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VI.E.4

MEMORANDUM

TO: Member Associations
Medical Advisory Panel
Dust Measurement Advisory Panel

20 July 1979

FROM: Sir Neville Stack

AIA/20/1/10

ASBESTOS/SMOKING/LUNG CANCER RELATIONSHIPS

Enclosed is a copy of a paper prepared by the Health and Safety Consultant of Cape Industries Limited, which I am sure you will find of interest.

T R Stack

Sir Neville Stack

C.C. [redacted]

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PLAINTIFF'S EXHIBIT UC-1614

ASBESTOS/SMOKING/LUNG CANCER RELATIONSHIPS.

It is becoming increasingly apparent that statistics relating to the Asbestos/Health issues are being used in differing ways by different people and the conclusions which are being drawn can often be very confusing.

In the U.K., for example, the HSE have recently revised the way in which they relate death certificates to asbestos related disease. Whereas, in years prior to 1977 statistics were taken from the reports of the Registrars General for England, Wales and Scotland and the figures were based on cases where the underlying cause of death was assigned to asbestos, the current breakdown which is to be published in the HSE stats for 1977 will be based on numbers of death certificates mentioning particular diseases known to be related to asbestos exposure. Cases will be included if the relevant disease or diseases are mentioned in either Part I or II of the "causes of death" section and will not therefore correspond with figures published by the Registrars General.

The interrelationship of Smoking/Asbestos/Lung Cancer is particularly interesting and very important to everybody concerned with occupational health problems. Here again the literature has pitfalls and particularly in relation to North American data there is a need for some caution. The recent official US publication "Asbestos - An Information Resource" displays the misuse of epidemiological data which has been recently seen in American official publications. After misquoting uncritically the Martischnig (1977) study, it reverses Selikoff's figures to give completely the opposite picture. Referring to Selikoff's series of asbestos insulation workers, it states:

"Of 1,457 workers who never smoked regularly, 4 lung cancer deaths were observed while 1.08 were expected". (Expected death rates based on those for US non-smokers). Selikoff's actual figures (copy attached) were: of 1,457 non-smoking workers, one died from lung cancer whereas 4.4 deaths were expected. Selikoff's estimated figure for expected lung cancer deaths did not take smoking habits into account. If we accept the official US figures for an age-adjusted estimate of expected lung cancer in that cohort of 1,457 non-smokers to be 1.08, Selikoff's finding of 1 case would be in agreement with the absence of additional risk.

Selikoff himself has consistently said that any additional risk in non-smokers, if it exists at all, is sufficiently small as to be insignificant. It is of interest that a recent paper on laryngeal cancer in asbestos workers found no cases among non-smokers.

At the recent "Asbestos '79" Symposium held in London, Dr. Nicholson a fellow worker of Prof. Selikoff at Mount Sinai produced a series which he indicated showed a multiplicative risk of lung cancer for asbestos workers in the "non smoking" category. Whilst his series was based on a study of insulation workers between 1967-77 I presently lack information on the derivation of the control group of non asbestos working non smokers. When a detailed examination of the study is available it seems unlikely that it will radically contradict the earlier work of Selikoff upon the same series of insulation workers.

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The following table lists all the series known to our Group Medical Adviser, giving figures of lung cancer incidence which separate non-smokers from smokers:

Sources	No. of Cases of Cancer of the Lung	
	Non-Smokers	Smokers
Doll (1971)	0	24
Berry & Newhouse (10/72)		
- males	0	40
- females	2	19
Braun (1958)	0	12
Selikoff & Hammond (1975 - 1)	0	45
(1975 - 2)	1	179
Elwes & Simpson (1971)	0	24
Peto & Doll (1977)	0	6

These figures do not, of course, provide any basis for comparison with non-asbestos working non-smokers, but they do serve to show how very rare lung cancer is in non-smokers, even in those exposed to the very high dust levels of 30 or more years ago.

A review of the situation is provided by Saracci of the IARC headquarters at Lyons in The International Journal of Cancer 1977. Two other studies relating smoking to lung cancer in asbestos workers are frequently quoted: that of Meurman (1974) for Finnish anthophyllite workers and Martischnig (1977) for cases of lung cancer admitted to a British hospital. Both, however, whilst confirming the probability of a synergistic effect between smoking and asbestos in producing lung cancer, fail to provide evidence of risk for non-smokers.

