

To: Medical Advisory Panel

AIA/20/1/4/HAS
6 June 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Extracts from annual reports of the South African National Centre for Occupational Health - for 1982 p.32/4 and for 1983 p.14/5 - showing a total of 1228 confirmed mesothelioma cases over a period of 26 years.

00984

926070 Asbestos Tissue Reference Panel and the National
Mesothelioma Register

B. Coldstein and I. Webster

The panel and the register have been in existence for approximately 16 years. With a few exceptions the panel has met in Johannesburg once or twice a year to examine and discuss cases referred to it as mesotheliomas. More recently slides and details of current cases have been circulated to panel members and this has resulted in earlier diagnosis and also allowed more time for discussion of difficult cases at the panel meeting as it has not been necessary to re-examine cases on which there has been agreement.

After the last panel meeting when 108 cases were reviewed the findings are as follows:- (The cases of mesothelioma have been collected over the last 25 years)

MESOTHELIOMA REGISTER

AS AT OCTOBER 1982

Occupation	Number	Percentage
Mining	235	20,9
Cobbing	45	4,0
SAR & H	70	6,3
Transport	7	0,6
Lagging	35	3,1
Building	22	2,0
Other Industries	62	5,5
Asbestos Cement	13	1,2
Environment	178	15,9
Unkown/Not available	<u>453</u>	<u>40,4</u>
	1120	99,9

00985

An additional: 2 cases were diagnosed as probable mesothelioma
 2 cases were diagnosed as possible mesothelioma
 5 cases were diagnosed as 'other tumours'
 2 cases were deferred for further investigations
 2 cases were diagnosed as 'no tumour'
 2 cases had inadequate tissue

PREVALENCE OF MESOTHELIOMA IN INDUSTRY

<u>Exposure</u>	<u>No</u>	<u>%</u>
Environmental	178	26.7
Mining	235	35.2
Hand cobbing	45	6.7
Railways	70	10.5
Other industries	62	9.3
Lagging	35	5.2
Building	22	3.3
Asbestos cement	13	1.9
Transport	7	1.0
	<u>667</u>	<u>99.8</u>

The percentages have been adjusted to the first decimal place. Although hand cobbing has been prohibited by regulation from the Government Mining Engineer cases giving a history of this occupation still present themselves. It is hoped that eventually the cases in which there has been environmental exposure will become less because of the prohibition of stacking to atmosphere in the mills, fencing of dumps, and prohibiting the use of asbestos tailing as a road base.

Cases from the South African Railways and Harbours should also become less as asbestos is no longer used for lagging on the remaining steam locomotives in use.

00986

Although there are claims that cases of mesothelioma do not occur in certain asbestos cement manufacturing industries 13 cases have been associated with exposure to asbestos in this industry in South Africa.

South African experience is that the majority of cases of mesothelioma are associated with exposure to crocidolite, a few to amosite and possibly an occasional case to chrysotile.

The present Panel consists of:

Prof. C.J. Uys

Prof. I.W. Simson

Prof. I. Webster

Prof. B.D. Middlecote

Dr. B. Goldstein

A study of the lymphocytic infiltration of pleural mesothelioma has been made during 1982, correlating its presence, to a significant degree, with favourable or unfavourable prognosis and survival in patients drawn from the Mesothelioma Register of the N.C.O.H.

The article appeared in the S.A.M.J. in September 1982 and drew numerous requests for reprints from all over the world.

A more extensive follow-up of all survivors in the study will be made later in 1983.

The morphological assessment of lymphocytic infiltration could be incorporated into an overall prognostic index (after Pomerany) in the total assessment of mesothelioma biopsy, or operative specimens.

00987

The panel and the register have been in existence for approximately 17 years. With few exceptions the panel has met in Johannesburg once or twice a year to examine and discuss cases referred to it as possible mesotheliomas. Slides and details of current cases have been circulated to panel members at intervals and this has resulted in earlier diagnosis and also allowed more time for discussion of difficult cases at the panel meeting as it has not been necessary to re-examine cases on which there has been agreement.

At the last panel meeting 119 cases were reviewed. One case was a deferral from the previous meeting and was now diagnosed as an adenocarcinoma. Another case was deferred as the slides were not available.

The remaining 117 cases were diagnosed as:

Definite mesothelioma	106
Probable mesothelioma	2
Possible mesothelioma	4
Other tumour	2
Inadequate tissue	3

NATIONAL CENTRE FOR
OCCUPATIONAL HEALTH
JOHANNESBURG

The definite and probable mesotheliomas registered so far were then classified according to occupation.

MESOTHELIOMA REGISTER
*AS AT OCTOBER 1983

<u>Occupation</u>	<u>Number</u>	<u>Percentage</u>
Mining	255	20,8
Cobbing	45	3,7
SAR & H	72	5,9
Transport	8	0,7
Lagging	37	3,0
Building	22	1,8
Other Industries	74	6,0
Asbestos Cement	15	1,2
Environment	190	15,5
Unknown/Not available	510	41,5
	1228	

*These cases have been collected over the last 26 years
The present Panel consists of:

Prof. C.J. Uys
Prof. I.W. Simson
Prof. I. Webster
Prof. B.D. Middlecote
Dr. B. Goldstein

00988

AIA/3/16/ADM

4 September 1984

**ASBESTOS INTERNATIONAL ASSOCIATION
CONFERENCE LIST - NO. 34**

1984

Expected to attend

4-8 June	International Symposium on Research on Work Related Diseases arranged by Nordic Institute of Advanced Occupational Studies, Helsinki - Hanassari Cultural Centre, Espoo, Finland. Symposium Secretary: Dr. Antti Teperi, Nordic Institute, Hartmaninkatu No.1, 00290 Helsinki. Half a day reserved for asbestos in section "Respiratory Diseases".	
8 June	Occupational Exposure Limits; London.	
20 June	"Asbestos & Asbestos Substitution in Building Materials"; Mr. H.D.S. Hardie invited to speak. University of East Anglia, UK.	
24-27 June	International Symposium on Prevention of Allergic Diseases; Florence. A section reserved for occupational lung diseases due to chemical agents & dusts; 3 papers on asbestos accepted. Organising Secretary: Dr.M. Ricci, via G. Modena 19, 50121 Firenze.	
8-21 July	International Seminar on "Environmental Impact Assessment"; University of Aberdeen, Scotland.	
10-13 July	International Conference on Environmental Contamination; London.	Chatfield/B Commins/ Doll/E Meyer FRG/ W Nicholson/Penney/ Robock/A K Sheils/Toft Murray
9-14 September	21st International Congress on Occupational Health; Dublin.	MAP Members; SCAP Members Bouige/Costa
18-19 September	Meeting on Lung Burden Studies by Electron Microscopy; Oxford.	MAP Members (DAP Members?)

(papers on asbestos in water)

00978

1984

Expected to attend

17-20 September	12th International Congress of the International Research Association on Water Pollution and Aquatech 84; Amsterdam.	van 't Haaff
18-21 September	European Symposium on Fires in Buildings; Luxembourg. Organised by CEC/DGIII & European Association of Professional Fire Brigade Officers.	
21 September	Occupational Exposure Limits for Toxic Substances - their legal status and practical implications; London	
24-27 September	International Symposium on "The Transport & Handling of Dangerous Goods by Sea & Associated Modes"; Havana, Cuba.	
1-3 October	3rd International Workshop on "The In Vitro Effects of Mineral Dusts"; Hochschwarzwald, FRG.	
1-4 October	UK National Society for Clean Air, 51st Conference. Session on Asbestos includes "The Risks of Exposure" (Dr. M.J.Gardner), "Safeguarding the Nation" (S. Grant) & "Advising the Citizen"; Brighton.	
3-5 October	International meeting on "Risk Assessment of Occupational Exposures in the Harbour Environment" - Scientific bases for the prevention of environmental health hazards; Genoa. Speakers include Dr. B. Bedrikow (ILO), Drs. Saracci & Tomatis (IARC) & Dr. Selikoff.	Costa
6-7 October	XVII Symposium - Developing Issues and New Legislation in Environmental Affairs; Brussels. (International Professional Association for Environmental Affairs).	

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00979

1984

Expected to attend

23-25 October	Air Pollution: Implications & Challenges. International Conference sponsored by the Department of Health and Welfare of the Republic of South Africa; Pretoria.	Robock
28 Oct.-1 Nov.	Arab Water Technology Exhibition; Dubai. (Supported by the United Arab Emirates, Middle East Water & Sewage Journal & International Drinking Supply Sanitation Decade.)	
30 Oct.-2 Nov.	AIA 5th Colloquium on Dust Measurement Techniques and Strategy; Johannesburg.	Baunach/Bouige/DG/Hart/ Ignatow/Jooste/Lebel/ Meeks/Nash/Robock/Selles/ Tucker/Vanherle/Verill
12 November	"Hazard Communication Rules, Regulations and Good Practices" - Workshop with discussion includes a session on Labelling Practices in Europe; London	
13-15 November	"Toxic Substances, Human Health and International Regulations" - Workshop with discussion covering EINECS & TOSCA Inventories, Preparations Directive, 6th Amendment etc.; London	
14-16 November	World Industry Conference on Environmental Management - sponsored by world industry and the United Nations Environment Programme (UNEP); Paris.	
date not arranged	OECD International Conference on the Economy and the Environment.	
last quarter 1984	Energy & Pollution in the Cement and Building Materials Industry. Meeting to be organised by Arab Union for Cement & Building Materials (AUCBM); Libya.	

00980

1985

Expected to attend

12-17 April "Industry and the Environment" is the theme of the 3rd European Biennial of films on Environment (European Cultural Foundation). Will comprise a European audio-visual presentation and European forum whose theme will be "Ways in which to reconcile the needs of industry with the demands of the environment"; Dortmund.

22-26 April International Fair specialising in Water Distribution (partly concerning pipes in different materials): Berlin.

22-28 April Within framework of "Wasser Berlin 85" international experts will meet for events including a congress, an exhibition "Water & Us" & specialised conferences of the International Association of Water Distribution; Berlin.

7-9 May AIA Biennial Conference, London.
6-26 June **ILO Conference - Geneva**
25-30 August XV World Congress on Diseases of the Chest; Sydney, Australia

AIA Members
Lesage

2-6 September 6th International Symposium on inhaled particles 1985 (British Occupational Hygiene Society); Cambridge, UK.

1986

June

ILO Conference - Geneva

cc Member Associations
Governing Council)
Executive Committee) at meetings
MAP)
DAP)

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ASBESTOS INTERNATIONAL ASSOCIATION

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68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

MEMORANDUM

TO: MEMBER ASSOCIATIONS
FROM: DIRECTOR GENERAL

OUR REF.:
AIA/20/1/23/HAS
15 August 1984

Subject: SUMMARY OF MAIN FEATURES OF MEDICAL SURVEILLANCE AT THE
WORKPLACE - AIM No. 4/83

Enclosed is a copy of AIA Information Memorandum (AIM) No. 4/83 -
"Summary of Main Features of Medical Surveillance at the Workplace".

