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Asbestos and Health

THE advisory committee set up in 1976 "to review the risks to health arising from exposure to asbestos" produced its final report¹ on Oct. 25. It recommends that blue asbestos (crocidolite), including products containing it, should no longer be imported into the United Kingdom and that the control limits for exposure to dust from white (chrysotile) and brown (amosite) asbestos should be reduced from 2 fibres/ml to, respectively, 1 and 0.5 fibres/ml. Where work with crocidolite imported in the past is unavoidable—for example, during the demolition of buildings or railway vehicles or in ship-breaking—the present control limit of 0.2 fibres/ml should continue to be enforced. The report implies that these recommendations should represent but "the first stage of a continuing effort to reduce exposure". Thus the committee hopes that legislation will be drawn up not only making it a requirement to reduce exposure to asbestos dust to the minimum practicable *within* the control limit but also requiring potential or actual users of asbestos to consider, as knowledge and technology develop, whether effective substitutes safer than asbestos have become available. Another point that has been taken into account is that improvements in dust-measuring techniques in the past decade have led to a de-facto tightening of the standards set in 1968 of between two and five fold. This means that, for example, the new standard for chrysotile will represent a tightening not of two fold but of from four to ten fold, compared with the standard of 2 fibres/ml as measured in 1968. The stringency of the recommendations stems mainly from the committee's view of the epidemiological evidence, which suggests that, at least as far as lung cancer and mesothelioma are concerned, there may be no safe threshold of dose of fibre below which no increment of risk occurs. A linear relationship between dose and response fits present data well and is therefore a reasonable working hypothesis.

The report is likely to be criticised both by that section of industry which has to face the cost of meeting the more stringent standards and by those environmentalists who regard prohibition as the

only acceptable solution to asbestos-related disease. Such criticisms are inevitable and, in a subject which generates so much heat, will doubtless be acerbic. The recommendation that the control limit for amosite asbestos should be stricter than that for chrysotile may also stimulate debate, since no other country has so far made such a distinction between amosite and the other commercial fibres. The reason for this recommendation is that the committee accepted the epidemiological evidence that amosite is probably more commonly a cause of mesothelioma in man than is chrysotile, though less commonly than is crocidolite. Electron-microscopic evidence published since the report was prepared provides further support for this view.² Samples of lung from male North American mesothelioma patients have been found to contain more amosite fibres than samples from controls, but similar amounts of chrysotile. The amount of crocidolite found was negligible, presumably because this material has been little used in the North American insulation industry.³

The report gives cautious acceptance to the idea that alimentary-tract cancers may sometimes be caused by asbestos in conditions of heavy industrial exposure, but it points out that the evidence on this matter is not unanimous. Since the report was prepared the results of three further surveys (of crocidolite miners,⁴ crocidolite gas-mask workers,⁵ and asbestos cement workers⁶) have been made known, none of which shows an excess of alimentary-tract cancers among the workers concerned. These findings raise the possibility that a factor additional to asbestos must be present in the industrial environment for alimentary-tract cancers to appear in excess among exposed workers.

A consideration of the benefits of asbestos to health was unfortunately not within the committee's terms of reference. It would have been interesting to know, for example, how many serious burns and how many injuries and deaths from road traffic accidents are prevented annually by the use of asbestos in fire-resistant boards and fire-fighting equipment and in brake linings. Are there equally effective alternatives to asbestos for these purposes? If so, can they be made as safe or safer to use in the workplace? An answer to these questions is urgently needed.

2 McDonald A. Mineral fibre content of lung and occupation in mesothelioma tumours. Paper submitted to the Conference on Biological Effects of Mineral Fibres, Sept. 25-27, 1979. I.A.R.C., Lyon.

3 Selikoff IJ, Hammond EC, Churg J. Mortality experiences of asbestos insulation workers. In Shapiro HA ed., *Proceedings of International Conference on Pneumoconiosis*, Johannesburg, 1969. Cape Town, Oxford Press, 1970.

4 Holdo MSF, Woodward N, Armstrong BK, Musk AW. Monitoring of cancer risk in previous crocidolite miners in Western Australia. Paper submitted to the Conference on Biological Effects of Mineral Fibres, Sept. 25-27, 1979. I.A.R.C., Lyon.

5 Jones JN, Wignall BK, Mabbey RJ, Aggarwal A. The tracing of personnel who were exposed to asbestos dust in a wartime gas mask factory. Paper submitted to the Conference on Biological Effects of Mineral Fibres, Sept. 25-27, 1979. I.A.R.C., Lyon.

6 Hughes J, Weil H. Lung cancer risk associated with manufacture of asbestos cement products. Paper submitted to the Conference on Biological Effects of Mineral Fibres, Sept. 25-27, 1979. I.A.R.C., Lyon.

1 Advisory Committee on Asbestos. Vol. 1, final report. Vol. 2, papers commissioned by the committee. Health & Safety Commission, 1979. H.M. Stationery Office. 25 each volume.