

# Occupational Exposures to Pollution

S. A. HALL, MB, DPH, DOHyg., Occupational Hygienist,  
London School of Hygiene and Tropical Medicine

Two features that distinguish occupational exposures to impurities in the environment are their wide variety and localised intensities.

Chemical pollutants are the largest in number: many arise from the dust, fume or vapours of common materials and still affect large numbers of people in some industries. Others are derived from synthetic compounds: out of about 1½ million new compounds synthesised in the past century, over 300 are fairly commonly used in industry. By comparison, physical pollutants such as noise, heat and other radiations are more easily and precisely evaluated; they still affect large numbers of workers in certain industries. Exposure to biological agents affect relatively fewer people, mainly those in agricultural occupations.

Uncontrolled and intense exposures at work may produce occupational diseases that are more familiar to most of us in textbook descriptions than in everyday practice. Sometimes their easy recognition is obscured by resemblances to other diseases (as with metal fume fever) or by non-occupational exposures (as with chronic bronchitis). Even when clinically bizarre outbreaks occur in small working groups, recognition is sometimes delayed, as in two recent outbreaks of acrylamide neuropathy (Garland and Patterson, 1967; Lloyd Davies, 1970). Difficulty in recognition may be greater when different practitioners are responsible for the medical care of individual members of a working group.

Some pollutants may temporarily lower physical or mental efficiency rather than produce recognisable disease, and increasing attention is being paid to them because of their subtler effects on work performance.

## MECHANISMS OF TOXICITY

Industrial pollutants feature in a number of advances in biochemical toxicology that shed light on disease mechanisms (Barnes, 1969).

The actions of most poisons are fairly well understood in terms of three mechanisms: non-specific, as with simple asphyxiants; reactive, as with direct irritants; and site-specific, in terms of the tissue affected, and ultimately the cellular component, antibody system or enzyme involved. Present studies on the association between chronic carbon disulphide exposure and coronary heart disease form an interesting example. A fourth mechanism, lethal

synthesis, in which a relatively unreactive substance is formed into a toxic metabolite, most commonly within the liver or by gut flora, is recognised more frequently as knowledge of metabolic pathways increases. Thus, the petrol anti-knock compound tetra-ethyl lead is harmful not in its original state, but after conversion in the liver to triethyl lead.

The study of carcinogenesis and mutagenesis has recently been enriched by the combination of two techniques; detection of mutagens by observing effects in bacterial cultures, and the simultaneous injection of bacteria and a test compound in experimental animals to detect the lethal synthesis of mutagenic metabolites. The early results of such host-mediated assays have shown clearly, what was previously suspected, the close identity of mutagenic and carcinogenic properties in several such compounds (Legator, 1970). This new method may greatly assist the assessment of new and old compounds in relation to possible human occupational exposures.

#### DIAGNOSIS AND IDENTIFICATION OF NEW RISKS

Occupational poisonings are sufficiently uncommon events in this country for the newer opportunities for investigation to be underemployed. For many compounds, much is known from animal experiment, but little for man. The role of specific enzymes in man is well established for lead poisoning (Goldberg, 1968) and for organophosphorus insecticides (Barnes and Edson, 1960) but not for many other pollutants. Opportunities to increase fundamental knowledge, perhaps bearing upon treatment or the detection of susceptibles, should not be ignored.

The recognition of new risks depends greatly on the acumen of individual clinicians, well exemplified for the occupational carcinogens from the early observations of Percival Pott down to the more recent ones of Macbeth *et al.* (1967) for nasal cancers in furniture makers. One case of an uncommon disease in a fairly small working group is not very improbable, whereas two or more cases are. This essential application of Poisson probability to the recognition of other possible occupational diseases is easy, if there is enquiry into the relevant exposure.

#### SAFE LIMITS FOR CONTROL

Despite the use of increasingly large numbers and quantities of potentially toxic materials in modern industry, most occupational diseases remain uncommon in this country. This is due to the wide application of engineering methods of control, such as the segregation or enclosure of dangerous processes, local exhaust ventilation systems and, sometimes, more basic changes such as the substitution of innocuous or less risky substances for toxic ones.

While the exact identification of risks and advice on control has been a medical responsibility, most methods of control (as with the limitation of waterborne disease in the nineteenth century) have been engineering ones.

There is today no occupational hazard that cannot be controlled by substitution or engineering design. But the speed of application of this knowledge is greatly influenced by socio-legal and economic pressures. Prevention of pollution is too often viewed as non-productive expenditure. The cost of ill-health is less easily computed than the expense of control, which can often add around 5 per cent to the capital costs of a new enterprise. Here is an area of medical responsibility where epidemiology rather than invective needs to be applied.

There is clearly a need for closer collaboration with the engineering profession, although much has been achieved. There is still room for misunderstanding or failure in communication, and it is essential that we should speak the same language about the precise risks involved, particularly the determination of safe limits of exposure to potentially harmful materials and agents.