The information has been compiled from details supplied by Members of
the Medical Advisory Panel and we ask you to peruse this Summary and
let us have any information which can be included to make the
Summary more helpful to Members.

We look forward to hearing from you.



for DIRECTOR GENERAL

cc. MAP

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RECEIVED

AUG 29 1984

H. C. LEWINSOHN, M.D.

UCC 000770

Summary of Main Features of Medical Surveillance at the Workplace

COUNTRY	GOVERNMENT REGULATIONS	SURVEILLANCE RESPONSIBILITY	MEDICAL EXAMINATION	FREQUENCY	REMARKS
ARGENTINA		Industry			
AUSTRALIA					
AUSTRIA					
BELGIUM	Yes	Industry	Chest X-ray, lung function		Individual medical files
CANADA	Yes, by each Provincial Govt.	Industry and Govt. approved physicians	Chest X-ray, lung function test, full medical examination	at least every 12 months	Provincial jurisdiction
CHILE		Industry			
DENMARK					
FINLAND					
FRANCE	Yes	Industry, also any Physicians	Chest X-ray lung function		
GERMANY	Yes	Government approved Physicians	Chest X-ray lung function		
GREECE					
INDIA	None	Industry	Chest X-ray lung function		
IRELAND					
ISRAEL					
ITALY	Yes	Industry	Chest X-ray lung function		

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COUNTRY	GOVERNMENT REGULATIONS	SURVEILLANCE RESPONSIBILITY	MEDICAL EXAMINATION	FREQUENCY	REMARKS
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JAPAN

KENYA

MEXICO

NETHERLANDS

NIGERIA

NORWAY

PARAGUAY

PERU

SOUTH AFRICA

SPAIN

SWEDEN

SWITZERLAND

TURKEY

U.K.

URAGUAY

U.S.A.

MIDDLE EAST

EEC & ILO

U.K.	Only in some cases	Industry, in some cases Government	Chest X-ray Lung function		
URAGUAY		Industry			
U.S.A.	Yes - OSHA	Industry	Chest X-ray Lung function		
MIDDLE EAST	None	Industry	Chest X-ray Lung function		
EEC & ILO	U N D E R D I S C U S S I O N				

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ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

MEMORANDUM

TO: Medical Advisory Panel

FROM: Director General

OUR REF.:
AIA/20/1/23/HAS

2 August 1984

The Medical Surveillance of Asbestos Workers (Draft AIA Information Memorandum No. 3/83)

You have received comments on the above draft (from Drs Lambert, Lewinsohn and Morin) since it was first circulated under cover dated 1 December 1983.

Please find enclosed some comments from Dr. Goffe which we send rather belatedly!

Also enclosed is a short note by Dr. Mansour on a related subject - training the asbestos worker in health awareness.

Would you please be prepared to consider the draft AIM 3/83 at the next MAP Meeting on 17/18 September, using as the discussion paper Dr. Lewinsohn's review sent to you under cover dated 24 January 1984.

TN Stack

Sir Neville Stack

Encs. 2

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AUG 6 1984

H. C. LEWINSOHN, M.D.

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(248)

Dr. T. R. P. Goffe,
Occupational Health Department.

Mr. H. Hardie,
Turner & Newall PLC,
Trafford Park,
Manchester.

TRPG/ON

22nd December 1983.

THE MEDICAL SURVEILLANCE OF ASBESTOS WORKERS

Thank you very much for the opportunity to comment on this draft document. It seems to me that it does not contain anything new, probably not meant to, and as such is really a statement of current practice, give or take a procedural detail here and there.

My passing comments are:-

- (a) 2.1 (d) After referring to "cessation of employment medicals", no details or guidance is offered as to the aims of carrying out this procedure. For example, my recommendation would be to provide a summary report for the G.P. with guidance for his further surveillance of the individual.
- (b) There is no comment on the format of medical records. Standardised record card(s) might be helpful in standardising the medical surveillance procedures. In particular it is my own personal view that it is essential to have a recording procedure which allows easy comparison between current observations and previous ones to ensure immediate recognition of significant change. In current practice, this is not usually done.
- (c) It would be useful to have a recommendation from the M.A.P. covering the recording on the medical records of exposure data. (Specifically asbestos exposure, but other occupational hazardous exposure should also be recordable). Again, a difficulty currently experienced is that this information is usually not available at the time of the medical screening.
- (d) There is no discussion of collection of mortality data.
- (e) There is no discussion of the value, advantages, drawbacks or general scope of computerisation of medical records.

Dr. T. R. P. Goffe

00936

Amiantit

MEDICAL CENTER
Tel 03 - 8573200

To: Dr. R.B.K. Tucker
18 Rockridge Road
Parktown, Johannesburg 2193
South Africa.

our ref. : CM-132/84 your ref. :

Dammam : 05 May 1984

Dear Ron,

Re: WORKER'S TRAINING

For the last 9 months I have been directly involved with worker's training in Amiantit Saudi Arabia.

The following program has been applied:

A. Pre-employment

1. Verbal worker information by the safety engineer and medical officer.
2. Leaflets in different languages explaining what is asbestos, its possible harmful effects, the increased risk of smoking and how to work safely with the material.
3. Audio visual training: A movie entitled "good housekeeping" is projected, then slides of different working conditions in asbestos cement manufacturing are shown and discussed. These slides include good and bad situations, acceptable and unacceptable working conditions. The emphasis is not only on what the worker should do but on what he is not allowed to do.

Contd.....P/2

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B. At periodical examination:

1 and 2 are repeated.

C. Audio visual training in itself never stopped. Once a week 50 workers are shown the slides and the film. The same workers will have the opportunity to have this training twice a year.

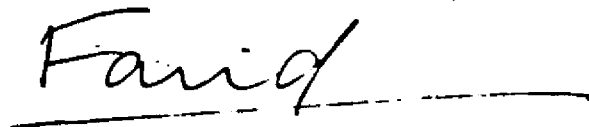
Fritz Baunach has sent me a set of interesting slides addressed to the worker. But they are directed more at the miner than the A/C worker. My feeling is that any audio visual should include:

1. General information
2. Specific information to the different asbestos industries.

Last September, I was approached by Camera Talks Limited of London for the production of an audio visual program specific to the asbestos cement industry. I referred them at the time to Mr. Crook of the Asbestos Information Centre - U.K.

I am sure the asbestos industry is "unfortunately" able today to provide us with slides of bad work practice which were supposed to have existed in the past but still exist in 1984 and could be used as a persuasive tool in a worker training program.

Yours sincerely,



DR. FARID MANSOUR

CC: Sir Neville Stack - AIA

00938

To: Medical Advisory Panel

AIA/20/1/HAS
21 March 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

RECEIVED

MAR 26 1984

With Compliments
Sir Neville Stack

H. C. LEWINSOHN, M.D.

Herewith copy of an article from "Occupational Safety & Health", March 1984:
"Lung Function Testing in Industry" by Dr. D. Courtney.

00939

UCC 000777

Lung function testing in industry

by Dr D Courtney

Introduction

Much attention has been given to occupational lung disease. This has been further heightened by the addition of occupational asthma to the list of prescribed diseases. Central to any assessment of lung disease is the need for objective measurement of lung function.

Lung function

The basic function of the lungs is to bring oxygen into close proximity to blood circulating in the body and to facilitate the transfer of oxygen from the lungs into the blood.

The lungs consist essentially of a branching structure of tubes (bronchi) along which air passes in and out of the lungs. These tubes terminate in air sacs (alveoli). These have very thin walls and come into close contact with blood vessels. It is at this point that oxygen is transferred from the air in the alveoli into the circulating blood.

There are about 200 million alveoli in each lung and the total surface area of the lungs where oxygen transfer can take place is approximately 80 square metres.

Clearly the two major situations in which lung function can be impaired is in the passage of air along the bronchi and also the diffusion of oxygen from the air

sacs into the bloodstream.

Use of tests

Lung function tests can be performed in a number of situations. At preplacement examinations an accurate baseline of an individual's lung function is useful for comparison at a later date.

If the person is likely to be exposed to any material which may have an effect on their lungs, such testing is very necessary. Follow up repeat examinations may be useful to detect any changes in lung function at an early stage. This may be done quite frequently initially to detect hypersensitive individuals to a particular substance and thus allow remedial action to be taken early. A typical example of such surveillance is found with workers exposed to isocyanates.

Lung function testing is also extremely useful in the investigation of individuals complaining of respiratory symptoms which may or may not be referable to some exposure at their work. Pre and post shift spirometry for a

number of days may be very useful.

It is important to ensure that any series of tests includes at least one weekend when the person is away from work. An additional side to such a regime is to use a peak flow meter. This can be issued to the individual who records his peak flow rates at regular intervals - about every two hours, both whilst at work and at home. This can often reveal clear changes in lung function related to a particular task or occurring at a particular time.

Types of tests

A variety of tests have been developed over the years in an attempt to measure the various factors involved in the efficient working of the lungs.

The elasticity of the lungs can be measured and gives an indication of how easily the lungs can expand and deflate. Measurements of the efficiency of the transfer of oxygen from the lungs into the blood stream can also be made. Whilst these are important tests they are complex and require both sophisticated equipment and highly trained technicians to carry them out.

The tests of lung function which can be carried out at the workplace with relative ease are those which measure various lung volumes. It is worth mentioning some of the more impor-

Above: A spirometer used for testing lung function.

