

FIRE SAFETY, SMOKE TOXICITY AND ACIDITY

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ABSTRACT

The question of the importance of smoke toxicity in relation to fire hazard or to the emission of acid gases in smoke is misdirected. Although roughly 2/3 of fire victims die as a direct effect of smoke toxicity, it is extremely rare that their deaths are caused by the inhalation of smoke from a specific very toxic material. In fact, probably well over 90% of fire deaths are the result of fires becoming too big (often so big that they extend beyond the room of origin) and thus resulting in too much toxic smoke. There is excellent correlation between blood CO concentrations in fire victims and the resulting fire fatalities; thus the toxic effects of other combustion products are negligible. More important than any individual combustion product is the fact that if a fire remains small it almost never causes fatalities. Therefore, fire hazard is directly related to the fire heat release rate (i.e. the fire intensity) with other issues (such as smoke toxicity) being of minor relevance. The toxic potency of the smoke of almost all individual polymers lies within a very small band, so that they are almost indistinguishable.

At flashover, every polymer will give off ca. 20% of its weight as CO: that is toxic enough to be lethal. The lethal toxic potencies of CO and HCl are similar and significantly lower than that of acrolein or HCN. In order to study the small toxic effects that can be added to the major heat release effect, the fractional effective dose of that toxicant must be calculated. It will be a function of the ratio of the concentration emitted and the lethal toxic potency. Studies show that CO concentrations regularly exceed its LC₅₀, while those of HCl and HCN rarely exceed a small fraction of their LC₅₀ and those of acrolein rarely exceed its LC₅₀. In other words there is so much more CO than anything else in fire atmospheres that CO is the big toxic killer in smoke. The lethal toxic dose associated with the smoke of virtually all commercial polymers are very similar; and are within a factor of 3, while lethal toxic doses for poisons can be orders of magnitude higher. Therefore, measurement of toxic gases is of very little interest from the point of view of fire hazard. It only helps in material development and in understanding the fire performance of any material or product. There are very few actual fire cases where smoke toxicity can have an important contribution: when people die in very small fires (often smoldering) that destroy minimal amounts of material. Acidity of solutions formed from smoke in fires is a function of acid gas release and it is often used as a synonym for smoke corrosivity (even if that is not fully correct). However, there is no relationship whatsoever between acid gases (or acidity of solutions) and smoke toxicity. “Declarations” of acid gas emissions are not associated with fire hazard but with the development of so-called property protection guidelines and the use of halogen-free materials. In fact, most assessments of smoke toxicity via “indices” tend to be ways of ensuring that materials are free of halogens. This may have a logical rationale in some limited applications for specific reasons, but has no general validity in fire hazard.

Toxic potency is a minor contributor to fire hazard and it is most critically dominated by CO concentration. Fire hazard and life safety are best served by ensuring that fires remain small, meaning that they need to exhibit heat release rates as small as possible.

INTRODUCTION

The toxicity of smoke in a fire is a function of four factors; the amount of materials burnt; the distribution of combustion products within the smoke; the individual toxic potencies of each combustion product found in the vapor phase; and the duration of exposure.

Clearly, the greater the amount of longer material burnt the greater the toxicity of the smoke. In fact although roughly two-thirds of fire victims die from the effects of smoke inhalation, it is extremely rare for the root cause of their deaths to be that the smoke comes from a specific very toxic material. Fire fatalities are usually the result of inhaling too much smoke of average toxicity. More than 83 percent of fire deaths in building fires in the United States occur in fires that have become very large so that they extend beyond the room of origin, and thus generate too much toxic smoke [1]. This means that very few people actually die in fires that are small and that fire deaths are rarely due to burning or heat effects, even in small fires.

CARBON MONOXIDE

All combustible materials release carbon monoxide (CO) when they burn [2]. Once a fire has reached flashover – the moment when every combustible in the fire area is burning and the temperature exceeds 500°C – roughly 20 percent of the mass lost from the combination of any material has been converted into carbon monoxide (CO). This is almost irrespective of fuel composition or ventilation [3-5]. Most fire fatalities occur only after flashover.

