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TOXICANTS IN COMPARTMENT FIRES

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Introduction

The effects of fires on victims are a combination of thermal hazard as well as exposure to toxic and / or optically – obscuring smoke . In many cases death is not primarily due to thermal hazards, it may results from the victim being trapped in the fire, either because irritant and optically- obscuring smoke prevents escape or because asphyxiant gases cause incapacitation Thus the victim remains in the enclosure under fire to die either from a fatal dose of toxic products acquired during the prolonged exposure, or from burns. One way of taking these factors into account would be to determine the dose defined as the product of concentration and time to exposure i.e. Ct, at which effects such as incapacitation due to asphyxia occur in small scale toxicity tests. These could then be applied to the fire hazard analysis to estimate the fractional incapacitating dose, rather than the fractional lethal dose. However, because of the differences in generating small-scale fire test atmospheres similar to those occurring in large –scale fires, an effective way of predicting toxic hazard would be to measure the concentration / time profiles of the important toxic products in the fire and to determine their effects from toxicity data derived from experiments on man and primates (and to a lesser extent also from rodents). Characteristics of the major asphyxiant and irritant fire products are discussed hereafter.

Asphyxiation by Fire Gases

Asphyxiant gases cause incapacitation mainly by affecting the central nervous system and to some extent, the cardiovascular system. In general, time to incapacitation means the shortest period of exposure of intoxicants at which severe incapacitation (i.e. loss of consciousness) takes place.. Most asphyxiant gases produce their effects by causing brain tissue hypoxia. Since the body possesses powerful adaptive mechanism designed to maximize oxygen delivery to the brain, it is usually possible to maintain normal body function up to a certain dose of asphyxiant, and the victim is often unaware of the impending intoxication. Once a point is reached where normal function can no longer be maintained, deterioration is rapid and results in death if exposure continues.

Asphyxiant Fire Products

The two major asphyxiant gases in fires are (1) Carbon monoxide (CO) and (2) hydrogen cyanide (HCN). Carbon monoxide is always present to some extent in all fires, irrespective of the materials involved or the stage (or type) of fire. Thus there is almost always some degree of risk of asphyxia from CO exposure. Hydrogen cyanide is always present to some extent when nitrogen-containing materials are involved in fires. These include materials such as acrylics, polyurethane foams, melamine, nylon and wool. Which are likely to be involved to some extent in most fires in buildings. Hydrogen cyanide is likely to be present at high concentrations in large, post-flashover fires. In addition, low concentrations of oxygen (less than 15 percent) and very high concentrations of carbon dioxide, CO_2, (greater than 5 percent) can have asphyxiant effects.

Carbon Monoxide

Carbon monoxide combines with hemoglobin in the blood to form carboxyhemoglobin (COHb), which results in a toxic asphyxia because it reduces the amount of oxygen supplied to the tissues of the body particularly brain tissue. Oxygen supply to the tissues is reduced because the amount of hemoglobin available for the carriage of oxygen (in the form of oxyhemoglobin) is reduced, and also because the ability of the remaining oxyhemoglobin to release oxygen to the tissues is impaired.

The affinity of hemoglobin for CO is extremely high so that the proportion of hemoglobin in the form of the carboxyhemoglobin

increases steadily as CO is inhaled. The toxicity of CO, therefore, depends upon the accumulated dose of carboxyhemoglobin, which is expressed in terms of the percentage of the total hemoglobin in the form of the carboxyhemoglobin,(% COHb).

There is little doubt that CO is the most important asphyxiate agent formed in fires because

1. It is always present in fires, often at high concentrations.
2. It causes confusion and loss of consciousness, thereby, impairing or preventing escape.
3. It is the major ultimate cause of death in fires.

To understand the effects of CO exposure on fire victims and to predict the likely consequences of a particular exposure, it is essential to know a number of features of CO intoxication. (To some extent these apply to an evaluation of the toxicity of any fire product.) It is necessary to

1. Determine which types of toxic effects occur at different levels of dose.
2. Determine the concentration / time relationships of those toxic effects, whether they occur immediately or some time after exposure, and whether short time exposure of high concentration has the same effect of a longer, low concentration, exposure.
3. Quantify the parameters that determine the rate of uptake and removal of CO from the body.

