PHYSIOLOGICAL EFFECTS OF COMBUSTION PRODUCTS AND FIRE HAZARD ASSESSMENT

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ABSTRACT

A major design requirement for any occupied enclosure is to ensure that occupants are able to escape safely in case of fire. The main cause of injury and death in fires is incapacitation resulting from exposure to fire effluent (smoke and gases). During fires, physiological effects dominate and determine time to incapacitation. The sequence of physiological hazards in developing fires can be applied to fire safety engineering design of buildings and to the evaluation of products for use in them, using appropriate fire data and calculation methods for assessment of time to incapacitation

INTRODUCTION

A major design requirement for any occupied enclosure (such as a building or transport vehicle) is to ensure that occupants are able to escape safely in case of fire. The main cause of injury and death in fires is exposure to toxic fire effluent (smoke and gases), while the next most important cause is exposure to heat. It is therefore necessary to ensure that the performance of the building and it systems, including any combustible structural products or contents, is such that occupants are able to escape before they are overcome by toxic smoke or heat. This is a regulatory performance requirement in the UK Building Regulations. Requirement B1 of Schedule 1 to the Building Regulations 2000[1] states that: "The building shall be designed and constructed so that there are appropriate provisions for the early warning of fire, and appropriate means of escape in case of fire from the building to a place of safety outside the building capable of being safely and effectively used at all material times". A similar objective is stated in the ISO fire engineering Technical Report (ISO/TR 13387-8 Fire safety engineering Part 8: Life Safety - Occupant behaviour, location and condition)[2]. “Should a fire occur in which occupants are exposed to fire effluent and/or heat, the objective of the fire safety engineering strategy is to ensure that such exposure does not significantly impede or prevent the safe escape (if required) of essentially all occupants, without their experiencing or developing serious health effects”.

Table 1: Fire causes - houses and flats

Frequency	Cause
25	Electrical
13	Chip pan
7	Grill/frying pan
5	Gas
5	Arson
4	Misuse of heating
4	Ignition by child
3	Chimney
3	Unknown
2	Cigarette
2	Cooking
1	Coal falling from fire
1	Overheating of wax
1	Paint stripping
1	Candle
1	Cooker, misuse of
78	SUM

Table 2: Causes – electrical fires houses and flats

Frequency	Cause	Cause detail
3	Electrical	Washing machine
3	Electrical	Tumble dryer
3	Electrical	Immersion heater
3	Electrical	Electric blanket
2	Electrical	Wiring
2	Electrical	TV
2	Electrical	Mains
1	Electrical	Toaster
1	Electrical	Stereo
1	Electrical	Iron
1	Electrical	Hi Fi
1	Electrical	Fridge freezer
1	Electrical	Fridge
1	Electrical	Tumble dryer lead
25	SUM	

In a detailed study of 100 domestic fires in the United Kingdom[3], by far the greatest single cause

of fire was electrical (32% in houses and flats), mostly arising from faulty appliances. The next biggest cause was related to cooking (22 fires = 28% for dwellings). If cooking fires and deliberate fires are excluded, leaving 51 fires due to other causes, then half of all accidental fires resulted from electrical faults. After electrical and cooking fires the next most important causes were gas appliances and deliberate ignition. Cigarettes and smokers materials generally were a very minor cause at 2.6%.

Electrical fires are detailed in Table 5. Half due were due to faults in domestic appliances and one third due to faults in supply or building services. These were of particular concern to occupants because they were completely unexpected, largely beyond their control and the cause was often never fully established. Occupants felt they needed to know why these fires had started and what they could have done to prevent them. They were some of the most serious and damaging incidents. In a number of major incidents in larger building electrical fires have presented major hazards. In some cases a key problem has been the size of the source fire, such as large fires in plant rooms in locations such as hotel basements, which have involved large volumes of toxic effluents spreading through a building via vents and stairwells. In other incidents involving fires in cable installations, both fire and fire effluents have spread between floors via ducts and plenum spaces. Particular issues with most fires involving electrical installations are the large amounts of smoke and toxic gases evolved in the effluent plumes, so that even small fires can lead to serious contamination of large buildings.

For these reasons it is particularly important that the potential physiological and toxic hazards of fires involving electrical installations are considered in parallel with the potential hazards from other combustible products in building and transport design

Since fire hazards are essentially time-based phenomena. The objective is to ensure as far as possible that should a fire occur, the occupants receive a timely warning and have suitable escape routes available for a sufficient time to enable escape before conditions deteriorate to such an extent that they can no longer use them. In fire safety engineering terms it is necessary to ensure that available safe escape time (ASET) is greater than required safe escape time (RSET) by an acceptable margin of safety[2].

The main tenability limits for ASET are conditions that cause incapacitation of occupants such that they are unable to escape.

Toxicity resulting from exposure to any substance arises from a combination of physiological effects on the function of body systems and pathological effects on body tissues. For the toxic effects of exposure to combustion products in fire effluent, particularly in relation to the survival, injury or death of fire victims, it is mainly the more immediate physiological effects that dominate at the fire scene. This is because physiological effects occur very rapidly, often within a few seconds, while pathological changes tend to occur over time scales of from hours to years. Physiological effects also occur in response to heat and smoke exposure in fires, interacting to some extent with the effects of toxic combustion products.

The acute physiological hazards in fires affecting escape capability are as listed below[4,5].

ACUTE SURVIVAL HAZARDS DURING FIRES

- Impaired vision from smoke obscuration
- Impaired vision, pain and breathing difficulties from effects of smoke irritants on eyes and respiratory tract.
- Asphyxiation from toxic gases leading to confusion and loss consciousness
- Pain to exposed skin and respiratory tract followed by burns from exposure to radiant and convected heat leading to collapse

These tend to be encountered more or less in the order shown, with exposure first to smoke, which is likely to be irritant, followed by asphyxia or burns, depending upon the type of fire scenario and the proximity of the person to the seat of the fire. Once a victim has become

trapped or incapacitated in a fire, then conditions usually become lethal within a further few seconds or minutes. This is because flaming fires grow exponentially, so that concentrations of smoke and toxic gases and the heat intensity increase rapidly, resulting in death either from asphyxiation or heat exposure depending upon the fire scenario. For this reason the key determinant of survival is incapacitation, while the lethal potency of fire effluent is of limited relevance.

Figure 1 shows an example of a typical set of time-concentration curves from a full-scale fire test of an item of upholstered furniture in the open lounge of a house[6]. In order to estimate the time when a room occupant would be incapacitated it is necessary to consider the developing hazards from each toxic gas, the smoke and heat and their interactions. For some toxic products (such as irritants and smoke particulates) the effects depend mainly upon the immediate concentration to which the subject is exposed, while for others (asphyxiant gases and heat) the total exposure dose is the most important feature (expressed in terms of varying functions of concentration x exposure time), so that effects are delayed until a sufficient dose has built up in the body to produce a given physiological effect[5,7]

Other important considerations are the nature of the physiological effects and the relationships between exposure concentration (or exposure dose) and the type and severity of effect. The intensity of physiological effects of stimulation of sense organs such as hearing, vision or pain reception tend to show a progressive increase, logarithmically related to the intensity or concentration of the stimulus. Thus walking speed in smoke is inversely proportional to the smoke optical density[8], while the painful effects of irritant gases lie on a continuum from mild eye and nasal irritation at low concentrations to intense pain, involuntary eye closure and breathing difficulties at high concentrations[5,7,9]

Asphyxiant gases act by limiting the supply or use of oxygen in the body tissues (tissue hypoxia). Since the body systems are designed to tolerate a degree of hypoxia (for example during exercise), the effects of low doses or asphyxiants are minor, with little change as the body dose increases other than some degree of reduction in exercise tolerance, and subtle sensory or cognitive effects, until a critical threshold level is reached above which a sudden severe and dramatic loss of functionality occurs. At this point subjects pass rapidly from near normal behaviour and function to collapse and loss of consciousness[5,10,11,12,13]

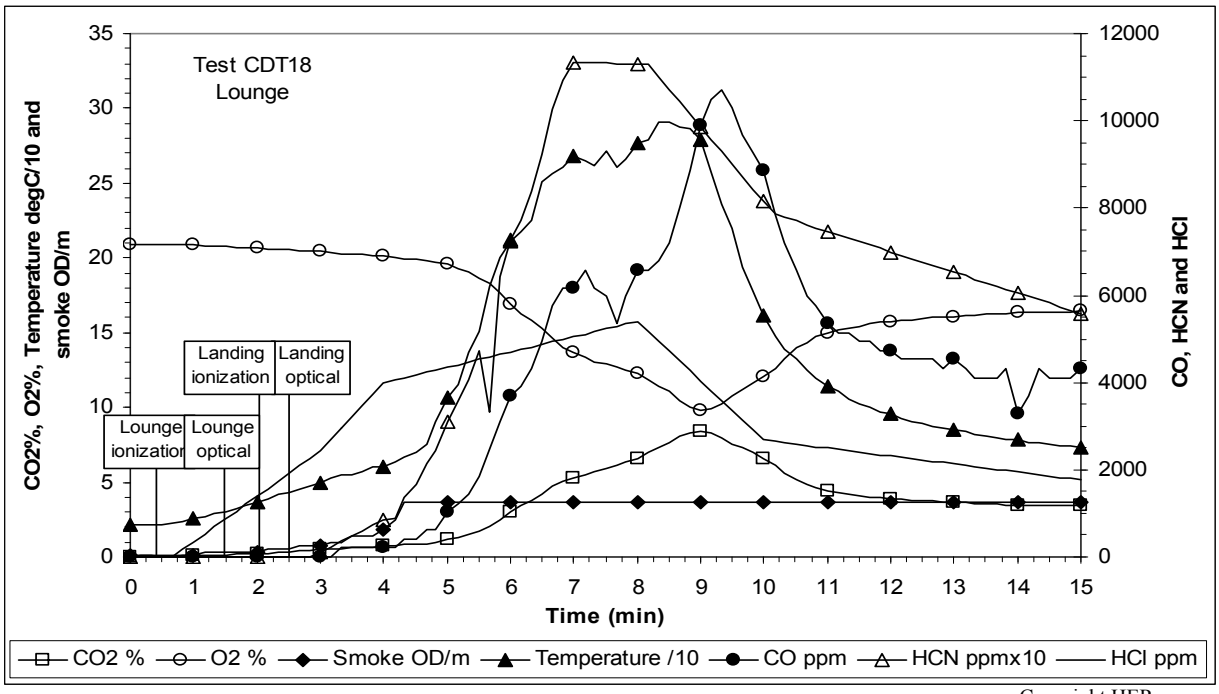


Figure 1: Example of time-concentration curves for smoke, toxic gases and temperature at head height in the domestic lounge of a house during an armchair fire. The doorway to the hall is open but the house is otherwise enclosed. The time for triggering of smoke detectors is shown

BASIC REQUIREMENTS FOR LIFE SAFETY HAZARD ANALYSIS

Life safety hazard from fires in buildings (or other enclosures) therefore depends upon the performance of a dynamic system involving interactions between the building, the fire and the occupants.

In order to determine the ASET time for any system it is necessary to determine

- The time-concentration (or time-intensity) curves for the major toxic products, smoke and heat in the fire at the breathing zone of the occupants, which in turn depend upon:
    - Fire growth curve (mass loss rate of the burning fuel [kg/s] and its dispersal volume [kg/m^3] with time)
    - The yields of the major toxic products (kg/kg) and heat (kj/kg) (for example kg CO per kg of material burned).

These terms can be measured directly in full-scale tests or calculated using appropriate fire dynamics computations, with appropriate input data including reaction-to-fire properties and data on product yields under a range of fire conditions.

- The concentration/time/physiological effect relationships of these products in terms of the physiological/toxic potency of the products and heat (the exposure concentration [kg/m^3]), or exposure dose (kg·m^{-3}·min or ppm·min) required to cause toxic effects (and the equivalent effects for heat and smoke obscuration).
    - Concentrations, doses (or heat intensity) likely to impair escape efficiency due to behavioural and/or physiological effects
    - exposure concentrations or doses likely to cause incapacitation or prevent escape due to behavioural and/or physiological effects
    - Lethal exposure concentrations or doses

These terms can be calculated by the application of appropriate physiological methods (Fractional Effective Dose methods) outlined in this paper and presented in detail in the SFPE Handbook of Fire Protection Engineering[5] and other publications[14,15,16]. A simplified method is present in ISO 15371[17].

