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Analysis of toxic load calculations and fluctuation concentrations modeling for the assessment of atmospheric accidental release

ABSTRACT

Computing the effect zones around industrial facilities requires using modelling tools. For the specific case of accidental scenarios involving toxic cloud dispersion, it is necessary to make use of acute toxicity threshold values. Those thresholds can be given in terms of concentration or in terms of toxic load. The toxic load is commonly evaluated by extensions of the Haber's law that corresponds to an integration in time of concentration. The improvements achieved in the atmospheric dispersion modelling now enable to predict the intermittency of the toxic cloud. However, such new approaches raise questions about the relevance of the toxic load evaluation, knowing that many parameters which characterize biological reality and the whole process of the dose-response should be taken into account. To achieve a deterministic model, it is necessary to pool research efforts between the communities of toxicologists and the atmospheric dispersion modellers.

This work consists in confronting the level of knowledge of the fluctuation toxic cloud and biological parameters in the context of the deterministic modelling of consequences generated by an accidental toxic cloud.

CONTEXT AND OBJECTIVES

For the specific case of accidental scenarios involving toxic cloud dispersion, characterizing the effect zones around industrial facilities requires using modelling tools and make use of acute toxicity threshold values. A common practice consists in calculating toxic effects with the total inhaled dose. This total dose is estimated by integrating the concentration evaluated by atmospheric dispersion modelling.

The toxic load is commonly evaluated by extensions of the Haber's law, that corresponds to an integration in time of concentration, intended to account for this effect by using an exponent (example : n > 2 for ammonia) to the concentration inhaled. There are several extensions of the toxic load model that have been proposed to take into account that a toxic cloud is always intermittent in the real life. Indeed the cloud concentration is always varying due to many phenomena: atmospheric turbulence, momentum of the release, etc.

A lot of scientific efforts have been produced to better assess fluctuations in the concentrations; however the inhaled total dose is estimated without taking into account physiological process that modify the accumulation process (Hilderman, 1999) of the total dose. In the same time there is a lack of knowledge to assess physiological response for very short time, i.e. less than 10 minutes. Therefore, difficulties exist in regulatory guidelines for very short time and for some chemical to determine load toxic thresholds.

The objective of this work is to estimate by a simple kinetic model, on the basis of concentration time series example, the effective arterial blood concentration that is expected to exceed a toxicological value. For each of this external concentration signal, the arterial blood concentration is calculated and compared to the safe target arterial blood concentration. This comparison allowed analysing effects of an external atmospheric concentration on the dynamic of the effective arterial blood concentration.

MATERIAL AND METHOD

In order to illustrate intermittency impacts, we choose a Halocarbon-extinguish agent (2,2-dichloro-1,1,1-trifluoroethane or HCFC-123) which is a gaseous compound under normal aircraft operating conditions. Halocarbons are relatively non toxic at recommended use concentrations (Tabscott and Speitel, 2002). No observable adverse effect level (NOAEL) and the lowest observable adverse effect level (LOAEL) are given in the Table 1 for 2,2-dichloro-1,1,1-trifluoroethane.

			Maximum safe 5-min human exposure concentration		Target arterial concentration, B_{safe} (mg/L)
Agent	NOAEL (%v/v)	LOAEL (%v/v)	(%v/v)	(mg/L)	(mg/L)
HCFC-123	1.0	2.0	1.28	78.9	69.9

Table 1: NOAEL and LOAEL concentrations, maximum safe 5-min human exposure concentrations, and target arterial concentration for HCFC-123.

DESCRIPTION OF KINETIC MODEL

Toxicokinetics (TK) is the quantitative study of factors that control the fate of chemicals within the body (Reddy et al., 2005). Toxicokinetic models provide a set of equations that simulate the time courses of chemicals and theirs metabolites in various tissues throughout the body. The internal dose results from all toxicokinetic processes that occur in the body, divided into the processes of absorption, distribution, metabolism and elimination (see Figure 1).

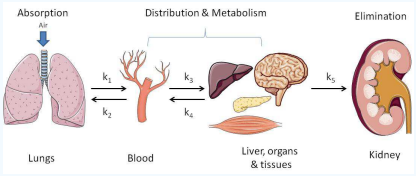


Figure 1 : Kinetic model of halocarbon transport in humans

The toxicokinetic model proposed by Lyons & Speitel (2010) predicts the blood concentration histories of human exposed to time-varying concentration of gaseous halocarbon fire-extinguishing agents such as HCFC-123. This model has been developed in the context of short term exposure (0-5 minutes). In the present work, this model has been used to predict the arterial concentration of HCFC-123 with different scenario of exposure. MCSim 5.0.0 (Bois and Maszle, 1997) has been used to implement the toxicokinetic model and to perform simulations.

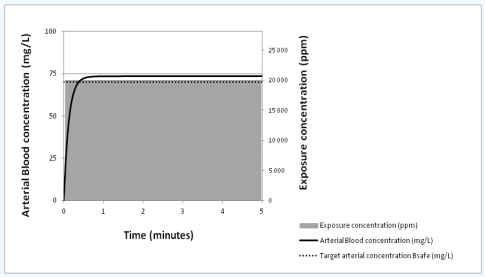
In order to compare exposure concentrations to an internal level, a target arterial blood concentration at which cardiac sensitization occurs for a group of dogs exposed to the LOAEL (20 000 ppm exposure) for 5 minutes has been determined (Lyon & Speitel, 2010). The target arterial blood concentration was evaluated at 69.9 mg/L (see Table 1). This target arterial concentration has been shown to be the same for dogs and human (Vinegar et al., 1998) and provides the link for predicting safe 5 minutes human exposure concentration (Lyon & Speitel, 2010).

RESULTS AND DISCUSSION

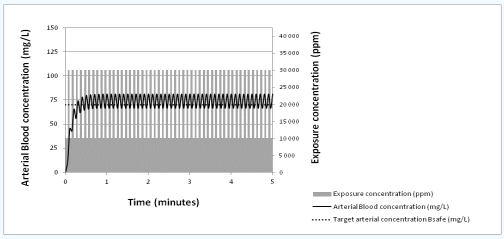
Three time series of external concentrations were taken as input of the toxicokinetic model. These time series correspond to three scenarios of atmospheric concentration exposure with the same mean concentration of 20 000 ppm exposure, corresponding to the LOAEL concentration, and the same toxic load, D(x), corresponding to a non-steady time varying concentration :

$$D(x) = \int_0^T C(x,t)dt$$

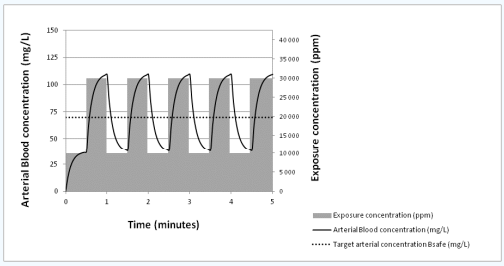
First condition exposure : steady state exposure (LOAEL) of 20000 ppm



Second condition exposure : repeated exposure to 10 000 ppm during 3 s with exposures to 30 000 ppm during 3 s



Third condition exposure : 10 000 ppm during 30s then 30 000 ppm during 30 s



Although the mean external concentration and external dose are equivalent for the three scenarios, we observed that the peaks of arterial blood concentration exceed to 5%, 20% and 50% the target arterial concentration respectively. This result can be explained by the influence of the peak exposure time which differs according the three scenarios.

These preliminary and academic results emphasize the influence of the peak exposure time on the arterial blood concentration for short total exposure times and the need to correctly asses it by atmospheric modelling.

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