.

3.2.1.1 Hydrogen chloride (HCl)

Hydrogen chloride (HCl) is considered to be the most important halogen acid gas which may be evolved during combustion [6]. Due to the chlorine content in many commonly used materials, including plastics and polymers such as polyvinylchloride (PVC), combustion of these materials will be likely to result in the generation of HCl. In an experimental situation, PVC has been shown to release as much as 50% of its weight in HCl upon combustion [31].

HCl is a strong sensory and respiratory irritant, with the main targets being the eyes, skin, nose, mouth, throat and trachea. Exposure to HCl at 35 ppm has been reported by one study to induce sneezing, chest pain, hoarseness, laryngitis and a feeling of suffocation [28, 32]. Inhalation of HCl at around 50 ppm has been reported to be strongly irritating to the eyes, nose and throat, including pain, coughing, inflammation and oedema of the upper respiratory tract and concentrations of around 100 ppm have been described as being extremely irritating and excruciatingly painful to the upper respiratory tract [2, 6]. Exposure to HCl in the range of around 50 to 100 ppm is considered to be barely tolerable [28]. Due to the high water solubility of HCl, irritation of the upper respiratory tract occurs predominantly. However, pulmonary irritation can occur following exposure to higher concentrations of HCl resulting in damage to the alveolae and pulmonary oedema [10]. Inhalation of high concentrations of HCl has also been associated with corrosive burns to the eyes, nose, mouth and throat, ulceration of the nasal septum, tachypnoea, bronchoconstriction and laryngeal spasm which may lead to suffocation [2, 6, 32]. Exposure to high levels which are sufficient to produce pulmonary toxicity may lead to the development of Reactive Airways Dysfunction Syndrome (RADS), which is a form of irritant induced asthma. Brief exposure of humans to concentrations of HCl in the region of 1000 to 2000 ppm can be regarded as being dangerous and may potentially be fatal [2].

3.2.1.2 Hydrogen bromide (HBr)

Hydrogen bromide (HBr) is another gas in the group of halogen acids which may be a product generated during combustion, particularly of synthetic polymeric materials. Flame retardant compounds containing halogens are commonly added to many plastics and polymers to impede its ability to burn, an example being brominated flame retardants such as decabromodiphenyl ether (deca-BDPE) used in high impact polystyrene (eg, television casings) [33, 34]. Upon combustion of such flame-retarded

polymeric materials, the bromine present is likely to be liberated, yielding the irritant gas HBr [4, 35].

Only very limited data are available on the irritant effects of HBr, which have not been studied as extensively as HCl. The effects are expected to be similar to those caused by HCl at comparable concentrations [6]. Exposure of six volunteers to HBr at 5 ppm resulted in nasal irritation in all of the subjects and throat irritation in one of the subjects [36]. Exposure to concentrations between 1300 and 2000ppm has resulted in death [37]. It has been suggested that the sensory irritation produced by exposure to HBr at 200 ppm may be sufficient to slow escape from a hazardous combustion environment [38].

3.2.1.3 Hydrogen fluoride (HF)

The combustion of synthetic fluorine-containing polymers, such as PTFE (polytetrafluoroethylene), PFA (perfluoroalkoxy), FEP (fluorinated ethylene-propylene) and PVDF (polyvinylidene fluoride), leading to the liberation of fluorine on decomposition, is likely to result in the production of the halogen acid gas hydrogen fluoride (HF) [39, 40].

Similarly with the previous halogen acid gases (HCl and HBr), HF is a strong sensory irritant and corrosive gas, which upon inhalation for 1 hour at concentrations of just 0.5 ppm has been shown to cause irritation and corrosion of the mucous membranes of the nose, mouth and throat. Inhalation of higher concentrations is likely to result in injury to the lungs, with the onset of pulmonary oedema which may be delayed 24-48 hours after exposure [41, 42]. Studies in volunteers showed some relatively low sensory and lower airways irritancy at 0.2 - 2.9 ppm including chest tightness, soreness, coughing, expectoration and wheezing. At 3.0 - 6.3 ppm more severe effects were seen [43]. HF is the most potent irritant of the halogen acid gases based on animal lethality data (AEGL-3), but has equivalent potency to HCl and HBr based on sensory irritancy (AEGL-2) (table 2). However, the generation of HF during combustion is likely to be in considerably smaller quantities than the production of HCl.

3.2.1.4 Sulphur dioxide (SO_2)

Sulphur dioxide (SO_2) is an inorganic irritant gas which is commonly produced upon the combustion of fossil fuels, but may also be formed during the thermal decomposition of any sulphur-containing compounds such as vulcanized rubber used in the manufacture of tyres.

SO_2 is a respiratory irritant and may cause tightening of the airways. Individuals with asthma are significantly more sensitive to SO_2 than people who do not have the condition. WHO suggest that exposure to 0.4 ppm may lead to significant narrowing of the airways in those suffering from asthma. In most, the effect would not be expected to be large, but, some individuals may be clinically affected [44]. Inhalation of SO_2 at 1 ppm for 1-6 hours has been shown to increase airway resistance and decreased forced expiratory volume and forced expiratory flow in healthy patients [45]. SO_2 is readily

absorbed by the mucosa of the upper respiratory tract, with irritation of the nose and mouth being the most common effect following inhalation [45, 46]. At concentrations of around 10 ppm SO_2 causes moderate to severe eye irritation with lachrimation [45]. The irritation effect of SO_2 is due to its conversion to sulphuric acid in the presence of water upon the mucous membranes of the upper respiratory tract end eyes. Sulphur dioxide also stimulates pain / irritant receptors which are present as fibres in the epithelium [47]. Exposure to high concentrations of SO_2 can be fatal, due to asphyxiation caused by blockage of the upper respiratory tract as a result of severe irritation [28].

3.2.1.5 Oxides of nitrogen (NO_x)

Nitrogen oxides such as nitric oxide (NO) and nitrogen dioxide (NO_2) are commonly present as mixtures in combustion atmospheres and can be denoted collectively as NO_x [48]. Oxides of nitrogen are likely to be generated during the combustion of any nitrogen-containing materials, with the formation of each being dependent upon the availability of oxygen in the fire environment. Nitrogen present as N_2 is a major constituent of air, therefore, during combustion NO_x may also be formed due to oxidation of this atmospheric nitrogen provided that the temperature of combustion is sufficient [4, 48-50]. Burning coal, wood, tobacco, oil and gas all generate nitrogen oxides. Nitrogen oxide is first formed and then oxidised to nitrogen dioxide.

