Journal of Hazardous Materials, 9 (1984) 235—240
Elsevier Science Publishers B.V., Amsterdam — Printed in The Netherlands

Short Communication

AN ASSESSMENT OF THE EFFECTS OF FLUCTUATIONS ON THE SEVERITY OF POISONING BY TOXIC VAPOURS

D.J. RIDE

Chemical Defence Establishment, Porton Down, Salisbury (Great Britain)

(Received February 21, 1984; accepted March 12, 1984)

It has long been the practice to relate the degree of poisoning of an average individual through inhaling toxic vapours to the total exposure to poison. Thus, if the average concentration of vapour in the individual's atmospheric environment is $\bar{\chi}$ and he is exposed to it for a time T then the symptoms of toxicity are judged to correlate with the product $\bar{\chi}T$. At least some acknowledgement is made of the tendency of vapour concentrations to vary with time in the free atmosphere by defining the term "dosage" as $\int_0^T \chi(t)\,dt$, which is a more generally expressed form of the quantity of available poison.

Implicit in the above approach is the concept that the severity of the symptoms of poisoning is independent of the length of time of exposure, which ignores the facts that breathing rates may not remain constant and bodily mechanisms of detoxification exist. The inadequacy of the concept of dosage is well illustrated by the case of hydrogen cyanide (HCN) vapour: the body can effectively detoxify relatively low, continuously administered quantities of inhaled HCN, but high concentrations provoke rapid and deep cycles of inhalation leading to the acquisition of a total quantity of poison greatly in excess of that indicated by the dosage. Research on war gases by the Japanese [1] demonstrated the effectiveness of increasing the concentration of HCN while retaining constant dosage levels. This was achieved by submitting animals to various intermittent concentrations for a constant period. In general the higher concentrations (and by implication, those with the greatest intermittency) produced the most severe toxic effects.

Aim

The purpose of this short communication is to demonstrate that dosage calculations and the neglect of naturally induced fluctuations in the atmospheric concentrations of some toxic vapours lead to an underestimate of their potential hazard.

Method

Recognition of the inability of dosage values properly to account for the — often competing — physiological processes which occur during intoxica-

tion has prompted consideration of a quantity, here called the "dosement", where the generally exacerbating effects of higher concentrations is modelled by weighting the concentration with an exponent n, $n > 1$. Thus,

$$\text{dosement} = \int_0^T \chi^n(t)\, dt$$

Values of n are considered to be constant for given substances. The value of n for HCN which produces the best correlation with toxic effects is given by Ballantyne [2] as 1.8. Griffiths and Megson [3] have conducted exhaustive searches of the literature for values of n appropriate for chlorine and ammonia. They were interested in producing probit relationships of the form

$$Pr = a + b \ln(\chi^n T)$$

where Pr is a measure related to the percentage of a population suffering a given level of poisoning effects. They discovered published values for n of 2.0 and 2.75 for ammonia and 2.75 for chlorine. They included the effects of intermittency, γ, defined here as the fraction of time during which concentrations fall below a measured level, by means of a simple mass conservation relationship substituted in the probit equation:

$$Pr = a + b \ln\left[\left(\frac{1}{1-\gamma}\right)^{n-1} \bar{\chi}^n T\right]$$

Using the published values of n they consider the changes in Pr arising from postulated values of γ. Thus, an LCt(5) value* for ammonia with no intermittency becomes an LCt(90) when γ is 0.7, for one of the probit relationships quoted in Ref. [3].

Further substantiation of this effect may be obtained from calculations performed on a model of concentration fluctuations due to Ride [4]. Ride models the fluctuations by considering the passage of an idealised cloud composed of clean air in which are suspended, in a uniformly random manner, identical spheres of air contaminated to the same level. Relationships necessary to define a two-parameter probability density function are derived from the model and evaluated for experimental data given by Jones [5]. A subsidiary but important feature of the model is the way in which measurements of cloud characteristics taken by instruments with one time period may be related to other responders, including the human lung, with different periods.

Following Ride, the peak concentration, χ_τ, observed for a time resolution of τ may be written

$$\chi_\tau = \bar{\chi}\,[k(\sigma_\tau/\bar{\chi})^\lambda + 1]$$

where $\sigma_\tau/\bar{\chi}$ is the intensity of fluctuations measured with a response time of τ, and k and λ are experimentally determined constants which parameterise

*The LCt(x) is the value of dosage which it is estimated would produce x% of deaths in a population exposed to it.

the combined effects of the cloud structure and its response to atmospheric turbulence and transport. Similarly

$$1 - \gamma_\tau = [k(\sigma_\tau/\bar{\chi})^\lambda + 1]^{-1}$$

where γ_τ is the intermittency observed with a response time of τ. Thus

$$\int_0^T \chi^n(t)\,dt = \chi_\tau^n\,(1 - \gamma_\tau)\,T = \bar{\chi}^n T\,[k(\sigma_\tau/\bar{\chi})^\lambda + 1]^{n-1}$$

That is to say, the dosement with a fluctuating concentration is equal to the dosement assuming a steady, average concentration enhanced by a factor which increases as the intensity of the fluctuations increases. This factor will never be less than unity whatever the experimentally derived values of k and λ.

Results

For the data of Jones, $k = 11$ and $\lambda = 1.5$. These values have been employed to compute the enhancing factor for postulated values of $\sigma_\tau/\bar{\chi}$, using values of n appropriate for hydrogen cyanide, ammonia and chlorine, and with $\tau = 1$ s, an assumed value for the human lung. The results are shown in Table 1 below. Values of $\sigma_\tau/\bar{\chi}$ for Jones' data are between about 1 and 2.

