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A Stochastic Model for CO Toxicity in Building Fires

FCRC Project 4
Fire Safety System Design Solutions
Part A – Core Model & Residential Buiidings

Fire Code Reform Research Program
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Background

The Fire Code Reform Research Program is funded by voluntary contributions from regulatory authorities, research organisations and industry participants.

Project 4 of the Program involved development of a Fundamental Model, incorporating fire-engineering, risk-assessment methodology and study of human behaviour in order to predict the performance of building fire safety system designs in terms of Expected Risk to Life (ERL) and Fire Cost Expectation (FCE). Part 1 of the project relates to Residential Buildings as defined in Classes 2 to 4 of the Building Code of Australia.

This Report was relevant to the project activities in support of the Model's development and it is published in order to disseminate the information it contains more widely to the building fire safety community.

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Comments

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A STOCHASTIC MODEL FOR CO TOXICITY IN BUILDING FIRES

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1 Introduction

It is by now a well established fact that the overwhelming hazardous toxicant in fires is carbon monoxide (CO) ([4]). The mechanism by which CO acts on mammals is by competing with oxygen for the hemoglobin in blood and tying it up as carboxyhemoglobin (COHb), rather than as the normal oxyhemoglobin. The hemoglobin fraction tied up as COHb is normally expressed as percent COHb (which means the percentage of the total hemoglobin present as COHb). We shall denote it for short by COHb. Lack of oxyhemoglobin leads to hypoxia (a reduction of the amount of oxygen supplied to the tissues of the body) leading to eventual death by asphyxiation.

The hemoglobin fraction is determined by the amount of carbon monoxide inhaled. The exact relationship will be discussed later in the paper.

Traditional wisdom has set a value of 50% COHb as the threshold level for potential human lethality.([1, 6]), in the sense that a COHb of more than 50% will inevitably lead to death and that if a fatality is autopsied and its COHb is less than 50%, CO poisoning cannot be the sole source of death.

However, Debanne et *al* ([4]),after an exhaustive study of available literature, have come to the following conclusions:

1. There is generally no need to look for an additional source of lethality in fire atmospheres if the COHb is above 20%.

2. The population of fire fatalities contains more individuals that are at a high risk of succumbing to carbon monoxide poisoning (namely the very young and the elderly) than does the population as a whole.

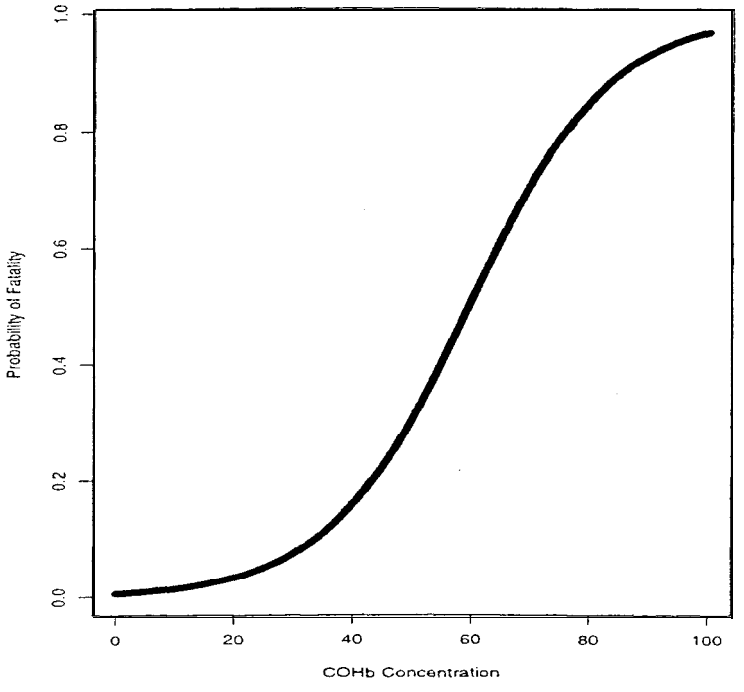
A typical example of the distribution of the COHb in fire fatalities with COHb above 20% is given in Table 1. It is derived from a data base compiled by the University of Southern Mississippi ([4]). The number of fatalities was 961.

It is clear that there does not exist a unique COHb threshold of lethality. Rather, the response of the population exposed to CO can be represented by what is known as a

COHb%	0-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-100
%population	0	6.5	6.5	11	17	17	23	16	5

Table 1: COHb Distribution in Fire Fatalities.

Figure 1: Typical bioassay curve.



bioassay curve, which represents the fraction of the population which dies when the COHb percentage reaches various levels. The bioassay curve can be thought of as the cumulative proportion of deaths when a very large population is exposed to increasing COHb levels.

A typical shape for a bioassay curve is given by Figure 1.