Thus, the smoke from fires that have reached flashover contains a baseline toxicity from carbon monoxide. The smoke toxicity from fire effluents other than CO is of little consequence, since there is enough smoke toxicity from carbon monoxide to cause fatalities. Under conditions where flashover is not achieved, smoke toxicity is calculated (with the N-gas model [6]) by adding the contributions to overall smoke toxicity from each individual toxicant found in the smoke (or in the vapor phase).

MASS LOSS MODEL

In a simplified approach, the overall smoke toxicity can be calculated under the general assumption that all smokes are similar in toxicity. This means that the overall smoke toxicity of most materials or products is very similar, and not that every component in smoke has the same toxic potency. With this approach, it is sufficient to assess an overall mass loss, and the criterion for the concentration time product (Ct) for lethality can then be considered to be 900 g min/m³ [7]. This is consistent with various calculations that have been made by summing up abundant data from multiple sources [8-10]. In general, the values of toxic potency of smoke have been found to range between 15 and 30 g/m³, leading to Ct products of 450 to 900 g min/m³ (for a 30 minute exposure, which is typically used in smoke toxicity tests). With the lesser toxicity (i.e. higher value) criterion in a normal size room that has a volume of about 36 m³, lethality results following an exposure to the smoke resulting from burning no more than some 10 kg in just over 3 minutes (actually, a mass loss of exactly 10.8 kg over the 3 minutes).

N GAS MODEL

The overall smoke toxicity can also be calculated in more detail as a fractional effective dose (FED) using the formula shown in Equation 1. In Equation 1, \sum_1 (from 1 to n), corresponds to the summation of the effects of each one of n toxic gases, \sum_2 (from t₁ to t₂), corresponds to the summation of the relative concentration-time effects of the toxicants at each individual time increment (usually 1 min). C_i is the average concentration (in ppm) of the toxic gas "i" over the chosen time increment Δt and (C t)_i are the specific lethal exposure dose (concentration-time product, in ppm•min).

Fractional Effective Doses (FEDs) can be determined for each toxic gas (from 1 to n) at each discrete increment of time. The time at which their accumulated sum exceeds the lethal toxic dose represents the time available until lethality sets in with the actual concentrations measured. With this analysis, smoke toxicity depends both on the concentrations of toxic gases and on the intrinsic toxic potency of each component of smoke. This analysis is also based on the concept that the effects of both asphyxiant gases and irritant gases are a function of their dose (i.e. concentration and duration of

exposure) and not simply of their concentration (as shown in studies of baboons and rats) [11-13].

$$FED = \sum_1 \sum_2 [C_i * \Delta t] / (C t); \quad \{\text{Equation 1}\}$$

Typical lethal toxic potencies (in volumetric units of parts per million, ppm, for 30 minute exposures) for the major toxicants are [5, 14, 15]:

LC ₅₀ CO:	4,000-5,100	Units: ppm
LC ₅₀ HCN:	150-200	Units: ppm
LC ₅₀ HCl:	3,700	Units: ppm
LC ₅₀ HBr:	3,000	Units: ppm
LC ₅₀ HF	2,500-2,900	Units: ppm
LC ₅₀ Acrolein:	90-200	Units: ppm
LC ₅₀ O ₂ (oxygen depletion):	- 54,000	Units: ppm

IRRITANT GASES

In one document, ISO TS 13571 [16] the issue of smoke toxicity of individual toxicants is incorrectly addressed by separating asphyxiants from irritants. Asphyxiants are properly addressed in that document by using the N-gas model, whereby the fractional effective dose (or FED) for toxicity by asphyxiants is the summation of the exposure dose of the individual toxic gases, based on their individual concentration at each time period, just as in Equation 1.. The concentrations used in that document are somewhat different from those shown above, but the general concept is reasonable. However, ISO TS 13571 assumes that heat and irritant gases have no effect on FED for asphyxiants, a statement which is patently incorrect and is not based on any published scientific work. Thus, ISO TS 13571 calculates an FED based on bioassay exposures of asphyxiants which looks at the concentrations of asphyxiants in isolation of everything else, particularly in isolation of irritant effects, heat effects and lack of visibility effects. Abundant work has shown that the N-gas model should not be limited to asphyxiants and that the effect of irritants is also dose-related and should be added to the FED equation, including work by the FAA [17-18], by NIST [6] and by SwRI [11,19-20]. However, ISO TS 13571 invents the bizarre concept of “incapacitating concentration” for dealing with irritants. This concept eliminates exposure time considerations, so that incapacitation occurs only after adding the effects of exposure to a toxic concentration at every time period. This means that as soon as a critical concentration of an irritant is reached, the victim is instantly incapacitated.