Nitrogen oxides are less soluble than most irritant gases and are therefore more likely to reach the bronchioles and alveoli following inhalation giving rise to pulmonary damage [51].

Nitric oxide (NO)

Of the nitrogen oxides present in combustion effluents, nitric oxide (NO) is expected to be present only in close proximity to the fire or in oxygen-limited atmospheres.

The possible health effects arising from exposure to NO have been relatively little studies, but it is recognised to be significantly less active as an airways irritant than NO_2 [52]. NO is irritating to the eyes and upper respiratory tract. Deep inhalation can result in the delayed onset of pulmonary oedema occurring a few hours post-exposure and may be aggravated by physical exertion [53]. Controlled exposure of healthy human volunteers to NO at concentrations above approximately 20 ppm (24.6 mg/m^3), have demonstrated a significant increase (~10%) in total airway resistance [48].

Nitrogen dioxide (NO_2)

Nitrogen dioxide (NO_2) is most likely to be present in smoke as it moves away from the fire and when sufficient oxygen is present as NO will be converted to NO_2 in air. Nitrogen dioxide is an irritant and an oxidant which produces inflammation and oedema of the lungs if inhaled in high concentrations.

The potency of NO_2 as an irritant is approximately 5 times greater than NO [6]. The irritant effect of NO_2 is due to the conversion of NO_2 in the presence of water into nitric acid (HNO_3) and nitrous acid (HNO_2) in the mucous membranes of the respiratory tract [4, 51]. Low concentrations of NO_2 may cause cough, headache, difficulty breathing, nausea, vertigo and fatigue [54]. NO_2 has been shown to cause significant increases in airway resistance in healthy individuals at exposure concentrations as low as 2.5 ppm [48]. Exposure to high concentrations of NO_2 have resulted in sudden death due to severe constriction of the airways and larynx [55]. Severe pulmonary oedema may occur within a few hours following removal from exposure to NO_2 [54, 55]. During pulmonary oedema a decrease in the partial pressure of arterial oxygen may also be observed due to impairment of the diffusion capacity [55]. When CO_2 is present in conjunction with NO_2 there is an increase in the toxicity when compared to NO_2 alone. Studies in healthy individuals have shown a threshold of effect of around 2 ppm. Individuals with asthma are more sensitive, with a threshold being around 0.2 ppm [56].

3.2.1.6 Phosphorous pentoxide (P_2O_5)

The combustion of any compounds containing phosphorous may lead to the formation of phosphorous pentoxide (P_2O_5) and phosphoric acid. Many traditional flame retardants contain halogen groups such as bromine, however, there has been an increasing drive to develop halogen-free flame retardants [57, 58]. Phosphorous is a common constituent of many halogen-free flame retardants, such as isopropylated triphenyl phosphates. These are incorporated into polyurethane foams to increase the resistance of the material to burning. Some halogen containing flame retardants, such as tris(2-chloropropyl) phosphate, may also incorporate phosphorous to enhance the flame retardant effect [58]. Such phosphorous-containing flame retardants are incorporated into casings and housing of electronic components (eg, televisions) in place of halogenated compounds.

Phosphorous pentoxide is corrosive to the eyes and upper respiratory tract, as it readily reacts with moisture in the mucous membranes to form orthophosphoric acid. Inhalation of P_2O_5 may also lead to pulmonary oedema the onset of which, may be delayed for a few hours following removal from the exposure [59, 60].

Table 2: Worker Exposure Limits (WEL) and Acute Emergency Guideline Levels (AEGL) for inorganic acid combustion products.

	WEL [29] (ppm)		AEGL – 2 / AEGL – 3 [30] (ppm)				
	LTEL	STEL	10 min	30 min	60 min	4 hr	8hr
HCl	1	5	100 / 620	43 / 210	22 / 100	11 / 26	11 / 26
HBr†	-	3	100 / 740	43 / 250	22 / 120	11 / 31	11 / 31
HF	1.8	3	95 / 170	34 / 62	24 / 44	12 / 22	12 / 22
SO_2†	-	-	0.75 / 30	0.75 / 30	0.75 / 30	0.75 / 19	0.75 / 9.6
NO†*	NR						
NO_2†	-	-	20 / 34	15 / 25	12 / 20	8.2 / 14	6.7 / 11
P_2O_5	Data not available						

WEL = Workplace Exposure Limits, LTEL = Long Term Exposure Limit (8 hour Time Weighted Average), STEL = Short Term Exposure Limit (15 minute reference period), AEGL – 2 = Acute Exposure Guideline Level – 2 (The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape), AEGL – 3 = Acute Exposure Guideline Level – 3 (The level of the chemical in air at or above which the general population could experience life-threatening health effects or death). NR = Not recommended due to insufficient data. † = Interim AEGL. * values for NO_2 should be used for emergency planning.

3.2.2 Organic irritants

The combustion of organic compounds will also be likely to result in the formation of organic irritant products in the fire effluent. Many organic irritants may be evolved upon combustion, however, the formation of these products will depend largely upon the nature of the material involved. The incomplete combustion or pyrolysis of materials including; wood, fossil fuels, synthetic and natural polymers and foodstuffs will give rise to the formation of a range of aldehydes [61]. Of this group, acrolein and formaldehyde are most commonly considered, although it is highly unlikely that these will be the sole organic irritants which may be present in fire smoke and may also include compounds such as acetaldehyde and butyraldehyde [28].

3.2.2.1 Acrolein

The combustion of cellulose based materials such as wood, cotton and paper have been demonstrated to evolve significant quantities of acrolein in the fire smoke [62]. Acrolein generated during the combustion of wood may represent up to 13% of the total aldehydes in the fire effluent [61]. Studies to investigate the amount of acrolein

generated during combustion have measured acrolein at up to 50 ppm in wood smoke, and 60 ppm in smoke generated during the combustion of cotton [28]. Acrolein has also been shown to be generated from polyurethane foams in much greater quantities during incomplete combustion. The complete combustion of polyethylene foam was shown to evolve acrolein in the range of 2 – 23 ppm, whilst incomplete combustion (smouldering fires) generated between 76 – 180 ppm acrolein [63].