Experiments have been carried out by low level exposure on humans. As CO inhalation affects carboxyhemoglobin concentrations which varies at various CO concentrations., the data are available on the symptoms experienced in humans at various carboxyhemoglobin concentrations at rest. Toxic effects vary depending upon the activity of the victim e.g. loss of consciousness is predicted at approximately 40 percent COHb for the victims at rest. Death is predicted when COHb concentration is 50-70%, however it can occur even at lower concentrations in case of subjects with compromised cardiac function. During the experiments conducted on animals by various researchers, the following were observed:

A. The concentration of COHb which affects the psychometric function of victim depends upon whether he/she is at rest or active. During early stage of CO uptake, the effect of exposure is minimal as COHb concentration builds up gradually till it reaches 40% in case of inactive or sedentary animals. However, COHb concentration higher than15-20% is good enough to show effect in active animals. O'Donnell et al. have reported similar results of no effects upon psychomotor performance at levels of up to 12 percent COHb in humans. Stewart et al. reported the first symptoms (consisting of a headache) to occur at 15 to 20 percent COHb. However a sedentary persons feel no effect of COHb upto 40%, while those engaged in light activity, are affected seriously even at 25 – 35% COHb. In one study involving human, although a sedentary subject could perform such tasks as writing, even at the exceptionally high level of 55 percent carboxyhemoglobin, the subjects collapsed and became unconscious immediately when attempting to rise and walk. Therefore, a victim in a bed or chair attempting to escape not only would be in danger of a rapid collapse due to continued CO uptake, but even if no further uptake occurred the ability to perform even light work or exercise would be severely impaired. Even the simple act of rising from a horizontal to an upright position could precipitate loss of consciousness. This is because the rate of uptake of CO depends on the respiration (respiratory minute volume) and hence the activity of the subject. When the subject becomes active the blood carboxyhomoglobin is likely to increase rapidly to an incapacitating value due to increased respiratory minute volume (RMV).

B. When victims are exposed to high concentration of CO, effect is significant and incapacitation is so rapid that by the time a victim becomes aware that he or she is affected; effective action is probably not possible. For active animals there was a sudden rapid decline in behavioral task performance accompanied briefly by signs similar to severe alcohol intoxication, which resulted in state of deep coma immediately.

These findings may explain why deaths from CO derived from defective heating appliances are so common. Survivors of such situations often report that they or other victims that died, experienced headaches or nausea, but had no idea of the causes, so they did not attempt to leave the area until overcome by toxic fumes.

Hydrogen Cyanide

Hydrogen cyanide (HCN) has been measured in the blood of both fatal and nonfatal fire victims. However, the dynamics of HCN uptake and removal from blood are poorly understood, so it is difficult to relate the HCN exposure to the blood cyanide level from samples collected after a fire.

Although the ultimate effects of HCN exposure (consisting of unconsciousness with cerebral depression) are similar to those produced by CO, the pattern of toxicity during the early stages is very difficult. While the onset of CO intoxication is slow and insidious, HCN intoxication tends to be rapid and dramatic. During one experimental exposure on animals it was found that when exposed to HCN, the immediate effects were relatively minor, consisting of slightly raised ventilation as with CO, but at some time during the 30-minute exposure period, there was a marked increased in respiration (hyperventilation). The RMV increased up to four times and the animals lost consciousness just after one to five minutes of start of the hyperventilation stage accompanied by EEG signs of severe cerebral depression, loss of muscle tone, and marked effects upon the heart and circulation, including a significant decrease in heart rate, arrhythmias and changes in the EKG waveform indicative of cardiac hypoxia. The hyperventilatory episode was caused by the stimulatory effects of cyanide upon respiration. The cyanide taken in via inhalation caused hyperventilation which increased the rate of HCN uptake and in turn provided a stronger hyperventilatory stimulus. Once the animals became unconscious the hyperventilation subsides and they went into a slow decline for the remainder of the exposure. This led eventually to a cessation of breathing in some cases, which would have proved fatal if exposure had not been terminated. It was, therefore, possible for an animal to survive a continuous HCN exposure for some time after the point of incapacitation. Once exposure was terminated the recovery was rapid and almost complete within five to ten minutes.

The pattern of incapacitation for HCN is some what different from that produced by CO in that the effect occur more rapidly, as unlike CO, HCN is not held almost exclusively in the blood, but is carried rapidly to the brain. Although the accumulation of a dose is one factor, the most important determinant of incapacitation with HCN appears to be the rate of uptake, which in turn depends upon the concentration of the HCN in the smoke and the subject's respiration. Thus in the animal experiments it was found that at HCN concentration below approximately 80 ppm the effects were minor over periods of up to one hour, with mild background hyperventilation. At concentrations above 80 ppm up to approximately 180 ppm, hyperventilation with subsequent unconsciousness occurred at some time during a 30-minute period. There was a loose linear relationship between HCN concentration and time to incapacitation. Above 180 ppm the hyperventilation began immediately with unconsciousness occurring within a few minutes. Data on human exposures to HCN are limited but Kimmerle quoted some approximate data showing a similar effect in humans, with incapacitation occurring after 20 to 30 minutes at 100ppm HCN and after 2 minutes at 200 ppm, death occurring rapidly at concentrations exceeding approximately 300 ppm.