This approach is reminiscent of denigrated concepts in use many years ago when people talked about “instant clampdown” resulting from PVC [poly(vinyl chloride)] smoke and developed “correction” factors when dealing with the toxicity of PVC smoke to make it look worse [21-22]. The concept of “incapacitating concentration” is particularly unrealistic for people who have worked in a chemical research laboratory, where it is not uncommon for emissions of irritant gases (e.g. hydrogen chloride) to occur. However, there is no evidence that incapacitated researchers are found throughout chemical laboratories. It is worthwhile remembering also that human exposures to various toxic gases (especially including irritants) have been conducted in Europe, in the late 19th century and early 20th century [23-27]. All of that work was summarized in a modern publication [28]; it clearly showed how researchers were able to continue being active and alert during exposure to high concentrations of irritants. Some of the experimental results of that work on humans are worth repeating:

- * Lehmann 1886 [23]: A 30 year old man was exposed for 12 minutes to 600 ppm min of HCl. He had available a gas mask which he could use to breathe if conditions became intolerable. He found working in the room absolutely impossible after 12 min, part of which was spent outside. He had some irritation of the respiratory system (nose, larynx), irregular respiration solely through his nose, chest pains (needle-like sensation), shortness of breath, no eye irritation and no acid taste.

* Matt 1889 [24]: Three experiments were conducted, designed to address safe work-place concentrations of HCl and did not involve concentration which were incapacitating or lethal

On 12/13/1888 three men were exposed to 100 ppm min of HCl for 10 min. They experienced cold and acid sensation in nose, mouth and throat, no effect on their eyes, a slight discomfort in their larynx, trachea and lung, as well as some secretion and coughing. One man had slight head and chest pains. All men recovered immediately on leaving the HCl atmosphere.

On 12/18/1888 one man was exposed to 1,050 ppm min of HCl for 15 min. He experienced little eye irritation, some irritation on breathing through the mouth, somewhat artificial respiration, irritation in the nose, throat, larynx, trachea and sternum, including “scratching” feelings and coughing. he had to exit the room because of chest pains. He experienced slight headache on termination of the experiment, which disappeared very rapidly.

On 12/13/1888 one man was exposed to 1,500 ppm min of HCl for 15 min. He experienced slight irritation in his eyes, abundant tear secretion, a strong feeling of coldness and irritation in the nose, mouth, throat, larynx and trachea. His respiration was enhanced and his salivation increased. He experienced strong coughing and a sensation of heat in the head, forcing him to exit the room. On reentry, he experienced abundant coughing and catarrh. After the experiment he felt a slight headache and catarrh, which disappeared soon.

As a result, the author recommended the following **work-place** limits for HCl:

10 ppm	Work unhindered
10-50 ppm	Work possible but uncomfortable
50-100 ppm	Work impossible

* Lehmann et al. 1908 [25]: A man (one of the authors, Dr. J. Yamada) breathed from a bottle containing progressively higher concentrations of HCl for periods of 20, 20 and 5 min respectively, and exhaled into another bottle. The inhaled doses were 4,021 ppm min, 4,107 ppm min and 5,170 ppm min. There were no ill effects whatsoever on the subject.

In more recent times, it has been shown that irritants (such as HCl or acrolein) do not cause incapacitation of baboons (primates very similar to humans) or of rats at dose levels so high that the victim eventually dies of inhalation toxicity after the exposure. This is a complex concept, but is critical: when animals have been exposed to doses of irritants at levels where they died a few days after exposure, they were still capable of performing the necessary avoidance responses to escape the exposure, thus not being incapacitated [11]. Interestingly, it has also been found that incapacitation from asphyxiants occurs at levels very similar to those leading to lethality, and not at levels an order of magnitude lower [29].

Thus, the type of statement frequently made regarding the inexistence of data on human exposure (or primate exposure) to irritants is misleading. This should not be used as excuse for presenting other concepts that have not been validated by experiments. Moreover, the pungent odor of most irritant gases (and their low odor detection level, often in the order of 1 ppm, [30]) means the warning appears at levels much lower than those at which any effect occurs. This is usually not being considered.