In conclusion we feel it can be said definitely that lung cancer in a non-smoking asbestos worker is a rare event - so rare that calculation of the relative risk is not possible on figures at present available. It must also be remembered that figures published up to now relate to the much higher exposures of byegone days, and for smokers at least there is evidence of an increasing dose-response for lung cancer as there is for asbestos. But the issue is of some importance since it is clearly relevant to know whether asbestos can by itself produce lung cancer (as it can produce mesothelioma).

Evidence which will go a long way to answering the Asbestos-non smoking lung cancer issue is probably already available on the HSE-EMAS computer as a result of the long term EMAS epidemiological study of asbestos workers in the U.K. and efforts are presently under way to try and persuade the enforcement authorities to devote resources to retrieving this fundamentally important information.

A 18065

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Asbestos Study Is Begun On 150 Types of Products

WASHINGTON, Aug. 31 (AP) — The Government is starting an investigation of nearly 150 types of consumer products to see whether they pose any danger caused by asbestos, a substance that when breathed can cause cancer.

The effort is being initiated by the Consumer Product Safety Commission, which is working on the wording of a request for information from scientists, manufacturers and the general public on asbestos in other types of products. Many of the product types among the nearly 150 are known to contain asbestos in at least some brands.

However, asbestos is not considered dangerous unless its fibers are released in a way that people can inhale them. Thus, not all products containing asbestos necessarily pose a health problem. Breathable asbestos fibers have been associated with cancer and other diseases in numerous scientific studies. Once inhaled, the fibers can become lodged in the lungs for life.

Publication to Be Delayed

The commission will publish the list of product types in the Federal Register. However, publication is being delayed until the Environmental Protection Agency is ready to announce its own inquiry into the impact of asbestos on the environment. The announcement is expected in October.

Following is a list of asbestos-containing consumer products in a draft prepared by the commission. The commission said in a footnote, "The inclusion of a product on this list does not mean that all brands or models of that product contain asbestos."

Asbestos Paper Products: Accoustical ceiling tile, lamp sockets, burner mats for gas stoves, roofing felts (outer layers), pipe and boiler covering, vinyl sheet flooring backing, radiator top insulation, appliance heat shielding for slow cookers, heat shielding for hair dryers, paper sheets for heat insulation; millboard for these uses: television and other electronic switch plates, electric switch boxes, meter reinforced gaskets for air-cooled engines, electrical washers, linings for ovens and other devices, wall protection behind heat-generating products, floor protection under wood and coal stoves, soldering and welding blocks, iron rests, heat shielding for toasters, heat shielding for rotisserie broilers, fireproof wallboard, metal-clad fire doors and partitions, tent grommets, stove pipe rings.

Cloth and Woven Products: Flexible air conductor for heating, cooling and ventilating equipment; appliance wiring for barbecue fire starters, broilers, curling irons, electric blankets, hair dryers, heating pads, ranges, slow cookers, toasters, irons, deep-fat fryers and electric fry pans; awnings, candlewicks, catalytic heater mantles, cigarette lighter wicks; cord for these uses: seals in high temperature gaskets, valve stem packings, insulation for glass handling tools and braided wall steam hose, theater curtains; felt for these uses: reinforcements in plastics, gaskets, reinforcement in asbestos tapes, secondary insulation in high-temperature wire and cable, asphalt impregnated roofing felts, piano and organ felts; heating pads (element insulation); ironing board pads and covers, lamp and lantern mantles, pipe and boiler covering, pot holders and oven mitts; these flame resistant garments: gloves, hats, helmets, hoods, mittens, overgaiters, sleeves, suits, umbrellas, aprons, arm protectors, blankets, boots, caps; smokers' bibs; coal and wood burning stoves, tape for pipe insulation, braid and rope for packing, motion picture screens; tent grommets.

Asbestos Cement Products: Water, sewer and septic drain field pipe; airduct pipe; these sheet products: roofing clapboard, siding, shingles, interior walls, boiler and furnace baffles, bulk sheeting, welding shields, baking sheets, blackboards, laboratory table tops, linings for vaults, safes, humidifiers and filing cabinets.