Acrolein is the most potent of the irritants. It is severely irritating to the respiratory tract and eyes and is a potent lachrymatory agent. Concentrations of acrolein in the region of 0.5 to 5 ppm have been shown to cause the onset of lachrimation and eye irritation in humans within a 10 minute exposure period [64]. Acrolein at 1.2 ppm has also been reported to cause lachrimation in humans within just 5 seconds of commencing exposure [65]. Volunteers exposed to a steadily increasing concentration of acrolein reported nose irritation at 0.26 ppm, throat irritation at 0.43 ppm and a reduction in respiratory rate at 0.6 ppm [66]. Exposure to acrolein may result in the onset of pulmonary oedema which may not manifest until a few hours post exposure [67, 68]. The onset of pulmonary oedema is due to inflammation in the respiratory mucosa resulting from protein denaturation caused by acrolein [10]. Eye irritation resulting from exposure to acrolein has been observed at concentrations as low as 0.06 ppm, and nasal irritation has resulted from exposure to 0.15 ppm. A reduction in respiratory rate has been observed in male volunteers exposed to acrolein at 0.3 ppm for 40 minutes. Respiratory effects including coughing, chest pain and difficulty breathing has been reported following exposure to 0.26 ppm acrolein. Most individuals are unable to tolerate acrolein in air at above 2 ppm for more than 2 minutes [69]. Exposure to acrolein at concentrations above 10 ppm has been shown to fatal within just a few minutes [28].

3.2.2.2 Formaldehyde

Formaldehyde is likely to be generated under similar conditions of combustion to acrolein. The combustion of wood has been shown to evolve formaldehyde in the fire effluent at up to 80 ppm and the combustion of cotton has been shown to generate up to 70 ppm formaldehyde [28]. Formaldehyde is also commonly present as a smoke product from the combustion of many polymers and plastics, particularly during incomplete combustion [70].

Formaldehyde is a potent sensory irritant, causing mild to moderate irritation of the upper respiratory tract and eyes at concentrations in the range of 0.2 to 3 ppm [71]. In both normal subjects and asthmatics, no significant clinically detrimental effects were seen in lung function at up to 3 ppm for 3 hours [72]. Formaldehyde inhalation at high concentrations will give rise to respiratory effects including bronchospasm, dyspnoea, respiratory depression and laryngeal spasm. Inhalation of formaldehyde at high concentrations may also cause the onset of laryngeal and pulmonary oedema which may be potentially fatal and may be delayed for a few hours post-exposure [70, 73]. Upper respiratory tract irritation has been reported following exposure to formaldehyde in the range of 0.1 – 25.0 ppm, and lower respiratory tract and pulmonary irritation resulting from exposure to 5.0 – 30.0 ppm. Pulmonary oedema, inflammation and

pneumonia has been reported following exposure to formaldehyde at 50 – 100 ppm, and at concentrations greater than 100 ppm may be fatal [52].

Table 3: Worker Exposure Limits (WEL) and Acute Emergency Guideline Levels (AEGL) for organic irritant gas combustion products.

	WEL [29] (ppm)		AEGL – 2 / AEGL – 3 [30] (ppm)				
	LTEL	STEL	10 min	30 min	60 min	4 hr	8hr
Acrolein†	0.1	0.3	1.44 / 6.2	0.4 / 2.5	0.10 / 1.4	0.10 / 0.48	0.10 / 0.27
Formaldehyde†	2	2	14 / 100	14 / 70	14 / 56	14 / 35	14 / 35

WEL = Workplace Exposure Limits, LTEL = Long Term Exposure Limit (8 hour Time Weighted Average), STEL = Short Term Exposure Limit (15 minute reference period), AEGL – 2 = Acute Exposure Guideline Level – 2 (The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape), AEGL – 3 = Acute Exposure Guideline Level – 3 (The level of the chemical in air at or above which the general population could experience life-threatening health effects or death). † = Interim AEGL.

3.2.3 Other inorganic irritants

The combustion of many polymeric materials will also be likely to generate additional irritants to those previously described. These irritants are inorganic by nature, but do not fall within the previous category of inorganic acid gases. Such inorganic irritants commonly derived upon combustion of natural and synthetic materials include ammonia, chlorine and phosgene.

3.2.3.1 Ammonia (NH_3)

Ammonia (NH_3) is likely to be present in the fire effluent upon the combustion of any nitrogen containing materials including wood, coal, paper and household waste. Ammonia may be generated in significant amounts during the incomplete combustion (pyrolysis) of low rank coals (containing water and impurities due to inadequate pressure, heat or time during formation) and biomass [50].

Ammonia is severely irritating and corrosive to the respiratory tract and eyes and causes lachrimation and respiratory distress [74]. Ammonia reacts with the water present in the eyes and mucous membranes of the respiratory tract to form ammonium hydroxide, resulting in necrosis of the cells with which it comes into contact due to its alkaline properties [75]. Exposure to ammonia at 50 ppm results in the immediate onset of moderate irritation to the eyes, nose and throat [75]. Exposure to higher concentrations of ammonia in the region of 400 ppm have been demonstrated to cause severe irritation of the nose and throat, 500 ppm ammonia has been shown to increase the rate of breathing and concentrations of 1700 ppm are reported to induce coughing [74, 76].

Exposure to ammonia at high concentrations above 1500 ppm may be associated with the onset of pulmonary oedema, which may be delayed for up to 24 hours post-exposure [75, 77]. Inhalation of very high concentrations of ammonia (~ 5000 ppm and above) may be rapidly fatal due to obstruction of the airways [75].

3.2.3.2 Chlorine

The generation of chlorine during combustion is likely to occur in similar situations to those in which HCl is formed, with both products likely to be present simultaneously. The combustion of any chlorine-containing compounds, including plastics, polymers and synthetic rubbers such as polyvinylchloride, and bleaches such as sodium hypochlorite, may result in the presence of chlorine in the fire effluent.