FLASHOVER

It is interesting to note that the toxic potency (LC_{50}) value of carbon monoxide assessed from experimental data is about 5 g/m^3 , for a 30 minute exposure. Therefore, the LC_{50} of post-flashover smoke (20 percent by mass of which is CO) cannot be higher than a value of about 25 g/m^3 , for a 30 minute exposure, irrespective of the other substances present in the smoke. It has been shown that bench-scale toxicity data will only reflect real-scale fires when the allowable error band is a factor of 3 [14]. Thus, if the toxic potency of flashover smoke is multiplied by a factor of 3 (since toxic potency cannot be

predicted better than that), normal smoke toxicity materials have a smoke toxic potency not exceeding 8 g/m³ (for 30 minutes) [14]. So, materials with a toxic potency lower than 8 g/m³ (or an LC₅₀ higher than 8 g/m³) will become of no consequence at flashover since the toxicity of the flashover fire atmosphere itself is larger than that of those materials. In simpler language, this means that the only materials that need to be considered for toxicity at flashover are those with an LC₅₀ lower than 8 g/m³, and they are very uncommon.

FIRE FATALITIES, CARBOXYHEMOGLOBIN AND TOXIC GASES

A pair of studies were made in the United States involving more than 5,000 fatalities and covering:

- a period between 1938 and 1979 in a localized area (Cleveland, Ohio);
- and a broad countrywide study in the early 1990s [3-4].

The studies demonstrated that there is an excellent correlation between fire fatalities and levels of carbon monoxide absorbed in the blood as carboxyhaemoglobin (COHb) and that the distribution of COHb concentrations was identical (when comparing populations of the same type, following a statistical multi variant analysis) between fire and non-fire deaths (e.g. defective space heater) . The studies also showed that whenever high levels of hydrogen cyanide were found in blood, high levels of COHb were also found, indicating that hydrogen cyanide is of minor consequence in the overall study of fire fatalities. Finally, the studies showed that fatalities can be linked to COHb levels as low as 20 percent and that it is likely that any COHb level above 30-40 percent is lethal. The work also reconfirmed that any hydrogen chloride found during fire fatality studies cannot be assigned to fire gases (or smoke) as it cannot be distinguished from the stomach acid naturally present. The overall conclusion of this work, the most extensive ever conducted, is clear: fire fatalities are overwhelmingly associated with the carbon monoxide generated when fires become big, and other causes of fire deaths are of minor importance. Similar conclusions were obtained earlier by other authors, with smaller data bases [31-33].

Looking at toxic potency of smoke data, Figure 1 indicates that the toxic potency of the smoke of virtually all individual polymers is within such a narrow band (in toxicological terms) as to be almost indistinguishable [34]. In particular, the smoke toxicity of poly(vinyl chloride) has been studied extensively and found to be quantitatively similar to those of most other polymers [28]. As discussed earlier, that work reviewed also hydrogen chloride toxicity studies, including some done by exposure of animals and people, in the late 19th century and early 20th century to hydrogen chloride alone or by their exposure to smoke containing hydrogen chloride. The critical issue is that what was studied was the behavior, and whether incapacitation or lethality occurred, rather than looking for hydrogen chloride itself in autopsies (which cannot be detected). The exposures of rats and baboons made in the 1980s at Southwest Research Institute showed that both rats and baboons were not incapacitated at huge concentrations of hydrogen chloride (and in fact sometimes they died a long time after exposure, but they were able to perform the escape functions that they were taught to do, to escape from their exposure) [11, 28]. This means that the toxic potency of hydrogen chloride is, in fact, such that exposed primates are not incapacitated at concentrations that may eventually kill them.

Two investigations were conducted in the United States in the late 1970s, wherein fire fighters were sent into buildings equipped with gas monitors [34-36]. In the fires investigated, carbon monoxide was almost inevitably present, with the maximum concentration found being 7,450 ppm (approx. 150% of the 30 min LC₅₀). The three other most common gases found in fires in those studies were (in descending order of number of times the gas was found, and shown in relation to the 30 min LC₅₀):

- acrolein (with a maximum concentration of 100 ppm, approx. 50-80% LC₅₀);
- hydrogen chloride (with a maximum concentration of 280 ppm, approx. 8% LC₅₀) and
- hydrogen cyanide (with a maximum concentration of 10 ppm, approx. 6% LC₅₀).