Other data suggest that human victim might be able to survive higher concentrations of HCN for shorter periods. McNamara suggests 539 ppm as the 10-minute LC_{50} for humans, and there is a report of a survival from an accidental exposure to 444 ppm. An experimental human exposure to 530 ppm HCN was survived without immediate symptoms for 1.5 minutes, although a dog exposed at the same time suffered respiratory arrest. It is observed that dogs are more susceptible to cyanide poisoning with HCN (as with CO) as body size influences time to incapacitation. Similarly, a human would be able to tolerate exposure to given concentration longer than a cynomolgus monkey. Physical activity also results in more rapid uptake in adults during HCN and CO exposure, and uptake would be more rapid in children because of their small body size. The above animal studies show that data on effect of exposure of toxic gases can be used to model for humans, erring slightly on the side of safety.

Another difference between CO & HCN exposure exists in the rate of inhaled incapacitation dose. The inhaled concentration in case of CO varies almost linearly with time i.e. incapacitation dose, Ct product is constant & approximately 27000 ppm.min for all CO concentrations. While Ct product remains constant i.e. 2000 ppm.min at 100 ppm and it is 400 ppm.min at 200 ppm, with very rapid incapacitation at higher HCN concentrations. This deviation from Haber's rule (which predicts a constant Ct product) was recognized by Haber himself in 1924, when he stated that the Ct product for HCN depended upon the exposure concentration. The exact reason for this is not known, but appears to be related to relationship between the rate of uptake of HCN and the dynamics of its distribution between the different body fluid compartments. The effect is to render concentrations in the range greater than 150 ppm more toxic than would be predicted from the effects of longer exposures to lower concentrations.

The accurate data on the concentration of HCN which causes incapacitation or death, is not available due to poor understanding of the dynamics of cyanide uptake, dispersal and metabolism in the body, and the inadequate database of blood cyanide measurements from both injured and dead fire victims. In most of fire victims, Carboxyhemoglobin is the only blood toxin routinely measured in fire victims, while cyanide concentration in most of cases is measured posthumpously during post mortem), a day or more after exposure. It is observed that concentration of HCN uptake depends upon the rate of respiration so HCN uptake rate is greatly reduced when the subject loses consciousness (or dies) and the cyanide in the plasma disperses throughout the body fluids, leaving a low immediate post-exposure plasma concentration. Also cyanide decomposes rapidly in cadavers, by approximately 50 percent in 1 to 2 days, and may subsequent decrease further or even increase slightly in stored blood while during exposure the HCN concentration is much higher as observed after the incapacitation or death.

For these reasons, blood cyanide concentrations measured in fire victims are often relatively low, but when blood samples are obtained immediately after exposure, higher toxicologically significant or life- threatening levels are detected. It is suggested that, in freshly obtained whole blood samples, levels of 2.0 to 2.5 µg CN/ ml should be considered capable of causing incapacitation and 30µg CN/ ml should be considered lethal, while for samples not taken and analyzed immediately after exposure, these concentrations/ effect ranges should be at least halved, depending upon the time of storage.

Hypoxia

Apart from the tissue hypoxia caused by CO and HCN, hypoxia in fires can also be caused by exposure to low oxygen concentrations. To some extent, a lowered oxygen concentration in the inspired air or a lowered oxygen concentration in the lungs (during exercise for example) is a normal physiological occurrence; there are compensatory mechanisms that tend to maximize the supply of oxygen to the brain. When a subject is placed in a hypoxic situation, there is a reflex increase in cerebral blood flow and also, up to a point, the unloading of oxygen from the blood is more efficient at lower arterial and venous blood oxygen concentrations. These factors compensate to a large degree for any decrease in the oxygen concentration of the inspired air. When cynomolgus monkeys were exposed to atmospheres containing 15 percent oxygen no deleterious effects occurred beyond a slight increase in the heart rate.