Exposure to chlorine can cause severe irritation to the eyes, nose, throat and upper respiratory tract. Concentrations of chlorine in the range of 1 to 3 ppm are associated with mild irritation of the eyes and the mucous membranes of the nose and throat, coughing, difficulty breathing and headache [32, 78, 79]. Moderate irritation of the upper respiratory tract and eyes is caused by exposure to chlorine concentrations of 5 to 15 ppm. Exposure to higher concentrations of chlorine in the region of 30 ppm will result in immediate chest pain, vomiting and coughing, with the onset of toxic pneumonitis and pulmonary oedema resulting from exposure to 40 to 60 ppm chlorine [78, 79]. The onset of pulmonary oedema may be delayed for several hours post-exposure [80]. Exposure to chlorine at concentrations greater than 430 ppm for 30 minutes can be fatal, whilst concentrations above 1000 ppm may be fatal in just a few minutes due to respiratory failure [78-80].

3.2.3.3 Phosgene ($COCl_2$)

Phosgene is likely to be evolved from the combustion of any chlorinated organic compounds, for example, chlorinated solvents such as chloroform and plastics and polymers such as polyvinylchloride. The requirement for oxygen for the formation of phosgene during the combustion of materials containing chlorine and carbon, suggest that it is likely to be formed in the greatest quantities during fire situations where ventilation is not a limiting factor.

Phosgene is a potent irritant, particularly of the lower respiratory tract. An acute exposure to a concentration of 3 ppm results in irritation of the eyes and upper respiratory tract [81]. Phosgene will also penetrate into and irritate the deep lung tissues following inhalation, due to its relative insolubility in the mucous membranes of the upper respiratory tract with onset of lung damage at exposures of greater than 300 ppm min^{-1} [80]. Exposure to phosgene can cause fatal pulmonary oedema at concentrations above 150 ppm min^{-1} [81]. The toxicity of phosgene following an acute exposure in the range of 30 to 300 ppm min^{-1} will typically display three phases. The first stage is the reflex syndrome characterised by irritation to the eyes and respiratory tract, pain, difficulty breathing and coughing which occurs during exposure and usually subsides once withdrawn from exposure. The second phase is a clinical latent phase in which the

exposed individual experiences no symptoms and generally feels recovered. The duration of the latent phase may be between 30 minutes to 24 hours and depends upon the severity of phosgene exposure, with a shorter latent period as the concentration or duration increase. The final phase of phosgene toxicity is the clinical oedema stage, which involves the manifestation of pulmonary oedema and is associated with shortness of breath, coughing, cyanosis, shock and respiratory arrest [80, 81]. In cases of phosgene exposure in the lethal range (above 300 ppm min^{-1}), the latent period may be truncated or non-existent, with immediate onset of pulmonary oedema [81]. Exercise is particularly dangerous in people exposed to phosgene as it exacerbates the adverse effects. Thus, the exertion required to escape a combustion atmosphere where phosgene is present can cause collapse, as can helping other casualties, such as carrying people.

Table 4: Worker Exposure Limits (WEL) and Acute Emergency Guideline Levels (AEGL) for inorganic irritant gas combustion products.

	WEL [29] (ppm)		AEGL – 2 / AEGL – 3 [30] (ppm)				
	LTEL	**STEL**	**10 min**	**30 min**	**60 min**	**4 hr**	**8hr**
Ammonia	25	35	220 / 2700	220 / 1600	160 / 1100	110 / 550	110 / 390
Chlorine	0.5	1	2.8 / 50	2.8 / 28	2.0 / 20	1.0 / 10	0.71 / 7.1
Phosgene	0.02	0.06	0.6 / 3.6	0.6 / 1.5	0.3 / 0.75	0.08 / 0.20	0.04 / 0.09

WEL = Workplace Exposure Limits, LTEL = Long Term Exposure Limit (8 hour Time Weighted Average), STEL = Short Term Exposure Limit (15 minute reference period), AEGL – 2 = Acute Exposure Guideline Level – 2 (The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape), AEGL – 3 = Acute Exposure Guideline Level – 3 (The level of the chemical in air at or above which the general population could experience life-threatening health effects or death).

3.3 Complex molecules

The combustion of many organic materials including fossil fuels, natural polymers such as wood and synthetic polymers such as plastics, will not only give rise to the asphyxiant and irritant gas compounds mentioned previously. In many cases, the combustion of organic materials, particularly if it is incomplete, may also give rise to more complex molecules in the smoke plume which may typically include longer carbon chains and multiple carbon-rings. The acute toxicity of these compounds is generally low and may not pose a direct health hazard during exposure. However, some of these compounds, in particular those from the polycyclic aromatic hydrocarbon groups, are recognised mutagens and carcinogens. The risks from single (acute) exposure are very small (and unquantifiable). Others give rise to concern because of possible effect in the reproductive system (eg, dioxins) [82-84].

3.3.1 Polycyclic aromatic hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a large group (over 100) of organic compounds containing a minimum of two fused benzene rings. Some of the most commonly known PAHs include benzo[*a*]pyrene, naphthalene and anthracene [82]. PAHs may be evolved in the fire effluent from all combustion processes, with the largest quantities likely to be generated during the slow, incomplete combustion of organic materials [82, 83]. PAHs are present as complex mixtures rather than as single individual compounds.

There is very little information regarding the adverse health effects following an acute exposure to PAHs, either from experimental data on individual compounds or from mixtures. Much of the data regarding the acute toxicity of PAHs relates to reports of accidental exposure of naphthalene [82]. Naphthalene may give rise to headaches, nausea, vomiting, confusion, profuse sweating, nose, throat and eye irritation and corneal damage following acute exposure to vapours. The onset of acute haemolytic anaemia, particularly in individuals with glucose 6-phosphate deficiency, is a characteristic feature of acute exposure to naphthalene, which may be delayed post-exposure [82, 85].

The health effects following exposure to mixtures of PAHs focuses on the fact that many of the compounds are known to have the potential to be carcinogenic, based on animal experiments or data from occupational exposures to mixtures of PAHs (eg, coke oven workers) [82]. In most cases the carcinogenic compounds also have mutagenic potential and they are considered 'genotoxic carcinogens'. An exception is naphthalene which is an animal carcinogen, but has no significant mutagenic potential. Some commonly known PAHs which are classified by IARC as being probably carcinogenic to humans (group 2a), include benzo[*a*]pyrene, dibenz[*a,h*]anthracene and benz[*a*]anthracene [86]. PAHs which are classified as possibly carcinogenic to humans (group 2b) include naphthalene, indeno[1,2,3-*cd*]pyrene and benzo[*b*]fluoranthene, whilst compounds including chrysene, fluorine and anthracene are not classifiable as to their carcinogenicity in humans (group 3) [86]. The experimental data relating to carcinogenicity of PAHs is mainly from chronic (long term) exposure studies in animals. Any risks from a single acute exposure are likely to be very small.