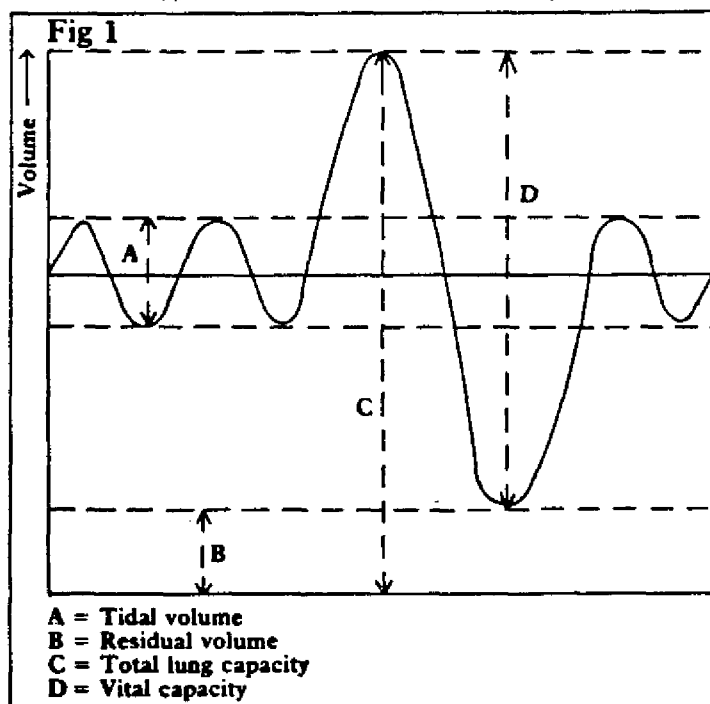
tant lung volumes as measured by a spirometer (Fig. 1). The total lung capacity is the volume of air in the lungs at the end of a full inspiration. The residual volume is the volume of air left in the lungs at the end of a full expiration and the volume is a finite and measurable volume of air, ie the lungs, even at full expiration are never completely empty. The tidal volume is the amount of air inspired or expired during a normal breath.

The vital capacity is the volume of air exhaled from full inspiration to full expiration.

Selection of tests

In selecting any test there are four main requirements. These are:

- 1) Acceptability - this factor is particularly important at the workplace and means that the test should be easy to carry out with the minimum of equipment and trained personnel and additionally should produce the minimum of discomfort and inconvenience to the subject.
- 2) Objectivity - the test should as far as possible be independent of the motivation, emo-





mental state and personality of both the subject and the operator.

- 3) Repeatability – consistent results should be obtainable at different times for the same subject.
- 4) Discrimination – this means that any test should be able to discriminate clearly the cause of any symptoms.

Equipment

There are two pieces of equipment which most closely fulfil these requirements and are therefore most useful in the investigation of lung function at the workplace. First is the spirometer which may be of two basic types – the piston spirometer or the wedge spirometer (Photo above). Both work on the principle that when the individual exhales fully into the machine a measure is given of both the total volume of air expired. A typical graph from a spirometer is shown in Fig 2 overleaf.

The two most useful volumes in practice are the Forced Vital Capacity (FVC) which is the

volume of air exhaled by a forced expiration after full inspiration and the forced expiratory volume in the first second (FEV_{1.0}) which is the volume of air expelled in the first second of a forced expiration after a full inspiration.

The other useful instrument is the peak expiratory flow meter (Photo 2). This gives actual rates of flow of the air passing along the bronchi.

Technique

In order that any results obtained may be reproducible it is important to carry out the test according to a set protocol. In the case of the spirometer the test can be carried out either with the subject sitting or standing, though the latter position is the most common and useful position. The entry point of the breathing tube should be adjusted to just below the level of the subject's mouth by raising or

lowering the spirometer. The subject should have the test carefully explained. They should fill their lungs as full as possible, place the mouthpiece of the spirometer in their mouth, ensuring a good seal and then should exhale as hard and as fast as possible. This should be repeated twice and these are known

as static tests since the carriage holding the chart does not move. Three dynamic tests are then performed. The technique for the subject is identical but in this case the operator depresses a switch which allows the chart to move across the spirometer as soon as air enters the machine.

Careful positioning of the chart in its carriage is important to ensure that the stylus starts at the correct point.

Results

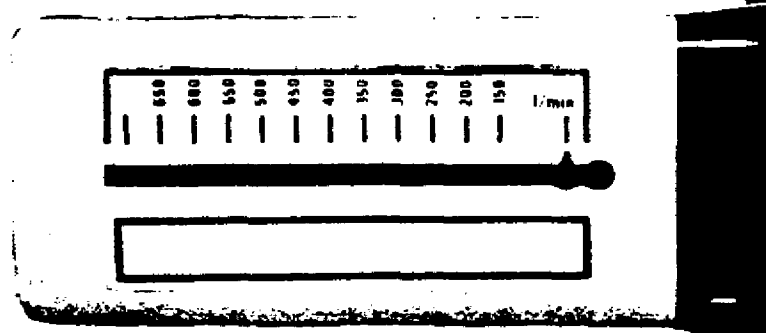
The best of the three traces should be taken and the FVC and FEV_{1.0} measured. These should be measured at body temperature and pressure saturated with water vapour (BTPS) which is to be found in the right side of the chart and adjusts the volume measured to allow for body temperature.

The two volumes give a measure respectively of the capacity of the lungs and the resistance in the bronchi to the flow of air. The FEV_{1.0} may also be calculated. This simply expresses the

Continued over



Below and right: A personal lung function tester which can be used by the patient.



Pictures courtesy of Visiograph Ltd.

Lung function testing in industry

Continued

proportion FEV_1 divided by FVC expressed as a percentage.

Two basic types of lung function abnormality can be distinguished. These are an obstructive pattern where the FEV_1 is reduced whilst the FVC remains relatively normal and a restrictive pattern where both FVC and $FEV_{1.0}$ are reduced (Fig. 2). The former is characteristic of obstruction of the airway, commonly found in asthma, whilst the latter is found in diseases where there is fibrosis of the lung tissues such as in the pneumoconioses.

Interpretation of results

By simply looking at a trace from a spirometer it is impossible to decide whether or not that trace is normal for that individual without further information. Lung volumes are influenced by a number of factors such as the sex of the individual, their age, their ethnic origin, their body

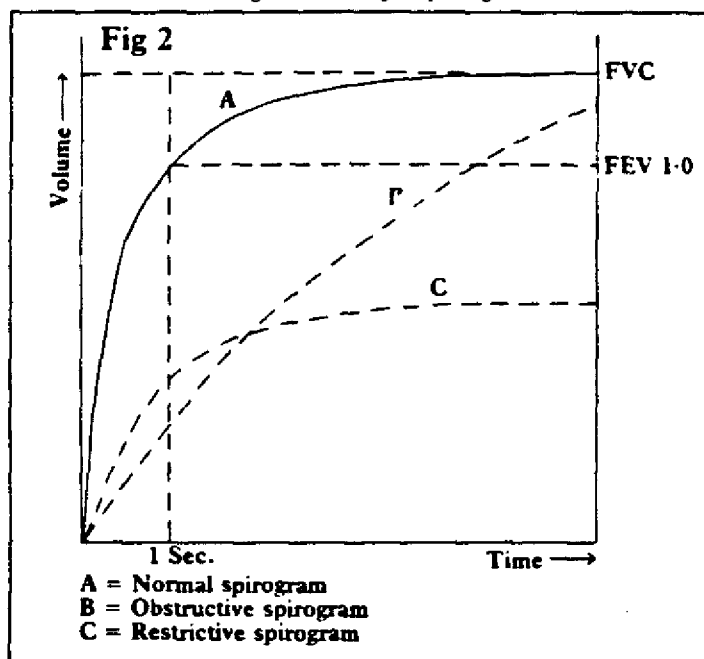
size and to a lesser degree the time of day when the test was taken.

The expected, or predicted lung volumes for any individual may be calculated from prediction equations or normograms. This allows a prediction to be made of what would be the normal lung volumes of that individual taking into account the factors mentioned above. Only when these predicted volumes are known and compared with the actual measured lung volumes can an interpretation be made as to whether or not the volumes obtained and thus the lung function could be considered abnormal.

Conclusion

Provided certain basic precautions are taken, lung function testing can usually be carried out at the workplace. It can be of help in the diagnosis of occupational and non occupational lung

disease as well as being an essential tool in the continuing surveillance of both individual and group lung function.



LABOURER'S LAW

A History of Occupational Health and Safety Legislation

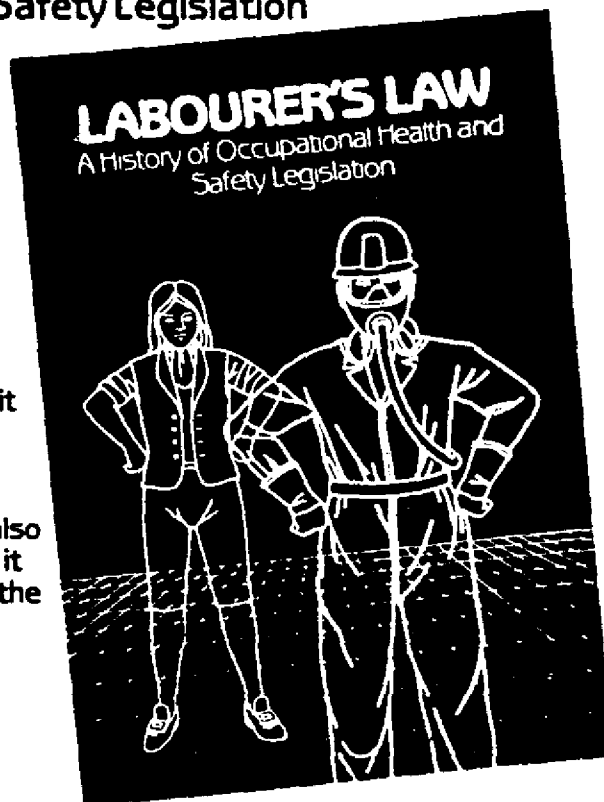
The reasonably healthy and safe working conditions that the British worker enjoys today are the result of a long, hard struggle. **Labourer's Law** tells the story of the 200 year battle to improve working conditions by outlining the progress of Health and Safety Legislation since the Industrial Revolution.

Managers, Safety Officers and workers in general will find that it provides them with all the background knowledge they need to fully appreciate the position they now occupy. But the book is written in such a highly readable style that it should also prove entertaining and informative to those with a general interest in social history. **Labourer's Law** seeks primarily to record the development of safety legislation, but in doing so also reflects the development of British culture making it a valuable reference for anybody who cares about the origins of the society in which they live.

Members Price (including carriage) £2.70

Orders, quoting ref. no. 15212 to:

The Royal Society for the Prevention of Accidents,
Cannon House, The Priory Queensway,
Birmingham B4 6BS.



To: Scientific Advisers
Medical Advisory Panel
Executive Committee

AIA/1/5/2/POL
10 April 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Herewith copy of a report by Dr. R. Murray on a visit to U.S.S.R.

COS43

RECEIVED
APR 17 1984
H. C. LEWINSOHN, M.D.

31 circ to [unclear]
[unclear] [unclear] [unclear]
[unclear] [unclear] [unclear]

5 APR 1984

1/5/2

ASBESTOS IN THE U.S.S.R.

I visited the Soviet Union from 11-18 March 1984 with the primary object of attending a meeting on Whole Body Vibration organised by Prof. Izmerov, under the auspices of the Scientific Committee on Physical Environmental Factors of the Permanent Commission. In the course of the visit Prof. Izmerov took me to visit Dr. Burgasov, the Deputy Minister of Health of the Soviet Union and I raised with him (as I had raised several times in correspondence with Prof. Izmerov) the possibility of going to Sverdlovsk.

