NOW OR LATER? A NUMERICAL COMPARISON OF SHORT AND LONG-TERM HAZARDS

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For fifteen years hazard analysis has been used in the chemical industry for comparing the risks to employees from various acute hazards: that is, accidents that produce immediate effects, so that we can decide which of these hazards should be dealt with first. Chronic industrial hazards, those that take a long time to produce their effects, have not been considered in the same way. The author, a Professor at the Department of Chemical Engineering, Loughborough University of Technology, Loughborough, makes an attempt to compare the risks to the lives of employees from the two sorts of hazard. Results suggest that the allocation of resources between the two types of hazard is probably not out by more than an order of magnitude.

n the Loss Prevention Symposium held at Newcastle n 197[illegible] presented one of the first papers[1] on the use of numerical methods for comparing the different risks to which employees in the process industries are exposed. Since then such methods have been used for setting priorities between different acute risks and the literature on the subject is now extensive[2-10]. In this paper I suggest an extension of these methods to hazards which take a long time, typically several decades, to produce their effects.

Consider a substance X, the product of an industrial process, which can cause harm in two distinct ways:

(A) It may leak out of the plant, vaporise, mix with air and be ignited, thus injuring or killing people by fire or explosion. Very small leaks do not matter; the hazard arises only if the leak exceeds several kg and is unlikely to be serious unless it exceeds 1 tonne (though smaller leaks can be serious in confined spaces).

(B) Exposure of employees to small quantities of the vapor for long periods for many years may cause industrial disease which may lead to premature death. In general, we are not concerned with the occasional large release but with the small quantities present in the atmosphere as the result of minute leaks from joints, glands, sample and drain points, maintenance operations and so on. (However, for some materials occasional large doses may produce long-term effect, or may cause sensitisation). The Threshold Limit Value (TLV) gives the time-weighted average concentration for a normal 8-hour workday or 40-hour workweek, to which nearly all workers may be repeatedly exposed, day after day, without adverse effect. It does not, of course, provide a sharp division between safe and unsafe conditions.

We try, of course, to prevent both sorts of leak and we spend a lot of money and effort in doing so. How does our success in overcoming hazard (A) compare with our success in overcoming hazard (B)? We do not know. Should we put more effort into preventing the occasional big leaks or more effort into preventing the continuous small leaks? We do not know. Usually different people, using different criteria and financed by different budgets are responsible for dealing with the two hazards. Finding a way of talking to them both at the same time is like finding a way of communicating, at the same time, to people who speak different languages. This is attempted in this paper.

The problem is not just one of comparing risks. It is made more difficult by the fact that the risks are different and are felt to be different by those who are subjected to them. Suppose that the probabilities of an employee being killed by the acute hazard (A) and the chronic or long-term hazard (B) are equal. An employee might feel that hazard (B) leaves him with an extra 20 or more years of life and therefore further resources should be spent on reducing the risk from hazard (A). On the other hand another employee might feel that a fire or explosion is soon over while industrial disease may mean years of worry, wondering whether or not he will contract it, possibly followed by many years of illness and reduced quality of life.

From society's point of view, though they may kill many people, long-term hazards will kill them over a long period of time and will not produce the same trauma or public outcry as one that kills many people at a time.

In one important respect, the problem of long-term effects differs from all other industrial problems—often the size of the problem is not known. As we shall see later, we do not know how many people die or suffer from industrial disease (of all sorts, not just the legally prescribed diseases), because the same diseases usually also have non-occupational causes. Before we spend resources on, say, reducing the usage of raw materials or energy, improving product quality, or preventing accidents, we start by asking ourselves what is the present usage, quality, accident rate, etc., and what is the scope for improvement. When dealing with toxicological hazards we not seem able to do this.

A Personal Note

I have some experience of industrial accidents but little knowledge of toxicology. In trying to compare the two I may be accused, like everyone else who tries to compare two subjects normally considered apart, of dabbling in a field in which I am no expert. I admit the accusation; but if we want to knock a hole in a wall we have to start from one side.

Some may argue that immediate deaths and delayed deaths are so different that they cannot be compared. However comparing different things is what management is about. Managers have to set priorities between different sorts of tasks. The question is not whether we compare short and long-term hazards but whether we do so openly and explicitly or inwardly, using unknown (to ourselves as well as others) criteria.

Let us look at some examples, starting with those where the size of the problem is known.