3.3.2 Dioxins / dibenzofurans

Polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are large groups (75 dioxin isomers, 135 furan isomers) of polychlorinated tricyclic aromatic compounds which have very similar structures. PCDDs and PCDFs are formed mainly during the incomplete combustion of any materials containing carbon, oxygen and chlorine and are therefore commonly found as emissions in most fire effluents [87-89]. The pyrolysis of organochlorine polymers such as polyvinylchloride, has been shown to give rise to the formation of both PCDDs and PCDFs at temperatures ranging from 500 to 700°C [90-92]. Once formed, PCDDs and PCDFs are relatively resistant to

thermal decomposition and are only destroyed after heating to 800°C for a prolonged period [87]. PCDDs and PCDFs present in combustion effluents are most likely to be present as complex mixtures. The amounts of dioxins and dibenzofurans produced during combustion will depend largely on the material involved. Data on the total emissions of PCDDs and PCDFs from municipal solid waste incinerators suggest that the amounts are very small, between the range of a few to several thousand ng / Nm^3 [87]. However, it has been demonstrated that the formation of PCDDs and PCDFs from the combustion of chlorine-containing plastics increased dramatically in the presence of copper (Cu) in the form of electrical wire [93]. The combustion of plastics containing polychlorinated biphenyls (PCBs) was known to be a source of PCDFs and PCDDs, although the use of PCBs has been restricted since the late 1970s [94]. However, fires involving PCB-containing materials, such as electrical transformers and capacitors manufactured prior to the beginning of the 1980s could potentially form PCDFs and PCDDs in the combustion effluent. The most commonly known of the PCDDs is 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) as it is one of the most toxic and most extensively studied of the dioxins [95]. Many of the other isomers are very much less potent than TCDD and have low toxicity, eg, the fully chlorinated derivative. The toxicity of mixtures of TCDD is in terms of TCDD equivalents, with TCDD being the standard. The most toxic PCDFs are those which have chlorine atoms substituted at the 2, 3, 7 and 8 carbon positions, particularly 2,3,4,7,8-pentachlorodibenzofuran (2,3,4,7,8-pentaCDF) and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) [96].

The most commonly documented adverse health effect following exposure to dioxins is the gradual onset of chloracne which may appear within days or even months post-exposure [87, 88, 95]. Chloracne is typically characterised by comedones, epidermal cysts and inflamed papules with hyperpigmentation [88]. The distribution of chloracne differs from that typically observed with adolescent acne, and is more commonly associated with areas of contact. Some additional adverse health effects which may occur following acute exposure to dioxins include transient hepatotoxicity, hypertension, hyperlipidaemia and possible peripheral and central neurotoxicity [88]. Studies in firefighters involved in PCB transformer fires (considered to be the worst case scenario, with respect to dioxin formation) did not detect any observable health effects after 1 year post-exposure, although PCDD levels were slightly elevated [87]. Data from animal studies indicates that dioxins have adverse effects on the reproductive system including teratogenicity, and that the most sensitive and consistent effect is upon the developing reproductive system in the male offspring. Exposure to chlorinated dibenzofurans appears give rise to similar adverse effects as that observed following exposure to 2,3,7,8-TCDD, however the toxicity of these dibenzofurans and other chlorinated dioxins is of much lower potency. There have been no reports of human fatalities due solely to acute exposure to dioxins [95]. However, 2,3,7,8-TCDD has been evaluated as carcinogenic to humans (group 1) by the IARC [97]. Polychlorinated dibenzo-*p*-dioxins other than 2,3,7,8-TCDD and polychlorinated dibenzofurans are not classifiable as to their carcinogenicity to humans (group 3) [97]. However, TCDD and other dioxins do not have significant mutagenic properties and prolonged exposure is likely to be necessary for the promoter effects to induce carcinogenicity. Risks from single exposure, if any, would be expected to be very low.

3.3.3 Isocyanates

The thermal decomposition of polyurethane foams and plastics is known to evolve isocyanates in the combustion effluent due to dissociation of the urethane monomer [98, 99]. Polyurethane foams are widely used and may therefore be present as materials in many combustion situations. Flexible polyurethane foams are used in many common domestic materials including furniture, bedding and carpet underlay, semi-flexible foams are employed widely in motor vehicle interiors, whilst rigid foams are commonly used as insulation for central heating tanks and pipes and in appliances such as refrigerators [100]. The amount of isocyanates formed in combustion effluents is expected to be greater from polyurethane foams which contain unreacted isocyanate. The combustion of polyurethane will not only yield isocyanates, but is also likely to result in the formation of greater yields of aromatic compounds such as benzene and toluene [98, 100]. The isocyanates which may be present as products of combustion will vary depending upon the composition of the material involved and the combustion conditions, but may include relatively simple compounds such as methyl isocyanate, or more complex compounds such as toluene diisocyanate.

Mehtyl isocyanates is a severe irritant to eyes and mucous membranes. Studies in volunteers indicated eye irritation and lachrymation in all individuals exposed to 1 ppm for 5 minutes. Nose and throat irritation was reported in some at this exposure level. A large amount of data are available on the effect of methyl isocyanates resulting from the Bhopal accident which resulted in over 250 deaths. The most frequently reported symptoms were burning/watering of the eyes, coughing, respiratory distress from pulmonary congestion, nausea, muscle weakness and CNS involvement secondary to hypoxia. However, insufficient details are available regarding the levels of exposure producing these effects. There is also evidence that methyl isocyanate produces allergic sensitisation reactions. Animal studies indicate that exposure to pregnant animals at 2 ppm produces fetotoxic effects (reduced body weight) whilst having no effects on the maternal animals, indicating that an acute exposure may harm the unborn child [101].

Toluene diisocyanate is severely irritating to the eyes and throat with exposure to 0.5 ppm for 30 minutes producing lachrymation. Exposure to 1.3 ppm was intolerable for 10 minutes; several hours later cold-like symptoms with cough persisted. Studies in pregnant animals indicated no adverse effects on development at exposure levels that were not toxic to maternal animals [101, 102]. Data from experimental exposure have clearly established that toluene diisocyanate is a potent allergic sensitiser following inhalation exposure, producing an asthma-like reaction. Persons previously sensitized would be expected to have such reactions following exposure to very low levels of toluene diisocyanate [102].