Unfortunately, said the Minister, it would be necessary to have some five months notice of such a visit as there was an atomic power station in the area. I forbore to point out that I had already written to Izmerov on the subject when the vibration meeting was first mooted in October last year.

However, in view of my interest, he was prepared to invite a number of scientists from Sverdlovsk to meet me. Accordingly on 15th March I met Dr. B. Gurvich, Community Physician of the Sverdlovsk Region, Dr. Yelovskaya, Dr. N.A. Senkievitch, Dr. A. Rashevskaya, Professor in Advanced Medical Studies, Dr. Malenenkova, Dr. G. Dimova, Dr. Gledkhova, Epidemiologist, Dr. N. Pylov, Oncologist, Dr. Grigorian, Radiologist, Dr. V. Ostopkovich, ENT specialist, Dr. F.M. Kogan, Asbestos expert.

Allowing for the vagaries of translation and the problem of several people talking at the same time, the following is an account of our meeting.

I thanked them for coming (and have since written to the Minister thanking him for making it possible and reiterating

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my wish to visit Sverdlovsk) and gave them a brief rundown on the situation regarding asbestos in Western countries, in particular the high emotional temperature amounting to panic, but referring also to the increasing incidence of mesothelioma as evidenced by the Annual Reports of the DHSS.

Dr. Gurvich said that there was no panic in the Soviet Union. Asbestosis had considerably diminished. Some cases were still noted at the annual medical check-up, but these were among workers who had been employed for more than 20 years when conditions did not comply with the standard of $2\text{mgm}/\text{m}^3$ total dust as they do (for the most part) now. (Incidentally dust bronchitis is also recognised as an occupational disease). The majority have retired before evidence of asbestosis appears. No cases of cancer had been recognised at annual examination though the incidence is greater than in the general population. There has not been the same drop in cases of cancer as compared with asbestosis. The duration of exposure of lung cancer cases was longer. There was no epidemiological evidence of a multiplicative effect of asbestos exposure and smoking, though he thought that this might possibly exist.

Regulations governing the use of asbestos exist for all branches of industry using the material. The improvement of working conditions took place between 1958 and 1965 and 80% of workers now in favourable conditions started off in unfavourable conditions.

Professor Kogan addressed himself to the problem of mesothelioma. He said that the production was mainly chrysotile; there were only tiny deposits of amphiboles; mesothelioma was very rare, the same as that in the general population, but in the light of the evidence from MacDonald about the incidence of

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mesothelioma in a population exclusively exposed to chrysotile, he had started an investigation. He thinks that MacDonald is wrong, but it could be that mesothelioma is higher than in the general population in some branches of the industry. He will inform me of the results.

He stressed that there is no fear among the general population and the average man in the street does not know the meaning of asbestos. Professor Rashevskaya agreed and said that the average doctor did not know either. There was no publicity on television or in the press.

It could be argued that the Soviet press is just as skilful at suppressing information as ours is to fan the flames of fear and that the absence of exposure to amphiboles reduces the risk of mesothelioma, but the difference is nonetheless striking.

Russian scientists are associated with and familiar with the work of the IARC. They recognise the fact that asbestos is not acceptable in the West and they accept the evidence produced by the IARC about MMMF which they regard as valid. They are puzzled, however, about neighbourhood cases. With the Western evidence they would have expected the prevalence of lung cancer in towns around the asbestos mines to be higher, but in fact it is lower than in other cities.

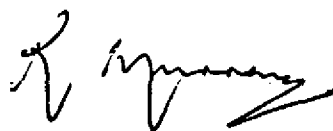
Dr. Senkievitch and Prof. Rashevskaya had worked as clinicians in the asbestos area for 30 years. They had seen cases of asbestosis but very few. It was difficult to get radiographs of asbestosis to show to students. The relationship of smoking and lung cancer is well recognised and almost all workers smoke. Cases of cancer associated only with asbestos exposure are very

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rare.

Compensation for asbestos related disease is not a problem. There are no possibilities for Soviet workers to make a claim against their employers (i.e. the State) for negligence. They were very interested in my description of a recent case in which a man is suing an electricity board for mesothelioma contracted, he said, as a result of exposure to asbestos for one week in 1947. They were particularly interested in peritoneal mesothelioma and the possible routes whereby asbestos fibres could affect the peritoneum. I will try to find some literature for them which might help their understanding, though I think this is very difficult.

Prof. Kogan gave me a paper published in the *Achivum Immunologiae et Therapiae Experimentalis* 1982, 30, 277. This is a journal published in Poland by the Ludwig Hirszfeld Institute of Immunology and Experimental Therapy. The Royal Society of Medicine gets this journal but had not yet received the particular number in which this paper appears. I attach a copy which I think it would be worthwhile circulating to the MAP and SCAP.



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To: Scientific Advisers
Medical Advisory Panel

AIA/20/1/16/HAS
10 April 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Herewith copy of paper " Changes in Occupational Morbidity Among Workers in
the Asbestos Mining Industry in Relation to the Improvement of Working Conditions"
by F.M. Kogan et al.

00948

RECEIVED
APR 17 1984
H. C. LEWINSOHN, M.D.

POLAND.

LUDWIG HRSZFELD INSTITUTE OF IMMUNOLOGY
AND EXPERIMENTAL THERAPY

ARCHIVUM IMMUNOLOGIAE ET THERAPIAE EXPERIMENTALIS
1982, 30, 277

PL ISSN 0004-069X

CHANGES IN OCCUPATIONAL MORBIDITY AMONG WORKERS IN THE ASBESTOS MINING INDUSTRY IN RELATION TO THE IMPROVEMENT OF WORKING CONDITIONS*

by

F. M. KOGAN, A. G. DEMINOV, N. A. GUSELNIKOVA and V. B. GURVICH

Institute of Labor Hygiene and Occupational Diseases, Popov Str. 30, 620014 Sverdlovsk, USSR

The improvement of technology and dust suppression measures in asbestos industry has decreased the dustiness at work places from 100 mg/m³ to 3–6 mg/m³. The asbestos morbidity decreased to single cases per thousands of annually examined workers and no cases were detected in shops where the dustiness was lower than the MAC (2 mg/m³). The lung and gastric cancer risk during the period 1950–1960 was higher than that of the population of the adjacent town. In the subsequent period no significant decrease of this risk was observed, which was probably due to the long latent period and incomplete efficiency of dust suppression means. Among the general town population; owing to the normalization of environmental conditions, the lung tumor morbidity in the last decade decreased to the level found in the general population of the entire republic. In the occupational pathology not only the thin and long fibres, but also the fibrogenic particulates of lizardite and antigorite — the major components of the dust, generating in this industry must be considered. In dose-response relationship more attention should be paid to the pattern of inhalation: continuous or intermittent.

Recent publications on occupational morbidity among asbestos workers are hardly optimistic. Despite of some improvements of working conditions in the British asbestos-processing industry, the number of new asbestosis cases increased from 20 in 1951 up to 189 in 1976¹. According to TRATTNER et al., 17% of all deaths during the coming ten years in the USA will be caused by asbestos-related tumors¹¹. In compliance with estimates of the US competent examiners (NIC, NIEHS, NIOSH), at least 40% out of 4 mln workers, with exposure to asbestos, may die of asbestos caused cancer; hence, expected mortality rate among the general population will not be greater than 8 to 9%. As to the forecast, up to 75 thousand people will die yearly, because of asbestos caused diseases particularly 13 to 18% will die of cancer².

Many authors pointed out the increasing occurrence of lung gastric and laryn-

* This paper was scheduled, however, not presented during the conference.

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geal cancers and also of pleural and peritoneal mesotheliomas among the workers with different kinds of asbestos exposure who were employed in various industries even, when asbestos exposure lasted less than one month (for reference see⁶).

We assume, however, that it is possible to reduce markedly the risk if the engineers and industrial hygienists will cooperate together.

We have developed and introduced a program of measures for reducing of dustiness at asbestos mines and mills. In ore extraction processes special attention was paid to mechanization of jobs dealing with extraction and transportation of mining masses. Due to the improvement of sealing facilities of cabs, spraying of excavating faces, preventive maintenance of dust traps, etc., the dust content in excavators, drilling machines and trucks cabs was sharply reduced. Dust content in ambient air of open pits has been reduced at present by means of dust-suppression solution with which roads are regularly sprayed. At most of the working places the dust content has been reduced from 38–74 mg/m³ down to 2–13 mg/m³.

Asbestos mills which were constructed before the second world war were gradually eliminated. Newly built mills are outfitted with dust-proof equipment (crushers, screens, separators, dust removers). Extra-long conveyers were enclosed and aspirated. Highly effective installations are provided for dust cleaning of the aspiration air. Particularly, in this country highly effective cleaning of dusty air was introduced at the world-largest asbestos concentrating mill in bag-hose strainers of enormous extension. Test results indicated that the residual dust content in filtered air had not been in excess of 0.3 to 0.6 mg/m³, i.e. it had been less than MAC for in-flowing

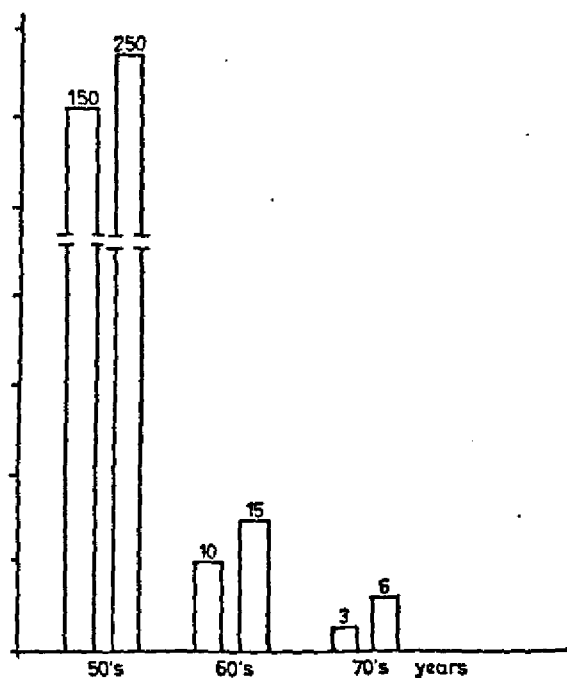


Fig. 1. Average values of dustiness in asbestos mills during the last three decades (mg/m³).

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air. After being cleaned, air is directed back into the shop together with heat generated by working machines and electromotors; thus the microclimate may be normally ensured.

