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PULMONARY ASBESTOSIS

pneumoconioses or dust diseases, asbestosis would be the most serious from many points of view. Now a relatively short exposure—months instead of years in silicosis—and while the morbidity rate, as judged by radiological means, has been much reduced by measures to lessen the inhalation of asbestos in factories, it can still be very high—44.5% in workers with 20 or more years' exposure and 20% with 10-15 years' exposure.¹

Reduction in frequency of pulmonary tuberculosis in the post-war era, and the equally striking increase in it in most countries, makes it difficult to assess the relative liability of present-day sufferers from asbestosis and these two complications. Clearly the data obtained 20 years ago have little validity now, while lung carcinoma has such a range of frequency in different countries that conclusions based on world averages are hardly acceptable. In the case of pulmonary tuberculosis: British figure for the association of this disease with asbestos in the years 1932-39 was 57.1% in the same period the figure was 57.1% in silicosis.² But in one part of Germany,

recent findings published in 1960 show that as a complication of silicosis was still very high—34.6%—while the association with asbestosis was 17%, little more than twice the tuberculosis figure for the whole population in that region.³

Lung-cancer rate in silicosis is not appreciably higher than in the general population, but in asbestosis it is estimated to be about ten times that of similar cases, both in Britain⁴ and in Germany.⁵

In recent years the matter has been extended by the discovery of asbestosis by most pathologists of malignant mesothelioma as an entity, and the even more tardy appreciation that mesothelioma of the pleura may be associated with asbestosis.^{6,7}

Occupations where pneumoconiosis of mineral origin is concerned, those affected are normally the miners and the workers handling the material in bulk. In these there is no danger as a rule to those who deal with manufactured goods containing the raw material. With asbestos it is otherwise. In Canada, where three-quarters of the world's production of asbestos is concentrated, the miners would appear to suffer little and show no significantly increased cancer of the lung. In South Africa at least, the miners, and even those in the vicinity of the mines, may develop asbestosis and pleural mesothelioma.⁸

Cases of asbestosis, however, result not from mining but from employment in asbestos factories. Today asbestos is used in so many industries and is a constituent of so many manufactured goods that the occupations where asbestosis is a potential hazard are too many to list here. Workers at risk include such unlikely ones as the garage worker employed in under-body car

spraying,⁹ and more obvious ones such as those employed in lagging pipes with asbestos, or those concerned with the manufacture of asbestos cement, roof and floor tiles, ceiling boards, etc., or with the use of them, such as builders and builders' labourers.

While the modern use of asbestos expands the field of potential inhalers of asbestos fibres, the risk of asbestosis is limited, and it is unlikely that these occupations will lead to a marked increase in pulmonary asbestosis. The effect of the inhalation of a small quantity of asbestos fibres, insufficient to produce pulmonary asbestosis, but possible in many of the occupations indicated, has been put on quite a different footing by two recent papers by Thomson and his colleagues in this *Journal*.^{10,11} In the first paper¹⁰ Thomson described 6 cases of mesothelioma, pleural and peritoneal, where there was no diffuse pulmonary asbestosis of the ordinary type, but only a few small foci of asbestosis at the bases of the lower lobes. It was stressed that this limited basal asbestosis produced no signs or symptoms and was likely to be overlooked at autopsy, unless sections from the lung bases were examined microscopically. In only one of these cases was there a history of exposure to asbestos.

This paper was followed by what appears to be the first investigation into the extent to which urban dwellers are exposed to the inhalation of asbestos fibres. Thomson *et al.*¹¹ examined the bases of lungs for asbestos bodies by a simple smear technique, and found that more than a quarter of the subjects over the age of 15 years in the autopsy services of the Groote Schuur Hospital, Cape Town, showed asbestos bodies, supporting their contention that the inhalation of asbestos fibres was now an urban hazard. They did not find that it was a significant hazard today, and were more concerned with what might happen in the future. They emphasized that the world's production of asbestos is increasing at a staggering rate, and is now eight times what it was 35 years ago, and that today asbestos-containing products are ubiquitous, especially in urban areas.

Thomson *et al.*¹¹ also drew attention to a simple but important fact which seems to have been overlooked so far from the medical and public health points of view. This is that the very reason for the use of asbestos in industry—its resistance to heat, acid, alkali, oxidation and reduction; in fact, its virtual indestructibility—is the reason why it may become progressively more dangerous to man. Wind and water may disperse asbestos fibres, but they cannot destroy them. Asbestos is now accumulating on the surface of the earth, mainly in the cities, at the rate of 2,400,000 long tons per annum; motor vehicles are discharging asbestos dust from brake drums, clutches, silencers and under-body coatings, and modern buildings may contain asbestos products from the roof to the basement. The indestructibility of asbestos makes this to some extent cumulative, and in effect we may be creating such

an asbestos environment in cities that in the future basal asbestosis may become almost universal in urban dwellers and mesothelioma of the pleura and peritoneum may become common tumours.¹²

These observations and opinions are new and we await confirmation of these findings from other countries; they may well be confirmed as regards the frequency of asbestos bodies in the lungs of urban dwellers. It would seem a hard turn of fate that the avoidance of ordinary pulmonary asbestosis might lead to an increased frequency of mesothelioma of the pleura or peritoneum, presumably because the patient lives longer and the carcinogenic action of asbestos has a longer time to act. We hope the fears for the future expressed by Thomson *et al.*¹² are unjusti-

fied, but it may be desirable to pay heed to their warnings, at least to the extent of restricting unnecessary and possibly dangerous uses of asbestos.