Inhalation of isocyanates can lead to the delayed onset of potentially fatal pulmonary oedema which may develop 12 to 48 hours post exposure [101, 102].

Table 5: Acute Emergency Guideline Levels (AEGL) for methyl isocyanate.

	AEGL – 2 / AEGL – 3 [30] (ppm)				
	10 min	**30 min**	**60 min**	**4 hr**	**8hr**
Methyl isocyanate	0.40 / 1.2	0.13 / 0.4	0.067 / 0.2	0.017 / 0.05	0.008 / 0.025

AEGL – 2 = Acute Exposure Guideline Level – 2 (The level of the chemical in air at or above which there may be irreversible or other serious long-lasting effects or impaired ability to escape), AEGL – 3 = Acute Exposure Guideline Level – 3 (The level of the chemical in air at or above which the general population could experience life-threatening health effects or death).

3.3.4 Perfluoroisobutylene (PFIB)

Perfluoroisobutylene (PFIB) is formed during the thermal decomposition of fluorine containing polymers, such as PTFE [103-105]. A study of the combustion products from PTFE has identified that PFIB is produced at temperatures greater than 475°C [104]. PFIB may therefore be present as a product in the effluent following thermal degradation of polymers such as PTFE at relatively low combustion temperatures.

Inhalation of PFIB is extremely irritating to the respiratory tract. The adverse health effects of exposure to PFIB are similar to those of phosgene, however the toxic potency of PFIB is approximately ten times greater than phosgene [106, 107]. In addition to the sensory and respiratory irritant effects, exposure to vapours of PFIB may give rise to headache, cough, chest pain and dyspnoea. Inhalation of sufficient amounts of PFIB can also lead to the onset of potentially fatal pulmonary oedema, which may be delayed up to eight hours post exposure [106, 108].

3.3.5 Particulate matter (PM)

Particles of organic and inorganic matter are likely to be released during all types of fires involving organic materials, particularly under conditions of incomplete combustion. Particulate matter (PM) is usually comprised of a complex mixture of different particle types and particles sizes. Many of the types of PM derived from combustion are unlikely to cause adverse health effects [109]. However, some PM particularly the smaller more respirable particles, can cause significant adverse effects [110]. These particles which are below 10 μm in diameter (commonly denoted as PM_{10}) are able to pass through the upper respiratory tract and can be deposited within the airways. Particles with diameters less than 2.5 μm ($PM_{2.5}$) may be respired deeper within the lungs and can be deposited within the alveoli [111]. Ultrafine particles are defined as particles which have a diameter of less than 100 nm in at least one dimension. Ultrafine particles have the potential to migrate from the site of deposition, through membranes into the blood and are then able to infiltrate other target organs [109].

Knowledge of the health effects of PM_{10} comes mainly from epidemiology studies relating to the effect of air pollution. A large number of such studies have shown that PM_{10} levels are associated with effects on health even at very low levels. These include increased daily deaths, increased admissions to hospital in patients suffering from heart and lung disorders and a worsening of conditions in those with asthma. It is believed that these effects on the respiratory and cardiovascular system are caused predominately by the very small particles usually referred to as ultrafine (or nanoparticles), ie, those smaller than 100 nm in diameter.

Upon deposition in the lungs, some PM and especially the ultrafine particles produce free radicals which may provoke oxidative stress. This oxidative stress can contribute to inflammation in the lung tissue, and may exacerbate any pre-existing lung condition such as asthma [110, 112]. Ultrafine particles which are able to migrate from the lungs into the blood have also been implicated in causing adverse effects upon the cardiovascular system [113]. There are two main theories as to how effects on the cardiovascular system may be induced. One is that the particulates set up an inflammatory response in the interstitium of the lung and this provokes an increase in the likelihood of blood to clot or atherosclerotic plaques to rupture, which could result in myocardial infarction [110, 113]. This hypothesis is true for sustained exposure, such as ambient air pollution, but may play a less significant role following exposure to ultrafine particles in smoke. An alternative hypothesis points to a reflex effect on the heart with receptors in the airways and lungs [113].

In contrast to many of the other combustion products highlighted in this review, the degree of adverse effects observed following exposure to PM is not directly related to the external exposure concentration, but may be more closely related to the size and surface area of the particles deposited at the target organ [114]. PAHs are some of the most common toxic compounds present in combustion derived PM, which may also include nitrogen substituted polycyclic aromatic hydrocarbons (nitro-PAHs) and nitro-PAH lactones, many of which are highly mutagenic and carcinogenic [83]. Particulate matter may also include contamination with metals such as lead, arsenic, cadmium and nickel [115, 116].

3.4 Health issues for vulnerable groups

Individuals who are most at risk from exposure to hazardous combustion products are those which have pre-existing respiratory diseases, such as asthma or chronic obstructive pulmonary disease. The presence of an existing respiratory condition increases the susceptibility of the individual to the adverse effects of exposure to asphyxiant gases such as CO [14]. Acute exposure to smoke containing mixtures of asphyxiant and irritant gases is therefore likely to exacerbate these conditions.

Pregnant women are particularly at risk following exposure to smoke, as unborn infants are particularly susceptible to carboxyhaemoglobin due to CO. Following exposure to CO, the foetal circulation would be expected to have a greater concentration of carboxyhaemoglobin than the maternal circulation due to differences in the uptake and elimination of CO. This increased level of COHb in the foetus compared to the mother,

may cause potentially serious hypoxia to the foetus at COHb levels which are less harmful to the mother. Newborn infants and children may also be at increased risk of adverse effects such as hypoxia, respiratory irritation and pulmonary oedema following exposure to hazardous combustion products. Infants are more susceptible to toxicity since the hazardous products present in the fire effluent are likely to result in toxicity to infants and children at lower concentrations than those required to cause similar effects in adults. Other combustion products such as methyl isocyanates give rise to concern because of adverse effects following in utero exposure.

Elderly individuals exposed to hazardous combustion products would also be expected to be at greater risk of potentially life threatening health effects due to conditions associated with age, such as reduced in lung function. Any reduction in the utilisation of oxygen therefore increases the individual from being at risk to hypoxia resulting from exposure to asphyxiant gases such as CO, HCN and low oxygen concentration.