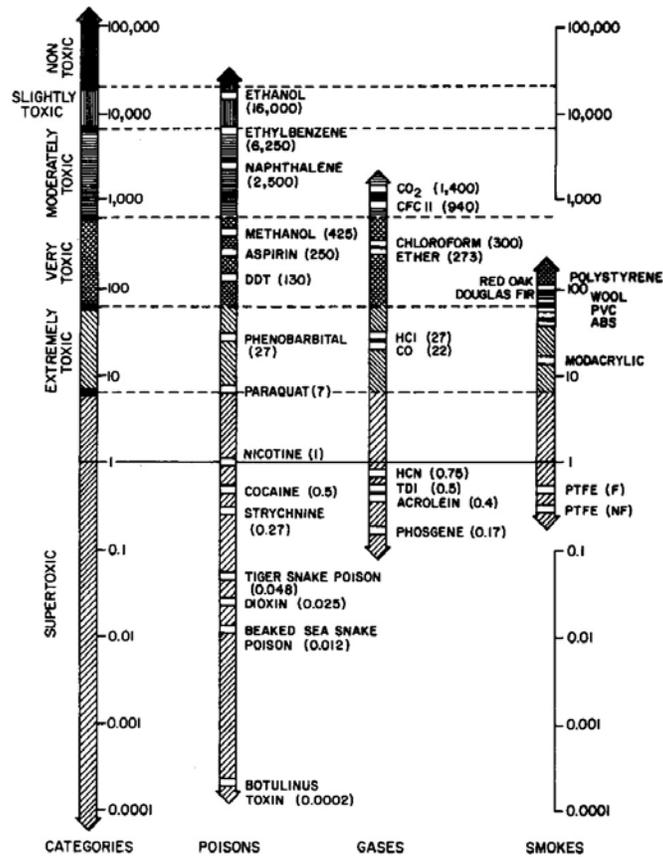


Figure 1: Toxicity Scales (inspired by Steven C. Packham)

FIRE HAZARD IMPLICATION

The analysis above indicates that all smoke is exceedingly toxic and fires can lead to lethality fairly rapidly if enough material burns. It also shows there is relatively little difference in values between the toxic potencies of some of the major gases in smoke, namely carbon monoxide (an asphyxiant gas formed by the combustion of all materials); hydrogen chloride (an irritant gas, and an acid gas, formed by the combustion of chlorine-containing materials); and hydrogen bromide (an irritant gas, and an acid gas, formed by the combustion of bromine-containing materials). However, some minor gases, including acrolein (a non-acidic irritant gas formed by the combustion of cellulosic materials, e.g. wood and paper, or polyolefins) or hydrogen cyanide (an asphyxiant gas formed by the combustion of nitrogen-containing materials) are significantly more toxic. This analysis also shows carbon monoxide is the major toxicant responsible for fire fatalities (and, in fact, no other toxicant needs to be searched for in post-flashover fires); and that hydrogen chloride from smoke cannot be identified in fire fatalities. Finally, this study again confirms that people will usually die in fires only after the fire has grown to be very big.

A conclusion to be drawn from this analysis is that fire hazard is directly related to the rate of heat release of the fire (i.e. the intensity of the fire) with other issues (such as smoke toxicity) being of much lesser relevance [37]. This is further enhanced by a study of fire hazard associated with several fire retarded and non fire retarded products [38]. In the study, the use of the fire retarded products was associated with an average 4-fold decrease in rate of heat release and led to a three-fold decrease in smoke

toxicity in the room, and to a 10-fold increase in tenability time (even though the ignition source used was more than three times as intense). Insufficiently low levels of flame retardants that do not make a substantial difference to the heat release rate of the final product do not affect toxic hazard [39]. This clearly shows that considerable improvements in smoke toxicity will be obtained by decreasing the heat release rate of the materials/products considered, almost irrespective of the actual toxic potency of the materials/products involved. Toxic hazard is a direct function of heat release rate, therefore, any measurement of toxic gases is of relatively little interest from the point of view of fire hazard.

WHEN SHOULD TOXIC GASES BE MEASURED?