However, a time is reached where these compensatory mechanisms fail; a 10 percent oxygen atmosphere produced a marked cerebral depression in monkeys. In humans hypoxia due to lack of oxygen (hypoxic hypoxia) has been studied extensively, particularly hypoxia that occur at high altitudes. As in monkeys there is little effect down to 15 percent O_2, beyond a slightly reduced exercise tolerance, but at approximately 10 percent O_2, effects suddenly become severe. It is possible, however, to identify a number of degrees of physiological and behavioral decrement, and for low-oxygen hypoxia certain signs can be related to particular exposure concentrations. Hypoxic

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conditions are also observed at different altitude. As altitude increase, oxygen concentration decreases. From experiments in humans at different altitudes, the effects have been classified into four phases as follows:

1. Indifferent phase (20.9- 14.4 percent O_2): In this phase, no major effect is found on the human being except minor effects on visual dark adaptation and beginnings of effects on exercise tolerance towards 15 percent O_2 . Such conditions are observed at heights of 3000 m above sea levels when oxygen concentration varies from 20.9-24.4%.
2. Compensated phase (11.8-14.4percent O_2): Slightly increased ventilation and heart rate, slight loss of efficiency in performance of complex psychomotor tasks and short term memory, some effects on judgment. Maximal exercise work capacity is reduced. Such conditions are observed at 3,000- 4,500 m height above sea level
3. Manifest hypoxia (4,500-6000 m or 9.6 - 11.8 percent O_2): Degradation of higher mental processes and neuromuscular control, loss of critical judgment and volition, with dulling of the senses. Emotional behavior may vary from lethargy and indifference to excitation with euphoria and hallucinations. Marked increase in cardiovascular and respiratory activity. This is the region likely to be particularly dangerous during the fire exposures, representing the catastrophe point as a victim passes from this stage into the fourth stage at approximately 10 percent O_2 (or COHb or blood cyanide concentrations producing an equivalent degree of brain hypoxia)
4. Critical hypoxia (6000-7,600m or 7.8-9.6 percent O_2): Rapid deterioration of judgment and comprehension leading to unconsciousness followed by cessation of respiration and finally of circulation at death.

In case of sudden exposure of low- oxygen environment, no immediate effect is observed on the physiological status or behavioral task performance of victim due to physiological compensatory mechanisms. As the severity of an exposure increases, a certain point is reached when tissues hypoxia becomes critical and deterioration becomes very marked and very rapid, usually leading to unconsciousness. This end point, therefore, marks the sudden change in a potential fire victim from a condition of near normality to a condition in which escape would not be possible.

Hyper capnia: At the concentrations of approximately 5 percent and above, carbon dioxide is itself an asphyxiant. However, the change in the degree of incapacitation is more gradual for elevated CO_2 concentration (hypercapnia) than that for hypoxia. For approximately 3% CO_2 concentration, breathing becomes rapid and upto 6 percent, the respiration becomes difficult. It becomes severe at approximately 5 to 6 percent, when breathing become intolerable within 20 minutes and fails to satisfy intense longing for air or much discomfort, with severe symptoms like headache and vomiting. Symptoms of dizziness, drowsiness, headache, and fatigue start to occur at concentration above 7 percent, with danger of unconsciousness occurring within a few minutes increasing from 7 to 10 percent. When the exposure concentration approaches to or becomes more than 10 percent, loss of consciousness occurs earlier (over a period of a few minutes), within 2 minutes at 10 percent CO_2 in humans.

Interactions between Toxic Fire Gases

Although data on the concentration /time/dose relationships of the dangerous and lethal asphyxiant effects in humans of individual fire gases are necessarily limited, they are adequate for the construction of a usable incapacitation model. However, the effect of interaction between combinations of these gases on time to incapacitation in fires is an area that requires further investigation, as very little information is currently available. The best that can be done currently is to suggest likely degrees of interaction based on physiological data from individual gases and on such experimental data for gas combinations as do exist.

Effect of Carbon Dioxide on effects of CO, HCN, and Low- Oxygen Hypoxia

Hyperventilation increases due to carbon dioxide exposure which causes increase in the rate of uptake of other toxic gases and thus decrease the time to incapacitation (or the time taken to inhale a lethal dose), in proportion to the increase in ventilation. This happens most likely to be most important with respect to CO intoxication, particularly for a subject at rest, and also to some extent for active subjects. It is found that RMV and hence the time to incapacitation is largely influenced by the concentration of CO present as shown in the table below.

