# Estimating Data for Incapacitation of People by Fire Smoke

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**Abstract.** Fire hazard and risk analyses establish the basis for providing conditions of safety for people, including those that are more sensitive to fire smoke than others. For this purpose, this paper develops a method for estimating, from information on lethal and incapacitating exposures for rats, smoke toxic potency values for incapacitation of smoke-sensitive people. For those engineering applications where the mix of combustibles is unknown, generic values are derived of the concentration of smoke that would incapacitate smoke-sensitive people in 5 min: 6 g/m<sup>3</sup> for a well-ventilated fire and 3 g/m<sup>3</sup> for an underventilated (e.g., post-flashover) fire. These values are estimated with significant assumptions in their derivation, resulting in an estimated uncertainty of about a factor of two. Further, there is a wide range of smoke toxic potency values reported for various combustibles, and some of these will lead to values significantly higher or lower than these generic figures.

Key words: fire, smoke, smoke toxicity, incapacitation, lethality

#### 1. Introduction

A fire hazard or risk analysis of an occupancy establishes the basis for providing conditions of safety for the people within [1]. Since most fire fatalities result from smoke inhalation [2], a key consideration in such an analysis is the potential for people to be overcome by the toxic fire effluent. The estimation of the onset of life threatening conditions involves such factors as the toxic potency of the smoke to people, the rate at which smoke is generated and transported, the movement of people to and through the generated smoke, etc. The focus of this paper is on estimating the first of these factors.

When performing an analysis of the fire safety level of an occupancy, it is common practice to use published  $LC_{50}$  of  $IC_{50}$  values<sup>1</sup> for materials similar to those that might be involved in the fire. There are no reported measurements of the toxic potency to people of the smoke from burning materials or end products. There are, however, many published measurements of the lethal and incapacitating potencies of smoke for laboratory rats [3]. For those who are performing fire hazard analyses, it would be useful to be able to "convert" information from the database on the rat into estimates of the effects on people.

For fire hazard analysis, the most critical of these effects is incapacitation, defined in ISO/TS 13571 [4] as the inability to take effective action to accomplish one's own escape from a fire. As such, "incapacitation" encompasses a range of possible conditions, including unconsciousness, severe physically distress, or inability to detect where to go to escape. These different conditions will result from different degrees of exposure to fire smoke,

making difficult the identification of a single value for input into a hazard analysis. The common feature of these exposures is that they are all between those that cause death and those that make it difficult, but not impossible, to escape. The analysis in this paper encompasses the level of threat AEGL-2 developed by the team currently producing the Acute Exposure Guideline Levels (AEGLs) for Hazardous Substances [5]. AEGL-2 is defined as "the airborne concentration of a substance at or above which it is predicted that the general population, including susceptible individuals, could experience irreversible or other serious, long-lasting effects, or *an impaired ability to escape* (my italics)." Use of this definition enables use of their sizable effort in compiling toxicological data on people and test animals. However, their application is substantially different from this one, and most of their analysis is not pertinent to estimating the effects of exposure to fire smoke.

Fire hazard analyses rarely lead to fire safety provisions that intend to protect only half of the occupants (while laboratory measurements are nearly all for 50% survival of the test animals). Thus, were median toxic potency values used, some safety factor would be applied in order to "save" a larger fraction of the population, including many who are more sensitive to smoke than the average person. When multiple safety factors are included in a hazard analysis, they can lead to the exclusion of desirable building designs that in fact do provide the desired degree of personal safety. Thus, it would be useful if an estimation method for smoke toxic potency for people also included consideration of those people with more than the average smoke sensitivity, i.e., also led to an  $IC_{sens}$ , the concentration of smoke that leads to the incapacitation of normal and smoke-sensitive subjects exposed to that smoke for a specified time. The definition of "smoke-sensitive" must also be specified.

This paper develops such an estimation method. This entails approximations, and the information on which to base this extrapolation is far from definitive. The analysis is directed at obtaining order-of-magnitude factors and estimated uncertainties at the current state of an imperfect art. Nonetheless, it provides the practicing engineer with some basis for including smoke toxic potency in safety analyses.

#### 2. Assumptions

Fire smoke contains a mixture of narcotic and irritant gases, and incapacitation results from exposure to this combination. As shown in the prior paper in this volume [3], the incapacitating exposure to fire smoke for rats is about half the lethal exposure, and this factor of about two is similar whether dealing with pure narcotic gases or the complex mix of gases in the smoke from a burning material or end product. Thus, we assume that the factor of two holds for incapacitation from irritant gases. Since, as shown below, irritant gases account for a modest fraction of the cause of incapacitation, the results are not very sensitive to this assumption.