In order to evaluate the fire safety of any built environment, or any combustible material or product used in a built environment it is therefore necessary to consider the overall fire performance of the system, either by performing a full-scale fire test (or series of tests) on the end-use system or by modelling the full-scale fire conditions, using suitable fire dynamics models run with a input data on a range of performance parameters obtained from a set of small or large scale fire tests.

Problems in representing "toxicity" of a burning material or product as a single test result constant

It should be evident from this that for systems containing combustible materials, no single small-scale test can possibly supply all the data necessary to evaluate the full-scale fire performance. It should be equally evident that representing "toxicity" as a single parameter for which data can be obtained directly from any small-scale test represents a gross oversimplification of a more complex reality. Even if the time-varying nature of both fire conditions and physiological effects are ignored, it is not possible to represent the "toxicity" or "toxic potency" of the combustion products from a material or commercial product by a single number because the yields of

individual toxic gases responsible for the overall toxic effects vary considerably between different combustion conditions. Combustion products consist of a mixture of individual toxic gases and particulates that vary in concentration throughout the fire exposure. The yields of each toxic species from any one burning material vary considerably (by up to several orders of magnitude) depending upon the combustion conditions, which in turn depend upon the varying fuel/air ratios and temperatures in the fire. Taking PMMA as an example, Figure 2 shows how the yield of carbon monoxide varies by a factor of approximately 50 as combustion conditions vary from well-ventilated (phi<1) to vitiated (phi >1) in two bench-scale apparatus (the ASTM E2058 flammability apparatus and the ISO TS19000 tube furnace[18,19]). Similar variations in toxic product yields with phi have also been shown to occur with other toxic combustion products from different materials including smoke particulates, total organics, hydrogen cyanide and oxides of nitrogen[20, 21]. These two test methods have both been developed specifically to measure the yields of toxic species from materials and products over a range of combustion conditions occurring in full-scale fires. The data obtained can then be used as part of the input needed for toxic hazard assessment or full-scale fire scenarios.

Although it is considered to have limited relevance to full-scale toxic hazards, small-scale tests such as the tube furnace test have been used to calculate figures for the overall toxic potency of the combustion product mixture obtained under any specific test condition. Toxic potencies estimated from small-scale test methods are usually quoted in terms of lethal toxic potency or a toxicity index number. The lethal toxic potency expressed as the LC_{50} is the mass loss concentration of a material (g/m^3) capable of causing death in 50% of a group of exposed rats after a 30-minute exposure period, with 14-day post-exposure observation period. Historically this was measured directly using animal exposures but is now usually estimated from the measured concentrations of toxic gases in a test using a calculation method. Toxicity indices are usually based upon published IDLH (immediately dangerous to life or health) values for specific toxic gases. For both types of potency estimates a simple additive model is used for the contributions from each individual toxic gas component, thereby ignoring the different time-related physiological effects and the interactions occurring between the different components. Figure 3 illustrates how the calculated LC_{50} concentrations for different materials decomposed in the tube furnace vary with different combustion conditions, depending upon the varying yields of the main toxic species[22]. In practice the lethal toxic potencies for common materials over different combustion conditions vary by a factor of around 100, while one group of materials can have a toxic potency approximately 1000 times greater than average under certain thermal decomposition conditions[5,23].

There are therefore two major problems in attempting to derive toxicity performance from existing small-scale toxicity test methods.

- Firstly most test methods reproduce only a single combustion condition, for which the relationship to combustion conditions existing in any particular type of stage of full-scale fires has often not been established
- Secondly the concept of an overall toxicity value for the mixed combustion products from any material or product has little meaning in relation to full-scale fire hazards, because it ignores the time-based nature of both fire atmospheres and the effects of the different toxic components.

In order to evaluate the fire safety of any built environment, or any combustible material or product used in a built environment it is therefore necessary to consider the overall fire performance of the system, either by performing a full-scale fire test (or series of tests) on the end-use system or by modelling the full-scale fire conditions using suitable fire dynamics models run with input data on a range of performance parameters.. It is then necessary to apply suitable algorithms to calculate the time at which different physiological ASET endpoints occur.

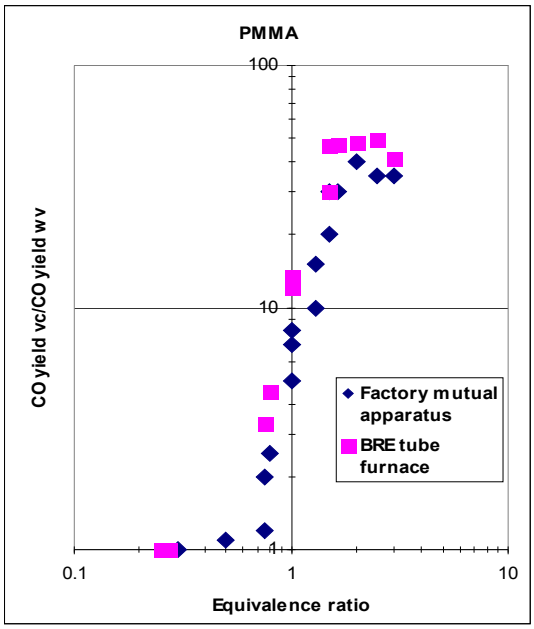


Figure 2: Variation of CO yield with combustion conditions in the ASTME2058 and ISO TS1900 test methods

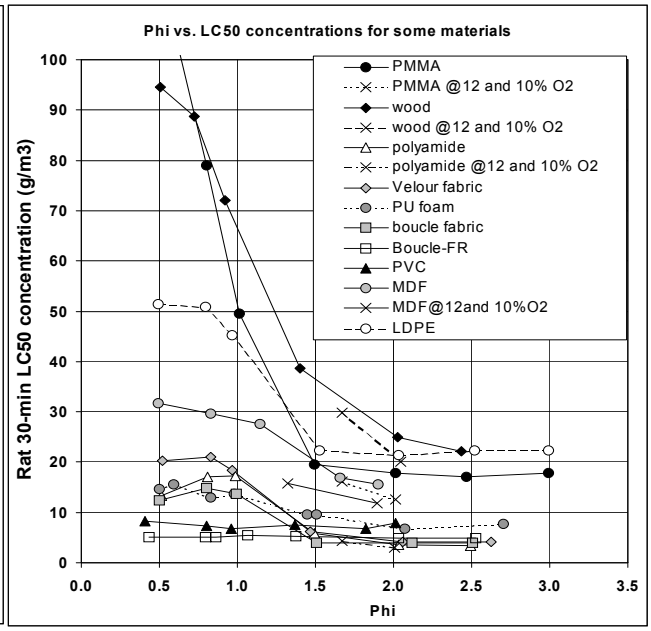


Figure 3: Variations in lethal toxic potency (LC_{50}) concentrations with combustion conditions in the ISO

Methods for estimating time to incapacitation for the main toxic gases, smoke and heat in fires (Fractional Effective Dose models [FED models]) have been in existence for somewhat longer than suitable fire dynamics models have been available for their application, but the increasing sophistication of fire modelling methods now makes the wider application of such methods feasible in addition to their direct application to measured results of full-scale fire tests

This paper outlines the physiological basis of the FED models for which a detailed description is provided in the SFPE Handbook[5] and other references, and a more simplified form in ISO 13571[17]. Some options are then presented on suitable approaches to product evaluation in relation to potential toxic hazards with specific comments on cable fires

METHODS OF FIRE HAZARD ANALYSIS BASED UPON PHYSIOLOGICAL ALGORITHMS

Fire hazard assessment is based upon a “step through” approach whereby the extent of the hazard is calculated for each successive minute (or other appropriate time interval) during the fire, until the point is reached where different hazard endpoints are predicted

The behavioural and physiological effects of exposure to toxic smoke and heat in fires combine to cause varying effects on escape capability, which can lead to physical incapacitation and permanent injury or death.

For a basic fire engineering design there are two main considerations:

- The time at which the concentration of effluents reaches a level such that safe escape is compromised due to:

  a) behavioral and physiological effects of exposure to heat and toxic smoke on escape capability;

  b) in the absence of direct exposure, behavioural effects caused by seeing fire effluents on escape behaviour.

The effects of exposure to smoke result from impaired vision due to the optical opacity of smoke and from the painful effects of irritant smoke products and on the eyes and respiratory tract. Occupant movement speed and wayfinding ability is impaired. Behavioural effects of seeing or being immersed in smoke result in a proportion of occupants being unwilling to approach smoke or heat-logged areas or escape routes.

- The time at which the exposure dose of asphyxiant toxic gases or heat reaches a level at which occupants is are likely to become incapacitated - such that they cannot save themselves and are likely to die within a minute or so unless rescued.

In a regulatory or design evaluation context several different considerations exist. In flaming fires the effluent is buoyant, so tends initially to fill enclosures from the ceiling downwards. This presents a potential opportunity for occupants to escape before the smoke layer descends to near head height, but occupants are still exposed to heat radiation from the hot fire effluent in the upper layer. Larger building such as shopping malls are often designed with smoke venting or extraction to maintain the upper layer above a minimum clear layer height of 2.5 m with a maximum upper layer temperature of 200°C. Occupants are considered to be willing and able to escape in clear air under such a layer and the downward heat radiation is considered tolerable. Above this temperature the downward radiation is capable of causing pain and burns within a short period. While such layering may be maintained for some minutes in large open structures, in most fires some degree of smoke mixing to near floor level occurs as effluent moves through building spaces and as the fire grows.

EFFECTS OF EXPOSURE TO NON-IRRITANT AND IRRITANT SMOKE

Smoke contains a mixture of gases, liquid droplets and solid particles capable of exerting a range of adverse physiological effects on escaping occupants. The size and composition of soot particles varies with the nature of the burning fuel and the combustion conditions. The main solid component under well-ventilated combustion conditions is soot particles comprised mainly of carbon. These cover a range of sizes but a considerable proportion is small and highly respirable. As combustion becomes less efficient these particles become mixed with a wide variety of organic compounds many of which are irritant to the eyes and respiratory tract. These compounds adhere to the particles and may also be present in vapour or liquid droplet form. If the burning fuel contains hetero-elements such as halogens, nitrogen or sulphur, then irritant acid gases are also present[5].

The first physiological effect: impairment of visibility, occurs even before a subject is immersed in smoke. Seeing smoke-logged areas, for example in escape routes, may impair escape behaviour and wayfinding.

In situations where smoke is mixed down to near floor level, some building occupants may move through dense smoke in some situations, but in other situations people may be unwilling to enter smoke logged escape routes, turn back or be unable to find an exit. Where heat is not an issue, the immediate effects of smoke depend upon the visibility distance, and the sensory irritancy of the smoke if people are exposed directly. For such situations it is necessary to set tenability criteria for design purposes, depending upon the level of adverse effects on occupants considered acceptable or non-acceptable

In a number of studies of fires in buildings, a proportion of people (approximately 30 %) were found to turn back rather than continue through smoke-logged areas[5,24, 25,26]. The average density at which people turned back was at a “visibility” distance of three metres. This represents an optical density ($OD \cdot m^{-1}$) of 0.33, (extinction coefficient 0.76) and women were more likely to turn back than men. A difficulty with this kind of statistic is that, in many fires in buildings, there is a choice between passing through smoke to an exit or turning back to take refuge in a place of relative safety such as a closed room. In some situations, people have moved through very dense smoke when the fire was behind them, while in other cases people have failed to move at all.

Behaviour might also depend on whether layering permits occupants to crouch down to levels where the smoke density is lower and whether low-level lighting is used to improve visibility.

Based upon considerations such as these in relation to parameters such as the size and complexity of the building, it is possible to set design limits for optical density of smoke. As an approximate guide it might be assumed that occupants will not use an escape route if the visibility in that route is less than three metres (OD/m = 0.33, extinction coefficient 0.76). However, if they enter an escape route contaminated to this optical density and become exposed to the smoke, then their ability to progress depends upon both the optical density and the irritancy of the smoke.

Figure 4 shows the effects of exposure to non-irritant smoke and irritant wood smoke on walking speed, and walking speed in darkness, derived from the work of Jin[8]. For Jin's study, volunteers walked along a corridor filled either with non-irritant theatrical smoke or irritant smoke generated using a bee smoker. The fuel consisted of wood chips, and although the exact decomposition conditions are unknown, they most likely involved a mixture of smouldering and non-flaming oxidative decomposition. This would produce a smoke containing a range of irritant organic species at significant concentrations. For non-irritant smoke there is a more or less linear relationship between walking speed and OD/m, and at around 0.55 (representing about 2 meters visibility), peoples' behaviour was observed to change from walking as if in the light to walking in darkness. Under these conditions people stopped walking directly along the corridor and started to feel their way along the walls using their hands. In an unfamiliar building subjects move very slowly for fear of tripping over obstacles or falling down a stair.