As has been discussed above, it is rarely important to measure individual toxic gases for hazard assessment purposes. The measurement of toxic gases is mainly helpful in material development and in understanding the fire performance of any material or product, especially before bringing it to market. There are some very rare cases where smoke toxicity can have an important contribution in fires. That is the case when people die in very small fires (often smoldering) that destroy minimal amounts of material. Fortunately, statistics indicate that such fire cases are extremely rare and a more complete investigation may be needed for each individual case.

There is another reason to measure gases emitted during burning, and it is not related to fire hazard or to fire safety, but to the ability to sell materials. Many specifications and requirements demand that materials be certified as lacking in a certain component, typically halogen elements. Thus, measurement of combustion products is often associated with “passing” certain specifications for material composition.

ACIDITY AND SMOKE CORROSIVITY

The acidity of solutions formed from smoke in fires is a function of the release of acid gases, typically hydrogen chloride, hydrogen bromide and hydrogen fluoride, as well as organic acids, such as acetic acid or formic acid. Acid gas emission (often assessed by measuring the pH, i.e. acid gas concentration, or the conductivity of aqueous solutions) is often used as a synonym for smoke corrosivity.

That is, however, not fully correct as it has been shown that smoke corrosivity does not fully correlate with acid gas content of solutions.

* For example, it has been shown that solutions that are very alkaline (i.e. the opposite of acid on the pH scale) can be very corrosive [40], especially to copper (see Figure 2). The data in Figure 2 show that smoke from alkaline (nitrogen-containing) materials such as nylon and wool is significantly more corrosive to copper than smoke from halogenated materials (a standard PVC wire and cable compound, a low halogen PVC compound and a Neoprene compound). The data also shows a lack of correlation between the corrosion of the metal and the acidity of these three acid materials.

* In another example, it has been shown that soot deposits can cause bridging and corrosion of electrical and electronic circuit integrity [41], causing failures.

Thus, although high acidity is usually an indicator of some degree of propensity for corrosion, acidity does not correlate with smoke corrosivity. Moreover, most assessments of acidity are unable to take into account the effects of different realistic combustion conditions.

ACIDITY AND SMOKE TOXICITY

However, irrespective of the issue of whether acidity is a good surrogate for smoke corrosivity, suggestions have been made recently that acidity is a good surrogate for smoke toxicity. That is clearly

not a technically defensible position. If this were true, that would mean that alkalinity (and the smoke from some nitrogen-containing materials forms alkaline solutions) must equate a lack of smoke toxicity (or perhaps even a favorable effect!).