The mentioned above measures caused that dustiness in various shops of asbestos mills was sharply reduced from 150–250 mg/m³ in the 50's down to 3–6 mg/m³ (mean values). However, the latter levels are still above the MAC – 2 mg/m³ (Fig. 1), it must be stressed that all the work leading to the improvement of working conditions was conducted simultaneously with an increasing quantity of processed mining mass in tens-time figures. In the meantime, short-time rises of dustiness were noticed which happened due to out-of-time elimination of troubles, derangements in aspirating systems, careless waste removal.

Dust content in the atmospheric air of the neighbouring city was also considerably reduced. In the 50's the dust content in the air at a distance of 500 m, and also at distances of 1000 and 1500 m considerably exceeded the acceptable values⁴. At present the mean dust content in the atmospheric air of the neighbouring city is limited, on average, to the acceptable values (0.5 mg/m³). No dust concentration in excess of the acceptable value has been found at distances of 1 to 3 km (the mills are located from the city at greater distances).

: As the anti-dust measures complex program has been put to life, the rate of occupational disease — asbestosis — of the workers is considerably reduced. Yearly many thousands of workers of "dusty" professions undergo medical surveys. As com-

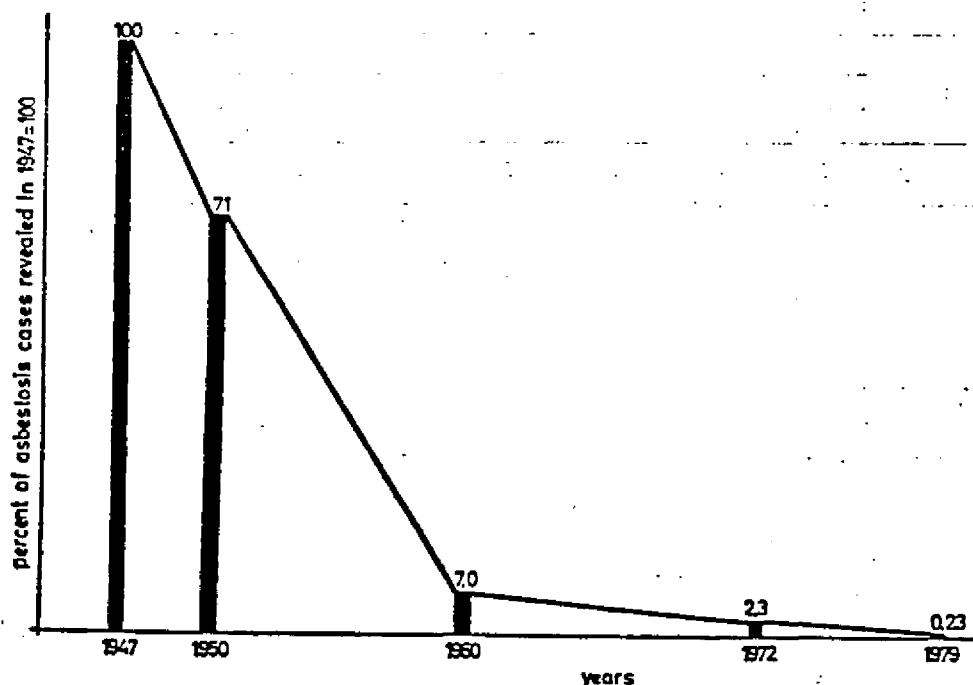


Fig. 2. The occurrence of asbestosis among the asbestos mill workers (number of asbestotic cases diagnosed in 1970 was taken as 100%).

pared with the 1947 data, the extent to which asbestosis had spread was sharply reduced to the beginning of the 60's. During 1974–1979 the number of newly diagnosed asbestotic cases was about ten times lower than in the 1963–1972 period (Fig. 2).

Thus, the morbidity due to asbestosis is now lower in the USSR than in other regions (Canada, Rhodesia, Cyprus, SAR). Recently, no cases of asbestosis of 2nd and 3rd degrees have been detected; asbesto-tuberculosis cases have been considerably reduced. At present asbestosis is rarely revealed, mainly in workers with long-term exposure to asbestos (15–32 years).

Scientific and technical progress in asbestos industry as well as in other industries, was accompanied by an increasing number of workers who ensure adjustment, cleaning and repair the equipment. Our investigations have proved that repair work on the equipment causes an intermittent exposure.

The mortality caused by malignant tumors in asbestos mines and mills workers was much higher than in the population of the neighboring city. These differences are probably due to a long-term effect of considerable dust concentrations which were especially high in the first ten post-war years.

Table 1 illustrates risk indices i.e. relations between observed and expected death rates of cancer for workers employed in asbestos mines and mills during two periods: 1) 1948–1967 and 2) 1968–1979.

Table 1. Relations between observed and expected mean-year mortality rates (standard mortality ratios) of cancers of different locations during two time-periods

Tumor	Men				Women			
	1948–1967		1968–1979		1948–1967		1968–1979	
	extr.	concent.	extr.	concent.	extr.	concent.	extr.	concent.
Pulmonary	3.9	4.3	2.9	5.8	3.9	2.9	9.4	9.7
Stomach	2.6	3.6	2.8	1.9	2.8	6.0	5.3	5.0
Enteric	2.9	—	2.4	2.8	1.9	2.6	1.9	10.6
Uterus	—	—	—	—	1.5	5.1	7.4	7.6
Others	3.5	3.5	3.1	2.9	1.1	2.9	2.3	2.1
Total	3.3	3.4	2.7	3.4	2.1	4.7	3.9	4.4

The risk index (RI) of cancer mortality of all locations for men miners has lowered from 3.3 to 2.7 and for women employed in various occupations in mines it has risen from 2.1 to 3.9 (Table 1). At mills the lung cancer risk index has risen for both women (2.9 and 9.7) and men — (4.3 to 5.8). At asbestos mills the gastric cancer risk index has gone down for both women and men, but it has risen for women employed at mines and is still unchanged for men miners (2.6 to 2.8). Among women employed at mines and mills the uterus cancer risk index has also risen.

Anti-dust operating conditions were widely initiated at asbestos mills at the beginning of the 60's; therefore, workers with long exposure worked at these mills under reduced but, unfortunately, not normalized dustiness. This is the reason why no trend is observed for reduction of lung cancer risk indices. The stomach cancer

risk indices are either reduced or stabilized. Maintenance workers who are exposed to dust intermittently show somewhat lower rates than those employed in main occupations.

Our results are in compliance with those published in Great Britain⁸. Workers who were employed in an asbestos mill for 10 to 19 years under conditions of reduced but not normalized dust content had the lung cancer risk index two times higher for men and three times for women, as expected.

NICHOLSON stated that the decrease of morbidity due to asbestosis not accompanied by the reduction of a risk of cancer⁷. It is worth noting that, in the 50's the lung cancer mortality for the population of the city close to the mills was, as a rule, considerably higher as compared with the data specific for the region (2.2 times, in average, during 1955—1962). During the last 15 years after efficient anti-dust measures had been introduced, the death rate for the population became lower (by 20 to 25%). Thus, the lung cancer death rate as measured for "check" groups was considerably reduced, which resulted, to a certain extent, in an increase of the lung cancer risk index among asbestos workers.

Despite of considerable reduction of air dustiness and thus of risk of asbestosis, a risk of malignant tumors is still alive among workers employed in asbestos industry. The prevention of asbestosis and the high oncologic risk call for a stable reduction of dust content in ambient air down to the MAC. However, the specialists in occupational medicine have no common view on the criteria of establishing this value.

In this country the MAC for airborne dust in the working area which contains more than 10% of asbestos — is 2 mg/m^3 . Clinical and hygienic examinations carried out at the Moscow asbestos-textile plant proved rightfulness confirmed the MAC accepted in the USSR — 2 mg/m^3 : no asbestosis disease case was detected in 100 workers who had worked under conditions of dust content less than 2 mg/m^3 for at least 20 years.

In the majority of the Western countries the MAC value for asbestos dust content is, as known, 2 fibres longer than $5 \mu\text{m}$ per 1 ml of air as measured through an optical microscope with $\times 400$ magnification. Examiners completely disregard short-size fibres and grains of the ore, which constitute the most part of particles in extraction and concentration processes.

This attitude would be rightful, if absolute biological inertness of serpentine ore grains was undoubtedly proved. In publications of STANTON et al.¹⁰, POTT⁹ and others it was stated that only the fibres of not more than $0.2 \mu\text{m}$ in thickness and of more than $10 \mu\text{m}$ in length are especially biologically aggressive. Meanwhile, it is extremely hard to expose and count such fibres by the standard method with the use of an optical microscope, since, as known, $0.2 \mu\text{m}$ is the extreme limit of the optical microscope resolving power. As a result, most aggressive particles may be left uncoun-
ted. Besides, most of the particles are considered non-aggressive without sufficient grounds for it. We decided to research the fibrogenity of the grains of the ore containing not more than 4—5% of asbestos fibres or containing no such fibres at all. Ores which accompany chrysotile-asbestos contains up to 37% of chrysotile serpentine, 23% of lysardite serpentine, 10% of antigorite serpentine (the rest — for diorites).

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In experiments with white rats it was found that tracheal introduction of chrysotile serpentine, lysardite and lamellar serpentines, caused lung fibrosis which was proved not only by histological comparisons but by determination of the collagen quantity in lungs. Meanwhile, chrysotile serpentine containing 4–5% of chrysotile asbestos fibres causes fibrosis relatively more expressed (though insignificant statistically) as compared with lysardite and antigorite serpentines which do not contain such fibres (Table 2).

Table 2. The average absolute content of oxyproline in the white rat lungs after 9 months intratracheal instillation of asbestos ore samples

Sample	Average oxyproline content in $\mu\text{g}/100\text{ g}$
Chrysotile serpentinite	1419.7 ± 52.7
Lysardite serpentinite	1196.5 ± 46.4
Antigorite serpentinite	1154.1 ± 58.6
Control group	741.4 ± 51.6

Thus, it seems unreasonable, when determining the concentration and evaluating it, to disregard particles of the above mentioned components of airborne dust. It seems more reasonable in such case, to use the gravimetric method for evaluation of the dustiness in asbestos mines and mills, as well as the optical microscope evaluation of asbestos fibres.

As known, a dust exposure concept is widely used in epidemiological examinations, which characterizes both mean concentration value and exposure time. We think that it is insufficient. The recently conducted experiments⁴ were successful to illustrate that a considerably more exposed fibrogenic effect had been observed, in condition of equal dust exposure in an inhalation experiment, in animal lungs held in continuous exposure of less concentration of asbestos dust as compared with those animals which were subject to intermittent dust exposure (Table 3). Later, DAVIS drew similar conclusions⁵.