With the exception of ionising radiations, most other mutagens and carcinogens, and some infective agents, almost all other pollutants are

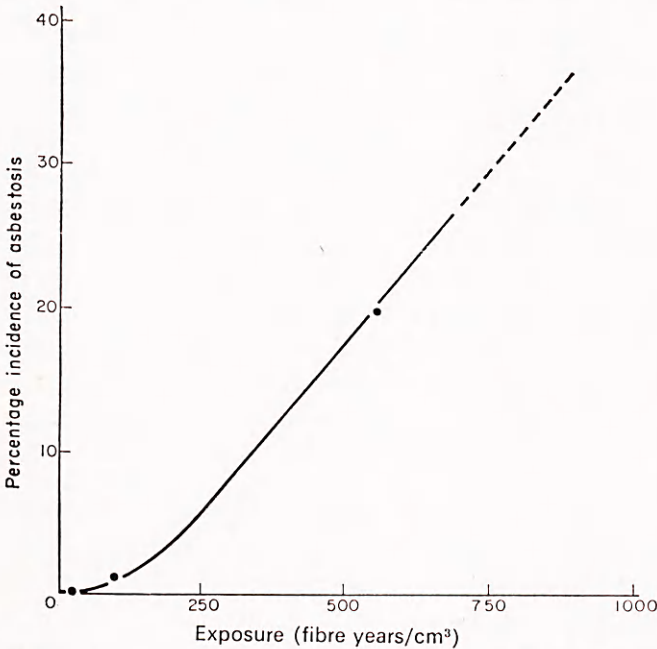


Fig. 1. Response to chrysotile asbestos exposure (*vide*, Roach, 1970). (Courtesy *The Annals of Occupational Hygiene*.)

harmless to the great majority of people at very low concentrations. For atmospheric contaminants, there exists an average concentration at which continuous exposure during a succession of 8-hour working periods over a period of (say) fifty years will be harmless to normal people. Such concentrations, termed Threshold Limit Values, have been established by epidemiological studies and animal experiment for over 400 industrial materials, and these values are currently used in the evaluation of engineering control measures in the USA and this country. The graph (Fig. 1) shows part of a dose-response curve to chrysotile asbestos dust exposure, with the development of signs of asbestosis in 2 per cent of the population from 100 fibre years/cm<sup>3</sup>. Allowing for a possible working life of fifty years, exposure above 2 fibres/cm<sup>3</sup> gives a detectable risk, and a Threshold Limit Value at this level has now been accepted in this country (Roach, 1970).

The criteria for evaluation, especially sampling criteria, obviously depend on the biological properties of the substance, in particular its rates of absorption, metabolism (or phagocytosis of particulates), and elimination by various routes. If there is a threshold below which no normal person is adversely affected, there must be a critical burden of the contaminant (or its metabolites) accumulated in the body. To be certain that this critical burden is not exceeded in a variable environment it is necessary to hold the average body burden well below the critical level so that the maximum excursions also remain below it. Roach (1966) showed that, for a large variety of atmospheric contaminants, if the rate of elimination of the substance is proportional to the body burden, then the sampling criteria are related to the biological half-time. This clarification has greatly assisted the rational evaluation of air sampling and testing compliance with Threshold Limit Values, particularly for respirable dusts (Roach *et al.*, 1967).

It is well known that marked differences exist between major industrialised countries in their criteria for permissible limits, and in the values laid down. There are understandable scientific reasons for these differences, even if they appear conflicting. They depend upon:

- (i) what effects are detected, e.g. a gross effect, like narcosis, or possible sub-optimal work performance;
- (ii) what reliance is placed on animal experiments in various species;
- (iii) the size of the human population in which effects are sought: for many industrial pollutants, only small populations can be observed, and the range of uncertainty is greater;
- (iv) the constitution of the population: their biochemical individuality in genetic, immunological and sometimes nutritional terms;

- (v) associated exposures: the problems of 'occupational bronchitis' (Gilson, 1970) and the synergic effects of asbestos and cigarette smoking (Selikoff *et al.*, 1968) are good examples;
- (vi) how 'appreciable risk' is to be defined when permissible limits are proposed: what proportion of the population are we prepared to exclude from our definition? In practice, the limit chosen tends to be related to the seriousness of the effect, but there is obvious opportunity for disagreement.

The review of these problems by the sixth session of the joint ILO/WHO Committee on Occupational Health (ILO, 1970) helped to resolve some of the existing differences in national standards for permissible limits. It must be emphasised that all established limits should be scrutinised critically in the light of new evidence. The Threshold Limit Values published by the American Conference of Governmental Industrial Hygienists, and accepted as a guide by the Department of Employment in this country, are revised annually; fresh evidence on the adequacy of existing limits and suggested additions are welcomed. Such evidence normally comprises accurate data on environmental concentrations as well as objective medical evidence of adverse effects.

#### RESEARCH ON SUSCEPTIBILITY

The pattern of response to most toxic agents varies in a large population and follows an S-shaped ogive with which we are familiar in pharmacology.