Second, we make the assumption that to extrapolate the toxic potency of the smoke from rats to the toxic potency for people, we can use a single asphyxiant gas as a surrogate for all asphyxiants and a single irritant gas as a surrogate for all irritants. Within these two groups, the gases are reasonably assumed to be additive, following Ref. 4. We choose CO and a strong halogen acid (HCl or HBr), respectively, because of their prevalence and because of the existence of draft AEGL compilations for CO and HCl [6, 7]. The toxic potencies of HCl and HBr are comparable [4]. Inhalation of carbon dioxide, which can increase the respiration rate and thus the ingestion of all toxicants, is handled separately.

Next, it is valuable to know what fraction of the smoke toxic potency is due to each of the two toxic components. This can be estimated from the relative concentrations of the two gases and their individual toxic potencies. In two room-scale studies involving the burning of a halogenated combustible, the CO concentration was significantly larger than the halogen acid concentration:

- Sheets of non-plasticized PVC (43% Cl mass fraction). The volume fraction of CO was about 1/6 that of CO<sub>2</sub>; the [CO]:[HCl] ratio was about 3 (accounting for some HCl losses) [8].
- A fire-retarded TV cabinet (about 12% bromine by mass) produced comparable volume fractions of CO and CO<sub>2</sub>. The volume fraction ratio of CO to HBr was about 10 [9].

Both of these items are toward the maximum of the halogen content in commercial products. Thus the ratio of CO concentration (or CO plus HCN, if the other principal asphyxiant gas is present) to halogen acid concentration from combustion of such products will generally be higher than these numbers. In reality, serious fires result from the burning of multiple items, many of which (e.g., wood items) are halogen-free. Thus, for the general fire that is capable of generating incapacitating smoke levels, a CO to halogen acid molar concentration ratio of 5 is a reasonable lower limit. Following the findings of Levin and co-workers that a small number of gases accounted for the lethality to laboratory rats within  $\pm 30\%$  for a variety of combustibles [e.g., [10]], we assume that the contribution of other irritants will be small relative to that of the halogen acids and that the contribution of other asphyxiants (e.g., HCN) will generally be small compared to the CO contribution.

In estimating an incapacitating toxic potency for people who are more sensitive to fire smoke than the "median" person, some definition of "smoke sensitive" is needed. Two examples that are often cited are adults with compromised cardiovascular function and small children. While these are by no means the totality of smoke sensitive people, inferred values for these groups will be used to indicate consideration of a fraction of the population distinctly greater than 0.5.

# 3. CO Toxicity

#### 3.1. Rat Lethality Data

Rounded rat LC<sub>50</sub> data assembled by the AEGL panel is compiled in Table 1.

#### 3.2. Human Data

Small children can be said to represent a smoke-sensitive but otherwise healthy subpopulation. As such, some showed post-exposure symptoms that would have impaired escape (had they occurred during the smoke exposure) at about 25% carboxyhaemoglobin

# TABLE 1Lethal Volume Fractions of CO for Rats for Various Timesof Exposure

Exposure time (min)	5	15	30	60	240
Volume fraction $\times 10^6$ (ppm by volume)	12000	8600	5000	4200	1800

(COHb) [11]. Using the Peterson-Stewart curves [12] and the input values for a 5-year-old child [6], this appears to result from, e.g., a 5 min exposure to about 0.0015 volume fraction (1500 ppm by volume), or one-eighth of the 5 min lethal exposure for rats.

Another smoke-sensitive sub-population is adult people with coronary artery disease. Here, the literature in the AEGL summary indicates that exposures resulting in about 5% COHb would lead to effects that (if they occurred during the smoke exposure) would seriously compromise evacuation. A similar calculation to the one for children indicates that this COHb level could result from a 5 min exposure to about 0.001 volume fraction (1000 ppm by volume), or about one twelfth the 5 min lethal exposure for rats.

Together, these estimates suggest using one tenth of the exposure lethal to rats in 5 minutes as the exposure that would incapacitate people in 5 min should provide protection for some fraction of the smoke-sensitive human population and thus over 50% of the total human population.

#### 3.3. Time Scaling of CO Exposure Data

Typically the interpolation/extrapolation from one set of exposure time data to other exposure times is done using an equation of the form:  $C^n$  t = constant. In the fire safety field, the exponent has most frequently been taken as unity [13]. For the CO data in Table 1, a value of n = 2 produces a reasonable fit (±20%) to the data. A similar dependence had previously been found for lethality due to CO potentiated by CO<sub>2</sub> [4]. Thus, the 5 min IC<sub>sens</sub> for people exposed to CO is about one fourth (one tenth × (30/5)<sup>1/2</sup>) of the 30 min rat LC<sub>50</sub>.

