cells resulting in the formation of carboxyhaemoglobin (COHb), with stability constant 200 times greater than that of oxyhaemoglobin, preventing the transport of oxygen from lungs to the body resulting in reduced blood oxygen capacity. This causes deterioration in mental and muscular performance. CO also combines with myoglobin in the muscle cells, impairing diffusion of oxygen to cardiac and skeletal muscles [2, 5].

Hydrogen cyanide is approximately 25 times more toxic than carbon monoxide. It owes its toxic effects to the cyanide ion formed by hydrolysis in the blood. Unlike carbon monoxide which remains primarily in the blood, the cyanide ion is distributed throughout the extra cellular fluid of tissues and organs and can react with the enzymes present. Two mechanisms have been identified for the incapacitation and lethal effects of cyanide. The first is by combination with the ferric ion in mitochondrial cytochrome oxidase, preventing electron transport in the cytochrome system and inhibiting the use of oxygen by the cells. The second mechanism is identified as brief stimulation, immediately followed by severe depression of respiratory frequency (tachypnea), lasting until convulsions, respiratory arrest and death (demonstrated in mice). It is not clear which of these mechanisms normally predominate. A first breath of HCN causes asphyxiation of the cells, as the enzyme responsible for using oxygen to supply energy to bodily tissues can no longer work. Then nausea, headache, dizziness, vomiting and weakness occur, together with skin irritation and rashes, eye irritation, and blurring of vision leading to permanent eye damage. As poisoning progresses, HCN starts to have an effect on the nervous system and inhibits heartbeat, decreases blood pressure, and delivers effects such as hallucinations, unconsciousness, and convulsions [2].

Irritants. In contrast to the direct effects of asphyxiant toxicants, the effects of exposure to irritants (hydrogen bormide, hydrogen chloride) are much more complex. Irritant gases such as hydrogen chloride (HCl), hydrogen bromide (HBr) produce signs and symptoms of both sensory and upper respiratory tract irritation, and of pulmonary irritation. Depending upon the concentration of an irritant and the duration of exposure (i.e. the dose) sensory/upper respiratory tract irritation stimulates the trigeminal and vagus nerve receptors in the eyes, nose, throat and upper respiratory tract causing discomfort, then severe pain. The effects may include tears and reflex blinking of the eyes, pain in the nose, throat and chest, breath-holding, coughing, etc. At sufficiently high concentrations, most irritants can penetrate deeper into the lungs, causing pulmonary irritation effects which may cause post-exposure respiratory distress and death, generally occurring from a few hours up to several days after exposure, due to pulmonary oedema then cause immediate incapacitation, mainly by effects on the eyes and upper respiratory tract, and longer term damage deeper in the lung [2].

Nitric oxide causes vasodilation of blood vessels in the lung area that actively participates in the exchange of oxygen and carbon dioxide. When inhaled at low concentrations, nitric oxide essentially induces vasodilation and especially pulmonary vasodilation and hence improves the blood supply in the lungs, reduces blood pressure and improves oxygen levels in the bloodstream. It passes quickly into the bloodstream, quickly reacts with the haemoglobin and is transported around the body. It can combine with oxyhaemaglobin to form methaemaglobin and nitrates but if the blood oxygen is low it can combine with haemoglobin to form nitrosylemoglobin. The inability of methaemoglobin to combine with oxygen can give effects similar to hypoxia. Coma and death can ensue at high methaemoglobin levels. Excessive levels of nitric oxide in blood have been shown to cause low blood pressure. However, it has been reported that tobacco smoke can contain up to 1000 ppm of nitric oxide but this does not produce acute mortality [5].

Fire hazard assessment

Death or incapacitation may be predicted by quantifying the fire effluents in different fire conditions in small-scale tests, using chemical analysis. Lethality may be predicted using equations, based on rat lethality data, presented in ISO 13344 [12]. Incapacitation (the inability to effect one's own escape) may be predicted using methodology and consensus estimate data in ISO 13571 [13].

The general approach in generating toxic potency data from chemical analysis is to assume additive behaviour of individual toxicants, and to express the concentration of each as its fraction of the lethal concentration for 50% of the population for a 30 minute exposure (LC_{50}) [12]. Thus an FED equal to one indicates that the sum of the actual/lethality concentration ratios of individual species will be lethal to 50% of the population over a 30 minute exposure. Since CO_2 increases the respiration rate, the Purser model, presented in equation, uses a multiplication factor for CO_2 driven by hyperventilation, V_{CO}, to increase the FED contribution from all the toxic species, and incorporates an acidosis factor A to account for toxicity of CO_2 in its own right [12].

$$FED = \left\{ \frac{[CO]}{LC_{50,CO}} + \frac{[HCN]}{LC_{50,HCN}} + \frac{[HCl]}{LC_{50,HCl}} + \frac{[NO_2]}{LC_{50,NO_2}} + ... + \text{organics} \right\} \times \times V_{CO_2} + A + \frac{21-[O_2]}{21-5.4}$$

$$V_{CO_2} = 1 + \frac{\exp\left(0.14[CO_2]\right)-1}{2}$$

A is an acidosis factor equal to $[CO_2] \times 0.05$.