Attention naturally focuses on those most susceptible in the population: the individuals affected by low doses of the contaminant, sometimes lower than the currently accepted Threshold Limit Values.

In this country, the increased susceptibility of coalminers with circulating rheumatoid factor to a characteristic form of pneumoconiosis (Caplan, 1953) has been further illuminated by the work of Allison *et al.* (1967) on the cytotoxic action of silica on the phagosomes within macrophages, and its immunological consequences.

A similar association of asbestosis and rheumatoid factor, first reported by Pernis *et al.* (1965) has been confirmed and also shown to be associated with circulating antinuclear factor by Parkes and Turner-Warwick (1970).

Investigations have been made, and are continuing, into the role of Type I and Type III allergy in occupational exposures to vegetable dusts (Pepys, 1969), enzyme washing powders (Flint, 1969; Newhouse *et al.*, 1969; Greenberg *et al.*, 1970), and a variety of sensitisers in the plastics industry.

Biochemical genetics is providing more examples of inborn errors of metabolism that may prove important in an occupational setting (Harris, 1970). Most defects are rare or uncommon autosomal variants, and it would be a

great help to be able to identify such susceptible individuals without prior knowledge of the exact enzyme defect. It is possible to do this, utilising such methods as first-cousin frequency in the parents of those affected, if cases are drawn from a population the size of Greater London, but populations exposed occupationally to a single class of pollutant are seldom so large. Otherwise, the recognition of susceptibles depends on devising and using suitable screening tests, as with detection of plasma cholinesterase deficiency.

Easier to investigate are the major polymorphisms, such as haemoglobin S and G6PD deficiency, which are present in some overseas and immigrant populations, and have an obvious bearing on anoxic and haemolytic risks, respectively, in industry.

Nutritional status bears further investigation in the overfed industrialised countries and the underfed developing ones (Sai, 1970).

#### CONCLUSION

No mention has been made of community exposures to pollution, which are of increasing concern. Some of the methodology for control of risks within industry may well prove useful when applied to these wider problems.

*This article is based on a paper read at the General Meeting of Members held at the Royal College of Physicians in November 1970.*

#### References

- Allison, A. C., Harington, J. S., Birbeck, M. and Nash, T. (1967) *Inhaled Particles and Vapours II*, p. 121. London: Pergamon.
- Barnes, J. M. (1969) *Brit. med. Bull.*, **25**, 219.
- Barnes, J.M. and Edson, E. F. (1960) *Modern Trends in Occupational Health*, p. 97. Ed. R. S. F. Schilling. London: Butterworth.
- Caplan, A. (1953) *Thorax*, **8**, 29.
- Flint, M. L. H. (1969) *Lancet*, **i**, 1177.
- Garland, T. O. and Patterson, M. W. H. (1967) *Brit. med. J.*, **4**, 134.
- Gilson, J. C. (1970) *Proc. Roy. Soc. Med.*, **63**, 857.
- Goldberg, A. (1968) *Sem. Haemat.*, **5**, 424.
- Greenberg, M., Milne, J. F. and Watt, A. (1970) *Brit. med. J.*, **2**, 629.
- Harris, H. (1970) *The Principles of Human Biochemical Genetics*. London: North-Holland Publishing Co.
- International Labour Office (1970) *Occup. Safety & Health Series*, **20**. Geneva: I.L.O.
- Legator, M. (1970) in *Environmental Chemical Mutagens* Ed. Hollaender. New York: Plenum Publishing Co.
- Lloyd Davies, T. A. (1970) in *Annual Report, HM Chief Inspector of Factories for 1969*, p. 63. London: HMSO.
- Macbeth, R. G., Acheson, E. D. and Hadfield, E. H. (1967) *Lancet*, **i**, 311.
- Newhouse, M. L., Tagg, B., and Pocock, S. J. (1970) *Lancet*, **i**, 689.
- Parkes, W. R. and Turner-Warwick, M. E. H. (1970) *Brit. med. J.*, **3**, 492.
- Pepys, J. (1969) *Hypersensitivity diseases of the lungs due to fungi and organic dusts*. Monographs in Allergy **4**. New York & Basel: Karger.
- Pernis, B., Vigliani, E. C. and Selikoff, I. J. (1965) *Ann. N.Y. Acad. Sci.*, **132**, 112.
- Roach, S. A. (1966) *Amer. Industr. Hyg. Assoc. J.*, **27**, 1.
- Roach, S. A., Baier, E. J., Ayer, H. E. and Harris, R. L. (1967) *Ibid.*, **28**, 543.
- Roach, S. A. (1970) *Ann. Occup. Hyg.*, **13**, 7.
- Sai, F. T. (1970) *J. trop. Med. Hyg.*, **73**, 294.
- Selikoff, I. J., Hammond, E. C. and Churg, J. (1968) *J. Amer. Med. Assoc.*, **204**, 106.