An important aspect of Jin's experiments is the difference between walking speed in non-irritant and irritant smoke. As Figure 4 shows, for a given optical density, walking speed was much slower in irritant smoke, with people walking as if in darkness at an optical density of around 0.2. A further problem in smoke is not just the visibility through the smoke, but the reduction in overall illumination. Lighting, including warning signs, are placed mainly at high level, so that when a dense smoke layer forms under the ceiling, the conditions in a building can become quite dark.

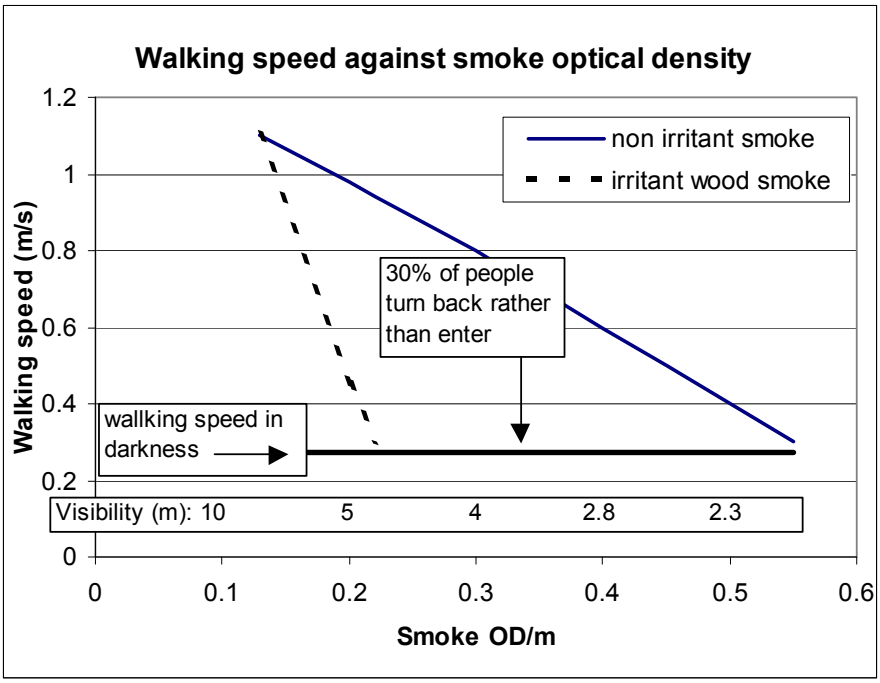


Figure 4 — Walking speeds in non-irritant and irritant smoke

Equations 1 and 2 for the relationship between walking speed and smoke optical density (OD·m^{-}

[1]), (together with all other physiological equations) are presented in the last section of the paper .

Based upon the finding that people move as if in darkness at a visibility of five metres in irritant smoke, and that smoke from most fires contains a variety of irritant chemical species, the generic design tenability limits shown in Table 3 are proposed, on the basis that concentrations exceeding these levels could impair or even prevent occupants' safe escape (for situations where the concentrations of acid gases are considered unlikely to be significant due to the fuel composition):

Another advantage of using these smoke concentration limits as tenability criteria is that the concentrations of asphyxiant gases present are most unlikely to exceed levels capable of causing incapacitation (loss of consciousness) in exposed occupants within 30-60 minutes.

Table 3 — Smoke tenability limits

Smoke density and Irritancy $OD \cdot m^{-1}$ (extinction coefficient)	**Approximate visibility** diffuse illumination	**Reported effects**
None	Unaffected	Walking speed 1.2 m/s
0.5 (1.15) non-irritant	2 m	Walking speed 0.3 m/s
0.2 (0.5) irritant	reduced	Walking speed 0.3 m/s
0.33 (0.76) mixed	3 m approx.	30 % people turn back rather than enter
Suggested tenability limits for buildings with: – small enclosures and travel distances; – large enclosures and travel distances.		$OD \cdot m^{-1} = 0.2\ \alpha_k\ 0.5$ (visibility 5 m) $OD \cdot m^{-1} = 0.08\ \alpha_k\ 0.18$ (visibility 10 m)
(where OD/m is $\log_{10}(I_o/I)$, the logarithm of the ratio of the intensities of light transmitted over a pathlength of 1 m from a light source to a receiver in the absence and presence of smoke, respectively. The light extinction coefficient α_k is ln (I_o/I)). For situations where smoke is expressed in terms of particulate mass concentration, these equate to approximately 0.7 and 0.3 g particulates/m^3 respectively (where particulates g/m^3 ~ 0.356 x OD/m).		

Although these generic values are useful, a problem with the prediction of the effects of irritant smoke on walking speed is that the irritant composition, and hence the level of irritancy, is very dependent upon the composition of the fuel and the decomposition conditions. Above certain concentrations, it is considered that exposure to irritant gases in smoke will severely impair and even prevent escape. For the majority of flaming fires, it is considered that the concentrations of mixed smoke irritants will be below this level provided the smoke optical density does not exceed $OD \cdot m^{-1} = 0.2$. Exceptions could be smouldering fires, for which the organic irritant yields tend to be high, and fires involving fuels giving off significant yields of inorganic acid gases (HCl, HBr, HF, SO_2, NO*x*). Between zero and the concentration causing incapacitation there will be a relationship between the irritancy of the smoke and walking speed (as demonstrated by Jin). In order to provide some indication of possible effects on walking speed between these limits expressions have been developed for any irritant based upon the concentration estimated to be very painfully irritant. The model is based upon a concept that at low concentrations an increase in irritancy will have a relatively minor effect as does smoke (for example walking speed in 10 m visibility smoke should be the same as in 100 m visibility smoke). There is then a middle range, over which an increase in irritancy is likely to have large effect on walking speed, and then a point where walking is slow and further increases in irritant concentration have less incremental effect. This concept is illustrated in Figure 5. This shows a general case for the

effect of exposure to any irritant gas or mixture or irritant gases on fractional walking speed. The x-axis show the fractional irritant concentration (FIC) where FIC = 1 represents incapacitation (e.g. 1000 ppm HCl) (see equations 3 and 4).

Unlike the situation for non-irritant smoke, the curve reaches a fractional speed of zero when FIC is 1.0. This is because painful effects on vision and breathing are predicted to be of sufficient severity to cause incapacitation and cessation of effective escape movements.

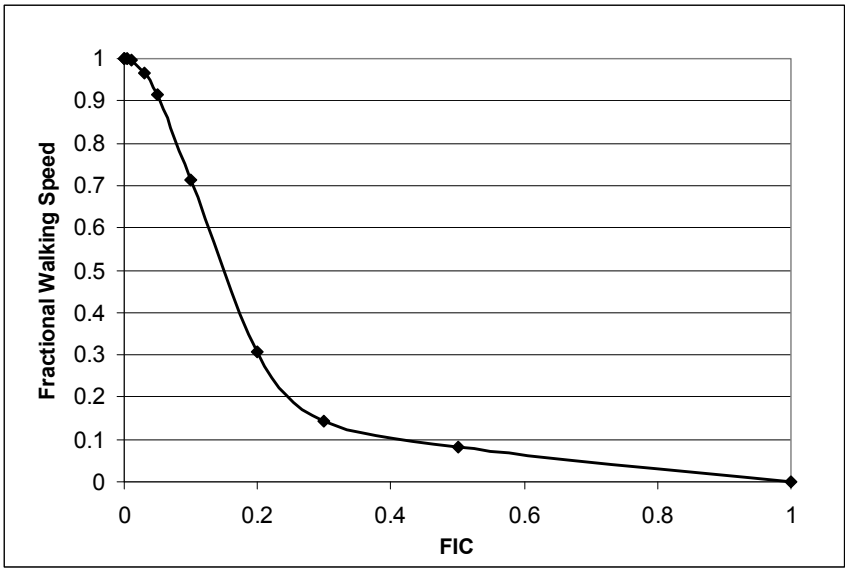


Figure 5: Estimated relationship between fractional walking speed and FIC for a sensory irritant

PREDICTING TIME TO INCAPACITATION BY IRRITANTS, ASPHYXIANT GASES AND/OR HEAT

Although the above smoke density limits may be used as design limits, they do not represent conditions under which escape is necessarily prevented. A design calculation should also evaluate predicted time to incapacitation due to the effects of heat or toxic gases. It might also be important to know the time between the visibility criteria being exceeded and that when incapacitation is predicted, especially for probabilistic design. The methods presented here are designed to predict time to incapacitation for an average person, after which a factor may be applied to allow for more susceptible members of the occupant population.

Time to loss of tenability due to smoke, irritants, asphyxiant gases and heat in fires is calculated using Fractional Effective Dose (FED) methodology as described in the following sections.

Fractional Effective Dose methodology for hazard analysis

As stated, some toxic or physical effects of exposure to combustion products occur almost immediately on exposure and the severity of the effect is proportional to the concentration of the substance and its potency. This applies to visual smoke obscuration and to the painful effects of exposure to irritants. For example, irritant smoke in the eyes or nose immediately causes pain (sensory irritation), reflex closure of the eyes and breathing difficulties.

For other substances, such as asphyxiant gases, the effect depends upon the dose inhaled. The effects therefore take some time to develop and depend upon the concentration inhaled and the time over which it is inhaled. The effects tend to more persistent that those of sensory irritation, since it take some time for the toxic material inhaled to be detoxified (for example

hydrogen cyanide) or expelled (for example carbon monoxide) An example of dose-dependent effects is collapse from asphyxia resulting from exposure to carbon monoxide.

In practice, for asphyxiant substances (and to some extent also for heat) a distinct threshold concentration or exposure dose can be identified at which serious effects are predicted. This is known as the effective concentration or exposure dose for a given toxic or physiological endpoint (for example, the exposure dose of CO required to cause loss of consciousness or the exposure dose of heat required to cause skin pain). For application to toxic hazard calculations the concept of Fractional Effective Concentration, or Dose, is used whereby the exposure concentration or dose at any point during a fire is expressed as a fraction of the exposure concentration or dose predicted to produce a given effect. For example the concentration of smoke present at any time during a fire can be expressed as a fraction of the concentration required to seriously impair escape capability.

Thus FEC_{smoke} (Fractional Effective Concentration of smoke) = 1 represents a smoke concentration considered capable of seriously limiting escape capability

where FEC_{smoke} = Concentration (of smoke) present in a fire at any time divided by the concentration considered to significantly affect escape efficiency

The exposure dose of CO can be expressed as a fraction of the exposure dose predicted to cause incapacitation .

Thus F_{Ico} (fraction of an incapacitating dose of carbon monoxide) can be expressed as:

$F_{Ico} = \frac{\text{conc. gas present} \times \text{time}}{\text{conc.} \times \text{time for incapacitation}}$	e.g.	$F_{Ico} = \frac{1000\ \text{ppm CO} \times 20\ \text{min}}{35{,}000\ \text{ppm.min}} = 0.57$

Although the FED calculations for different asphyxiant gases are based upon this simple concept, they are in practice more complex for a variety of physiological reasons, but the user is required to know only the concentration and exposure duration to perform the FED exposure dose calculation.

Another reason for expressing the concentration or dose as a fraction of an effective concentration or dose for each toxic product is that fire atmosphere contains a mixture of toxic products of differing potencies. In order to sum the effects of the different effluent components it is necessary to normalize them in terms of the effective dose. Furthermore, the concentrations of toxic products change with time during a fire, so that for constituents whose effects are dose-related, it is necessary to calculate the effective doses received, based upon the concentrations averaged over short periods of time, and then integrate these over successive periods. The aim is to calculate the time at which the summed effective doses reach unity, at which point the endpoint (such as incapacitation) is predicted to occur (see equation 5).

Effective concentrations and exposure doses for defined endpoints for smoke, heat and toxic effluent mixtures are presented in the following sections.

Effects of irritant fire effluents

Irritant chemical products in smoke constitute one of the parameters know to affect people in fires in several adverse ways[5,7]; affecting behaviour and use of escape routes, reducing travel speeds, causing painful incapacitation, and threatening post-exposure survival by effects on the lungs (bronchopneumonia, bronchiolitis, inflammation and oedema). It is therefore important to consider all these effects in a hazard analysis, taking into account that effects delaying or

preventing escape, while they may not in themselves be fatal, may lead to death from exposure to heat or asphyxiant gases. It is also necessary to consider the time-concentration curves for irritant and other gases during the course of a fire. If the material properties or combustion conditions are such that certain irritant compounds are not formed, or are rapidly cleared, or not released at a stage when occupants may be exposed, then a potential irritant hazard may not develop into a real one or may subside.