Copper Mass Loss from Corrosion Due to Warm Smoke

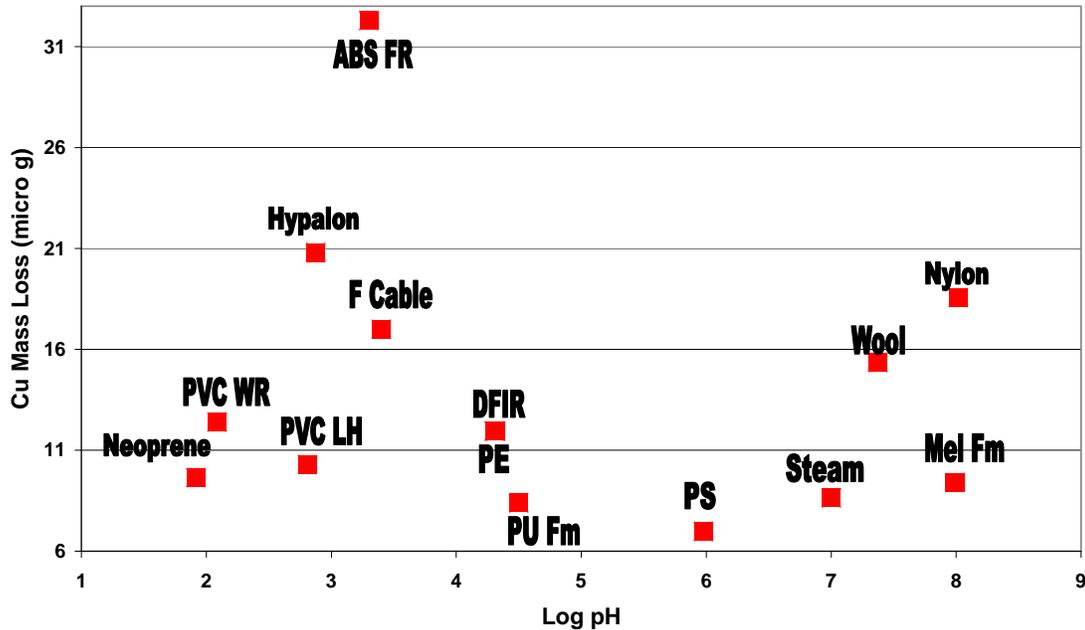


Figure 2: Exposure of Copper to Smoke from Various Materials [40]

As has been shown above, smoke toxicity is associated primarily with carbon monoxide, and secondarily with some other combustion products (including hydrogen chloride, hydrogen cyanide, acrolein, hydrogen bromide, hydrogen fluoride, carbon dioxide and low oxygen) while acidity is associated exclusively with the generation of acid gases. Of those gases listed above, only a few are acids, and two of the most prominent toxicants, carbon monoxide and acrolein, clearly are not. Thus, of course, any analysis of acidity will identify virtually only those materials that contain halogens in their composition (chlorine, fluorine, bromine). Therefore, it is worth pointing out some studies on one of the most widely studied polymeric materials, PVC.

- * NIST work [6] showed that the FED contribution of HCl in PVC smoke is negligible in full scale studies, although it is significant in all toxicity test studies. It also showed that the average gas concentration of CO tended to be of the same order as that of HCl in one toxicity test and , and 2.5-3 times higher in another toxicity test. This indicates that the relative toxic importance of HCl in PVC smoke would be, at most, comparable to that of CO, and in full scale fires, much smaller than that of CO.
- * Other work [20], which looked at 4 different PVC compounds and 3 test methods, found average HCl concentrations that were in most cases lower than those of CO. This again indicates that CO represents at least half the toxic load in PVC smoke.

These two examples alone show that the toxicity of the most notable material emitting acid gases cannot be directly related to the acidity of the resulting smoke. The same NIST work [6] also studied two other common combustibles: Douglas fir wood and a polyurethane foam. In both cases the yields of acid gas were negligible, and yet the toxic potency of the materials was broadly similar to that of PVC. Table 1 shows the results of a number of materials all using the same test method and it is clear that materials

that have little if any acid gases can exhibit similar levels of toxic potency as those that are highly acidic, like some of the PVC materials tested. A small difference in toxic potency, as discussed above, is not really an indication of a higher degree of fire hazard.

Table 1. ASTM E 1678 Radiant Toxicity Test Results, in mg/L [5]

Material	LC ₅₀	Material	LC ₅₀
ABS	17.8	PVC Jacket	53.1
Acrylic Fabric + Melamine Foam	9.6	PVC Low HCl	146.9
Ceiling tile	30.5	PVC Med HCl	86.2
Composite	20	PVC Profile	26
Cork	ca. 40	PVC Profile	20-30
Douglas Fir	100-200	Particle board	120-138
Douglas Fir	56	Rigid PU Foam	22
Flexible PU Foam	52	Rigid PU Foam	20-30
Melamine Foam	12.5	Vinyl Fabric	32
Nylon	36.7	Vinyl Fabric + Melamine Foam	26
Nylon Rug (Treated)	28.5	Vinyl Flooring + Plywood	82
Nylon Rug (Treated)	42.9	PVC FR Low Smoke 1	18.2
Nylon Rug (Untreated)	> 41	PVC FR Low Smoke 2	45.9
PVC Cable	36	PVC FR Low Smoke 3	35.8
PVC Insulation	33.4		
PVC Insulation	29.2		

Smoke Toxicity vs Acidity
(At Same Mass Loaded in Toxicity Test)