1 COAL DUST

This substance is not of great interest to the process industries but it is the cause of serious industrial disease—in 1975 out of 802 deaths from prescribed industrial disease in the UK, 643 were due to pneumoconiosis, most of it caused by coal dust—and it is one of the few substances for which the dose-response relationship is known. The National Coal Board has shown[11] that

$$P = \sin^2 (0.0704 x - 0.1201)$$

where

x = Mean respirable dust concentration in mg/m³

P = probability of developing category 2/1 pneumoconiosis (ILO classification) in 35 years

when calculating the sine the expression in the brackets is assumed to be in radians.

The equation is based on a statistical extrapolation of observations over ten years in twenty coal mines and mean coalface dust concentrations in those mines up to 8 mg/m³.

In 1970 the National Coal Board set standards which implied that long-term concentrations experienced by individuals would not exceed 4.3 mg/m³. The corresponding value of P is 9.42×10^{-4} per person per year. This would be equivalent to a fatal accident rate (FAR) of 50 or 100 deaths per 10^8 man per year if we assume that all cases of category 2/1 pneumoconiosis lead to premature death.

However, by no means all cases of category 2/1 pneumoconiosis lead to premature death. According to the Institute of Occupational Medicine[12] over 22 years men in the 25-34 year age group with categories 1, 2 & 3 pneumoconiosis had survival

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rates of 90.1% compared with 93.0% for those with category 0. The difference, they state, is statistically significant and suggests that the mortality risk for those with simple pneumoconiosis is about 60% higher than that experienced by young men with no radiological signs initially.

What happens after 22 years is not known. Let us therefore assume that ultimately one in three of those with simple pneumoconiosis dies prematurely (though the time of death may be many years ahead). The pneumoconiosis death rate is then equivalent to a FAR of 17 to 33 deaths per 10^5 men per year.

For comparison the FAR for all acute accidents in coal mines was 40 (80 deaths per 10^5 men per year) in the 1960s but fell to about 14 (28 deaths per 10^5 men per year) in the 1970s.

A coal miner joining the industry is thus about as likely to die from pneumoconiosis as from an acute accident, if past trends continue. This suggests that the Coal Board have got the allocation of priorities between short and long term hazards about right.

However, for a man aged 20-24 the total probability of death from all causes (including natural causes) is 10^{-3}/year[13]. If he is a coal miner, acute industrial accidents increase this by a factor of 3. Pneumoconiosis also increases his risk of death by a factor of 3 but this will probably not take effect for about 40 years by which time his total probability of death from all causes has risen by 20 times to 20×10^{-3} year. Looked at this way, the risk of death from pneumoconiosis does not seem as bad as the risk of death from acute industrial accidents.

On the other hand, it could be argued that our hypothetical coal miner may have looked forward to his retirement for 40 years and that death at the time of his retirement is worse than an early death.

This example shows that we cannot assume that probability of death is necessarily the right parameter for use in comparing immediate and delayed effects. Many people may consider that delayed death is preferable; others may take the opposite view. The National Coal Board's actions, imply that an immediate death is equivalent to a delayed death. Whatever our views, they seem to have got the balance between acute and chronic risks right to well within an order of magnitude. (Incidentally halving the coal dust concentration will decrease the incidence of 2/1 pneumoconiosis by about 20 times).

2. RADIATION

This is another area where good dose-response data are available though there is some doubt as to whether it can be extrapolated to low concentrations. For employees the agreed maximum dose is 50 mSv/y (5 rem/y) which is believed to give a risk of death of 5×10^{-4}/y (FAR 25 or 50 deaths per 10^5 men/y). This seems high compared with acute risks particularly as people exposed to radioactivity are presumably also exposed to normal acute industrial risks as well. If we assume that the FAR for these acute risks is 2 (as in the chemical industry and most UK manufacturing industries) then those who set nuclear standards are implying that an immediate death is about 12 times worse than a delayed death. Kinchin[14,15], a former head of the Safety and Reliability Directorate of the UK Atomic Energy Authority, has suggested levels of risk which are so low that action to reduce them further is not justified: his figure for delayed deaths is 30 times higher than his figure for immediate deaths.

Reissland and Harries[16] have suggested, taking radiation as an example, that loss of expectation of life should be used for comparing risks instead of the probability of deaths. Table 1, based on their data, shows that for a man aged 36 or more and exposed to 50 mSV/y the acute risks are worse than the chronic risks (assuming as before that he is exposed to the same acute risks as in the other industries).

However, as Griffiths[17] points out, loss of life expectancy is misleading. Consider a group of 1000 men. All of them could lose 10 days' life or one man could lose 10000 days (27 years) but the average loss of life-expectancy is the same. In no way can the two cases be considered similar. To quote Harold Wilson, "For the man who is unemployed, the unemployment rate is 100%".