A number of attempts have been made to provide guidance on effects and acceptable limit exposure levels to humans for irritants both in the context of industrial hygiene and emergency planning, and in the context of fire safety. The industrial context differs somewhat to that for fires, in that industrial concerns tend to relate to exposures to individual irritant gases, and where the specific gas constitutes the hazard. In the fire context a mixture of irritant gases and smoke particulates is present and the hazard consists of the combination of smoke and irritant gases with asphyxiant gases and heat. A detailed description of the effects of irritants and tenability limits is presented in Purser[5] in the SFPE Handbook. Industrial and emergency limits are usually expressed in terms of concentration over a given exposure period (US ERPG - Emergency Response Planning Guidelines[27], AEGL – US EPA Acute Exposure Guidance Levels for Hazardous Substances)[28]. These take into account both immediate irritant effects and lung damage. In the fire safety context, guidance has been provided on separate phenomena for irritant fire effluent mixtures by Purser: setting ASET tenability limits for impairment of escape capability and incapacitation, estimating the relationship between irritant smoke concentration and walking speed (RSET) and estimation of lethal exposure doses for irritant smoke[5,7.] Tenability limits and calculation methods have also been set in British Standard BS 7899-2 (1999)[29], with guidance on estimation of levels of irritancy likely to inhibit escape or cause incapacitation, lethal exposure doses for smoke irritant mixtures and effects of irritant smoke on walking speeds. ISO 13571 addresses only one aspect – concentrations of mixed irritants estimated to cause incapacitation (prevent escape).

Setting Tenability Limits For Painful Sensory Irritant Effects

The basic effects and mechanisms of acute sensory irritation are similar in mammals[30,31,32,33]. In humans, irritation is characterised by an irritant or painful burning sensation in the upper respiratory tract (nose, mouth throat and chest), and also the eyes and skin of the face. There is a reflex decrease in breathing rate, with pauses during which the breath is held in, and there may be some bronchoconstriction. Irritation of the cornea can result in blepharospasm (reflex eye closure). Setting tenability limits depends upon what degree of pain and distress is considered to constitute acceptable limits. In the firesafety context Purser 2002[5] and BSI[29] recommend two possible ASET limits for consideration for any specific irritant:

- A concentration predicted to impair escape of an average person
- A concentration predicted to cause incapacitation of the average person.

ISO13571 specifies concentrations of each irritant "expected to seriously compromise the ability of most occupants to escape". Allowing for the range of susceptibility in the human population a safety factor of 0.3 x limit concentration is recommended for normal populations.

In order to estimate limiting concentrations for any specific irritant it is necessary to consider the relationship between exposure concentration and the severity of the effects. In order to achieve this, consideration has been given to published data on sensory irritant effects in humans and animal models (rodents and primates) for a variety of irritant substances. A difficulty with achieving precise predictions of effects is that it is not possible for ethical reasons to carry out experimental studies on humans. The same stricture applies to a lesser extent to animal studies, especially those using non-human primates, and with animal studies there is the added difficulty of extrapolating from animal data to predicted effects in humans. The resulting data-base consists of limited human data from experimental and accidental exposures to individual irritant gases and irritant fire effluent, some very limited experimental data from experiments in non-human primates, and more extensive data on experiments using rats and mice. Very few of

these experiments are related to the endpoint of interest – compromise of escape capability.

One helpful consideration is that the range of effects of most sensory irritants are very similar, so that detailed understanding of the effects of any one irritant can be used to predict those of another within certain limitations. The main difference between different sensory irritants is their potency, so that for a given degree of painful stimulation, different concentrations are required for different individual substances. If the differences in potency of different substances are measured, then the relationship between concentration and severity of effects can be estimated for any specific irritant.

Mouse RD_{50} model

A good physiological model for measuring both the potency of any specific irritant and the severity of irritant stimulation is the mouse Respiratory Rate depression (RD_{50}) test, which makes use of the reflex decrease in breathing rate when exposed to an irritant atmosphere, which is common to humans and rodents. This reflex is particularly well developed and stable in rodents, especially mice, such that the percentage decrease in breathing rate is proportional to the log of the inhaled concentration.At this concentration the effects on breathing rate are almost instantaneous, the rate decreasing to around 30% of the pre-exposure level. Similar effects in primates breathing thermal decomposition products from wood, polypropylene and flexible polyurethane foam are shown in Purser[15]. At concentrations of most irritants producing a high intensity sensory irritant response, the effects occur almost instantaneously and are concentration related as anyone who has experienced the following will be able to confirm (eating a hot chilli or rubbing your eyes with chilli on your hands, bonfire or barbeque smoke blowing into the face, exposure to CS riot control gas)[5,7].

For most irritant substances, human exposure at the mouse RD_{50} concentration is reported as being very painful and disruptive. Table 4 shows the wide range of irritant potencies for different substances and the similarity with the mouse RD_{50}. A particularly good example is one sensory irritant for which the effects on symptoms and behaviour have been studied experimentally in humans – CS riot control gas. For CS, the RD_{50} concentration is 0.52 ppm and human exposure to 0.5 ppm is reported as very painful .This relationship is generally applicable, so that at concentrations for most irritants around the mouse RD_{50}, severe effects on behaviour and escape capability are likely to occur. CS is know to inhibit behaviours such at those important for escaping from a building during a fire incident, and there have been a number of incidents where CS released inside occupied buildings, resulted in severe effects on occupants, leading to deaths from crowd crush injuries during escape attempts. This is therefore not to say that the symptoms and behavioural effects in mice are identical to those in humans, but that the RD_{50} model provides a good analytical tool for the assessment of irritant potency in humans.

In estimating concentrations for different irritants likely to cause severe disruption of escape capability other data have been taken into account in addition to the mouse $RD_{50.}$ Table 3 illustrates the range of data available for HCl. Based upon these data it is considered that between 100 and 1000 ppm the effects on escape capability from an occupied building are likely to be severe (remembering that severity is proportional to log concentration). However for HCl and acrolein experiments have been performed in which baboons were capable of escaping from an exposure chamber using a simple door lever after 5 minutes exposure to concentrations of HCl as high as 15,000 ppm. Taking into account differences in sensitivity between humans and other primates, and the more complex scenario of escaping form a crowded building in irritant smoke, it is suggested that around 1000 ppm HCL would be likely to seriously impair escape capability and likely to cause incapacitation due to severe pain and breathing difficulties in most people, while around 300 ppm most people would suffer significant effects on escape capability and behaviour such as entering or turning back from contaminated escape routes.

Table 4: (for details see reference 4)

Sensory and Pulmonary Irritancy of Combustion Products				
Irritant	RD_{50} (ppm) mouse	Severe sensory irritancy in humans (ppm)	30-minute LC_{50} (ppm) mammal	LC_{50}/RD_{50}
	0.1-1.0			
Toluene diisocyanate	0.20	1.0	100	500
o-chlorobenzylidene – malonitrile (CS)*	0.52	0.5	150-400	529
α-chloroacetophenone (CN)*	0.96	6-50	300-400	365
	1.0-10			
Acrolein	1.7	1-5.5	140-170	91
Formaldehyde	3.1	5-10	700-800	242
Chlorine	9.3	9-20	100	11
	10-100			
Crotonaldehyde		4-45	200-1500	
Acrylonitrile		>20	4,000-4,600	
Penteneone			1,000	
Phenol		>50	400-700	
	100-1,000			
SO_2	117	50-100	300-500	3
NH_3	303	700-1700	1,400-8,000	16
HF		120	900-3,600	
HCl	309	100	1,600-6,000	12
HBr		100	1,600-6,000	
NO_2	349	80	60-250	0.4
Styrene	980	>700	10,000-80,000	46
Acetaldehyde	**1,000-10,000** 4946	>1,500	20,000-128,000	15
	10000-100000			
Ethanol	27,314	>5,000	400,000	15
Acetone	77,516	>12,000	128000-250000	3

The potential for causing sensory irritation spans six orders of magnitude, while that for causing death spans approximately three orders of magnitude. For substances down to NO_2 death is likely to be due to lung irritation, while for the remainder from styrene to acetone death is likely to be due to asphyxiation

*Substances not detected in combustion atmospheres. RD_{50} concentrations from Alarie

LC_{50} concentrations have been normalised to a 30-minute exposure time according to Haber's rule

Table 5: Irritants effects of exposure to hydrogen chloride	
ppm	**Effect**
1.8	*AEGL-1* [34]
3	*ERPG-1 Maximum concentration human exposure up to one hour without experiencing other than mild transient health effects of a clearly defined objectionable odour*[35]
<5	Minor nasal irritation can be detected below 5 ppm (the OEL)
20	*ERPG-2 Maximum concentration human exposure up to one hour without impairing ability to take protective action or develop serious health effects*
43	
50	**AEGL-2 for 30 minute exposure**
10 - 50	*IDLH (immediately dangerous to life and health)*[36] Perceived as irritant, but work is possible at up to approximately 50 ppm.
50 - 100	Strongly irritant, and some people report exposure to 100 ppm as being excruciatingly painful to the eyes and respiratory tract[37].
100	
	ERGP-3 Maximum concentration human exposure up to one hour exposure not life threatening. Also AEGL-2 for 10 minute exposure and AEGL-3 for 1 hour
140	Incapacitation in guinea pigs after 16.5 minutes
190	No obvious signs in baboons during 5 minute exposure
200	
210	
300	**Purser SFPE Handbook – predicted to impair escape in average person**[5]
309	
500	**AEGL-3 (lethal) human 30 minutes**
	ISO-13571 maximum concentration to prevent human incapacitation (allowing for sensitivity variations in the population)[17]
620	Mouse RD_{50}[34]
700	Hyperventilation (lung irritation) in sedated baboons and post exposure chronic lung injury after 15 minute exposure
900	
	AEGL-3 (lethal) human 10 minutes
	Severe upper respiratory tract and eye cornea damage following 15 minute exposure - rats[38,]
	Purser SFPE Handbook – predicted to cause incapacitation average person[5]
800-1000	Signs of severe irritancy in baboons during 5 minute exposure including eye rubbing, profuse salivation and blinking. Able to perform shuttle box escape
1000	
1000-2000	**ISO-13571 maximum concentration to prevent human incapacitation in average person**
1095	Brief exposure regarded as being dangerous to lethal to humans
1293	30-minute LC_{50} tracheal cannulated mice
	30-minute rat exposure (nose or mouth breathing) severe necrotizing rhinitis, turbinate necrosis, psudomembrane formation, ulcerative tracheitis, necrosis, polymorhonuclear leucocyte infiltration down to alveoli
2810	60-minute rat LC_{50}
3800	30-minute rat LC_{50} (representing an exposure dose of 114,000 ppm.min)
5000	
15,000	40% decrease in arterial blood oxygen in sedated baboons
	5 minute lethal exposure concentration in rats and baboons is around 15,000 ppm but baboons able to perform shuttle box escape from a chamber after 5 minute exposure

Thresholds and variations in sensitivity in humans

Based upon all this information, what can be developed to predict effects in humans during fires? It is important in this respect to recognise that all people are not the same, and that a considerable range of sensitivities can be expected to occur human subjects. Also, although the severity of irritant effects lies on a continuum related to the exposure concentration, it might be possible to recognise to important thresholds for effects related to fires:

- A concentration capable of seriously impairing escape capability and movement speed
- A concentration capable of causing incapacitation, such that the subject effectively cannot move.

The threshold concentrations shown in Table 4 are proposed for common fire irritants likely to severely affect escape capability in most humans. In order to allow for more sensitive individuals it is recommended that design limit threshold of 0.3 x these levels might be used. Since fire effluents contain a mixture of irritants it is necessary to consider how they work in combination. It is currently recommended that they should be considered additive (see equation 6).

Post-exposure lung inflammation and survival

In addition to incapacitating effects of sensory irritation, affecting escape capability, a proportion of inhaled irritants penetrates to the deep lung, and when a sufficient exposure dose is accumulated this can lead to inflammatory processes which can be fatal over periods of several hours to several days after exposure. A guide to concentrations and exposure doses of common irritant gases likely to cause incapacitation at the scene or death following exposure is given in Table 6 (see equation 7).