Viscous Matrix Products: Adhesives (glues and epoxies), air-duct cement for asbestos-cement air duct, buffing and polishing compounds, caulks and putties, floor tile cement and mastic, auto body filler, flashing cement, furnace cement, glazing compound for ceramics, pipe and boiler coverings, roof and driveway coatings, stains and varnishes, automotive metal deadener, automotive undercoating, refrigerant cements, automotive muffler repair compounds.

Products Subject to Inadvertent Asbestos Contamination: Driveway gravel, fertilizer and lawn care products, potting materials, talcs for noncosmetic or food use applications.

Miscellaneous Products: Sprayed accoustical and thermal insulation material, ammunition shell wadding, automotive mufflers, barbecue firebed materials in gas barbecue grills, boat hull repair kits, flower pots, clutch plate friction materials, brake lining friction materials, home hobby potters' kilns, pottery clay, asbestos powder, bulk fiber, reinforcement in molded plastics and rubber, automotive radiator sealant, vinyl asbestos floor tiles, abrasive wheels, aerial distress flares, molded plastics and phenolic laminates, paint, textured paint, cement, drywall and plaster patching compounds; artificial gas fireplace embering material, phonograph records.

These products were listed as possibly containing asbestos according to "consumer inquiries and other sources not verified by the commission:"

Appliances: Air conditioners, dishwashers, hand-held mixers, portable electric heaters, popcorn poppers, refrigerators, vacuum cleaners, waffle makers.

Miscellaneous Products: Carpet padding, fireplaces, instant pâpier-maché, light fixtures on railroad passenger cars, welding masks, file cabinets.

Underlined products are those which contain(ed) or may contain "Calidria" asbestos products.

JLM
9/10/79

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UC 1614

4.1.1979

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WITH COMPLIMENTS

Enclosed is a report by Dr. Kevin Browne on the medical evidence presented to the Advisory Committee on Asbestos, whose report was recently published.

AIA/1/7/POL

10 December 1979

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A 18067

For your information.
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Sir Neville Stack,
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5th December 1979

DEPARTMENT
- 6 DEC 1979

Dear Sir Neville,

For circulation to the MAP and to the Member Companies of the AIA I enclose an excellent synopsis produced by Dr. Kevin Browne on the medical evidence presented to the Advisory Committee on Asbestos whose report was recently published in the U.K.

I feel that this report cannot be improved upon. I would only add to Dr. Browne's final paragraph the caveat that we are likely to be faced with some of the biological effects of inhalation of asbestos dust for many years after the use of asbestos has either been closely controlled or eliminated. So great is the latent period between exposure and disease that many years must elapse before Dr. Browne's expectations are fulfilled.

Yours sincerely,

Dr. W.J. Smither

Enc.

cc. Dr. Kevin Browne

A 18053

The Advisory Committee on Asbestos asked Professor Acheson, one of its members, to provide a review of the available scientific evidence on the ill effects of asbestos in man, together with a critical commentary. His review appears under the above title and in fact constitutes more than half of the total report.

Asbestosis, Mesothelioma and Lung Cancer

Asbestosis is defined as fibrosis or scarring of the lungs to such an extent as to interfere with their ability to function normally. Three points are made; firstly that there is a wide range of individual susceptibility, secondly that there is a time lag of several years before any ill effects appear, and thirdly, in a proportion of patients asbestosis progresses even if the employee leaves the industry.

Mesothelioma is a very rare type of cancer which has been known for more than two hundred years, and which is often very difficult to diagnose with any certainty. While about one person in four in this country dies of cancer nowadays, only one in every two thousand dies of mesothelioma. Of these, perhaps four fifths occur in people who have at some stage in their lives been in close contact with asbestos, while for the remaining one fifth, as with so many cancers, no reason can be found. Because of the long time lag between first exposure and the appearance of the tumour - forty years is mentioned as an average interval - the incidence of mesothelioma is still rising (there were about 300 cases in this country in 1977) and the peak is not expected to be reached until the 1980's or perhaps even later.

