
Toxicological Mechanisms Of Fire Smoke

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Citation

H Savolainen, N Kirchner. *Toxicological Mechanisms Of Fire Smoke*. The Internet Journal of Rescue and Disaster Medicine. 1997 Volume 1 Number 1.

Abstract

Most of the fire victims die or are affected by smoke rather than by flames and the resulting burns. Asphyxia is the principal mechanism of the intoxication, mediated by oxygen deprivation, carbon monoxide inhalation and sometimes even by hydrocyanic acid inhalation. The other major mechanism is the inhalation of soot and irritating products of combustion. In this paper we review the mechanisms of smoke inhalation resulting from the combustion of some materials present in the modern building, such as mineral fibres, rigid polyurethane, polystyrene and cellulose fibres.

INTRODUCTION

There were some 467,000 residential fires in the USA during 1990 (Orzel, 1993). They caused over 4,000 deaths directly with over 20,000 injuries (Orzel, 1993). Smoke inhalation accounted for two thirds of the fatalities. While the number of fatal burn cases have decreased by 34 % in the USA as compared to the figures 1979-1985, there has been little changes in the smoke inhalation fatalities (Orzel, 1993).

The continuing toxicity of smoke may be related to the use of synthetic polymers as construction materials, for furniture and upholstery. One of the proximal toxicants is HCN liberated from the nitrogen-containing polymers, like polyurethane, acrylonitrile-butadiene-styrene copolymer or styrene-acrylonitrile copolymer. All of them are extensively used in the construction or for the furniture. Together with CO, CN⁻ ion has been principally implicated in the death of fire victims (Baud et al., 1991).

The inhalation of soot in the form of smoke aerosol is the second major clinical problem in the care of fire victims (Masanès et al., 1995). It seems that the carbon particles of the soot are persistently deposited on the alveolar and bronchiolar surface (Mitchelson, 1992) necessitating phagocytosis by lung macrophages for their removal (Moores et al., 1993). The carbon particles are toxic to the macrophages (Herlihy et al., 1995), possibly through the heavy metals in them (Mitchelson, 1992) through their polycyclic aromatic hydrocarbon (PAH) content (Gerde et al., 1991) or directly by inducing peroxidation through free radical mechanisms (Sagai et al., 1993).

The third major problem is the intense sensory irritation of the smoke. The irritants include notably acrolein (Table 1) and lead rapidly to functional impairment (Malek et al., 1987). Acrolein has a synergistic role in the toxicity of carbon particles (Jakab, 1993) in addition to its directly toxic lung effects (Hales et al., 1992).

The concentrations of the major toxic agents encountered in residential fires have been summarized in the Table 1. From the point of view of respiratory protection of fire fighters, the self-contained breathing apparatus is necessary means to provide safety (Burgess and Crutchfield, 1995). As to the risk factors in fatal residential fires, they include foremost risk-taking behaviour of residents and the heating equipment (Runyan et al., 1992). Modern houses built after 1976 have more often smoke detectors than older buildings clearly providing early warning (Runyan et al., 1992) possibly alleviating the toxic smoke hazard from burning polymeric material. Nevertheless, the choice of construction materials is a critical factor in the passive structural safety in case of fire.

EFFECTS OF SMOKE FROM TECHNICAL MINERAL FIBRES

Contamination of air with mineral fibres occurs in case of fire in buildings where the technical products have been used as construction material (Hoskins and Brown, 1994). Glass and rock wool fibres melt in the temperature range of 1,000 to 1,500°C so that in high-temperature fires they begin to loose their fibrous structure. Otherwise, the uptake of the fibres by the lungs follows the same rules as for fibres released in the air by other means.

In the fire situation, the mineral fibres are included in analysis of the particulate fraction (Table 1) whereas systematic studies for fibre counts in house fires have not been published in the literature. The possibility of inhalation exposure to mineral fibres occurs after the fire in the demolition or rebuilding of the remains.

Figure 1

Table 1: Common combustion product concentrations in residential fires

Contaminant	Concentration (ppm)		IDLH (ppm)
	Mean	Maximum	
Acrolein	1.9	98	5
Benzene	4.7-56	250	3,000
CO	246-1,450	27,000	1,500
HC1	0.8-13	280	100
HCN	0.14-5.0	75	50
NO ₂	0.04-0.7	9.5	50
SO ₂	2.3	42	100
Particulates*	232	15,000	n.a.

*Particulates are given as mg/m³. IDLH stands for a concentration defined as “immediately dangerous for life and health”. Data taken from Burgess and Crutchfield (1995). n.a. = not available.

As mineral fibres do not burn, the toxicity of off-gases from the technical products results most probably from binders, covers or vapour barriers (Table 2). Thus, the use of layers of fibrous minerals rather limits than adds to the toxicity of fire smoke as calculated by dose or by exposure time (Table 2). It seems that e.g. the glass wool can be classified as one the safest construction materials in this respect (Levin and Purdom, 1983).

Figure 2

Table 2: Comparative toxicity of smoke from burning construction materials

Material	LC ₅₀ (g)	LT ₅₀ (min)	CO (%)	HCN (ppm)	Remarks
EPS	5.8	11	1.95	--	Fluorocarbon blown.
PUR	7.5	17	1.20	100	
Cellulose	11.9	21	4.0	--	no flame retardant. Blown fibres for insulation 4.4 m building insulation pane with paper and vapour barrier
Glass fibres	35.7	25	n.a.	--	

LC50 indicates the amount of material to cause smoke concentration that is fatal to 50% of rats when burnt at 822°C. LT50 shows the time until death of the rats under same conditions. 1% equals to 10,000 ppm (part per million, vol./vol.). EPS denotes to expanded polystyrene and PUR stands for rigid polyurethane. Addition of a flame retardant prolongs the LT50 for EPS and PUR. Data taken from

Alarie, 1985 and Levin et al., 1987a.

n.a. = data not available.