Table 6: Exposure concentrations and exposure doses for incapacitation and lethal lung damage

Gas	Concentration predicted to impair escape (ppm)	concentration predicted to cause incapacitation (ppm)	Exposure doses predicted to be lethal to half the population (ppm.min)
HCl	200	1000	114,000
HBr	200	1000	114,000
HF	200	500	87,000
SO_2	24	150	12,000
NO_2	70	250	1,900
NO	-	>1000	~30,000
CH_2CHO (acrolein)*	4	30	4,500
HCHO (formaldehyde)*	6	250	22,500
* where the concentrations of acrolein and formaldehyde (or other important irritants) are unknown, a term derived from smoke density a term derived from smoke density 0.5 OD/metre may be used as an indication of irritancy likely to impair escape efficiency.and 90 OD/metre.min may be used as an indication of lethal organic irritant exposure dose			

Tenability limits and hazard calculations for asphyxiant gases

The main cause of incapacitation and death during and immediately after fires is exposure to asphyxiant gases. Incapacitation results from loss of consciousness due to the combined hypoxic effects of carbon monoxide, hydrogen cyanide and carbon dioxide, with some additional effects from low oxygen hypoxia, nitrogen oxides and inhaled irritants. Loss of consciousness prevents escape and further uptake of asphyxiants while comatose is likely to result in death within a further minute or so unless the occupant is rescued. The most useful tenability endpoint to work to is therefore considered to be loss of consciousness ($FED_{IN} = 1$), with design limits set to prevent this occurring. Since individual susceptibility varies in the population, this is

predicted to represent the median of the distribution of exposure doses resulting in collapse. Approximately 11.3% of the population is considered likely to be susceptible below an FED of 0.3. (see ISO 13571[17]). It will be necessary for the designer or regulatory authority to select an FED level suitable to protect vulnerable sub-populations in the chosen application (for example 0.3 or some other value depending upon the application).

The effects of combinations of asphyxiant gases causing incapacitation in fires are considered to be approximately additive, but a number of interactions need to be considered[5]:

- The FED for CO and HCN are considered directly additive as has been demonstrated experimentally
- NO and NO_2 (designated as NOx in mixtures) also act as asphyxiants, reducing oxygen carriage in the blood due to the conversion of haemoglobin to methaemoglobin. To this extent their asphyxiant effects can be considered additive with those of HCN and CO. However, methaemoglobin combines with HCN in the blood, thereby reducing its asphyxiant effect. NO_2 is also a potent lung irritant.
- The effects of irritants on lung function also cause some hypoxia and so an additive term is included consisting of the FLD_{irr}
- The main effect of carbon dioxide is to increase the breathing rate and thus the rate of uptake of CO and HCN. A multiplicatory term VCO_2 is used to calculate this effect.
- Low oxygen hypoxia will be additive with the overall effects, but is not increased by VCO_2 (in fact it is improved)
- The direct intoxicating effects of CO_2 are considered unlikely to occur before other effects so are normally ignored.

The overall equation for these relationships is Equation 8. For each of these gases it is necessary to obtain an expression for the fraction of a dose required to cause incapacitation. The derivation of these expressions is detailed in the section on asphyxiation by fire gases in the SFPE Handbook[5]. Basically, the exposure dose acquired over any period of time during a fire is expressed as a fraction of the dose required to cause incapacitation for each asphyxiant component. These are then summed and corrected for VCO_2 to provide an overall F_{IN} for each time period.

A complication is the extent to which the dose response effects of each gas follow Haber's rule and where it is necessary to allow for deviations from this oversimplification used by all small-scale toxicity test methods. Haber's rule is that any given physiological or toxic endpoint (for example loss of consciousness due to asphyxiation), occurs at a constant c x t exposure dose, irrespective of whether the subject has been exposed to a high concentration for a short period or an equivalent lower concentration exposure for a longer time. This effect is illustrated in Figure 6, which shows time to incapacitation for primates exposed to constant concentrations of CO or HCN for different exposure periods of up to 30 minutes. For exposures to CO, the animals were largely unaffected until they achieved and exposure dose of approximately 27,000 ppm.minutes, at which point they showed signs of confusion and lethargy followed by loss of consciousness within less than a minute. The exposure dose for incapacitation was constant for exposure concentrations of 1000-8000 ppm, providing the smooth curve shown. This contrasts strongly with the effects of exposure to HCN, whereby exposure to concentrations of around 200 ppm and above resulted in loss of consciousness within two minutes (c x t = 380 ppm.minutes), while a much large exposure dose over a long period was required at lower concentrations (c xt = 2610 ppm.minutes at 87 ppm). Cyanide is also much more potent that CO by a factor of around 20 or so.

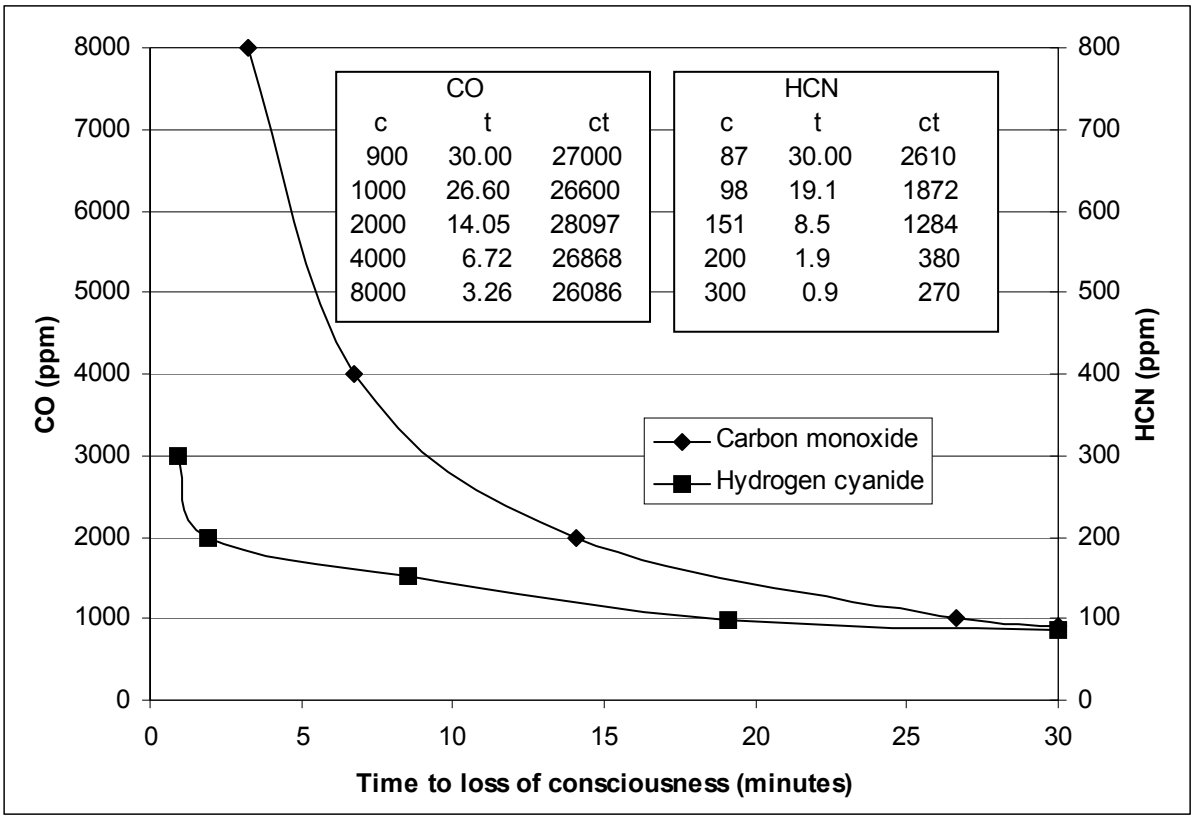


Figure 6: Time to incapacitation for cynomolgus monkeys exposed to different concentrations of CO or HCN. CO exposures were for active animals exercising in a chamber, HCN exposures in resting animals

This behaviour of HCN is important because it means that a short exposure to a high concentration (above around 150 ppm) will cause rapid collapse of a fire victim, who then remains incapacitated, inhaling more HCN and CO which may then lead to death. This variation of exposure dose required for a given endpoint is yet another reason why use of small-scale toxicity test methods or toxicity indices based upon a constant exposure period is likely to lead to error, while the time-based FED methodology allows for these effects in calculating time to incapacitation. Similar dose-response effects deviating from Haber's rule occur for low oxygen hypoxia and heat[5]

Another important aspect of time to incapacitation for fire victims, especially with regard to CO intoxication, is the level of physical activity. A person at rest breathes only around 8.5 litres of air each minute, while a person engaged in light activity such as walking breathes around 25 l/min, so the rate of CO uptake is much more rapid in active subjects. Also, due to the energy and oxygen demands of physical activity, an active subject is likely to collapse at a lower blood %COHb level than a sedentary subject.

However, the effects are based on data for healthy young adult animals or humans. The exposure dose or concentration causing incapacitation therefore represents the maximum in a statistical distribution of subjects' responses surrounding that exposure dose or concentration, that is, the mode, or most frequently expected exposure dose for an exposed population. Individual exposure doses or concentrations for the response would, in practice, be statistically distributed around the mode in a probability curve. The overall human population contains a number of subpopulations, which exhibit greater sensitivity to various fire effluent toxicants, principally due to compromised cardiovascular and pulmonary systems.

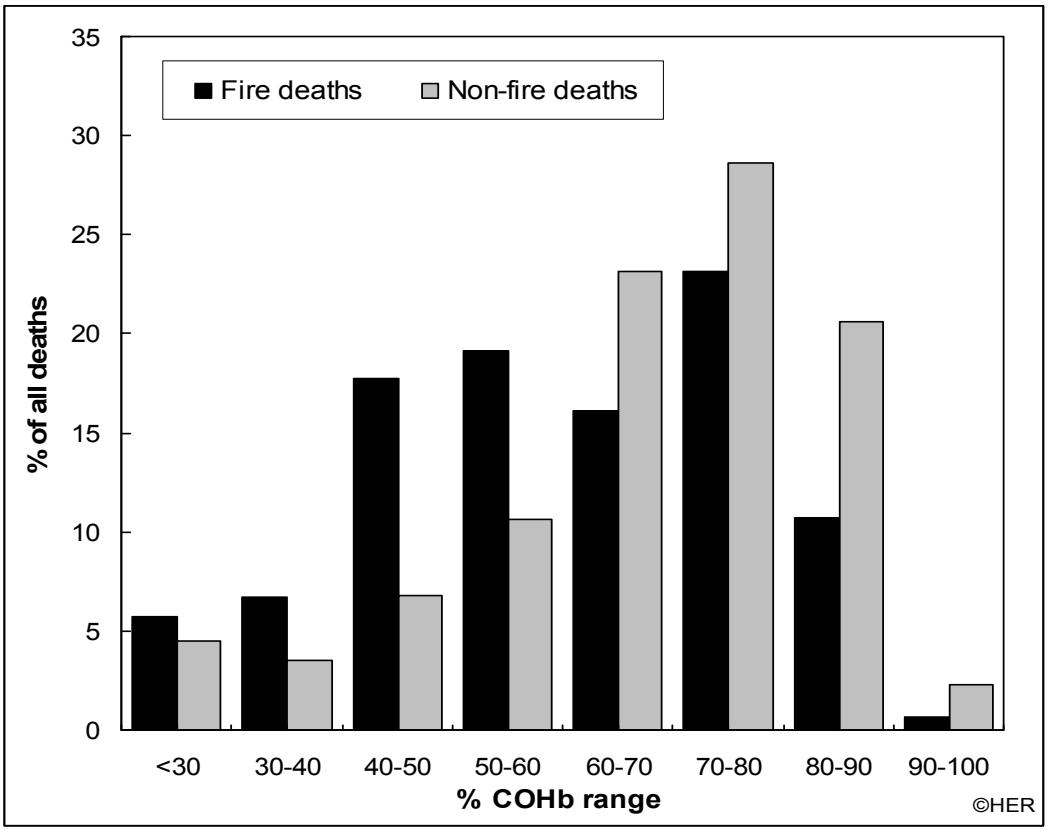


Figure 7: Distribution of fatal %COHb in non-burned fire victims and non-fire CO poisoning cases (After Nelson)

This variability of susceptibility in the population is illustrated in Figure 7, which shows the distribution of %COHb in non-burned fire victims and non-fire CO poisoning cases in the United States (data compiled by Gordon Nelson). The Figure illustrates the range of sensitivity in the exposed human population. The mode is between 70-80% COHb. Note that the fatal levels in fire victims are somewhat lower than for CO poisoning cases. This may reflect the effects of other toxic fire gases such as hydrogen cyanide.