Table 3. The average content of oxyproline in the rat lungs after 9 months inhalation of chrysotile containing dust in permanent and intermittent exposure (at equal total dust masses)

Regime of inhalation	Oxyproline content in μg per 100 g
Permanent every day $75\text{ mg}/\text{m}^3$	1615.3 ± 80.6
Intermittent ($150\text{ mg}/\text{m}^3$ every 3rd day)	1375.3 ± 65.0
Control group	976.6 ± 91.2

It was also shown that fibrogenic effect of dust upon animals of both sex is practically the same.

All this should be taken into account when evaluating the fibrosis risk in asbestos workers under different working conditions.

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CONCLUSIONS

1. A complex program of anti-dust measures sharply reduced the dustiness in airborne air in working areas at asbestos mines and mills.
2. The reduction of exhausts into air of the neighbouring city caused a reduction of dust content in the city air down to the MAC.
3. Asbestosis morbidity workers employed has been greatly prevented. But no trends are observable yet for reduction in malignant tumor death risk indices among asbestos workers. The oncological mortality for the adjacent city population is lower than that of the mean value for all the region during many years.
4. When evaluating the epidemiology of asbestos-caused diseases, apart from thin and long fibres, short-length fibres and the content ore dust should be taken into account. Besides, an important information is if the dust exposure conditions are continuous or intermittent.
5. The asbestos MAC — 2 mg/m^3 — reliably safeguarded against asbestosis during two ten-year periods. It is also useful to perform examinations to determine the oncologically safe level of air contamination with asbestos-containing dust.

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To: MAP

AIA/20/1/4/HAS
25 April 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Herewith copies of comments from Dr. Murray and Dr. Rao re "New Scientist" item on the US National Research Council study on "Non Occupational Health Risks of Asbestiform Fibres". Also enclosed is an extract from AIA/NA News & Notes which indicates where copies of the report can be obtained.

00956

From: AIA/NA News + Notes, February 29, 1984

(20/1/4)

ACADEMY ISSUES REPORT ON NONOCCUPATIONAL
HEALTH RISKS OF ASBESTIFORM FIBERS

A committee of the National Research Council - National Academy of Sciences issued a 334-page report earlier this month on "Nonoccupational Health Risks of Asbestiform Fibers." This study was conducted under a contract from the Environmental Protection Agency. Lester Breslow, School of Public Health, University of California, served as chairman of the committee.

The report is replete with stress on the highly uncertain extent of risk from nonoccupational exposure to asbestiform fibers in air to human health. For example, at one juncture the statement is made that estimating the extent of health risk from such low exposures is "fraught with uncertainty." The committee then proceeds to make a quantitative estimate of the risk of excess lung cancer and mesothelioma that "might" occur in persons breathing low levels of asbestos in the air. Based on the committee's estimated level of exposure to 0.0004 f/cc for a 73-year lifetime, "approximate" risk of 10 excess deaths in a million is predicted (nine mesotheliomas and one lung cancer). The committee then emphasized: "Because of the great reliance on assumption and on clearly deficient exposure and effects data, the committee views these risk estimates as guides to the qualitative assessment of non-occupational health risks from asbestos and asbestiform fibers - not as definitive estimates of the amount of disease to be anticipated."

A part of the committee's mandate from EPA was to also evaluate the public health risk from fibrous minerals other than asbestos and from the man-made substitutes, such as rock wool and fibrous glass. The concern is that the asbestos look-alikes also might be carcinogens because they have many of the same physical properties as asbestos. The term "asbestiform fibers" includes all of them for purposes of the study.

Results of scientific studies of the man-made asbestiform fibers, mostly fibrous glass, have been "equivocal," the report pointed out. These materials have not been in use nearly as long as asbestos; concentrations in the workplace have been lower, and fewer workers have been exposed. Studies done before 1980 did not, in fact, suggest any serious health risks at all. But the studies done after 1980 begin to tell a different story. Most of them report "excess" lung cancer deaths; that is, more lung cancer deaths than one would expect to find if the fibers were not implicated. The evidence is neither clear nor consistent, however, and the results of long-term mortality studies are not yet in.

Copies of the Academy's report may be obtained from the National Academy Press, Room 700, 2100 Pennsylvania Ave., N.W., Washington, D.C. 20418/(202) 334-3113 at a cost of \$22.50 each.

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DR. ROBERT MURRAY OBE, B.Sc. FRCP (Glas) Hon. D.Tech (Brad)
DPH, D.H., FFOM, Hon. FIOSH
Consultant in Occupational Health

120 TEMPLE CHAMBERS, TEMPLE AVENUE, LONDON EC4Y 0DT

TELEPHONE 01 353 0518

Re: RM/SR/29/5
Your ref

13th March 1984.

Sir Neville Stack
Asbestos International Association
68 Gloucester Place
London W1H 3HL

15 MAR 1984

Dear

re: Asbestos Risks - New Scientist - 16 February 1984

Thank you for sending me this typical extract from New Scientist.
I like "cancer - though probably not very often". It is like
saying that the maid servant was only slightly pregnant.

I do not know the study by the National Research Council in the
United States and I think it would be important to have a look
at this study before commenting on the "marked increase" among
young children.

Kind regards

Yours sincerely

Dr. Robert Murray

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PLEASE ADDRESS
REPLY TO A.C. DIVISION

Our Ref: AC:AV:Dr:84

Date: April 3, 1984.

The Director,
Asbestos International Association,
68, GLOUCESTER PLACE,
LONDON W1H 3HL

12 APR 1984

Dear Sir,

Enclosed are my comments on "Asbestos Risks" from 'New Scientist' 16th Feb '84, circulated through AIA circular No. AIA/20/1/4/HAS at 1st March, 1984.

Mesotheliomas in children were described before commercial use of Asbestos and none of them were proved to be Asbestos related on the basis of postmortem studies. (K. Browne).

Latent period for Mesothelioma in Asbestos exposed population is 30 years, hence, if Mesothelioma recorded in young children could not be due to Asbestos exposure. (Shortest latent period for Mesothelioma in Asbestos workers are 13.3 yrs - Hobbs 1980).

Further details, for the basis to say children's lungs may be specially sensitive to result in Mesothelioma are required.

Hence, I will appreciate if you can provide a copy of study by the National Research Council U.S., which makes the above statement.

Thanking you,

Cordially yours,

DR. S. P. VIVEKCHANDRA RAO
Medical Surveillance Unit.

Dr/rs.

00959

REGD. OFFICE: SANATNAGAR, HYDERABAD-500 018. (INDIA)

UCC 000797

To: MAP

AIA/20/1/2/HAS
AIA/20/1/4/HAS
25 April 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Herewith copies of two letters dated 16 April from Dr. S.P.V. Chandra Rao re:
(1) Article in 'The Practitioner' by Dr. M.L. Newhouse: "Asbestos Related Diseases"
(2) Second article by Dr. G.H.G. McMillan "Asbestos Related Diseases" in
'The Safety Practitioner' of February 1984.

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UCC 000798



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PLEASE ADDRESS
REPLY TO A.C. DIVISION

Our Ref: AC:MSU:84

Date: 16.4.1984

The Director General,
Asbestos International Association,
68, Gloucester Place,
LONDON W1H 3HL.

Dear Jimmy,

This is regarding Dr.G.H.G.McMillan's second article on "Asbestos related Diseases" appeared in "The Safety Practitioner" of February 1984.

First line of the article says, almost all Mesotheliomas are due to Asbestos exposure. As you are aware, this is not so, hence this point may be discussed with Convenor, Chairman and members of MAP, and the necessary action may be taken.

But the same article also says that there is no risk of Lung Cancer or Mesothelioma due to Asbestos in Urban or rural air pollution. This is a good point for Industry but further details are needed for argument sake.

Best regards,

Yours cordially,

(Dr.S.P.VIVEK CHANDRA RAO)

00961

REGD. OFFICE: SANATNAGAR, HYDERABAD-500 018. (INDIA)



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Tel: 262401 (10 LINES) Telex: 0155-210 & 561 Grams: ASBESTOS

PLEASE ADDRESS
REPLY TO A.C. DIVISION

Our Ref: AC:MSU:84

Date: 16.4.1984

The Director General,
Asbestos International Association,
68 Gloucester Place,
LONDON W1H 3HL.

Dear Jimmy,

Having read the article "Asbestos related Diseases" by Dr.M.L.Newhouse published in 'The Practitioner', September 1983, circulated to members of MAP, I enclose my comments. Please take the opinion of Convenor, Chairman and other members of MAP and further action may be taken accordingly.

Referring to signs of Asbestosis, Dr.Newhouse is of the opinion, "Examination often shows clubbing". But in view of MAP members, and the document "Criteria for Diagnosing Asbestos related Diseases" by MAP, clubbing of fingers may be seen in case of Asbestosis, but it is not often.

Regarding radiological diagnoses of Asbestosis, Dr.New house opines, blunting of the costophrenic angles usually occurs in early cases of Asbestosis.

Blunting of costo phrenic angles is due to pleural reactivity, which is only an indication of exposure to Asbestos. Moreover, Asbestosis an interstitial lesion, and blunting of costophrenic angles a pleural lesion, hence it does not form a part of radiological diagnoses of Asbestosis.

As regards, pulmonary function test results, article mentions that signs of constriction are seen, which means both restrictive and obstructive. If the author anticipates obstructive changes also a further explanation is needed like in which stage of the disease, and the role of smoking in it.

While discussing prognosis, "Asbestosis may progress after exposure ceases", here a further positive information to be added like, further progress of the disease depends upon the severity of Asbestosis at the time of diagnoses, and also it is the individuals susceptibility that determines it.

(contd.. 2..)

REGD. OFFICE: SANATNAGAR, HYDERABAD-500 018. (INDIA)

00962



HYDERABAD ASBESTOS

CEMENT PRODUCTS LTD.

Sanatnagar, Hyderabad-500 018.

Tel: 262401 (10 LINES) Telex: 0155-210 & 561 Grams: 'ASBESTOS'

PLEASE ADDRESS
REPLY TO A.C. DIVISION

Our Ref: AC:MSU:84

Date: 16.4.1984

: 2 :

Commenting on effect of smoking in Asbestos workers and relating to Lung cancer, it is rated as 50 times higher, when it is only 5 times in non smoking Asbestos workers. This may not hold good for the present working conditions, and higher rate of Lung cancer is seen as the dose of Asbestos increases. (Mc Donald study).

The above comments are not to make mountains out of Mole Hills, but one who follows this criteria for diagnosing Asbestos related diseases will not be self sufficient.

with regards,

Cordially yours,

(Dr. S.P. VIVEK CHANDRA RAO)

REGD. OFFICE: SANATNAGAR, HYDERABAD-500 018. (INDIA)

00963

UCC 000801

ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

Your Ref. Tng/Hlg

Our Ref. AIA/13/COMP

Telephone : 01-486 3518/9

Telegrams : Intag London

Telex : 298618 INTA G

Mr. Göran Truedson,
Svenska Brossbandsfabriken AB,
88020 Langsele,
Sweden.