EFFECTS OF SMOKE FROM RIGID POLYURETHANE

Rigid polyurethane begins to degrade already at 250°C (Rosenberg and Savolainen, 1986). The degradation products include the isocyanate monomer (MDI, or methylene diphenyl isocyanate), the corresponding amine (MDA, or methylene dianiline), CO and HCN (Rosenberg and Savolainen, 1986; Orzel et al., 1989). The release of CO and cyanide is degradation temperature-dependent (Zitting et al., 1982). The co-exposure to HCN and CO show synergistic rather than additive effect in the toxicity of the smoke in a rat model (Esposito and Alarie, 1988; Prager et al., 1994). If the experimental polyurethane samples were proofed with flame retardants, the toxicity of smoke was slightly diminished or remained unchanged (Hilado et al., 1976; Hilado and Huttlinger, 1980a).

The fire smoke from rigid polyurethane contains much aerosol (Zitting et al., 1982). Most of this is liquid droplets which contain remaining polyols (Orzel et al., 1989) and most likely short oligomeric chains of the original polymer (Skarping et al., 1994). Accidental exposure to pyrolysis products from rigid polyurethane has caused respiratory irritation and fever (Littorin et al., 1994) and constrictive pulmonary disease (Voumard et al., 1995). Mice experimentally exposed to smoke from polyurethane showed extensive changes in the lung surfactant metabolism (Oulton et al., 1994).

As only polyurethane of the examined construction materials released HCN (Table 2) it is probable that it and other nitrogen-containing polymers have caused the HCN concentrations in the fire atmospheres (Table 1) and sometimes fatal cyanide doses in fire victims (Baud et al., 1991). It should be remembered in this context that almost invariably the victims have also been exposed to CO and have an important amount of soot in the lungs (Shusterman, 1993). This necessitates special treatment facilities and strategies (Crapo and Nellis, 1980). Long-lasting branchial hyperreactivity may result from exposure to fire smoke (Kinsella et al., 1991; Moisan, 1991).

EFFECTS OF SMOKE FROM POLYSTYRENE

Thermal degradation of polystyrene causes temperature-dependent emission of CO and aerosol (Pfäffli et al., 1978) Free styrene and benzaldehyde are also released. The aerosol

contains e. g. oligomeric polystyrene chains (Pfäffli et al., 1978). Styrene is moderately neurotoxic in animal models (Savolainen and Pfäffli, 1977; Savolainen et al., 1980). The off-gases of thermally degraded polystyrene caused a decrease of isolated liver cell glutathione content in an in vitro experiment (Zitting et al., 1980). Based on this, it is probable that the most important aspect of polystyrene smoke toxicity is its aerosol and CO content.

Burning polystyrene causes a dense black smoke (King, 1975) which impairs visibility and may hinder rescue work.. The health effects of smoke particles from polystyrene are not known to the detail while in a comparative study they were assessed to be less harmful than smoke from wood, cork, leather or rubber (Hofmann and Oettel, 1968). The toxicity of polystyrene smoke is most clearly associated with the concomitant CO exposure (Larsen et al., 1994). The authors raise also the question as to the potentiation of CO effects by a simultaneous co-exposure to CO₂. A synergistic toxic effect has been shown also in earlier independent studies (Levin et al., 1987b). One of the mechanisms seems to include changes in the hemoglobin oxygen-binding capacity.

In conclusion, the fire smoke toxicity from polystyrene seems mainly to be determined by the CO given out from the material. Its toxicity in terms of the smoke lethality is comparable to that of polyurethane (Table 2).

EFFECTS OF SMOKE FROM CELLULOSE FIBERS

Cellulose is a polysaccharide and therefore its oxygen content is higher than of that of polyurethane or of polystyrene. The cellulose polymer begins to decompose at 250°C (Nousiainen, 1983). The initial products include various glucose and furan products with further formation of acrolein and other respiratory irritants (Morikawa, 1976). Acrolein is very toxic to lungs (Hales et al., 1992; Jakab, 1993). The furans are also toxic, and a model furan derivative, furfuryl alcohol, shows dose-dependent neurotoxicity in an inhalation exposure model (Savolainen and Pfäffli, 1983). There are no data on the roles of toxicity of furan derivatives in the fire victims.

The aerosol of the cellulose smoke contains free oxygen-related radical species (Lachocki et al., 1988). Depending on their stability, they probably add to the toxicity of soot particles deposited in the lung. This might provide one of the synergistic toxicological mechanisms for the interaction of acrolein and carbon particles (Jakab, 1993).

The flame retardants in the cellulose decrease the yield of smoke CO content in a dose-dependent fashion (Hilado and Huttlinger, 1980b). Whether they also facilitate the extinguishing of smoldering cellulose fire (Fielding et al., 1975) remains an open question.

CONCLUSION

Of the evaluated materials, the mineral fibres show less toxicity in case of fire than the other polymers. As mineral fibres do not burn the toxic smoke yield from the burning technical construction materials comes from resin, vapour barriers and covers attached. As to the other evaluated materials their fire smoke toxicity is grossly comparable to each other, while the mechanisms and proximal toxicants vary very considerably.

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