Co_2 Concentration	Respiratory Minute Volume (RMV)	Effect on RMV	Time to Incapacitation
<3%	-	Insignificant	t
3%	Doubled	Effect is doubled	t/2
5%	Tripled	Effect is tripled	t/3

There is a possibility that the effects on the time to incapacitation would not be as dramatic as this, since there is evidence that the presence of carbon dioxide may counteract the leftward shift in the oxygen dissociation curve caused by carbon monoxide, some what counteracting its deleterious effects. However, in the absence of experimental data on combination exposure it is best to ignore this possible beneficial effect, since the effect on uptake rate is likely to be dominant. A similar effect on uptake may also occur with HCN. With regard to low oxygen, carbon dioxide has been shown to have a marked beneficial effect on resistance to incapacitation. This is due to the hyperventilatory effect that increases the rate of oxygen uptake. This improves the delivery of oxygen to the tissues, counteracting the respiratory alkalosis that otherwise occurs. New evidence is currently being obtained from experiments on the effects of combinations of asphyxiant gases with CO_2 in rodents, that with severe exposures, post exposure lethality is increased by the presence of CO_2. When animals are severely affected and suffering from a hypoxia induced metabolic acidosis, this appears to be enhanced by the further acidosis effect of CO_2 inhalation, and the animals then fail to recover after exposure under conditions when they would otherwise be expected to do so. It is also to be expected that hyperventilation induced by CO_2 would increase the uptake of substances that irritate the lung, which also tend to cause toxic effects some time after exposure, and recent experiments in rodents are providing evidence that this is so, with increased deaths possibly caused by post exposure acidosis and increased lung damage. Exercise also causes a CO_2 driven hyperventilation, and there is new evidence that this may also cause deaths when rodents are exposed to irritants at normally sub-lethal concentrations.

Interactions between CO and HCN

Some studies have been made of interactions between CO and HCN, with varying results. On theoretical grounds little interaction is to be expected, since CO diminishes the carriage of oxygen in the blood and its delivery to the tissues, while HCN diminishes the ability to use oxygen once delivered to the tissues. It is therefore to be expected that either one or the other gas would constitute the rate- limiting step in oxygen supply and utilization. However the consensus view is that there is at least some additive effects between these two gases. Experiments in primates have shown that time to incapacitation by HCN is slightly reduced by the presence of near-toxic concentrations of CO; also, the rate of uptake of CO may be increased by the hyperventilatory effect of HCN. In these circumstances it is probably safest to assume that these gases are additive in terms of time to incapacitation and dose to death, and that incapacitation or death will occur when the fraction of the toxic dose of each one adds up to unity.

Interactions between CO and Low-Oxygen Hypoxia

The effect of CO and low oxygen Hypoxia is additive if both are present in toxic gases as both reduce the percentage oxygen in arterial blood. CO also has the hypoxia effect by reducing the concentration of O_2 in the gases because of combination of CO with O_2. When the subjects are at rest or at altitude they remain symptom free at low levels of CO saturation. However, in a hypoxic environment, exposure of the high concentration of CO has severe effect.

In case of irritant gas environment, the CO uptake reduces due to decrease in respiratory rate and RMV because of irritation in upper respiratory tract. This effect is observed in rats and mice. This marked, prolonged decrease in respiratory rate does not occur in humans or nonhuman primates; indeed in the primate smoke experiments, irritants products tend to increase rather than decrease ventilation (although not sufficiently to increase CO toxicity).

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In summary, data on interactions between the asphyxiant gases CO, HCN, low oxygen, and CO_2 are limited. The above paragraphs show that there is additive effect due to interaction of different ashphyxiant gases which should be incorporated in the incapacitation model to have proper safety. For this reason, it is proposed that the interactions should be quantified in the incapacitation model as follows:

1. Assume that CO and HCN are directly additive (1:1) on a fractional does basis (the evidence suggests that they are additive, but that the additive interaction may actually be less than unity)
2. Assumes that the rates of uptake of CO and HCN and their fractional doses are increased in proportion to any increase in ventilation (RMV) caused by carbon dioxide
3. Assumes that the fractional doses of CO and HCN, adapted for carbon dioxide, are additive with the fractional dose of low- oxygen hypoxia.
4. Assume that asphyxia by carbon dioxide is independent of that induced by CO, HCN, and hypoxia.
5. Assume that irritancy is independent of asphyxia, but that uptake of irritants is increased by carbon dioxide.

Irritant Fire Products

Incapacitation effect of irritant fire products is difficult to quantify as can be done in case of asphyxiants, which are clear-cut and well understood. Irritant fire products produce incapacitation during and after exposure in two distinct ways.