Infants, children and elderly individuals may also be less able to escape from an environment containing hazardous combustion products, thereby increasing the duration of exposure and thus increasing the potential for adverse effects.

Individuals who smoke are also likely to be at greater risk to toxicity following inhalation of combustion products as the baseline level of COHb is likely to be greater than would be observed in non-smokers. Therefore, the inhalation of combustion products at concentrations that do not cause significant toxicity to non-smokers may cause considerable toxicity to individuals that smoke regularly [1].

4 HAZARDOUS COMBUSTION PRODUCTS FORMED BY FUEL TYPE

The products formed during combustion processes can vary greatly depending upon the material involved. The compounds generated during each individual fire scenario should be viewed on a case by case basis due to the large number of variables affecting the products formed. However, general predictions of the products most likely to present a hazard to health can be made if the materials involved are known.

4.1 Fires involving polymeric materials

Polymeric materials such as plastics, resins, fibres and foams are likely to produce significant quantities of carbon monoxide upon combustion. The presence of a high level of carbon monoxide is, however, likely to be of most concern within the immediate vicinity of the fire, particularly if the fire is enclosed within a building and is ventilation controlled.

The combustion of polymers which contain large quantities of nitrogen, such as nylons, polyurethanes and polyacrylonitriles are likely to yield significant amounts of hydrogen cyanide (HCN), nitrogen oxides (NO_x) and ammonia. However, HCN, NO_x and ammonia

are not exclusively formed from the combustion of nitrogen containing plastics, but may also be present to a lesser extent during the combustion of non-nitrogen containing plastics due to the incorporation of atmospheric nitrogen. In this case, the incorporation of atmospheric nitrogen to form HCN and NO_x will be largely dependent upon its availability in the combustion environment and the temperature.

Many plastics contain a considerable proportion of halogens, which can be released as irritant gases or as more complex molecules. Hydrogen chloride (HCl) is released in large quantities during the combustion of plastic such as polyvinylchloride, with the majority of the chlorine present in the plastic being released as HCl. In addition to HCl, some chlorinated plastics may also release phosgene and small amounts of PCDDs and PCDFs. HBr is likely to be generated during the combustion of many plastics used for electrical applications due to the addition of bromine as a flame retardant. Fluorine containing polymers, such as PTFE are known to liberate HF upon combustion, and if the combustion is incomplete can also generate PFIB, which is an extremely potent irritant. Many more recent flame retardant plastics have been produced which do not contain halogens, but instead contain phosphorous. The combustion of these phosphorous-containing plastics may therefore yield oxides of phosphorous such as P_2O_5 and phosphoric acid. The combustion of polyurethane foams commonly used in furnishings, have been shown to generate isocyanates and their derivatives.

As the composition of polymers such as plastics, resins, fibres and foams can vary greatly, this will influence the products formed and it may not be possible to identify all of the types of plastic which may be involved during an individual combustion situation.

In general for fires involving plastics the greatest hazard to public health outside the immediate vicinity of the fire will be due to inorganic and organic irritant gases, particulate matter and other more complex organic compounds. The asphyxiant gases generated during plastic combustion are most likely to be of greatest concern to health within the same compartment / building as the fire.

4.2 Fires involving wood

The combustion of cellulosic materials such as wood, either as vegetation, such as in forest fires, or that used in construction and furnishings will be likely to lead to the formation of organic irritants, such as acrolein and formaldehyde. However, depending upon the conditions, relatively small amounts of PAHs, particulate matter and more complex exotic molecules may be formed, particularly during incomplete combustion, all of which may present a concern to public health.

Asphyxiant gases such as carbon monoxide will be generated during the combustion of wood, although its presence is only likely to be of concern in the immediate vicinity of the source of the fire. The formation of CO from the combustion of wood in the open environment, such as that seen in the case of forest fires, is less likely to be a concern than in building fires due to the greater availability of oxygen and the greater potential for dispersion of the fire effluent.

In addition to the hazardous products from the combustion of wood, burning of wood incorporating preservatives such as the heavy metal containing preservative, chromate copper arsenate (CCA) may lead to the liberation of heavy metals as oxides such as chromium trioxide and arsenic trioxide [117].

The main hazard to public health following exposure to products from wood fires is due to the generation of organic irritant gases. These irritant compounds are more likely to be present in the effluent plume away from the source than gases such as CO which are only expected to be present in significant quantities in the immediate vicinity / compartment of the fire.

4.3 Fires involving rubber / tyres

Fires which involve large quantities of rubber, such as tyre fires may give rise to the generation of significant yields of sulphur dioxide (SO_2) due to the high sulphur content resulting from the vulcanisation process.

The combustion of rubber is also likely to give rise to the formation of CO, organic irritants, inorganic irritants, PAHs, some complex organic molecules and particulate matter. Some rubber compounds contain organophoshate-based flame retardants which upon combustion may additionally yield phosphorous pentoxide (P_2O_5).

The compounds most likely to pose a hazard to the health of individuals outside the immediate vicinity of the fire, might be expected to be irritants such as SO_2 and organic irritants such as acrolein.

4.4 Fires involving oil / petrol

Fires involving oil or petrol in an external environment might be expected to undergo extensive combustion due to the high temperature and availability of oxygen, with carbonaceous particles being a prominent product. In this fire situation the generation of particulate matter may therefore be significant. Such a fire would also be expected to result in the generation of some PAHs, other complex compounds and organic irritants which would be present in the greatest quantities at the combustion source. If the temperatures are high enough, these more complex organic chemicals may be completely broken down. The health hazard that these compounds pose will be reduced as the distance from the source increases, but could be of concern for individuals directly exposed to the plume.

The combustion of petrol or oil, will most likely lead to the generation of irritants and particulate matter in the effluent plume, which may be expected to be the greatest hazard to the health of individuals outside the immediate vicinity of the source.

4.5 Fires involving hazardous chemicals (chemical / pesticide manufacturer / storage)

This scenario involves a fire at a facility concerned with the manufacture or storage of hazardous chemicals, such as an industrial chemical or pesticide manufacturer. In such a situation the hazardous materials present may be evolved unchanged during the fire. Specific examples of hazardous chemicals which may either be present unchanged or as hazardous decomposition products in a smoke plume include organophosphorous and organochlorine pesticides. The compounds and products arising during combustion at a location where hazardous chemicals such as pesticides may be present will be largely dependent upon the specific chemicals present at the source of the fire. Pesticides containing chlorine for example, are unlikely to undergo complete decomposition under most combustion conditions, giving rise to the presence of hazardous organochlorine compounds in the smoke plume.