It is customary to assume for the bioassay curve an equation of the form

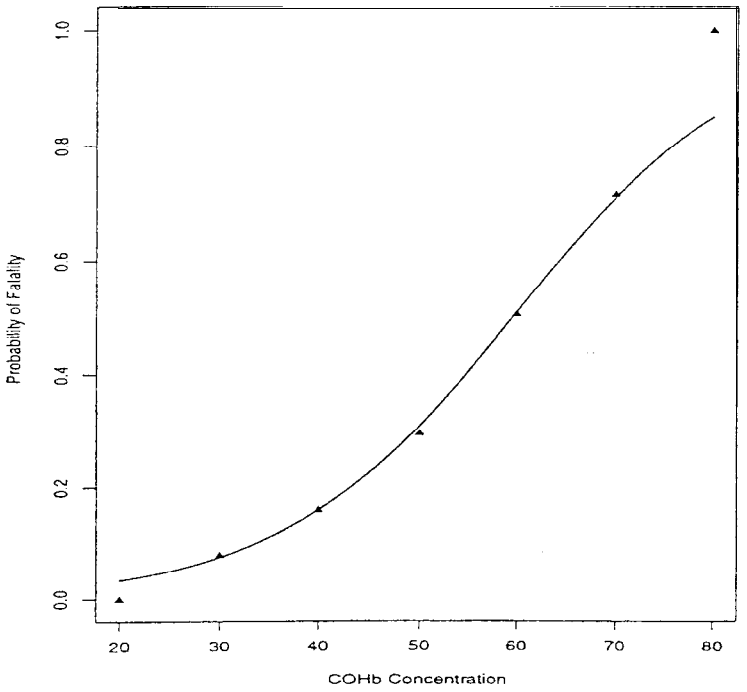
$$A(x) = \frac{e^{a+bx}}{1+e^{a+bx}}. \tag{1}$$

It is known as the logistic curve.

An important parameter of the bioassay curve is the value at which 50% of the population is dead. This is known as the $LC50$ value and is equal to -a/h for the logistic curve.

The difficulty with the data of Table 1 is that it gives the number of fatalities in various ranges of COHb percentage, but does not provide the total number of building occupants who ended up with the various ranges of COHb, only the number who did die. The total number is required in order to estimate the bioassay curve. An extensive search of the literature failed to locate any such data. In fact, we were unable to find a bioassay curve for COHb even for experimental animals, let alone humans. And in any case, such a curve would be quite useless, since what is needed is a bioassay curve which would reflect the

Figure 2: Logistic Curve fitted to Fatality Data.



However, the definition of l_i has to be modified for $p_i = 0$ *or* 1.
$l_i = \log\{\frac{1}{2}/(n - \frac{1}{2})\}$ when $p_i = 0$ with a complementary definition for $p_i = 1$.

Applying the methodology just described to the data of Table 2 we find the following estimates for a and b:

$$\hat{a} = -5.070 \quad (5)$$
$$\hat{b} = 0.085. \quad (6)$$

The corresponding logistic curve is plotted in Figure 2 together with the observed cumulative proportions.

3 Clinical presentation

Clinical observations show a fairly consistent relation between the COHb level and physical symptoms, as described in Table 3.(Kimmerle [5]).

Keeping in mind that the clinical observations refer to healthy adults; and that the population of fire fatalities contains, as noted above, a high proportion of the very young and the elderly, who are far more sensitive to CO poisoning, it appears that the proposed bioassay curve is consistent with the above table of clinical symptoms.

COHb%	20	30	40	50	60	70	80
Cum. Prop.	0	0.0 80	0.160	0.296	0.506	0.716	1

Table 2: Cumulative proportion of fatalities versus COHb.

-composition of a typical population of building occupants, including the very young and the elderly, as mentioned above. Such a population would probably be very different from an experimental population.

The best we can do at present is to make an educated guess at the number of occupants in each range of COHb. In the first place it seems reasonable to assume that large fires are likely to lead to inhalation of larger amounts of CO, and therefore to a higher COHb. It is a well established fact that large fires are less frequent than small fires. On the other hand, the number of people exposed to CO inhalation in large fires is greater than in small fires. Since these two factors operate in opposite directions, it may not be far from the truth to assume that there are approximately equal numbers of occupants in each COHb range.

A possible way of improving our knowledge in this area would be to carry out computer simulations of various sizes of fires and to use available statistical knowledge about the frequency of these fires and about the typical number and location of occupants to evaluate the number of occupants who would reach the various ranges of COHb.

One must also take into account known clinical results (discussed later in detail) which indicate that COHb values above 80% are lethal for all humans. So in attempting to estimate the bioassay curve, we shall consider only fatalities in the COHb range 20 – SO%, with a total population of 560. The modified cumulative proportions are given in Table 2.