## 4. HCl Toxicity

#### 4.1. Baboon Exposure Information

Kaplan [15] exposed baboons, generally presumed to be a good surrogate for humans, to 0.019% volume fraction to 1.7% volume fraction (190 ppm to 17000 ppm by volume) of HCl for 5 min. All were able to escape, despite significant trauma at the higher concentrations.<sup>2</sup> In separate tests, exposure of anesthetized baboons to 0.5% volume fraction and 1.0% volume fraction (5,000 and 10,000 ppm by volume, respectively) for 15 min produced significant drops in arterial oxygen pressure [16]. (Such an effect was not observed in exposures to 0.05% volume fraction.) Hartzell notes [17] that, if combined with exposure to CO, this drop could lead to incapacitation at modest COHb levels. Data on combined exposures were not developed. Since there are no data for exposures between 0.05% volume fraction and 0.5% volume (500 and 5000 ppm by volume) and since the 15 min exposures are three times longer than those from which none of the animals were incapacitated, we suggest that the HCl concentration that could lead to incapacitation in 5 min in the presence of CO is about 0.3% volume fraction (3000 ppm by volume).

There are no citations for relating incapacitation of the "median" person to include the sensitive fraction of the human population. The AEGL draft report [7] uses a factor of 3 for this, saying that the irritation "is not expected to vary greatly between individuals." This leads to an estimate that the HCl concentration that could lead to incapacitation in 5 min of smoke-sensitive people in the presence of CO is about 0.1% volume fraction (1000 ppm by volume).

As noted above, in a fire involving chlorine-containing combustibles, the HCl concentration is likely to be at least five times lower than the CO concentration. Thus, when the CO concentration is ca. 0.15% volume fraction, the HCl concentration would be under about 0.03% volume fraction (300 ppm by volume). This is well under the incapacitating concentration for smoke-sensitive people.

#### 4.2. Time Scaling of HCl Exposure Data

There do not appear to be reliable primate data to enable time scaling. The AEGL-2 summary indicates that n = 1. The toxicologists associated with ISO TC92 SC3 (Fire Threat to People and the Environment) agreed that sensory irritancy was almost instantaneous and thus not time-dependent. The baboon exposures cited above indicated incapacitation did not occur for 5 min exposures to HCl volume fractions up to 1.7% (17000 ppm by volume). Thus, the result of Section 4.1 is conservative.

### 4.3. The Effect of $CO_2$

As noted above,  $CO_2$  increases the inhalation rate of all toxicants and is thus generally applied as a multiplier. ISO/TS 13571 shows the multiplier is unity for the  $CO_2$  concentrations in well-ventilated fires (<0.02 volume fraction). For the post-flashover conditions in references 7 and 8, in a location where the CO concentration is about at the incapacitating level of ca. 0.001 volume fraction (1000 ppm by volume), the  $CO_2$  concentration is also under 0.02 volume fraction. This too leads to a multiplier of unity.

# 5. Estimation of Incapacitation Values

- For materials and products that do not generate strong acid gases, we can assume that CO (as a surrogate for asphyxiants) is the primary toxicant and use one fourth the 30 min rat LC<sub>50</sub> as the 5 min human IC<sub>sens</sub>.
- For materials and products that do generate strong acid gases, narcotic gases account for the majority of the combined incapacitating effect of narcotic and irritant gases. Based on the relative generation rates of CO and HCl cited above, one could use one fifth of the 30 min rat LC<sub>50</sub> as the 5 min human IC<sub>sens</sub>.
- Since the narcotic component dominates the IC<sub>sens</sub> values, the use of C<sup>2</sup>t as a time scaling formula is preferred.

It is hard to affix an uncertainty to these conclusions given the lack of uncertainty in the resources for analysis of the AEGL information and the other assumptions stated above. An estimate is that they are accurate to within  $\pm 50\%$ .

In Ref. 3 the authors estimated that, for an unknown mixture of combustibles, a generic value for the concentration of smoke that would incapacitate a rat of average smoke sensitivity in 30 min would be 30 g/m<sup>3</sup>  $\pm$  20 g/m<sup>3</sup> for a well-ventilated flaming fire and 15 g/m<sup>3</sup>  $\pm$  3 g/m<sup>3</sup> for a post-flashover fire. Incorporating the analysis in this paper leads to corresponding values for the concentration of smoke that would incapacitate smoke-sensitive people in 5 min: 6 g/m<sup>3</sup> for a well-ventilated fire and 3 g/m<sup>3</sup> for an underventilated fire. The user of these values needs to be mindful of three key factors:

- There is a wide range of smoke toxic potency values reported for various materials. Some of these have significantly higher or lower values than these generic figures.
- These generic values are estimated with significant assumptions in their derivation. An estimated uncertainty is about a factor of two.

• People involved in fires are often excited and physically active. Both of these can increase the intake of the smoke-laden air and increase its toxic potency. At present, there is no quantitative literature on these effects or any way to apply them to the population as a whole.

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#### Notes

- The LC<sub>50</sub> is defined as the concentration of smoke that results in the death of half of the test subjects during a particular exposure time and often a post-exposure observation period. The IC<sub>50</sub> is a similar measure of incapacitation. Incapacitation generally is measured as an inability to move or react.
- 2. The AEGL panel used rat data over the baboon data because the latter exposures are for very short exposure times minutes and they needed information on exposures up to 8 hours—a big extrapolation. For this application, use of the baboon exposures is more appropriate.

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