1. **Sensory Irritation:** During exposure the most important form of incapacitation is sensory irritation, which causes painful effects to the eyes and upper respiratory tract and to some extent also the lungs. Although exposure may be painful and thus incapacitating, it is unlikely to be directly lethal during exposure unless exceptionally high concentrations of irritants are present. For sensory irritation, the effects do not depend upon an accumulated dose but occur immediately upon exposure and usually lessen some what if exposure continues.
2. **Inflammatory Irritation**: It can cause respiratory difficulties and may lead to death within 6 to 24 hr after exposure. The effects do not show the sharp cut off of asphyxiation, but lie on a continuum from mild eye irritation to severe pain, depending upon the concentration of irritant and its potency. For inflammatory reaction, the effects is dependant upon an accumulated dose, approximately following Haber's rule, and there seems to be a threshold below which the consequences are minor, but when this dose is exceeded, severe respiratory difficulties and often death occur, usually 6 to 24 hr after exposure. However, for most sensory irritants the ratio between the concentration producing severe irritation and the dose causing death is usually large (15 to 500 times) for 30-min exposure times.

The low concentration of irritants has minor effects like producing mild eye and upper respiratory tract irritation resulting in slow down of the movement of occupant through building (as would simple visual obscuration) but the combined effects of eye irritation and direct visual obscuration may be more serious, and it has been shown that human volunteers moved more slowly through irritants smoke than through nonirritant smoke not only due to direct physiological effects but also due to psychological and behavioral effects such as the unwillingness of an individual to enter a smoke-filled corridor.

One of the main difficulties to predict the consequence of exposure to irritants is the poor quality of data available on the humans. Obviously very few controlled studies have been made on the effects of severe irritancy in human, so that most data are anecdotal, derived from accidental industrial exposures, with only a vague knowledge of exposure concentrations. It is not possible to quantify the incapacitation or lethal threshold in view of the following facts:

1. Reports of the severity of the effects are found very subjective, so that the term "severe irritation" could cover a wide range of sensations with varying degrees of actual incapacitation.
2. As sensory irritations covers a continuous range from mild eye and upper respiratory tract irritations to severe pain, there are no simple objective end points or thresholds.

During fire smoke exposure studies on humans, conflicting reports were observed. Some persons say that they went through dense smoke without experiencing any great discomfort, while others say that respiratory difficulties prevented them from entering smoke- filled areas. This seems to depend upon the type of fire. For example, the smoke from some well ventilated fires involving primarily cellulosic materials has been reported as irritant but not seriously incapacitating, while that from the some plastic materials (e.g., the interior of a burning car) was found to cause severe effects when only a small amount of smoke was inhaled. However, the effects can be mitigated by blinking or shutting the eyes and the effects on nose can be mitigated by mouth breathing and breath-holding. Also, it is known that people in emergency situations are often unaware of painful

stimuli. It is therefore likely that irritant smoke products do have some severe effects on the escape capability of the fire victims, but it is difficult at present to predict accurately the likely degree of incapacitation.

Conclusion

It can be observed from previous sections, exposure to ashphyxiants / high concentration of irritants can cause incapacitation and deaths. So in order to mitigate this hazard, following measures should be taken

1. For smoldering fires, it would be advantageous if materials were designed to self- extinguish, and if the formation of products other than CO during decomposition (such as oxidized hydrocarbon fragments or CO_2) could be encouraged. Early audible warning by smoke alarms may be particularly advantageous, as sound often appears to alert victims where the presence of irritant smoke or heat fails.
2. For early flaming fires where the victim is in the room of origin, any measure that limits the rate of growth once ignition has occurred will give a victim more time to extinguish a small fire or escape from a growing one.
3. For fully developed fires where the victim is remote from the point of origin, the most important mitigating factors are probably early warning and containment of the fire and gases within the original fire compartment.
4. For designing a system to be safe in hazardous situations in a fire, involves a whole range of factors, including fire development from ignition to the post-flashover spread of fire and smoke, toxicity, and the interaction of the fire with structure and with passive and active fire protection, as well as escaped- related factors, including detection, warnings, the provision of escape routes, way finding, physiological and behavioral impairment, and escape movements or rescue should be considered, and the ultimate evaluation of safety depends upon whether it is possible to ensure, by performing a life-threat hazard and risk assessment, that the occupants can reasonably be expected to have escaped before they are exposed to levels of heat and smoke that may endanger health and threaten life.

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