Lung cancer is accepted as occurring more often in asbestos workers than in the general population, although as the report states, because lung cancer is so common in the population as a whole, individual cases related to asbestos exposure are almost impossible to recognise with certainty. But the part played by smoking is very important. The question whether a non-smoking asbestos worker is more liable to develop lung cancer than a non-smoker elsewhere is still not settled. But all the series studied are in agreement that if there is a risk it is exceedingly small - one estimate quoted suggests that a non-smoking worker exposed to 1 fibre/cc of chrysotile all his working life ran the same risk as he would if he smoked three quarters of a cigarette per week. For smokers, however, the risk is appreciably increased at the sort of exposure level which prevailed 20 years ago, and estimates varying between two and eight times the risk for smoking non-asbestos workers are quoted.

Crocidolite, Amosite or Chrysotile

In the USA, it is generally held that there is not much to choose between the three types of fibre in terms of their harmful effects, and the report draws attention to this fact.

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In this country, on the other hand, crocidolite has for 10 years been regarded more adversely, and the report gives much attention to evidence distinguishing between the effects of the three types. A table sets out mortality figures from all the important published studies and relates them to the fibre types used in the industries to which they relate for asbestosis. Professor Acheson interprets them as suggesting that chrysotile may be the least harmful, crocidolite the most harmful and amosite in between, but he points out that differences in the quantity of dust inhaled may render these comparisons invalid. On equally uncertain evidence the same ranking is given for the relative risk of inducing lung cancer. For mesothelioma, however, the evidence is much stronger. The first major paper linking asbestos with mesothelioma was based on observations on crocidolite miners in Cape Province. The crocidolite miners in Western Australia also produced a high proportion of mesothelioma cases, as did gas mask assemblers both in this country (at Nottingham) and in Canada who worked with crocidolite filters. By contrast a search for mesothelioma cases at the Rhodesian chrysotile mines was unsuccessful; the same was true of the Ural mines, and among Canadian chrysotile miners less than one death in a thousand was attributable to mesothelioma. In manufacturing also mesothelioma seems rarer among chrysotile workers; a study is quoted of 20,000 T & N workers at Rochdale who entered after 1933, and among whom 10 mesothelioma cases were found - and even here some crocidolite had been used, raising the possibility that it could be responsible for the cases which occurred.

The greatest question-mark hangs over amosite. A thorough attempt to find cases of mesothelioma linked with the Transvaal amosite mines revealed only 4, compared with 83 from the Cape crocidolite mines; a fact which Professor Acheson regards as 'truly remarkable' especially as up to 1951 more amosite than crocidolite had been mined. The only strong evidence against amosite comes from Selikoff's study of the New Jersey factory where among men employed during wartime 2% had died of mesothelioma by 1973. The conditions in this factory were known to be extremely bad, and there is a faint possibility that some crocidolite was also used. In most other studies of workers in manufacturing and insulation, exposure was to at least two or all three of the fibres. Selikoff is quoted as saying that only chrysotile was used up to 1930 in the USA and during the 30's only a little amosite and almost no crocidolite was imported; on the basis of a further assumption about the greater use of amosite in shipbuilding he has produced figures purporting to show that the risk of mesothelioma is the same whether insulators use amosite or chrysotile. Professor Acheson is clearly reluctant to take these at their face value. He also raises the possibility, for which there is some support in the various studies, that mixtures of fibres may carry more risk than exposure to one type alone.

Fibre Size and Dose Response

The fate is discussed of fibres after they have been inhaled. Only fibres shorter than 200μ and thinner than 3μ diameter are thought to penetrate to the depths of the lungs, and the normal clearance mechanism of the lungs appears to be able to dispose of fibres shorter than 10μ.

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experiments in which fibres of varying chemical structure, length and diameter were injected into the pleural space outside the lung in animals showed that the degree to which they were capable of producing malignant tumours was related to the proportion of fibres in the size range 10-80μ with diameters less than 2.5μ. The curious coincidence by which the size range of fibre which is aerodynamically able to penetrate to the alveoli, the air sacs where blood is oxygenated, is almost identical with that which will produce malignant tumours when implanted in the pleural cavity outside the lungs in animals is passed over without comment. But attention is drawn to the fact that the ability to produce mesothelioma in animals extends to fibres with diameters as low as .01μ. The ordinary light microscope will not detect fibres much smaller than .3μ, so that current methods of counting may not provide a good estimate of the levels of potentially dangerous fibres in the environment. A return is made to this point in discussion of Pooley's election microscope studies of post-mortem lung tissue of Canadian chrysotile miners. He found tremolite in large quantities in the lungs of men diagnosed in life as suffering from asbestosis, and raised the question whether contamination by tremolite or other inorganic fibres may not be responsible for some of the disease attributed to chrysotile (or amosite?). It seems certain that the electron microscope will in future have to play a much larger part in fibre counting.