Fed assessment for building materials

The steady state tube furnace allows the ventilation to be controlled without affecting the rate of burning [14 ÷ 16]. The apparatus is able to investigate the production of smoke (aerosol or particulate matter) and combustion gases for different fire scenarios, but can also be used to study different characteristics such as ignitability and heat release by oxygen depletion [17].

The thermal insulation materials were selected to have comparable densities [18]. The fire toxicity of six insulation materials: glass wool (GW), stone wool (SW), expanded polystyrene foam (EPS), phenolic foam (PhF), polyurethane foam (PUR) and polyisocyanurate foam (PIR) were investigated under a range of fire conditions, oxidative pyrolysis, and well-ventilated flaming to under-ventilated flaming [19].

For the two fibrous materials in the flaming condition, stone wool and glass

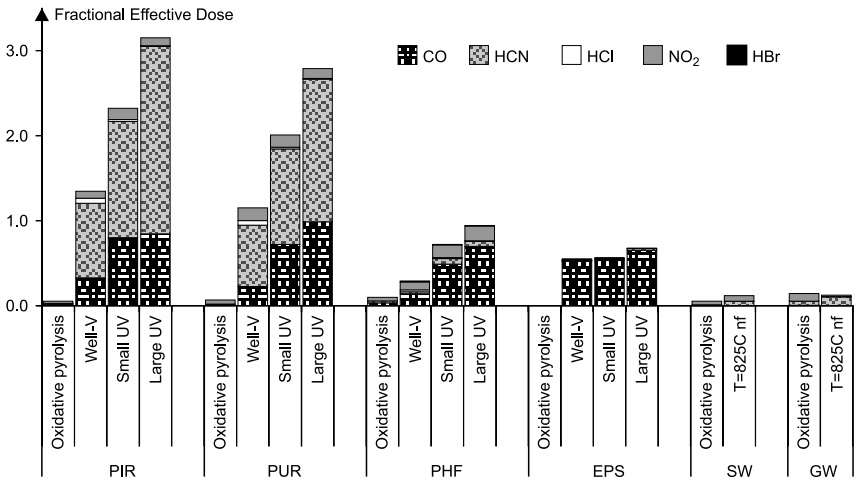

Fig. 2. FED for insulation materials (for oxidative pyrolysis and flaming conditions, except SW and GW non-flaming (nf)) [19]

wool, ignition was not observed even above 800 °C (nf). Both glass wool and stone wool are classified as non-combustible or limited combustibility depending on the binder content. While both lose small (~5%) quantities of pyrolysable binders, most of the mass will not burn and there is insufficient fuel for a flame to propagate through the bulk of the material, so their contribution to the fuel load is negligible. For the four foams, PUR, PIR, PhF and EPS, ignition and steady flaming was achieved for the two flaming fire conditions and for intermediate ventilation conditions [19]. Figure 2 shows the increase in carbon monoxide yield as the ventilation changes from well-ventilated to under-ventilated. In comparison to polymers without flame retardants, the CO yields for PIR and PUR in well-ventilated conditions are high [19]. For the two nitrogen containing polymers, PUR and PIR, the HCN yield is significant, both in well-ventilated flaming, and for under-ventilated flaming, and those materials have much greater fire toxicity than EPS or PhF. The glass wool and stone wool products show very low fire toxicity. The variation of NO_2 with fire conditions for the three materials shows less consistent trends although the phenolic foam seems to show a progressive increase as the fire condition becomes under-ventilated. For PIR and PUR, there is a slight decreasing trend with under-ventilation, which corresponds to the increased yields of HCN, and reduced availability of oxygen. HCl and HBr were close to limits of detection.

Conclusions

- Most fire deaths arise from inhalation of toxic gases, though the incapacitating effects of smoke and irritant gases also play an important role.
- Fire growth has been classified into a number of stages, from oxidative pyrolysis and early well-ventilated flaming, through to fully-developed under-ventilated flaming. The steady state tube-furnace (ISO/DIS 19700) provides an excellent method for exploring the relationship between combustion conditions and product yields.
- Assessment of fire gas toxicity is an essential component of fire hazard analysis. The toxicities of the effluents, showing the contribution of individual toxic components, are compared using the fractional effective dose model. The predicted toxicities show variations of up to two orders of magnitude with change in fire scenario. They also show changes of at least one order of magnitude for different materials in the same fire scenario. Finally, they show that in many cases CO, which is often assumed to be the most, or even the only toxicologically significant fire gas, is of less importance than HCl or HCN when chlorine or nitrogen is present in the compound. For polyisocyanurate and polyurethane foam there is a significant contribution from hydrogen cyanide resulting in doubling of the overall toxicity, as the fire condition changes from well-ventilated to under-ventilated.

The insulation materials showed an order of increasing fire toxicity, from stone wool (least toxic), glass wool, polystyrene, phenolic, polyurethane to polyisocyanurate foam (most toxic).

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