25 April 1984

Dear Göran,

Pleural Plaques - Compensation in Sweden
Medical Investigation Committee

Thank you very much for your notification of 12 March about compensation being awarded at present for pleural plaques in Sweden and that a Medical Investigation Committee would look into the policy after 1984.

There are a number of papers showing that pleural plaques:-

- (a) are an indication that asbestos fibres have been inhaled;
- (b) are not a cause of disability.

In a recent Report* the British Government stated that, in deciding whether lung cancer should be attributed to working with asbestos (and therefore attract compensation), the presence of pleural plaques would not be a factor in the judgement.

It would perhaps be useful to forward relevant Reports and Papers to the Swedish Medical Investigation Committee, and I would be happy to send them to you for onward transmission or to anyone concerned whom you might indicate.

Yours very sincerely,
Jimmy Stack

Sir Neville Stack

cc AIA/20/1/15/HAS

MAP

CAIC

* Asbestos Related Diseases without Asbestosis (Cmd 9184 March 1984)

Directors: Mr. E. van der Rest (Belgium); Mr. E. Costa (Italy); Sir Neville Stack (Director General & Secretary)
Reg. no. 1392454 England Registered office as above

00964

UCC 000802



SVENSKA BROMSBANDSFABRIKEN AB

Datum

8th March, 1984

Er brev

Vår beteckning

Tng/Hlg

Er beteckning

Sir Neville Stack
Asbestos International Ass.
68 Gloucester Place
London W1H 3HL
ENGLAND

12 MAR 1984

Ref: Info - Sweden

A medical investigation committee has been set up by the central Swedish Employers federation and the Central Workers- and Staff unions of Sweden, in order to investigate the medical harmful effects that individuals could achieve by working with asbestos.

It has also been decided between above mentioned parties that anyone who in working life has got pleura-plaques due to asbestos work would be compensated with a lumpsum of 10 000 SEK if the pleura-plaques has been detected between 31.12.78 - 31.12.84. After the later date the findings of above mentioned committee will influence the standpoint for compensation.

The compensation money will be paid from the insurance that is obligatory for all Swedish companies.

Best regards

Göran Truedson

(dictated by mr. Truedson and
signed in his absence)

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Postadress	Telefon	Telegram	Telex	Bankgiro	Postgiro
880 20 LANGSELE	0620-217 50		6151 SBFL	848-0097	9 93 98-0
Försäljningskontor					
435 00 MÖLNLYCKE	031-88 40 50	Svebrofa	209 59 SBFS		

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ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

MEMORANDUM

TO: All Member Associations
Members of Governing Council/Executive Committee
Members of EAC

FROM: Director General

RECEIVED

APR 17 1984

H. C. LEWINSOHN, M.D.

OUR REF.: AIA/1/POL

9 April 1984

Organisation Chart and Annexes

Enclosed are copies of the following Annexes, all dated 1 April 1984, which have been amended to incorporate recent changes:-

1. Annex A - Governing Council Members
2. Annex B - Executive Committee Members
3. Annex C - Member Associations & their Officers
4. Annex D - List showing Countries, Associations & Members of Governing Council and of Executive Committee
5. Annex E - EEC Advisory Council
6. Annex F - Dust Advisory Panel
7. Annex G - Medical Advisory Panel
8. Annex H - Building & Construction Advisory Panel
9. Annex J - Communications Advisory Panel
10. Annex L - Fibre Producers Advisory Panel
11. Annex M - Sealing Materials Advisory Panel
12. Annex N - Textiles Advisory Panel
13. Annex P - Scientific Advisers

Earlier copies should be destroyed.

Please let us know if there are any corrections which should be made to these lists.

Tn Stack

Sir Neville Stack

cc Annexes F, G, H, J, L, M, N, P to Panel Members concerned

00966

Annex G to Organisation Chart

ASBESTOS INTERNATIONAL ASSOCIATION
MEDICAL ADVISORY PANEL
(MAP)

Dr. R.B.K. Tucker, (Chairman) 18 Rockridge Road, Parktown, Johannesburg 2193.	SOUTH AFRICA
Dr. J. Lepoutre, S.A. Eternit, 2920 Kapelle-op-den-Bos, Belgium.	CIAB/CVA
Dr. B. Korsgaard, Dansk Eternit-Fabrik A/S, P.O. Box 763, DK-9100 Aalborg.	DENMARK
Dr. C. Raffaelli, Societe VALEO, BP No. 30, Conde-sur-Noireau.	FRANCE
Dr. F. Mansour, c/o Amiantit, P.O. Box 589, Dammam 31421.	SAUDI ARABIA
Dr. S.P. Vivek Chandra Rao, Hyderabad Asbestos Cement Products Ltd., Hyderabad 500 018 A.P.	INDIA
Prof. F. Capellaro, c/o Ferodo Italiana SpA, Corso Inghilterra 2, 12085 Mondovi (Cuneo).	ITALY
Dr. S.F. McCullagh, James Hardie Industries Ltd., GPO Box 3935, Sydney, NSW 2001, Australia.	SPAA
Dr. K. Browne, Cape Industries PLC, 114 Park Street, London W1Y 4AB.	UNITED KINGDOM

Annex G to Organisation Chart

Dr. Hilton C. Lewinsohn,
Assistant Corporate Medical Director,
Union Carbide Corporation,
Old Ridgebury Road,
Danbury,
Connecticut 06817.

USA

Corresponding Members:

Dr. M. Lesage,
Lesage, Boivin & Associates,
235 Boulevard Dorchester Est,
Suite 305,
Montreal, Quebec, H2X 1NB.

CANADA

Convenor:

Dr. R. Murray,
120-122 Temple Chambers,
Temple Avenue,
London EC4 YODT.
England.

AIA/1/POL

1 April 1984

00968

To: Medical Advisory Panel
Mr. A. Sharon

AIA/20/1/23/HAS
7 August 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Re: Paper by John R. Goldsmith "Health Hazards of Asbestos: Charting a Path for Israel"
(sent to you on 14 May 1984) - herewith copy of comments by Dr. K. Browne.

00969

Cape Industries PLC

114 Park Street London W1Y 4AB



Memorandum to Sir Neville Stack

From Kevin Browne

Subject Worker Health Hazards from Asbestos -
AIA/20/1/23/HAS

Date 3rd August, 1984

To 6/8

To answer the easy question first, I think Goldsmith is not intending to be understood as saying that all mesotheliomas are due to asbestos, which we know they are not. In this context I think he is implying that if a mesothelioma occurs in an asbestos-exposed worker (whose exposure was substantial and started at least 15 years ago) the odds are so high that it was due to asbestos exposure that it is not worth looking for any alternative.

The main question has no simple answer. A particular difficulty is that an asbestos worker's disability may have resulted from exposure to very high airborne asbestos dust levels 20 or more years ago, whereas his present work may involve exposure only to very low levels. Factors to be considered in addition are:

- his age - is he capable of retraining or adapting to an alternative job?
- his job - is he physically capable of continuing?
- his alternative - is other employment available?
- his mental state - would he be removed from a job he enjoys or is skilled at to one less pleasant or less challenging or satisfy?
- his respiratory state - can he tolerate respiratory protection if this is necessary in the job?

In short, all aspects of his present job and prospects if this is taken from him must be balanced against the 'no dust' ideal.

J.E. Tuck-Martin
PP KEVIN BROWNE

00970

To: Medical Advisory Panel (for information)

AIA/20/1/HAS
10 August 1984

ASBESTOS INTERNATIONAL ASSOCIATION

68 GLOUCESTER PLACE,
LONDON, W1H 3HL
TEL: 01-486 3528/9

With Compliments

Herewith copy of a letter dated 10 July to Dr. Kevin Browne about comments on asbestos and health questions.

00971

ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

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Your Ref.

Our Ref. AIA/20/1/HAS

Dr. K. Browne,
Cape Industries PLC,
114 Park Street,
London W1Y 4AB

10 July 1984

Dear Kevin,

Asbestos & Health Questions

We spoke the other day about certain comments on the asbestos and health problem which occur regularly in articles, discussions (the law courts!) etc. and which could with advantage be listed.

The comments are usually over-simplified statements of fact which are not easy to refute. They also tend to have a scare factor.

In many cases, while there is some truth in what is stated, there is usually a qualification or two omitted which if expressed could radically modify the impact of the statement.

The aim is to isolate the offending statements and then to see if there is sufficient evidence in the shape of learned papers to enable us to raise a doubt in people's minds by showing another angle to each statement; i.e. not necessarily a refutation, but more of an "ah, but have you thought of this point?".

It would be hopeless to go into detailed arguments from the learned papers but one should know of them and be confident that they support one's views and could if necessary be produced subsequently.

Attached are nine statements and on a separate sheet a few ideas in response to help start a process of argumentation.

I have picked the nine questions at random and have probably left out the important ones!

Do you think that you could support the responses?

I am copying this to Brian Commins who has expressed an interest.

Yours ever
Jimmy

Sir Neville Stack

Directors: Mr. E. van der Rest (Belgium); Mr. E. Costa (Italy); Sir Neville Stack (Director General & Secretary)
Reg. no. 1392454 England Registered office as above

00972

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AIA/20/1/HAS

ANNEX 1
to letter dated 10 July 1984

Some Statements

1. One fibre can kill (i.e. mesothelioma and cancer are not dose-related).
2. Only asbestos fibre causes mesothelioma.
3. All fibre sizes are equally dangerous.
4. Asbestos inhalation causes cancer.
5. Asbestos ingestion causes cancer.
6. Children have developed mesothelioma from asbestos exposure.
7. Working in asbestos factories causes thousands of cancer deaths in the UK (or US etc. etc.).
8. The general population is at risk from asbestos.
9. There is no threshold level for asbestos fibre.

C0973

ANNEX 2
to letter dated 10 July 1984

Some Comments on the Statements

1. (a) If one fibre kills, then we should all be dead since asbestos, a naturally occurring mineral, is everywhere and we have been inhaling fibres since birth;

(b) there are papers showing that asbestos-related diseases are dose related;

(c) this is the same logic as saying that one cigarette can kill.
2. Baris and others has shown otherwise.
3. There is increasing evidence that the longer fibres are the dangerous ones ($> 5\mu$).
4. Cancer-causing substances are now divided into "initiators" and "promoters". The second type (of which asbestos is one) cannot by themselves cause cancer.
5. This is now generally accepted to be untrue.
6. There are no documented cases of childhood mesothelioma.
7. (a) The proportion of cancer deaths due to occupation is 4%, of which that due to asbestos forms a part.