The difficulties in establishing a precise relationship between the quantity of fibres inhaled and resultant disease are discussed in detail. Dust counts, which in any case do not go back for very many years, were at first carried out on midget impingers and thermal precipitators before the present membrane filter came into use on any scale about 10 years ago and each method gives a different type of result. In addition, methods of counting have changed, so that a dust concentration reported as 2f/ml in 1968 would now be reported as between 4 and 10 f/ml.

There is good evidence for each of the three asbestos related diseases that, as common-sense would suggest, the more dust you inhale the more risk you run. But the question whether there is a threshold dose, i.e. a dose below which no risk is incurred, is less readily answered. Professor Acheson believes that the balance of probabilities is against there being a threshold dose in each case. However, he qualifies this in the case of asbestosis by adding that he believes there is a simple relationship between dose inhaled and risk within the range experienced in industry. This is a very important qualification to which I will return later.

A large number of studies are listed which provide evidence on dose relationship. Almost all are agreed that the relationship exists, but great discrepancies exist between estimates of the size of the risk incurred at any given dose. It is made clear that the dose response depends not only on the fibre count, but the method by which this is measured, the type of fibre, the presence of contaminants (such as tremolite), the amount of cigarette smoking (in the case of asbestosis and lung cancer but not mesothelioma) and lastly the diameter range of the fibre. It has been shown that crocidolite from Cape Province where many cases of mesothelioma have been found, is very fine, the size range being 0.09-0.8μ, while Transvaal Blue, which seems to have been responsible for far fewer cases, has a size range of 0.2-2.5μ.

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(for all purposes except asbestos cement), thereby producing fibres with a smaller mean diameter, and that if this were continued the reduction is risk associated with chrysotile may be lost.

Animal Experiments

Animal experiments have been carried out since before the war in an attempt to shed more light on asbestos effects in man. It has been found possible to produce lung fibrosis, lung cancer and mesothelioma in rats by exposing them to inhalation of asbestos dust. The early experiments encountered the same difficulties of variations in fibre count and fibre size of different samples, and current experiments are attempting to control these.

In these animal inhalation studies chrysotile has shown itself consistently to be at least as likely as crocidolite to produce asbestosis, lung cancer and mesotheliomas, while amosite consistently comes off best. Davis' conclusion is quoted that for a given average diameter, samples with the highest proportion of fibres $>20\ \mu$ in length are the most dangerous, and it is suggested again that fibre size rather than type may be the decisive factor. But these experiments have all exposed the animals to massive doses (measured in hundreds or thousands of fibres per ml) and have raised more problems than they have answered.

Other Cancer Risks

There have over the years been several reports of increased mortality in asbestos workers from cancer of the stomach, bowel, larynx, ovary and breast and these are listed and discussed in some detail. Again the results of various studies are not consistent and again it is Selikoff's series in which the important increases in mortality occur. The report concludes that while the information available is sketchy and other explanations for the figures can be given, a true increase in cancer of the stomach, bowel and larynx remains a possibility in insulation workers exposed to a mixture of fibres; chrysotile miners and manufacturing workers do not appear to run any extra risk, and evidence of excess risks for other organs is inconsistent.

Environmental Exposure

Such measurements as have been made suggest that levels of asbestos fibre in the general atmosphere, even in the dust of cities, are very low - less than one thousandth of the current 2 fibre/ml standard for industrial workers. Higher concentrations may be expected inside buildings in which asbestos has been used in the construction. Even here, however, in most circumstances fibre levels were almost always less than one hundredth of the current standard. Higher levels were reported where sprayed asbestos was exposed to wear and tear, and further studies are recommended. Asbestos cement, on the other hand, seems to be associated with even less risk of release of fibre to the environment.