2 Estimation of the parameters of the logistic curve.

Estimation of the parameters of the logistic curve has been extensively studied and the recommended procedure (Berkson [2] is *as* follows:

Let x_i be the upper limits of the COHb ranges and let p_i be the cumulative proportions of fatalities. Let n be the population size and let $q_i = 1 - p_i$. Let $l_i = \log(p_i/q_i)$. l_i is known as the "logit" of the proportion and can be thought of as the observed value of a random variable L_i. It is easy to see that, asymptotically, for large n,

$$\sim a + b\,x \tag{2}$$

$$\mathbf{Var}(L_i) \sim \frac{1}{np_iq_i.} \tag{3}$$

'Thus a and *b* can be consistently estimated by weighted least squares, minimizing

$$\sum np_iq_i(l_i - a - bx)^2. \tag{4}$$

COHb	Symptoms
O-10	None
10-20	Tension in forehead, dilatation of skin vessels
20-30	Headaches, pulsation in sides of head
30-40	Severe headache, ennui, dizziness, weakening of eyesight; nausea, vomiting, prostration.
40-50	Same as above,increasing in breathing rate and pulse, asphyxiation, prostration.
50-60	Same as above, coma, convulsions, Cheyne-Stokes respiration.
60-70	Coma, convulsions, weak respiration and pulse, death possible.
70-80	Slowing and stopping of respiration, death within hours.
SO-90	Death in less than an hour.
90-100	Death within a few minutes.

Table 3: COHb and physical symptoms.

4 Calculation of the COHb level from CO inhalation.

As mentioned above, the COHb level is determined by the amount of CO inhaled.The most accurate predictor of the COHb level in humans is the Coburn-Forster-Kane equation (CFK for short) (Coburn et *al* [3]). However, the CFK equation requires as inputs a whole range of parameters, including the volume of air breathed per minute (RMV), body size, exposure duration and parameters related to lung and blood physiology. The complexity of the CFK equation makes it unsuitable for use in a fire risk model, and we propose to use a simpler equation proposed by Stewart *et al* [7]. This equation has the further advantage that it lends itself to the determination of the eventual COHb level under exposure to varying CO concentrations. The equation reads

$$\%COHb = \int_0^t 3.317 \; x \; 10^{-5} \; x \; CO(u)^{1.036} \; x \; RMV \; x \; du \qquad (7)$$

where $CO(u)$ is the CO concentration in ppm in the inhaled air at time u. RMV is the volume of air breathed per unit time (L/min) and $(0, t)$ is the exposure interval of time.

Compared to the CFK equation, equation (7) gives very good results for high CO concentrations and conservative results for low CO concentrations. Since equation (7) is much simpler than the CFK equation, it is recommended for use in the fire risk model.

It must be pointed out, however, that there still remains a great amount of uncertainty in the derivation of the COHb level, due to the fact that the parameter RMV can vary within wide limits. Purser [6] reports that for a 70 kg human at rest the RMV is approximately 8. j L/min, while for slow running or walking up a 17% gradient the RMV can reach *50* L/min. The situation is further complicated by the fact that children will take up CO much more rapidly than adults, and that inhalation of small concentrations of carbon dioxide will stimulate breathing, so that, for example, at 3% CO_2 the RMV is approximately doubled.

5 Evaluation of expected number of fatalities.

The bioassay curve derived above can be used to evaluate the expected number of fatalities due to CO poisoning in a particular fire scenario, as follows:

Let there be n occupants in the building and let their COHb levels by the time they are rescued, or the fire has died down, be $x_1, \ldots, x_n$. The probability of death before the COHb level reaches x is p = $A(x)$, where $A(x)$ is given by equation (1). Thus the contribution of an occupant whose ultimate COHb level is x to the expected number of fatalities is 1 x p + 0 x (1 − p) = p. The expected number of fatalities N among the n occupants in the particular fire scenario will therefore be given by:

$$N = \sum_{i=1}^{n} A(x_i). \tag{8}$$

The global expected number of fatalities will be obtained by averaging N over all considered scenarios.

6 Incapacitation.

Incapacitation is expected to occur at lower levels of COHb than death. There do not seem to be statistical data for incapacitation in fires, but it appears from perusal of Table 3 that incapacitation occurs roughly at a COHb level 20% lower than the lethal COHb level This suggests that the bioassay curve relating to incapacitation can be taken to be a logistic curve with parameters a = -3.3 and b = 0.085.

Exact calculation of the mean time to incapacitation is difficult because it depends on the exact dependence of the COHb level on time. However: in view of the other uncertainties of the model, it appears to be sufficient to take it as the time at which the COHb level reaches the $LC50$ value; namely 3.3/0.085 = 39%.

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