Two of the largest such subpopulations are the elderly and the approximately 15 percent of children and 5 percent of adults who are asthmatic. The elderly, and particularly those with impaired cardiac perfusion, are especially susceptible to asphyxiant gases. Thus the average lethal carboxyhemoglobin (COHb) concentration in adults dying in fires or from accidental CO exposure is lower in the elderly. The results in Figure 7 show that some individuals died at COHb concentrations below 30 percent while others survived long enough to obtain blood concentrations above 90 percent COHb. Many fire fatalities occur at lower COHb concentrations than for cases of CO poisoning alone. This may partly reflect the influence of other toxic gases in addition to CO in fire atmospheres. Also, it has been shown in experimental studies that as little as 2 percent COHb significantly reduces the time to the onset of pain in an exercise test of angina sufferers. This could be very important when attempting to escape from a fire. A further complication with the CO lethality data is that in practice few people survive an exposure of more than 50 percent COHb if rescued and treated, even though much higher levels are found in the bodies of decedents. This is because uptake continues in comatose people until the point where respiration ceases. This is illustrated in data from a study by Pach[39] (Table 5), which shows the proportions of survivors and fatalities from a sample of 260 CO poisoning cases. The data show that survival is rare above 50%COHb, around 0.67 in the 40-50%COHb range, and that most people survive below 40%COHb. Equations for calculating the FEDs for CO and each of the other gases are numbers 9-12.

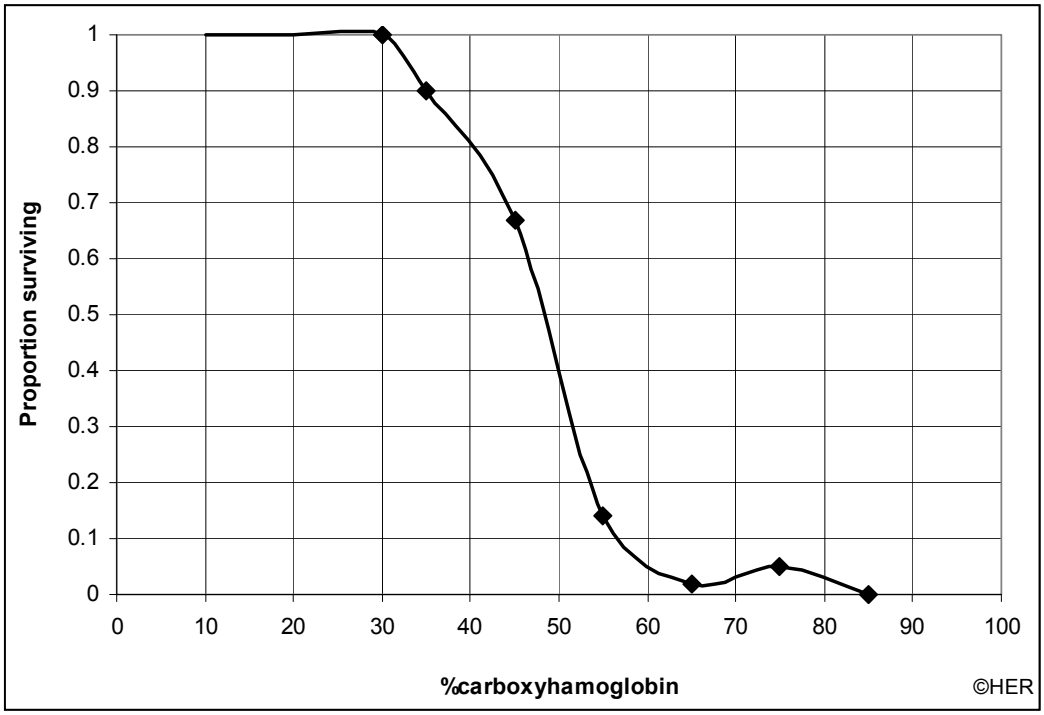


Figure 8: Proportions of survivors and fatalities in different COHb ranges from a sample of 260 CO poisoning cases (after Pach 1978)[39]

Tenability and hazard calculations for development of pain, incapacitation, injury and death from exposure to heat and burns

There are three basic mechanisms whereby heat exposure may lead to incapacitation and death in fires:

- Heat stroke (hyperthermia)
- Skin pain followed by body surface burns
- Respiratory tract burns

Hyperthermia involves prolonged exposure (approximately 15 minutes or more) to heated environments at ambient temperatures too low to cause burns. Under such conditions, where the air temperature is less than approximately 121°C for dry air or 80°C for saturated air, the main effect is a gradual increase in body core temperature. Hyperthermia leads to confusion and collapse. Exposure to air temperatures above approximately 121°C (or to radiant heat fluxes above 2.5 kW/m^2) leads to pain to exposed skin followed by body surface burns and hyperthermia if exposure is prolonged. Respiratory tract burns can also occur if exposure to heated air is sufficient to cause facial burns. For a basic engineering design it is proposed that a tenability endpoint for exposure to heat should be taken as the limits of heat tolerance due either to the onset of hyperthermia or to severe pain to exposed areas of skin (such as the head and hands). It is proposed that under such conditions exposed building occupants may be unable to escape (if affected by hyperthermia) or unwilling to move through painfully hot environments. It is known that in some fires people have moved through (and escaped from) scenarios under conditions hot enough to cause severe burns. It is suggested that severe incapacitation is likely to occur under conditions likely to cause second degree burns, and death under conditions likely to cause third degree burns.

There are also three basic heat exposure scenarios for building occupants during a fire:

1. Exposure to convected heat in a hot air environment.
2. Exposure to radiant heat direct from a fire or from a hot upper smoke layer
3. Exposure of a subject immersed in hot smoke – subjected to both radiant heat from hot smoke particles and convected heat from contact with hot gases in the hot smoke environment

Experimental human exposure data are available for the first two cases, from which time-tolerance curves have been obtained for exposure to convected or radiant heat. For direct exposure to radiant heat (for example from a fire or heater), empirical relationships between exposure time and effect have been measured by a number of authors. For these experiments heat radiation is expressed in terms of received heat flux (kW/m^2). See equation 13.

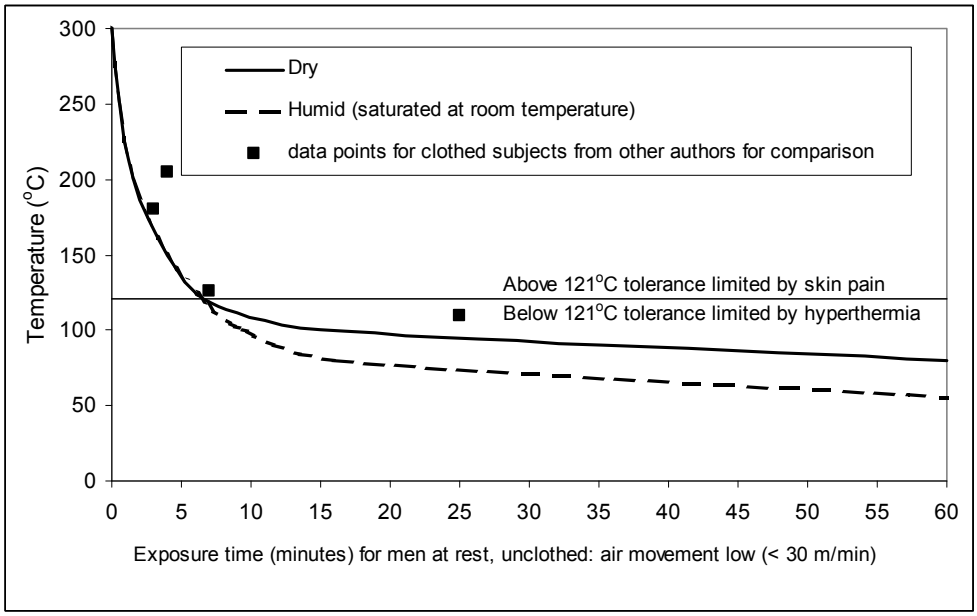
The tenability limit for exposure of skin to radiant heat is approximately 2.5 kW/m^2, below which exposure can be tolerated for at least several minutes. Radiant heat at this level and above causes skin pain followed by burns within a few seconds, but lower fluxes can be tolerated for more than 5 minutes. Above this threshold, time (minutes) to incapacitation due to radiant heat t_{lrad}, at a radiant flux of q kW/m^2 is given by Equation 13[5]. The effects of heat on an occupant response may depend upon the situation. The threshold for pain occurs at a value between approximately 1.33 and 1.67 $(kW.m^{-2})^{4/3}$.min. Second degree burns occur at 4.0-12.2 $(kW.m^{-2})^{4/3}$.min and third degree (full thickness) burns at appro16.7 $(kW.m^{-2})^{4/3}$.min.
A figure of 1.33 $(kW.m^{-2})^{4/3}$ is used to represent a tolerance threshold and 10 $(kW.m^{-2})^{1.33}$ a threshold for incapacitation and serious injury. For infrared radiation it is also proposed that 10 $(kW.m^{-2})^{1.33}$ min represents a fatal level for a vulnerable population (over 65 years of age) or a 1% fatality level for the average population, while 16.7 $(kW.m^{-2})^{1.33}$ .min represents a 50% probability lethal level for the average population.

Calculating effects of exposure to convected heat only

Some experimental data are available for exposure of unclothed or lightly clothed subject to hot air environments in terms of tolerance time to pain or hyperthermia. The Blockley[40] curve shown in Figure 9 is for dry air and humid air (saturated at normal room temperature), which is then heated. This is very different from the effects of air saturated with water at higher temperatures. Water is a serious potential problem in fires due to its high latent heat. If fire effluent contains water at above 100°C (i.e. steam) it releases considerable heat if it comes in contact with the skin or is inhaled. Although the hazards of contact with steam are well known, it may be less obvious that air saturated with water vapour at lower temperatures can be dangerous. The highest temperature at which saturated air can be breathed for more than a few minutes is 60°C. As an approximate guide, the volume concentration of water vapour in fire effluent is similar to the CO_2 concentration, which might reach as much as 10%. This is less than the concentration in saturated air at the breathable limiting temperature of 60°C. For this reason it is considered that the fuel-derived water vapour content of normal fire effluent is not likely to present a serious hazard.

An expression has been derived for exposures of up to two hours to convected heat from air containing less than 10% by volume of water vapour (see equation 14) .

As with toxic gases, the body of a fire victim may be regarded as acquiring a “dose” of heat over a period of time during exposure, with short exposure to a high radiant flux or temperature being more incapacitating than a longer exposure to a lower temperature or flux. The same fractional incapacitating dose model as with the toxic gases may be applied and, providing that the temperature in the fire is stable or increasing, the fractional dose of heat acquired during exposure can be calculated (see equations 14-16)..



Figue 9 Thermal tolerance to convected heat exposure for humans at rest, naked skin exposed. Adapted from Blockley[40].

OVERALL HAZARD ANALYSIS FOR A FIRE

Figure 10 shows an example of an overall hazard analysis for the furniture house fire illustrated in Figure 1. The fractional effective concentration or dose of each physiological effect is summed with time during the fire, with the endpoint for each effect reached at an FIC or FED of 1. For the example shown the first hazard to exceed the tenability limit is impairment of escape capability due to the effects of irritant organics and acid gases in the smoke products at 1.5 minutes. This is followed one minute later by the limit for optical obscuration by smoke particulates. The combined effect of these two hazards in to seriously impair or prevent escape. After five minutes it is predicted that an occupant would lose consciousness, primarily due to the asphyxiant effects of hydrogen cyanide, with death occurring within approximately a further 30 seconds. For this fire escape impairment due to the effects of heat exposure is predicted after six minutes, although several minutes further exposure would be required for burns to occur. Also shown for comparison is the predicted time to incapacitation if there had been no hydrogen cyanide present (after 7 minutes), this would be due to mainly to the asphyxiant effects of carbon monoxide and carbon dioxide. The exposure dose of irritants capable of causing potentially fatal deep lung inflammation some hours after exposure is only around 0.2 after 10 minutes, so this effect is predicted not to occur, although elderly subjects have been found to be particularly sensitive to potentially fatal bronchopneumonia following exposures of this kind in fires.