3. ASBESTOS

The UK Threshold Limited Value of 2 fibres per ml for white asbestos was set following a study[18] which showed that 1% of those exposed to this concentration for a working lifetime will develop asbestosis. If we assume that every case of asbestosis results in premature death, this is equivalent to a FAR of 10 (20 deaths per 10^5 men per year). This seems high compared with the acute risks to which asbestos workers are also exposed but actual doses are probably a good deal lower than threshold limit values and by no means all cases of asbestosis result in premature death. Unfortunately very little data is available on the ratio between actual and permitted exposures. (Coal dust is an exception[11], the ratio of average to peak exposure is about 1/2).

If we assume that the average exposure is one-fifth of the TLV, that is, 0.4 fibre/ml and if we also assume that the dose-response relationship is linear, then 0.2% of those exposed will develop asbestosis. This is equivalent to an FAR of 2 if all cases result in premature death (which they do not). The figure of one-fifth is just an example. I do not know the true figure, which will differ from one factory to another.

It must be remembered that asbestos can cause other diseases besides asbestosis but dose-response data are not available.

TABLE 1

Loss of expectation of life for various risks

Risk*	FAFR	Deaths per 10^5 men per year	Loss of expectation of life (days) Age at beginning of exposures (yrs) 20	30	40	50	60
Acute:							
UK Manufacturing industry	2	4	20	13.5	8	4	1
Chemical industry**	4	8	41	27	16	8	2
Chronic:							
Exposure to 50 mSv radiation per year	25	50	68	33	12	3	0.5

*Assuming exposure continues for rest of working life.
**and all premises covered by Factories Act.

4. CHEMICALS

For chemicals very little accurate dose-response data are available. Roach[19] points out that there are 612 substance in the 1976 ACGIH list of TLVs. Of these 63 TLVs are based on industrial surveys, of which only 8 involve more than 200 people, and 50 are based on laboratory studies on volunteers.

This sparcity of data is often quoted as a reason for not using hazard analysis[20]. However, TLVs are set despite the lack of data, and if there is enough information to set a TLV it should be possible to estimate a probability of death. The probability may err on the safe side; but even so, we can still compare this probability with that of acute accidents. Some TLVs are based on the concentration required to ask irritation, not risk to life. These are not the ones with which I am concerned. When a TLV is based on a risk to life, if it is possible to set a TLV it should be possible to estimate the risk to life.

In applying hazard analysis to acute problems it is also often necessary to use expert judgement to fill gaps in the data. If we break problems down into component questions (What was the dose of A? For how long did it continue? What is the effect of such a dose?), answering them with facts where possible and with expert opinion when no facts are available we are more likely to get a correct answer than if we try to answer the whole problem by expert opinion. Such opinions should be used as substitutes for unavailable data rather than as an alternative method of problem solving.

An attempt to compare acute and long-term risk has been made for workers exposed to benzene (Mountfield[21]). He shows that exposure to 10-1000 ppm produces FARs of 1-10 (2-20 deaths per 10^5 men per year), but that there is no evidence that exposure below 10 ppm produces any excess risk. This is confirmed by a recent

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extensive review of the subject by Frieder[22].

This brings us to the question of linearity on which expert opinion is divided[23]. Some writers believe that the effect of a toxic chemical is proportional to the dose and that even one molecule (or one photon of radiation) can cause disease, though the probability is small[24]. Others consider that there is a threshold dose below which there is no effect, the body's natural defense mechanism coping with the alien material only when the body's defense mechanism is overwhelmed does disease result. It may be that the response is linear for some substances or effects (e.g. carcinogens) but that there is a threshold for others (e.g. coal dust).

The argument cannot be settled epidemiologically as at low doses the effects are too small to be detectable above the background noise, that is, the naturally occurring incidence of the disease.

In most cases a TLV may be exceeded but only for a short time. A man should not be exposed to twice the TLV for 4 hours and then to a nil concentration for another 4 hours. This implies that the dose-response data is not or may not be linear at these concentrations, and that low doses produce less effect than would be expected by extrapolation from high doses.

However, in making comparisons of the type discussed in this paper it would be prudent at the present time to base them on a linear hypothesis.