Reviewing the evidence for actual health damage, the report considers it unlikely that the public is at risk from asbestosis (although one or two cases of pleural thickening may have occurred, these are certainly known to occur in areas in Europe where mineral fibre occurs naturally in the soil).

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Similarly, a lung cancer risk is considered unlikely in ordinary circumstances. The mesothelioma risk is dealt with in more detail owing to reports which arise from time to time that it has occurred following extremely brief exposure. There are certainly a small number of cases attributable to domestic exposure dating from the days when wives brushed the dust off their husband's overalls, and there does seem to have been a higher incidence in the neighbourhood of shipyards, perhaps for the same reason. But it is concluded cautiously that with modern practice no danger exists.

Asbestos fibres have also been found in drinking water and manufactured food and drink. The main evidence on which any conclusion can be based comes from Duluth, on the banks of Lake Superior, where the water supply was contaminated with waste asbestos fibres dumped in the lake. No excess of cancer deaths has been found. Similarly, animal experiments in which quantities of asbestos were eaten did not suggest any harmful effect.

Conclusion

The advisory committee's final recommendations are based closely on the conclusions of Professor Acheson's report, and do not need to be repeated here. But it is worth setting down the basis on which his figure was arrived at. He points out that in the case of occupational exposure in ionising radiation a health standard has been set (5 rems per year) which, if it was reached annually for 50 years, it was calculated would lead to an increase in cancer mortality in the exposed workers from 20% (the "natural" cancer incidence) to 22%. This is a decision which has been made and which has been accepted by society in general both in this country and internationally. If asbestos is accepted as a parallel situation, and if for chrysotile it is accepted that about half the mortality is due to lung cancer and half to asbestos, a 2% excess mortality (as for ionising radiation) would, on the b sis of estimates containing "very considerable uncertainties", result from fibre levels in the range from 5 to 0.4 fibres per cc. And out of this range he suggests 1 fibre/cc, nearer the lower end, as an acceptable compromise.

Comment

Professor Acheson's review was, I believe, substantially completed nearly two years ago, and there have been a few minor research developments since then. Tremolite is beginning to be regarded with much more suspicion, occurring as it does as a contaminant of some chrysotile mines, and the high proportion of mesothelioma cases in a Turkish village due to a form of zeolite, a naturally occurring non-asbestos mineral fibre, has given further impetus to research on how this type of tumour is caused. Cancer of the larynx is not now considered to be asbestos-related. As far as asbestos in food and drink is concerned, further careful animal studies have produced no ill effects. And finally the election mircoscope is beginning to make a big impact on studies of the type and quantity of asbestos in the lung, and in analysis of environmental exposure.

But the two aspects of asbestos-related disease, over which the report is most deficient have not been changed by new evidence. These are the influence of smoking and changes in industrial practice in this country.

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Taking smoking first, although this is mentioned briefly in connection with lung cancer, its influence on the development of asbestosis is ignored, and the major part it plays in the causation of these two diseases is not at all given the emphasis it deserves. On the second point, the absence of a threshold for the dose-response to chrysotile is mentioned 'within the range experienced in industry'. But this is the one vital issue which has not been faced; the dust range experienced in the industry in the 50's and 60's, on which the statistics of asbestos related disease are based, were probably between 10 and 1,000 times those to be found in the last 10 years. It is true that much space was devoted to dose response and the possibility of a threshold dose, but estimates were still made of low dose effects based on high dose experience, and a real examination of the evidence for the lungs' capabilities for clearing and the consequences of overloading was passed over.

Yet asbestosis has never been recorded with the minimal exposure found in the environment (as would be expected on the no-threshold calculation). There is a strong suggestion from the figures that much of the disease over which there is uncertainty, such as stomach cancer, only arises when there is massive exposure by comparison with modern experience. We know too that lung cancer becomes common in another type of severe lung fibrosis unconnected with asbestos but implying that at a certain stage a qualitative change may take place in the body's defences. This is also in keeping with what we know about the way the body's defences - the immune system - react. The system itself is extremely complex, but the analogy with a material which is stressed to breaking point is still applicable.

With all this in mind, and better evidence of true dose exposure from electron microscope studies, we can reasonably expect that modern control limits will relegate asbestos-related disease altogether to the past.

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