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Changing trends in US mesothelioma incidence

The paper by Weill and colleagues[1] requires to be read with great circumspection by those concerned with public health policy. It would be premature for them to conclude from it, that with the decline in US mesothelioma incidence, which the authors associated with the earlier decline in use of amphiboles, the asbestos problem has been largely solved. Expert opinion published by WHO, ILO, and IARC is not so sanguine as them about chrysotile, and it is increasingly acknowledged worldwide that its safe use is not reasonably practicable.

There is a danger of understanding from the authors' statement: ''Asbestosis and asbestos attributable lung cancer have been found to be linked...'' that they are asserting that the evolution of interstitial pulmonary fibrosis is a necessary stage in the carcinogenic process in the bronchus (or for that matter in the pleura or in the peritoneum). In the case of bronchial carcinoma, this was once held to be a self-evident truth supported by unproven mechanisms of varying degrees of ingenuity and vagueness, but it is of note that consensus was reached that the sequence was not necessary, by a group of experts covering the adversarial spectrum.[2]

The pathologist co-author Churg, having contributed significantly to the literature,[3] will be aware of the extensive and persuasive experimental evidence that all species of asbestos are equally potent inducers of bronchial epithelial metaplasia and malignant change in whole animals and in tracheal explants, dissociated from interstitial pulmonary fibrosis. He will also be aware of the tissue culture studies in which from 4 minutes onwards, fibres are seen to interact with the cell membrane, the cytosolic apparatus, and nuclear spindle, leading to various transformations.

Two statisticians are cited[4] as authority for the ''biological plausibility'' of the authors' contention that use of amosite and crocidolite related to temporal trends in US mesothelioma incidence. These statisticians in their review of the literature had relied on an earlier review[5] in support of the ''biological plausibility'' of chrysotile being a less powerful tumorogen than the amphiboles, but its author with her limited resources, balking at the task of covering the vast literature, in her turn had relied on earlier ''state of the art'' reviews. In the adversarial atmosphere pervading attitudes to asbestos, uncritical reliance on such reviews was imprudent. The two statisticians cited, took note of the experimental evidence for the carcinogenic potency of chrysotile being comparable with that of the amphiboles, but discounted it on the basis of it applying only to the brief life of rats, and of it being more rapidly ''cleared'' than amphibole; the significance of the events observed in the first 4 minutes in cell culture were not discussed.

Weill *et al* attribute the lesser rate of mesothelioma in the USA compared with the UK to amosite being the amphibole used most in the USA, and to its national population being four times larger. Official figures present US amphibole imports in tonnes for the years 1964 and 1965 respectively as: amosite 23 932 and 17 042; and crocidolite 21 163 and 17 042.[6] (UK imports of amosite in 1965 totalled 22 582 tonnes and crocidolite 3425.) They report the peak US mesothelioma incidence rate to have occurred in the early to mid-1990s, and attribute it to the reduction in amphibole use since its peak import in the USA in the 1960s.

National asbestos import tonnages are no better surrogates for the exposures of the population subsets at risk, than the total populations of the states and metropolitan areas with cancer registers are acceptable as denominators of those occupationally exposed. The derivation and interpretation of trends in mortality and cancer registration rates for malignant mesothelioma from such data for occupational sub-sets present problems.

Between 1900 and 2002, the USA exposed its workforce to a total of some 25 million tonnes of home produced and imported asbestos (all species), and exported a small proportion.[7] The accelerated flight from asbestos manufacture in the 1980s will be reflected in due course in asbestos worker mortality patterns, but service workers, construction workers, and bystanders will continue to be at risk. The Third Wave of Asbestos Disease[8] was no Selikoff bugaboo; pace Weill and colleagues' reassurance, public health policy makers require to maintain a watching brief for many years yet

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BOOK REVIEW

Analyses of hazardous substances in biological materials. Volume 9 Special issues: Marker of susceptibility

Edited by J Angerer and M Müller. Weinham: Wiley-VCH, 2004, pp 329. ISBN 3-527-27799-4

This is an unusual edition in a series of books devoted to methods of estimation of chemicals in workplace atmospheres. Protocols for genotyping CYP P450 1A1, 1B1, 2E1, N-acetyltransferase 2, glutathione S-transferase T1, M1 and P1, sulphotransferase 1A1 and 1A2, and phenotyping of glucose-6-phosphate dehydrogenase, N-acetyltransferase 2, and glutathione S-transferase T1 are presented. Each protocol is clearly set out with a discussion of underlying principles, quality control and sources of error. A short section on real time PCR genotyping for a number of polymorphisms is also included. The authors quite sensibly took the decision to repeat essential basics in each protocol (for example, preparation of gels for chromatography) so that each protocol can be read independently. It is inevitable that the methods proposed will become dated but that should not detract from the current value of this edition to bench scientists. A number of the preliminary chapters will be of value to students. Of these I found the section on polymerase chain reaction and background information on polymorphisms which preceded each protocol easy to follow and instructive. The editors do note that the book may contain minor typographical errors. Readers should note the lack of reference numbers in the bibliography for N-acetyltransferase 2 genotyping (although these are listed in numerical order) and the incorrect concentration for the Tris buffer concentrate in the CYP 1A1 gentoyping protocol (p. 74). The book is not intended to be a text on molecular epidemiology and the short chapter on evaluation of susceptibility is at most a very basic introduction. Overall, well worth purchasing by academic libraries for use by researchers and students.

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