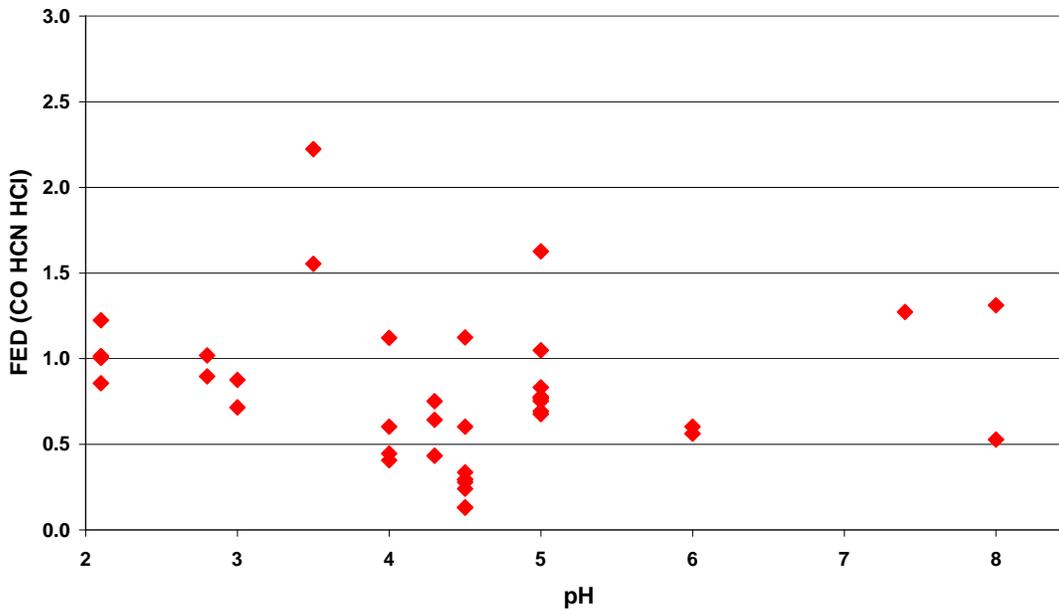


Figure 3: Comparison of Smoke Toxicity & Acidity for Various Materials

This work has clearly shown that an analysis of acidity cannot give a reasonable ranking or classification of materials or products in terms of smoke toxicity, since it will basically divide products among halogenated materials and non halogenated materials. As was shown above, this division is not a proper classification that is in any way related to smoke toxicity. It is even less associated with fire hazard.

Figure 3 shows a plot of a series of materials for which concentrations of CO, HCN and HCl were all measured in the same smoke toxicity test chamber (the one corresponding to the ASTM E 1678 test). The gas concentration data were taken from references 20, 38 and 42 and converted to a common mass of material loaded into the chamber: 40 mg/L. The acid gas data were taken from reference 40 and assessed by comparison for the materials not measured. The materials tested included halogen-free polyolefins, wood, styrenics and PVC. The Figure indicates that there is no correlation at all between smoke toxicity and acidity. The data also shows, just like Figure 1, that most polymeric materials have quite similar ranges of smoke toxicity, virtually irrespective of chemical composition.

IMPORTANCE OF ACIDITY

In summary, acidity is a poor representation of smoke corrosivity and is totally inadequate as a representation of smoke toxicity. In fact, even as a representation of smoke corrosivity, acidity simply looks at the “worst-case scenario” that can be associated with a material or product in that it does not consider differences in smoke emission or smoke decay (such as the well-known phenomenon of acid gas decay before transport through fire atmospheres [43]). Moreover, although smoke corrosivity (or acidity) may have some relevance to property damage it has no relation to life safety or even to smoke toxicity.

The use of acidity as the basis on which to assess toxicity of fire effluents may provide an illusion of life safety which is, in fact, incorrect, since the most common toxicant (carbon monoxide) is not taken into account.

CONCLUSION

As an overall conclusion, fire safety and fire hazard are associated primarily with the control of heat release and with ensuring that a fire remains small. On the other hand, smoke toxicity is, generally, no more than a small component of fire safety, except in some special circumstances. Therefore, fire safety is best served by monitoring and regulating heat release. Moreover, smoke toxicity is primarily identified with carbon monoxide, while several other gases are additional contributors: acrolein, hydrogen cyanide and hydrogen chloride primarily. Acidity (as an intended, albeit incomplete, surrogate for smoke corrosivity) may have potential utility as a property protection measure, but only when no thermal damage is to be expected. Acidity has no value as a monitor of improved life safety. Finally, acidity is totally unrelated to smoke toxicity and is not, in any way, to be looked at as a surrogate for smoke toxicity or as a fire hazard assessment measure.

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