Since hazards from toxic effluent are the main causes of incapacitations and death in fires it may seem sensible to regulate for toxic hazard or toxicity, with respect to both the overall design and performance of the built environment (such as buildings and vehicles) and the individual products used in their structure and contents. This may be achieved by means of prescriptive requirements supported by pass-fail performance or ranking of specific products in standard small or large-scale tests, or through performance-based (fire engineering-based) design using engineering calculations with appropriate test data inputs to assess the overall performance or built systems or products used in them.

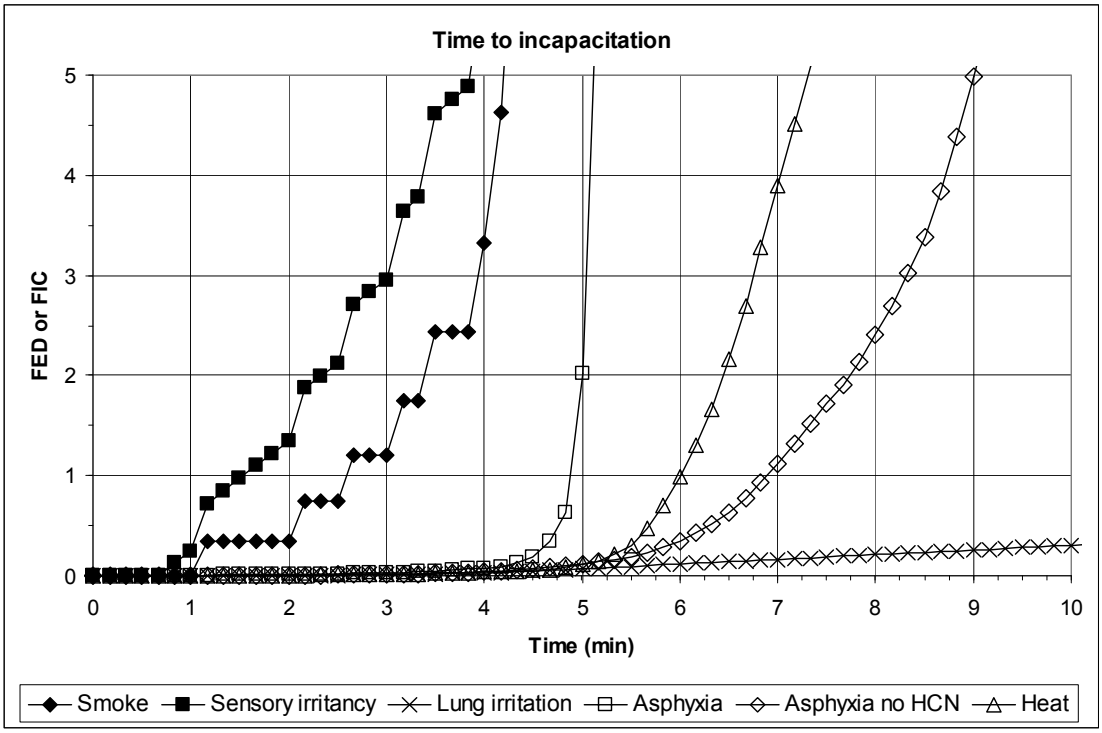


Figure10: Fractional effective dose analysis in term of times to incapacitation for an occupant of a domestic house lounge during an armchair fire.

METHODS FOR REGULATING TOXICITY AND TOXIC HAZARD

Historically, prescriptive guidance for fire safety has been based upon design requirements for aspects such as detection, warnings and provision for means of escape, combined with test-based fire resistance and reaction-to-fire performance of products and materials. Toxic hazard has been controlled indirectly, mainly by controlling reaction-to-fire properties such as ignitability, flame-spread and heat release rate using a battery of small-scale and medium-scale test methods. This approach works reasonably well because these tests either address fundamental properties, or fire properties for which small-scale behaviour is predictive of end-use performance. For example, materials incapable of supporting combustion due to a very low organic content, or products failing to support spreading flaming combustion under high radiant heat fluxes in small-scale tests, are unable or unlikely to support formation of a large, growing, hazardous fire in the end-use application. Because of this, they are unlikely to make a significant contribution to a rapidly developing fire hazard from either toxic smoke or heat.

The same cannot be said of small-scale tests for toxicity or toxic hazard, since both are in practice more complex outcomes of the performance of fire scenarios in the full-scale end-use system, and specifically:

- Small-scale toxicity tests cannot possibly replicate the complex, dynamic, full-scale fire hazard scenario
- Toxicity tests use very primitive fire models that generally have not been shown to replicate the decomposition or combustion conditions in different types and stages of full-scale fires, so they do not represent the range of yields and toxic product mixes occurring in such full-scale fires.
- Toxicity tests employ very simplistic toxic potency or toxicity index models, which in no way reflect the physiological effects and interactions of fire effluent mixtures
- The simplistic direct application of such index-based toxicity criteria is likely to result in sanctioning the use of products or materials that may be hazardous in end-use applications, while proscribing the use of other products or materials that might is some situations have a

high toxic potency, but in end-use have been shown to provide a good level of overall safety with respect to fire performance and toxic hazard.

In contrast to this, performance-based approaches to toxic-hazard assessment, as used in fire engineering-based design, have the potential for a realistic evaluation of end-use hazards both of entire systems (buildings and transport) and specific products in end-use applications.

Performance-based design for buildings, trains and airplanes, whereby available safe escape time is compared with required safe escape time for relevant fire scenarios, is increasing used for fire-safety design compliance. For ASET evaluations these make use of fire dynamics data and models, or full-scale tests, to obtain the time-concentration and intensity curves for design fires, in combination with physiological FED models (such at those described here and detailed in ISO 13571[17] or in the SFPE Handbook of Fire Safety Engineering)[5] ,in order to calculate times to loss of tenability

Although such approaches are not generally applied to the application or regulation of products used in building (or transport) structure or contents, it is proposed that standard methods could be developed for applying physiological FED models for this purpose. The main advantages to this approach would be that all products could be evaluated on a level-playing field with respect to their ultimate end-use performance, taking into account specified combustion conditions, and that the standards could be tailored for different potential applications.

An example might be a regulatory performance requirement for the use of a wall covering or an item of upholstered furniture in a domestic dwelling. The regulatory performance requirement could be expressed in terms of time available for escape for a specified fire scenario (or set of fire scenarios) in an enclosed room: for example a period of 5 or 10 minutes following application a defined ignition source.

A protocol might developed be along the lines of the following:

1. Reaction-to-fire tests performed using a specified small or medium – scale test such as the Cone Calorimeter[41], furniture calorimeter or Single Burning Item (SBI) apparatus[42]. These are used to obtain the heat release rate/mass loss rate curves for the fire.
2. Small-scale combustion tests are performed to measure the yields of a standard set of toxic fire effluents (kg/kg product mass loss) under a range of thermal decomposition and combustion conditions representing those occurring at in different stages and types of full-scale fires. Suitable test methods might include the ISO 19700 tube furnace method[18] or the ASTM E2508 flammability apparatus[21.]
3. These test data are then input into a specified fire calculation model (such as the NIST CFAST[43] zone model) using a specified standard domestic room (for example a 50 m^3 room with specified linear dimensions).
4. From this, a set of time-concentration and time-temperature curves are calculated for the smoke, toxic gases and heat in the defined fire scenario.
5. The standard ISO 13571 physiological FED model is applied to calculate the predicted time to loss of tenability.
6. Depending upon the tenability time obtained, the product could be specified for use in different applications. For example a longer time might be require for a care home than for student accommodation or general domestic use.

The advantages of this type of approach is that once the basic test data are input, it is simple to run a range or variety of cases, either for different applications of for different scenarios in any one application. For the domestic case it would be possible to specify a case involving an open fire room door with effluent spread into the greater volume of a specified standard apartment or house, with assessment of tenability for occupants in locations remote from the fire room. Or tenability in the room of origin could be examined for several different ventilation scenarios (such as enclosed room, open door, open window). In this way the applicability of a product could be evaluated quickly for a range of common dwelling fire scenarios. The same input data might also be used to test applicability to a hotel room case, with different spaces and

dimensions specified. Reference scenarios for different cases could involve full-scale fire tests or higher-level computational methods such as computational fluid dynamics models.

For the basic approach envisaged, the tests required to obtain data would consist of some methods commonly in current use and the calculation/modelling could be standardised so as to be performed within an hour or so.

As with any standard test method some degree of generalisation is necessary to represent the diversity of actual built environments by a small set of standard enclosures (or enclosure sets) and the diversity of possible fire scenarios by a small set of standard scenarios, but it is considered that such an approach could provide a far more realistic and balanced evaluation of actual end-use performance than currently used methods based upon unproven and overly simplistic small-scale toxicity tests and indices, which bear no real relationship to either the end-use fire conditions or their effects on occupants. It would also be possible as part of a performance-based assessment for a product producer to tailor the modelled scenarios to specific end-use applications where their product might show particular performance advantages.

PARTICULAR ISSUES IN RELATION TO CABLE FIRES

The time concentration curves for toxic gases and smoke, and the time intensity curves for radiant and convected heat in any fire depends upon the full-scale fire scenario, in terms of the disposition and rate of combustion of the fuels and their interaction with the fire enclosure, and it ventilation. When evaluating any product from a fire hazard perspective it is important that the testing and hazard assessment strategy is relevant to the end use fire hazard scenarios of interest.

In some installations, such as in plant rooms, cable runs may be open in the enclosure, in which case the basic fire scenarios are somewhat similar to those for other combustible fuels such as structural products and contents. The rate of combustion depends upon the reaction to fire properties of the cables (as installed) and the enclosure ventilation. In large enclosures, particularly if vents such as windows, doors or smoke extractors are open, then combustion tends to be relatively efficient, leading to rapid fire growth combined with relatively low toxic product yields for non-fire retarded fuels, and generally slower fire growth rates from fire-retarded fuels, but accompanied by higher yields of toxic products. In smaller enclosures, or where vents are mostly closed, then fires soon become vitiated, with the fire size controlled by the available ventilation. If vents are opened, then flashover may occur, but the fire is still vitiated and ventilation controlled. Under these conditions the yields of smoke and toxic gases are greatly increased and even small fires can rapidly fill open spaces within a building with lethal concentrations of smoke and toxic gases.

For cables in enclosed vertical or horizontal ducts, or in ceiling or floor plenum spaces, the potential fire scenarios are somewhat different form those in room-sized enclosure fires. Vertical cable runs are often in small enclosed spaces or ducts, often running up many floors in a building. The rate of spread for fire and the rates of production, and yields, of toxic products are likely to be very dependent upon the ventilation within the duct, the height of the run, and the efficiency of fire stopping between compartments. From a hazard perspective the extent of sealing of the cable runs within the ducts and the efficiency of fire stopping is of crucial importance, since in a well-sealed environment the potential for fire growth is limited and fire effluents are largely contained within the space of origin. Where containment is less efficient, a developing fire is likely to be extremely vitiated, with very high yields of smoke and toxic products and large volumes of toxic smoke spreading between floors and into occupied spaces at different locations. In situations where vertical runs have air flowing through them, the potential for more rapid fire growth and spread exists, and the extent of vitiation and toxic product formation depends upon the fuel/air ratio in the combustion zone. The preEN50399 test apparatus would appear to be a reasonable

approximation of an end-use application for vertical cable run fire scenarios[44], although for enclosed vertical runs the enclosure depth would often be much less than 2 metre depth in the test apparatus, perhaps providing a greater "chimney" effect. Also it might be relevant to consider a range of different ventilation conditions to evaluate different end-use applications.

Other important full-scale fire scenarios involve cable runs in horizontal unventilated or ventilated plenum spaces. As with vertical runs, the potential for fire growth and spread depends partly on the reaction-to-fire properties of the cable runs, especially the propensity for fire spread along horizontal cables, and partly on the size and extent of enclosure of the plenum space and its ventilation. As with vertical runs, an important aspect of potential hazards is the extent of containment within separate enclosures and fire compartments, the separation between the floor or ceiling spaces and the adjacent occupied rooms (including the probability of tiles being removed), and the efficiency of fire stopping or shutter activation. A consideration with small, vitiated fires is the extent of toxic effluent production before operation of heat-activated shutters. If significant fire spread and propagation occurs in plenum cable fires then the resultant fires are likely to be relatively small and slow growing if ventilation is very restricted, but producing highly toxic fire effluent, while more ventilated spaces may lead to larger fires, but in the restricted spaces within plenums, especially those with high fuel loads, then vitiated combustion with high toxic product yields is also likely to occur. The general position is that since most cable fires tend to occur in confined spaces with limited ventilation, they are likely to involve vitiated combustion with high toxic product yields, while the rate of fire development and ultimate fire size will depend upon the reaction-to-fire properties of the cables as installed and the available ventilation.