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5. Carver, P. (1952): *Arch. Industr. Hyg.*, 5, 262.
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BAKTERIELE GENETIKA

Daar was 'n tyd toe dit aanvaar was dat die klassieke genetiese wette en beginsels nie op mikro-organismes, en veral bakterieë, van toepassing was nie. Bakterieë reproduseer vegetatief en hul mikroskopiese grootte het hulle ongeskik vir sitologiese studies gemaak. Vir baie jare was dit aanvaar dat daar fundamentele verskille, beide geneties en fisiologies, tussen hoër diere en mikro-organismes bestaan en dat die wette wat op die een van toepassing is, nie vir die ander geld nie. Maar, gesien in die lig van die moderne fundamentele eenvormigheid tussen alle biologiese wetenskappe, is dit duidelik dat hierdie opvatting verkeerd was. Gemeenskaplike grond is gevind deur die intensiewe studie van bakteriële genetica wat slegs in die laaste twee dekades op die voorgrond getree het. Vandag is hierdie vak die brandpunt van die biologiese wetenskappe en het al selfs sover gevorder dat dit amper die teregende vraag „wat is lewe?“, kan verklar.

Jacob en Wollman¹ verdeel die historiese ontwikkeling van bakteriële genetica in sekere tydvakke: Gedurende die laaste helfte van die 19de eeu is mikrobiologie as eksperimentele wetenskap ingestel en aandag is hoofsaaklik bestee aan die isolasie en morfologiese beskrywing van nuwe spesies. Van 1900 tot 1940 is 'n groot aantal variasies van bakterieë geïdentifiseer, bestudeer en beskrywe, en hieruit is die vak bakteriële genetica gebore. In die laaste tydvak, beginnende 1940, word bakteriële oorerwing ontleed en gekoördineer in die lig van die klassieke genetiese teorieë. Klassieke genetica ontleed die aard van komplekse, dikwels nie-essensiële, eienskappe. In teenstelling daarmee analiseer bakteriële genetica op die molekulêre vlak en gee 'n beter begrip van basiese sellulêre meganismes. Ook dien dit as 'n handige instrument waarmee die struktuur en funksie van biologiese sisteme bepaal kan word. Treffende bewys, dat daar 'n chemiese basis vir oorerwing bestaan, is gelever toe 'n bakteriële eienskap omvorm is deur desoksieribonukleïensuur (D.N.S.), wat uit 'n pneumokokkale mutant² berei is. Soortgelyke ingrypende waarnemings is in die afgelope 20 jaar met bakterieë gedoen waarvoor verskeie faktore verantwoordelik is. Om slegs 'n paar te noem: Bakterieë kan, weens hulle ongeëwenaarde voortplantingsnelheid in 'n kort tyd tot enorme getalle aangroei. Die bakteriële sel se oorerwingsfaktore is vasgelê deur 'n enkele liniêre struktuur, die bakteriële chromosoom, bestaande uit D.N.S. wat die makromolekulêre patroon van die bakterium beheer. Bakterieë se genetiese materiaal is relatief onstabiel. Mutasies

kom spontaan voor in 'n gegewe gene in een uit honderd miljoen sel-verdelings. Eksperimentele na in bakteriële genetica tref deur hul eenvoud, akkuraasie en berekenbaarheid waarmee analiese uitgevoer kan word.

Die fundamentele probleme van genetica het hulle binne die bestek van die nie-genetikus geval. Van merkwaardigste bydraes op hierdie gebied is indertyd gemaak deur fisici en biochemici. In 1953 het Watson en Crick,³ Nobelprysweners van 1962, die struktuur van D.N.S., gebaseer op chemiese analiese en kristallografiese studies, wat allerweë as 'n biochemiese toetsaanvaar is. Hiervolgens sou D.N.S. bestaan uit twee nukleotied-kettings wat spiraalsgewys rondom 'n sentrale as gedraai is. Die ruggraat van elke spiraal bestaan uit fosfodiester-bande. Die basisse, op 'n gegewe vlak, vorm die een ketting, word met behulp van waterstofbindings aan die ander ketting. Waterstofbindings kan, weens simmetrie, net plaasvind tussen die basis-paare adenien en guaninsitronien sodat die een ketting dan die beeld van die ander is. Dit is verbasend watter helder biologiese gevolgtrekkings van hierdie model gemaak word: Dit lewer bewys van D.N.S. se molekulêre kassie; die aaneenlopende liniêre volgorde van basiese aanleiding tot 'n bepaalde genetiese kode en tot eienskappe, terwyl dit ook 'n verklaring vir mutasies aan die hand gee. Hierdie model dien verder as voorbeeld vir 'n nuwe studierigting, naamlik *molekulêre biologie*. Daargestel is hoofsaaklik deur die toedoen van bakterie- en virus-genetika. Hoe ver ons reeds vandag gevorder is, kan uit die klassieke sitologie blyk uit die feit dat bakteriële en virus-genetika is, wat weer op hulle hulle ontstaan aan weefselkulture te danke het.

Die sentrale posisie wat atoomfisika vandag in die fisiese wetenskappe beklee, kan ons vergelyk met die posisie van molekule biologie in die biologiese wetenskappe. Teoretiese teorie is die praktyk van môre. In hoeverre bakteriële en virus-genetika die natuurwette gaan beïnvloed, hang sukses waarmee die teorieë, wat sodanig verwerk is, in ander biologiese sisteme toegepas kan word.

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