In general, if the quantity of these specialised chemicals in a fire situation was small and that any emissions were well dispersed, there is unlikely to be any significant additional risk to that arising from the smoke from any large building fire.

4.6 Fires involving asbestos

Large scale fires in which the fabric of the building may contain asbestos, eg, from asbestos cement roofing give rise to significant concern by the public regarding exposure to asbestos. However, there is considerable data to show that providing appropriate clean-up procedures are followed, there is no significant public health risk resulting from the asbestos [118].

Table 6: Hazardous combustion products generated with respect to the material involved.

Material Involved	Fire Zone	CO	HCN	HCl / HBr / HF	NO_x	SO_2	P_2O_5	Organic Irritants, eg, Acrolein / Formaldehyde	Inorganic Irritants, eg, Phosgene / Ammonia	PAHs	Complex / Exotic, eg, PCDDs / PCDFs / Isocyanates / PFIB	PM
Polymeric Materials	1	+++	+++	+++	++	+	+	++	+	++	++	+++
	2	±	±	+	+	±	-	++	+	±	++	++
Wood	1	++	-	-	+	-	-	++	-	+	+	+++
	2	-	-	-	±	-	-	+	-	±	+	±
Rubber / Tyres	1	+++	+	+	+	+++	+	++	+	++	++	+++
	2	±	±	+	±	+++	±	++	±	±	++	++
Oil / Petrol	1	++	-	-	-	±	-	++	-	++	++	+++
	2	-	-	-	-	±	-	++	-	±	+	++

Zone 1 relates to the immediate vicinity / compartment of the fire. Zone 2 relates to the location immediately outside the source of the fire.

Key:

+++	Likely to be present in very high quantities
++	Likely to be present in high quantities
+	Likely to be present
±	May be present at low level
-	Unlikely to be present

4.7 Smoke behaviour

The generation of hazardous compounds are likely to pose less of a health hazard for individuals as the distance for the source of the fire increases, due to dispersion and conversion of the products as the smoke plume travels further from the source. The area affected by the plume of smoke varies, sometimes rapidly, depending upon the meteorological conditions, including wind speed, wind direction and atmospheric stability. This plume can be modelled by the Met Office using modelling software such as Chemet.

Some hazardous combustion products are produced in large quantities during limited ventilation, such as CO and NO but are readily converted into CO_2 and NO_2 respectively, as the availability of oxygen in the effluent plume increases. These compounds are therefore likely to only present a significant hazard to health for individuals who are in the same compartment (such as room or building) or immediate vicinity of the fire source (zone 1). For individuals in this zone, the fire environment may become lethal within a matter of minutes, due to heat and the rapid generation of toxic compounds at life threatening concentrations. In zone 1, the most important hazardous combustion products are the asphyxiant gases, CO, HCN, CO_2 and the low availability of oxygen as these may be fatal in just a few minutes. The hazard from these asphxiant gases can however be confounded by the presence of irritant gases and a high optical density of the smoke, since this may impede the escape from this zone, thereby prolonging the exposure duration. The asphyxiant gases produced from a well developed fire contained within a building may however, be fatal to individuals present in a location distant to the source of the fire. As the concentration of gases produced from the fire increases, the smoke will disperse throughout the building. As the concentration of the compounds within the smoke increases steadily, a lethal concentration may be reached without an individual becoming aware (which can often occur if the individual is asleep). For this reason individuals may be found in locations within the same compartment but, distant from the source of the fire who have been overcome by hypoxia, with no sign of fire damage in that location. Due to the immediate hazards of a combustion environment such as heat, flames and lack of structural integrity, in addition to the presence of asphyxiant gases at lethal concentrations, zone 1 of a fire is most likely to be of major concern to the emergency services.

The exposure of individuals to the fire effluent in the zone outside the immediate fire zone (zone 2) would be expected to be of most concern to public health. This zone is likely to cover many more people than zone 1, and it may not be immediately obvious that these individuals are at risk. The combustion products present in this area are less likely to pose an immediate danger to life, but should be considered as hazardous. Asphyxiant gases such as CO are likely to be present in the effluent at much lower concentrations than would be seen in zone 1, and are therefore likely to be less of a hazard to health, unless individuals are directly in contact with the effluent. The major immediate hazard to public health in zone 2 is therefore, expected to be exposure to irritants and particulates generated in the effluent. Low concentrations of the irritant gases may cause significant irritation of the eyes and respiratory tract, which may affect a large number of people who are not directly exposed to the effluent plume. The adverse effects resulting from exposure to these irritants are likely to be completely

resolved following removal from the exposure, with no long term sequelae. However, the generation of more complex products such as PAHs, dioxins, dibenzofurans and particulate matter are of concern, but are likely to present a significantly greater hazard from long term or repeated exposure than following a large single acute exposure. Any risk of carcinogenicity from PAHs or dioxins from a single exposure is likely to be very low.

5 MAIN CONCLUSIONS

The prediction of toxic combustion products is a complex area and there is the potential for generation of a huge range of pyrolysis products depending on the nature of the fire and the conditions of burning. Although each fire will have individual characteristics and will ultimately need to be considered on a case by case basis there are commonalities, particularly with regard to the most important components relating to toxicity.

- Asphyxiant gases (carbon monoxide, hydrogen cyanide and carbon dioxide) and low oxygen concentration are most likely to be of concern to individuals in zone 1 (within the fire compartment / building), but are less likely to pose a major hazard to public health to individuals in zone 2 (outside the immediate compartment of the fire) due to dispersion and dilution.

- The most common potential hazards to individuals in zone 2 from all considered sources are;
    - organic irritants, including acrolein and formaldehyde
    - complex molecules, including dioxins, dibenzofurans, isocyanates
    - particulate matter

- Scenarios in which the combustion is considered to be incomplete, due to low temperature, lack of ventilation and absence of flaming would be expected to form the greatest quantities of hazardous combustion products.

These generalisations may assist in rapidly identifying which hazardous combustion products are likely to be of most concern to public health during a fire depending upon the materials involved.

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