Physiological Calculation Equations

An equation for the relationship between walking speed and smoke optical density ($OD \cdot m^{-1}$) is given by:

Walking speed in non-irritant smoke (m/s) = 1.36 – 1.9 × smoke optical density ($OD \cdot m^{-1}$) [1]

Walking speed in irritant smoke (m/s) = 2.27 – 9 × smoke optical density ($OD \cdot m^{-1}$) [2]

An equation for the curve in Figure 5, for walking speed during exposure any individual irritant compound, or to the fractional irritant concentrations for a mixture of compounds, is given by:

$$F_{wvirr} = 1 - ((1 - e^{-(x/b)^2}) + (-0.2x + 0.2)/1.2) \quad [3]$$

Where
F_{wvirr} = fractional walking speed (1 = normal walking speed 1.2 m/s)
b = 160
x = FIC
The overall effect of exposure to an irritant smoke on walking speed (F_{wv}) would then be given by:

$$F_{wv} = 1 - (1-F_{wvsmoke}) - (1-F_{wvirr}) \quad [4]$$

Where:
F_{wv} = Overall fractional walking speed
$F_{wvsmoke}$ = Fractional walking speed due to smoke effects on visibility
F_{wvirr} = Fractional walking speed due to irritant effects for irritants compounds 1 to n

The general FED equation for summing exposure doses for each toxic product during each time period and then integrating with time is:

$$FED = \int_{t1}^{t2} \sum_{i=1}^{n} \frac{C_i}{(Ct)_i} \Delta t \qquad [5]$$

where:

C_i is the average concentration, of an dose related toxicant such as an asphyxiant gas "*i*" over the chosen time increment;

Δt is the chosen time increment, expressed in minutes (min)

$(Ct)_i$ is the specific exposure dose expressed as concentration × minutes, that would constitute an effective dose (i.e. an exposure dose producing the a defined endpoint such as preventing an occupant's safe escape)

The overall FIC for an irritant mixture is as follows:

$$FIC = FIC_{HCl} + FIC_{HBr} + FIC_{HF} + FIC_{SO2} + FIC_{NO2} + FIC_{CH2CHO} + FIC_{CH2O} + \Sigma FIC_x \qquad [6]$$

Where ΣFIC_x = FICs for any other irritants present.

The FED equation for the lethal effects of inhaled irritants (i.e FLD or Fractional Lethal Dose) is:

$$FLD_{irr} = FLD_{HCl} + FLD_{HBr} + FLD_{HF} + FLD_{SO2} + FLD_{NO2} + FLD_{CH2CHO} + FLD_{HCHO} + \Sigma FLD_x \qquad [7]$$

The overall FED equation for asphyxiant gases is :

$$F_{IN} = (F_{ICO} + F_{ICN} + F_{INOx} + FLD_{irr}) \times V_{CO2} + FED_{IO} \qquad [8]$$

Where:

F_{IN} = Fractional effective dose for incapacitation (loss of consciousness) due to asphyxiants

F_{Ico} = Fractional effective dose for incapacitation by CO

F_{Icn} = Fractional effective dose for incapacitation by HCN

Note: If necessary this can be corrected for the presence of other nitriles besides HCN and for the protective effect on cyanide poisoning of NO and NO_2. [CN] can then be calculated as: [CN]=[HCN]+[Total organics nitriles]- 0.66 x [NO + NO_2]

FI_{NOx} = Fraction of an incapacitating dose of NO + NO_2 (= [NOx ppm x t_{min}]/15000)

FLD_{irr} = Fractional lethal dose for irritants

Note: where significant concentrations of acid gases are present this term is calculated according to equation 22. Otherwise it may be expressed in terms of smoke optical density as OD/m x t(min)/90

VCO_2 = Multiplicatory effect of inhaled CO_2

F_{Io} =Fractional effective dose for incapacitation by low oxygen hypoxia

For a simple analysis the direct asphyxiant effects of NOx and those of NOx on HCN asphyxia may be ignored without significant error.

The expressions used to calculate the FEDs for each individual component are as follows:

For the effects of CO the FED is expressed in terms of %COHb. The denominator is the %COHb predicted to cause loss of consciousness in an active (escaping) person = 30% COHb

(or 40% COHb for a resting person). The numerator is the Stewart equation[45], by which the %COHb in the subject is calculated from the inhaled CO concentration in the fire, the exposure time and the volume of air breathed each minute:

$$F_{Ico} = 3.317x10^{-5}[CO]^{1.036})(V)(t)/D \quad [9]$$

Where:
[CO] = carbon monoxide concentration (ppm v/v 20°C)
V = volume of air breathed per minute (liters/min)
t = exposure time in minutes
D = exposure dose (%COHb) for incapacitation

The following values are taken for V and D:

Activity level of subject	V (l/min)	D %COHb
Resting or sleeping	8.5	40
Light work – walking to escape	25	30
Heavy work – slow running, walking up stairs	50	20

Note 1: This expression (the Stewart equation) was obtained from young adult male human volunteers. It is suitable for adults in situations where the CO concentration is high in relation to the blood COHb concentration (as for most flaming fires and short exposure durations). Where long exposures may lead to near equilibrium conditions, the Coburn Forster Kane[46,47] equation should be used since significant deviations from the Stewart equation may then occur. The Stewart equation somewhat underestimates uptake rates for children. Where more precise CO uptake calculations are required, differences in body size (including children) can be accommodated using the CFK equation. For a basic design, use of the Stewart equation is recommended. Differences in body size and other susceptibilities can be considered as allowed for in the overall safety margin chosen to accommodate more susceptible sub-populations (for example an FED 0.3 or other endpoint).

Note 2: As an alternative to using this expression the FED_{Ico} may be expressed as a CO exposure dose ratio. This method is used for ISO 13571[17]. For this method the FED_{ico} is expressed as CO ppm x t/35,000. This is approximately equivalent to a "light work" case for a subject breathing approximately 20 l air/minute.

Since occupants must at least walk in order to escape from a fire, the default case suggested is that for light work. However this could be varied according to the case. For example a sleeping person escaping from a basement might start by being at rest, then awaken and walk to a stair (light work) then climb the stair (heavy work).

For the effects of hydrogen cyanide and low oxygen hypoxia, the expressions are more complex, because the denominators are not constants. Exponential expressions have been developed to fit the time to incapacitation versus exposure concentration curve from experimental exposures in non-human primates (HCN) and humans (hypoxia), so that the Fractional Incapacitating Doses for HCN (F_{ICN}) and hypoxia (F_{IO}) as follows:

$$F_{I_{CN}} = \left[\left(\frac{\exp([CN]/43)}{220}\right) - 0.0045\right]t \quad [10]$$

Where CN = HCN concentration (ppm v/v at 20°C)
t = exposure time in minutes

$$F_{Io} = t/\left(\exp\left[8.13 - 0.54\left(20.9 - \left[\%O_2\right]\right)\right]\right) \quad [11]$$

Where $[\%O_2]$ = oxygen concentration (% v/v at 20°C)
t = exposure time in minutes
I = exposure dose for incapacitation

Similarly, a curve has been fitted to the effect of CO_2 on ventilation (breathing volume per minute) based upon human experimental data.

Ventilatory stimulation by CO_2: $VCO_2 = \exp([CO_2]/5)$ [12]

Where $[CO_2]$ = carbon dioxide concentration (% v/v at 20°C)

FEDs are calculated for successive short periods during the fire and then integrated with time in order to calculate the time when incapacitation is predicted (FED = 1). Due to the rapid (t^2) rate of increase of asphyxiant gas concentrations in most flaming fires, variations in individual susceptibility and uncertainties in prediction of incapacitating doses tend to have relatively minor effects on predicted times to incapacitation.

The tenability limit for exposure of skin to radiant heat is approximately 2.5 kW/m^2, below which exposure can be tolerated for at least several minutes. Radiant heat at this level and above causes skin pain followed by burns within a few seconds, but lower fluxes can be tolerated for more than 5 minutes. Above this threshold, time (minutes) to incapacitation due to radiant heat t_{Irad}, at a radiant flux of q kW/m^2 is given by Equation 13[5].

$$t_{Irad} = \frac{1.33}{q^{1.33}} \quad [13]$$

Where:
t_{Irad} = time to endpoint (pain in this case) in minutes
q = heat flux kW/m^2.

The effects of heat on an occupant response may depend upon the situation. The threshold for pain occurs at a value between approximately 1.33 and 1.67 $(kW.m^{-2})^{4/3}$.min. Second degree burns occur at 4.0-12.2 $(kW.m^{-2})^{4/3}$.min and third degree (full thickness) burns at appro16.7 $(kW.m^{-2})^{4/3}$.min.

A figure of 1.33 $(kW.m^{-2})^{4/3}$ is used to represent a tolerance threshold and 10 $(kW.m^{-2})^{1.33}$ a threshold for incapacitation and serious injury. For infrared radiation it is also proposed that 10 $(kW.m^{-2})^{1.33}$ min represents a fatal level for a vulnerable population (over 65 years of age) or a 1% fatality level for the average population, while 16.7 $(kW.m^{-2})^{1.33}$ .min represents a 50% probability lethal level for the average population.

The following expression has been developed for the mid-humidity case from Blockley[40] and other data. Tolerance time t_{tol} (minutes) is then given by:

$$t_{tol} = 2 \times 10^{31} \times T^{-16.963} + 4 \times 10^{8} \times T^{-3.7561} \quad [14]$$

Where T = room temperature, degrees centigrade

For Equations 13 and 14, the "Fractional Effective Dose" of heat received each minute is given by the reciprocal of the expression.

For situations when a subject is exposed to both radiant heat (for example from a heated upper layer) and covected heat from exposure to heater air, the overall heat dose received may be estimated by summing the radiant and convected fractions using Equation 34:

$$FED = \int_{t1}^{t2} \left(\frac{1}{t_{Irad}} + \frac{1}{t_{Iconv}}\right) \Delta t \qquad [15]$$

The doses acquired each unit of time are then integrated. The tenability limit is predicted when the FED for heat = 1.

A possible method for estimating tolerance time for the case of a subject immersed in hot smoke could be to calculate the total heat flux from the radiant and convected components of the smoke. On this basis it is proposed that the total incident flux to the skin of a person immersed in hot smoke is given by:

$$q = \varepsilon\sigma (T_i^4 - T_m^4) + h_c (T_i - T_m)/1000 \qquad [16]$$

Where:
q = heat flux kW/m^2
T_i = heat source temperature °K
T_m= material surface temperature °K
ε = emissivity (0.05 for a gas to 1 for a black body, perhaps 0.5 for smoke)
σ = Stefan Bolzmann constant (5.67 x 10^{-8} Wm^{-2}K^{-4})
h_c = convective heat transfer factor. For air this depends upon the flow rate past the object. It will be approximately 5-8 for slow moving air.

The first term in the equation represents the radiant component of heat flux and the second term the convected component of heat flux. Using this equation it is therefore possible to calculate total heat flux from the room temperature at body height. The radiant component is relatively small at low temperatures, and is negligible for hot air due to its low emissivity. However, for smoke the emissivity is likely to be must higher (around 0.5) so that at higher temperatures both components should be considered in order to calculate the total heat flux to the skin. In addition to the heat flux from the fire effluent enveloping a subject there is likely to be additional heat radiation from hot upper layers and/or directly from the fire. If the subject is in air (with a low emissivity), below a hot smoke layer, the only significant radiative heat flux sources are likely to be the upper layer, the fire or hot surfaces.

Time to the different heat effect endpoints for total heat fluxes in excess of 2.5 kW/m^2 is then given by equation 16 using the appropriate exposure dose endpoints as the numerator:

1.33 (kW.m^{-2})$^{1.33}$ .min (tolerance limit/pain/first degree burns)
10 (kW.m^{-2})$^{1.33}$ .min (severe incapacitation and second degree burns)
16.7 (kW.m^{-2})$^{1.33}$ min (fatal exposure with third degree burns).

or all the expressions in this section time to effect is estimated based upon measured effects on exposed skin of subjects for given exposures to hot air or different levels of incident radiant heat flux.

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