5. ALL INDUSTRY

Can we derive any useful information by looking at UK industry as a whole? For 1975-78 the average number of deaths from industrial accidents was 694/y and the average number of deaths from prescribed industrial disease was 925/y (1974-78)[25]. This suggests that over industry as a whole, accidents and disease are problems of comparable magnitude. However, about 800 deaths/y out of the 925 are due to pneumoconiosis, asbestosis and byssinosis, deaths restricted to a few industries and occupations. This suggests that acute accidents are the major problem.

However the figures for industrial disease include only those due to prescribed disease[26]. Some deaths will be due to non-prescribed diseases, but there is no consensus of opinion on the number. Considering cancer alone, one report[27] estimated that 1% of male deaths from cancer (that is, 0.3% of all male deaths or 1000 per year) might have occupational causes. Other reports[28,29] have suggested much larger figures. Thus Ref. 29 states that 5% of all male deaths in the US are due to occupational cancers while Ref 26 suggests 3-6% (10000-20000 per year) for the UK. These latter reports have been extensively criticised[31] "It [Ref. 29] shows how a group of reasonable men can collectively generate an unreasonable report"), and the Royal Society report seems more likely to be correct.

The 1000 male deaths from occupational cancer per year will include some of those due to prescribed disease; there will be some deaths due to non-prescribed diseases besides cancer, hence it seems that the total number of deaths per year due to occupational disease is perhaps between 1000 and 2000. The uncertainty in the data is illustrated by the fact that the TUC state "every year about 1400 people die as a result of occupational accidents and diseases"[32] while Farmer writes "on a conservative estimate (cancer) kills twice as many workpeople as industrial accidents"[33]. Death from occupational disease is thus a problem comparable with, perhaps rather worse than, death from industrial accidents and justifies appropriate allocation of resources.

Note that deaths from industrial disease reflect the working conditions of many years ago.

If we compare days lost due to industrial accidents with days lost due to industrial disease, we find that in 1976/77 accidents caused 12.2×10^6 lost days while *prescribed* diseases caused 0.5×10^6 lost days (Ref. 34). Even if we multiply by ten to allow for non-prescribed diseases, industrial disease, measured in this way, seems a smaller problem than industrial accidents.

CONCLUSIONS

This paper has explored, in a preliminary way, the possibility of using estimates of the probability of death for comparing acute and chronic hazards. The purpose of doing so is to help us decide whether reduction of acute risks or reduction of chronic risks should have priority. Results suggest that the allocation of resources between the two types of hazard is probably not too far out.

A major difficulty is knowing whether or not delayed death is worse than immediate death. On the one hand we have 20-40 more years life; on the other hand we have the worry of possible disease and years of illness before death. Perhaps these two effects can be offset and all deaths treated as equally undesirable?

We do need a new look at the data on which TLVs are based in order to estimate however roughly and with a safety factor to cover uncertainties) the probability of death at various exposures. Until we can do this we do not know if we are setting TLVs too low and thus spending on industrial hygiene resources that would be better spent on the reduction of acute hazards (or vice versa).

However the evidence suggests that in many industries the acute and chronic risks are now within an order of magnitude of each other. This may not seem very good but is not bad for problems of resource allocation. Nationally for example, our allocation of resources to medical care, road safety, industrial safety and discouragement of smoking bears no relation at all to either the relative risks or the costs of reducing the hazards[2].

FURTHER WORK

In this paper the relative size of short and long term risks have been compared and it has been assumed that if we can identify the higher risks we should give priority to their reduction. There is, however, another way of comparing risks. We can give priority to the expenditure which will save most lives per £1m spent. I have argued elsewhere[2] that this method should not be preferred as it leads to the toleration of risks which are high but expensive to reduce but that it may be useful as a secondary criterion.

In the present case I have no information on the relative costs of reducing short and long term risks. This is a subject worthy of further investigation.

Acknowledgements

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LOSS PREVENTION AND SAFETY IN THE PROCESS INDUSTRIES

Professor Kletz's paper was a plenary session at the recent Harrogate 4th International Conference (*The Chemical Engineer* last month). It forms part of a 3-volume set, IChemE Symposium Series Nos. 80, 81 and 82. The three volumes can be obtained from IChemE Sales, 165/171 Railway Terrace, Rugby CV21 3HQ at a price of £70.00, post free. Alternatively, No. 80 (in which Prof. Kletz's paper appears) can be purchased at £40.00, post free.

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the craft approach to a scientific approach and this has evolved many new drugs mainly of chemical origin and often with high potency and specific action.