TABLE 1

Enhancing factors calculated for some different intensities of fluctuation in concentration

Intensity of fluctuations	Enhancing factors		
	HCN, $n = 1.8$	Ammonia, $n = 2.0$	Chlorine, $n = 2.75$
0	1.0	1.0	1.0
1	7.2	12.0	77.4
2	16.1	32.1	433.2
5	47.3	124.0	4607.0

Discussion

The LCt values derived by Griffiths and Megson for ammonia, quoted earlier, and the factors in Table 1 both show dramatic increases in toxicity produced by fluctuations. The absolute values of the factors appear unrealistic for high intensities of fluctuation, and they are extremely sensitive to the precise value chosen for n. The reason lies with the definition of dosement. The values of mean concentration covered by these figures may span several orders of magnitude; it is unlikely that n retains a constant value or that the form of weighting sufficiently models the toxic process over this range. For

instance, breathing rates cannot increase indefinitely in response to higher concentrations. This fact alone precludes the figures from posssessing any absolute validity; however, they do indicate trends around those concentrations for which the values of n were determined. These trends are valid in showing that, for given values of concentration, toxic effects increase when the intensity of concentration fluctuations increases.

Even in the absence of reliable quantitative estimates, this last point is of great importance for the assessment of the hazard area created by a toxic cloud in the free atmosphere. First, all such clouds possess discontinuities and irregularities so that the downwind safety distances calculated from average values of concentration (e.g., from Gaussian models) must be increased to account for the presence of fluctuations. Secondly, the increase in the intensity of fluctuations as the distance from the centroid of the cloud increases (noted by many workers, e.g., Ramsdell and Hinds [6]) will to some extent compensate for the decrease in average concentration. This means that a given toxic effect will extend over a broader area within the cloud than is indicated from the width of the dosage contours. This effect is of particular importance in the assessment of the hazard area created by multiple sources, such as might occur in a major hazards accident. The source pattern will be random in nature and the cloud travel only partially correlated so that the concentration contour pattern will be complex. However, the increase in the intensity of fluctuations at the cloud edges which is a normal characteristic will be prolonged by the existence of overlapping clouds. The net effect will be a tendency for casualty contours to form far simplier patterns than the complex concentration contour pattern may suggest. Two conclusions emerge from this argument: effects of toxic releases tend to consistancy and hence to predictiveness; and simulation models often need not be too complex.

The level of need for complexity may be illustrated by the types of multiple-source models discussed by Calder [7]. Perfect correlation is assumed for the plume travel in these, so that the sole source of variance in the dosage pattern for a given combination of wind speed and atmospheric stability arises from the range of possible spatial distributions of the sources specified with respect to all relevant wind directions. In practice, even for a fixed disposition of sources, the magnitude of this variance is likely to be much greater than any additional variance introduced by relaxing the correlation of plume travel. In view of the enhancing effect of fluctuations on casualties and the spatial *reduction* of their variances, discussed above, it does not appear profitable to introduce realistic plume behaviour alone into the program, a conclusion further reinforced when the uncertainties in the parameters of the location and conditions of release are considered.

Two general philosophical points concerning the scope and validity of modelling are raised by this discussion. First, the pitfalls of combining two simple models — each of which is adequate over its range of application but which together produce inconsistencies by the interactions of the extreme

ends of the ranges — is exemplified by the instances where the dosement model is made to react with the probit model and the cloud fluctuation model, respectively. Secondly, the present inability to model adequately the toxic response to fluctuating concentrations for materials of the type discussed above demonstrates the impropriety of modelling more than the dominant physical features unless all the features are equally well understood, as is shown by the multiple-source model.

Conclusions

(a) Experimental results and trends in Table 1 demonstrate clearly that dosage calculations will result in serious underestimates of the severity of toxic effects and the size of the hazard area produced by atmospheric releases of toxic vapours with non-linear effects.
(b) While the dosement correlates more highly than the dosage with toxic effects in a population in steady concentrations, its unconstrained combination with other simple models may result in unrealistic numeric estimates of potential casualities.
(c) No model yet proposed gives other than relative trends of effective toxicity when toxic substances with non-linear effects are administered as time-varying vapour concentrations.
(d) It is pointless, from the point of view of quantitative risk assessment, to model physical processes which are not dominant contributors to estimates of the range of toxic casualties until a better understanding is achieved of the way in which concentration fluctuations enhance a substance's toxicity.

Acknowledgement

The work on which this paper is based was conducted under the supervision of Dr. P.C. Chatwin of the University of Liverpool who has provided valuable comments and suggestions.

References

1 British Intelligence Objectives Sub-Committee. Intelligence report Japanese Chemical Warfare, Volum 2, Appendix A: Fundamental research on effectiveness of toxic gases. HMSO Report BIOS/JAP/PR/394 (1946).
2 B. Ballantyne and P. Schwabe (Eds.), Respiratory Protection, Chapman and Hall, London, 1981.
3 R.F. Griffiths and L.C. Megson, The effect of uncertainties in human toxic response on hazard range estimation for ammonia and chlorine, Atmos. Environ., 18 (6) (1984) 1195—1206.

4 D.J. Ride, A probabilistic model for dosage. Proceedings of the IUTAM Symposium on Atmospheric Dispersion of Heavy Gases and Small Particles, Delft University, Delft, The Netherlands, 1983.
5 C.D. Jones, On the structure of intstantaneous plumes in the atmosphere, J. Hazardous Materials, 7 (1983) 87—112.
6 J.V. Ramsdell and W.T. Hinds, Concentration fluctuations and peak-to-mean concentration ratios in plumes from a ground-level continuous point source, Atmos. Environ., 5 (1971) 483—495.
7 K.L. Calder, Multiple-source plume models of urban air pollution — Their general structure, Atmos. Environ., 11 (1977) 403—414.