These can present many manufacturing problems and every firm that was visited recounted difficulties that have had to be overcome the "hard way", since the demands of the drugs developed by the scientific approach have not been matched by a corresponding development in the technology for the production of safe, clinically effective products. Furthermore, the Medicines Act has resulted in tighter regulatory control of products, processes and facilities.

For those unfamiliar with the pharmaceutical industry the report outlines scope and scale of the industry and indicates the numbers and disciplines of the personnel involved. Some of the regulatory controls imposed by the Medicines Act, 1968, are summarised particularly with regard to the "Qualified Person" (which affects persons having legal responsibility for supervising the manufacture of pharmaceuticals). In view of the wide range of activities of the pharmaceutical industry the range of products is reviewed in some detail, together with some of the special features and precautions that are called for and the processes and operations that are involved in their manufacture.

Reference has been made to the research emphasis of the pharmaceutical industry but much of this relates to the search for new drugs, evaluation of their pharmacological action and pharmaceutical development of a product suitable for patient use. Hence the work has a clinical bias, with a limited engineering input in the later stages.

This pattern shows also in the personnel, pharmaceutical interests having tended towards clinical pharmacy, with attention to the ultimate individual relationship between one drug and one patient. Equally chemical engineering has moved away from emphasis on specific unit operations to a more general systems approach and concern with transport phenomena. Generalisations are always fraught with danger and there are obvious exceptions but overall the pharmacist is 'product-oriented' while the chemical engineer is process-oriented.

The report concludes that an engineering approach to many of the problems could make an important contribution. However there is need to improve inter-disciplinary communication as well as carrying out engineering research on pharmaceutical topics. At this stage a requirement is seen for a chemical engineering "input" in terms of ideas, concepts and approaches, rather than detailed studies of specific topics.

The report concludes, therefore, that there should be some means of enhancing interdisciplinary co-operation and communication. After this, the recommendations for chemical/biochemical engineering research involvement would become of greater value.

In conclusion it must be emphasised that an engineer entering the manufacturing or development side of the pharmaceutical industry will need to make a conscious effort to ensure sufficient appreciation of the clinical characters of the product, which can often over-rule the conventional chemical engineering approach to processes.

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By Prof. P. N. Rowe

This Working Party meets every other year and combines its meetings with a two-day colloquium which is in the nature of a rehearsal for the International Symposia (ISCRE) which are also held in alternate years (Europe and North America alternately). The most recent meeting was in Copennagen, 8–9 September, and was attended by 15 Working Party members together with invited guests to a total of about 40.

The theme of the colloquium was "The application of CRE to industrial processes" and it was organised by Professor John Villadsen of the Danish Institutet for Kemiteknik with the help of local industry. The first morning session was at the Danish Technical University when the following papers were presented:

"Control of exothermic reactions in cooled reactors", *G. F. Froment, Belgium*

"Development of laboratory reactors at DTH, 1968–82", *J. Villadsen and H. Livbjerg, Denmark*

A back-mixing model describing mixing in one-phase continuous flow systems, *Th. Zwietering, Netherlands*

Reactions with gaseous reactants and a liquid catalyst, *H. Livberg and J. Hjortkjoer, Denmark*

The afternoon was spent at Haldor Topsoe A/S a privately owned catalyst manufacturing company that specialises in ammonia and methanol synthesis. We were shown their development and research laboratories later two papers were presented.

"The radial converter and its application to methanol synthesis", *Ib. Dybkjoer, Denmark*

"Aspects of fuel methanol production", A. Paggini, Italy.

The next day was spent at Novo Industri, a Danish company producing enzymes and pharmaceuticals. We were shown their laboratories and pilot plant including fermenters up to 2½ m³ capacity. Papers presented were:

"Use of enzymatic processes in industry", *S. Branner, Denmark*

"Multi-enzyme systems in membrane reactors", *C. Wandrey, West Germany*

"Immobilised biocatalysts in organic synthesis", *J. Tramper, Netherlands*

"Immobilised lipases for modification of oils and fats", *A. R. Macrae, UK*

"Optimisation of reactor assembly for 6-APA production from penicillin using immobilised enzymes", *L. G. Karlsen, Denmark*

None of these papers was preprinted but I could probably lead interested enquirers to further information.

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The formal Working Party Meeting was fairly brief. The letter of 4 May from the Chairman of the EFCE General Assembly was discussed and a detailed reply will be prepared. Preparations for ISCRE 8, Edinburgh, 10–13 September 1984, were reported. Organisation is well in hand and about 200 proposals for papers have been offered which is twice as many as can be presented at the meeting. ISCRE 9 is to be in Philadelphia, 19–21 May 1986.

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