(b) Deaths due to other occupations far exceed those due to asbestos work.
8. This is untrue. The levels experienced by the general population are about 3 orders of magnitude less than those at the workplace which are considered safe.
9. Common sense suggests there must be - see 1. However, it is difficult to prove.

CC974

~~SECRET~~

ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL, ENGLAND

MEMORANDUM

TO: MEDICAL ADVISORY PANEL

OUR REF.:

FROM: DIRECTOR GENERAL

AIA/20/1/16/HAS

4 September 1984

Subject: Cohort Study of the causes of death among asbestos
exposed repair shop workers - Ohlson et al (Orebo
railway workshop workers)

Enclosed is an English translation of parts of this study including
the Tables and Figures.

We also include a copy of the comments made by Dr K Browne.



for

DIRECTOR GENERAL

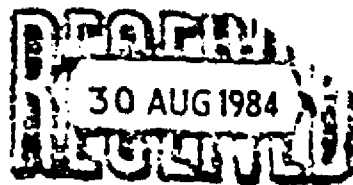
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RECEIVED
SEP 24 1984
H. G. LEWINSOHN, M.D.

90007

Cape Industries PLC

114 Park Street London W1Y 4AB



Memorandum to Sir Neville Stack

From Kevin Browne

Subject Orebo railway workshop workers study by Ohlson et al.

Date 28th August, 1984

This is an extremely good and valuable mortality study - one more piece in the epidemiological jigsaw. It shows increases in lung cancer appearing in workers exposed more than 25 years and after a latent period of more than 20 years with a suggestion of an increased gradient of risk in the higher exposures. It also shows definitely no increase in gastro-intestinal cancers.

I have only seen a very shortened translation of the original text, and therefore some of my comments may appear inappropriate in the light of the full text. However, these are the points to which attention should be drawn. Firstly, all the excess lung cancer deaths occurred in employees whose first exposure was before 1939 (table 6). It is true that most of the deaths occurred in this group because of the time lapse, but 5 deaths from lung cancer would still have been expected from post 1939 employees, whereas none have been observed. This effect is seen from another viewpoint in table 5; taking only men who had a minimum of 20 years since first exposure, and who had worked in areas of heaviest exposure, no increase in lung cancer risk is seen in the 10-25 years exposure category, and only in those who were exposed for more than 25 years (and were first exposed before 1939) was the risk increased. I am sure Dr. Ohlson and colleagues will have looked into this point and I would be very interested to know whether they have found any important differences, for instance in work practices, pre 1939 compared with post 1939.

In considering the significance of the increased lung cancer risk it is always desirable to have some information about the prevalence of asbestosis. My English text suggests that there probably was some. In the category of deaths from respiratory disease other than chronic obstructive in the tables, the SMR is not significantly raised, but one suspects that, bearing in mind the healthy worker effect shown and commented on elsewhere in the paper, this includes a small number of deaths in which asbestosis was a major contributory cause. The relatively small increase in lung cancer deaths despite a 50% incidence of pleural plaques, the small amount of asbestosis and 5 mesothelioma deaths (the same number as the excess of observed over expected lung cancer deaths in the whole cohort) give a pattern similar in many ways to that of the British dockyard workers.

Kevin Browne

KEVIN BROWNE

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COHORT STUDY OF THE CAUSES OF DEATH AMONG ASBESTOS EXPOSED REPAIR
SHOP WORKERS. PROJECT Nos. 79/342 and 81-1122 RESPECTIVELY.

Carl-Göran Ohlson¹
Birgitta Klaesson¹
Christer Hogstedt^{2,3}

- 1 Department of Occupational Medicine, Regional Hospital,
701 85 Örebro.
- 2 Section for Occupational Medicine, Research Department,
National Board of Occupational Safety and Health,
171 84 Solna.
- 3 Department of Occupational Medicine,
Karolinska Hospital,
104 01 Stockholm.

Department of Occupational Medicine,
Regional Hospital,
Örebro.

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AIA/20/1/16/HAS
(translated on 27.6.84)

No. 1239

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RESUME

This sample study has investigated risk of lung cancer for 3442 asbestos exposed repair shop workers during the study period 1939-1980. The study population was made up of collectively employed men, employed for a minimum of 3 weeks at the Swedish Railways main workshop in Orebro, where the repair of engines, motor carriages and passenger carriages takes place.

Asbestos has been used in the workshop as insulating material since 1902. Cumulative exposure for each sample member has been worked out on the basis of length of employment and dust contents for each department and year.

The life story has been researched in several steps, firstly through a search of the registers of National Insurance, county halls and church offices, of the NHI pension register, the regional hospitals secondary journal archives as well as the National Libraries address registers. 1053 had died, and the causes of death established through the SCBs register of causes of death. Only in 14 cases was the life story unknown, which gives a story percentage of the sample of 99.6%.

The total fatality for the entire sample was lower than expected as compared with the national average, SMR 0.82, whereas the relative risk for lung cancer was somewhat higher at SMR = 1.16. The total fatality as well as death caused by other forms of cancer was not affected by the length of employment. Through increased cumulative exposure it was established that the SMR for lung cancer increased by 0.27 for the lowest exposed to 1.62 for the highest exposed. Sample members, who at some time had been employed in departments, overhauling steam engines, have the highest excess fatality in lung cancer, SMR = 1.92 for members employed for more than 30 years.

The investigation therefore shows reasonable increase in risk for lung cancer. The risk can, however, be reckoned as trebled for the highest exposed, if it is taken into consideration that the sample's initial risk for lung cancer can have been substantially higher than for the country's men on average. The employment in workshops without contact with steam engine repairs has since the second world war not caused any significant increase in risk of lung cancer.

01046

BACKGROUND

Asbestos is a well known work environmental risk, which was noted already in the beginning of the 20th century. The number of scientific publications on the subject of the biological effects of asbestos fibres exceeds 3000 (1), but only a small number of epidemiological studies throw light on the risks from asbestos and cancer within groups exposed to asbestos during work. Information relating to the risk by different grades of exposure is only found in a dozen or so studies (2). The information on exposure is, however, scant and allows only rough evaluation of the connection between dosage and result.

As the conditions for exposure within industries, handling asbestos, have improved during recent years, the risk of future illness from dust in the lungs has decreased considerably, and the number of new cases of such illnesses can be expected to decrease steadily during the coming years. The risks of cancer illnesses, caused by asbestos, primarily lung cancer and mesothelioma, have therefore all the more come to the forefront.

In a noticeable American report the number of new cases of cancer, caused by asbestos, has been calculated for the period 1980-2020. These forecasts point to a steep increase and are based partly on information about the number of employees within industries, where exposure to asbestos is found, and partly on the fatality development in asbestos-related illnesses during past decades (3). The figures cannot be directly transposed to Swedish circumstances, but nevertheless they give a hint that the culmination of the fatality curve has not yet been reached. A doubtful factor in the calculations is that different types of asbestos and sizes of fibre seem to have different grades of illness-causing properties.

Several English investigations indicate that *Chrysotile* (white asbestos), which has had a complete dominating share of the total consumption of asbestos, has less cancer causing properties than ~~amphibious~~ ^{amphibole} asbestos types in the majority of production areas (4). The type of fibre seems for instance to be important to the risk of lung cancer. Even the type of industrial process has an influence on the health risks, which is shown in the very varied risk assessments in the literature. An investigation from an asbestos textile industry in South Carolina has thus proved ~~high excess risks~~ ^{high excess risks} of lung cancer despite the fact that only ~~chrysotile~~ ^{chrysotile} was used there (5). The mechanical processing of fibres and the resulting fibre sizes can possibly explain such an increased pathogenesis. Such a hypothesis even has animal experimental support (6).

The purpose of this study has been to show the lung cancer risks from asbestos exposure in workshop industry with a variation in length of employment and various dust contents. During the development of the project work an evaluation of the various types of asbestos and their importance to health has stood out as significant. On the other hand the project has aimed at showing the differences in fatality patterns between the various employment samples and between short and long term employees.

01047

DISCUSSION

The studies show a reasonable increase in fatality from lung cancer within the sample compared with the expected values based on the national average. This fatality increases with the dust content in a regular pattern which is seen as an expression of a dose/response relationship between asbestos exposure and risk of lung cancer. During the discussion the use of national average for the calculation of expected values, the significance of smoking, and the validity of the ways of exposure will be discussed.

Expected values from the national average

The sample as a whole has a total fatality which only comes up to about 80% of the national average. Such a general below average fatality within work active groups is normally found in sample investigations and called "healthy worker effect" (17), but as a matter of fact means a failing validity in the comparison (20). The demand for a good health level at the time of employment has for long been a rule, which furthermore has diminished the comparison with national values.

The brittle comparison is shown in the SMR for lung cancer for the lowest exposure categories, which lie among 0.5. Amongst employees employed for a period under one year the especially significant below average fatality can be attributed to short-term employees who are later transferred to completely unexposed supervisory jobs. An even more important explanation to the general below average fatality can be the relatively low incidence rate for lung cancer in the borough of Örebro, which for the years 1975-1980 was 72% of the national average (21).

A maximum risk estimate of $SMR = 1.92$ (table 7) should therefore be adjusted upwards, allowing for the lower risk level for lung cancer, from which the sample started. Roughly calculated the highest exposure categories can therefore be said to have approx. three times as high a risk of lung cancer compared with unexposed Swedish Rail workshop employees.

01048

Smoking habits and lung cancer

The smoking habits of the sample are important to consider when the risk of lung cancer is discussed. During earlier decades smoking was prohibited in the departments. Information is, however, missing as to whether the smoking habits of the sample are different from the national average. Within a sample of employees at the end of the 1970s whose lung function was examined with regard to pleuraplaque, 36% stated that they had never smoked, which would be over and above the general population's proportion of "never" smokers.

Another measure of smoking habits during previous decades can be the fatality from chronical obstructive lung diseases, which mainly reflects the exposure to cigarette smoke. Tables 208 show a pronounced below average fatality in this diagnosis, which probably indicates that the sample as a whole during earlier decades had a higher proportion of non-smokers than the national average.

Asbestos exposure and smoking work together as risk factors for lung cancer in a way which is normally called multiple synergy (19). As the role of exposure to asbestos in the risk of lung cancer is evaluated, consideration should be given to this tendency to under evaluation, as the sample can be presumed to have been smoking less than the national average.

Measure of exposure and dose response

SMR for lung cancer is higher in the highest exposure category, as the categories are based on dust intensity (3 or 4 dust points) or cumulative dust exposure (point years) compared with the exposure measurement years of employment. Dose response reaction is therefore more distinct when the measurement for asbestos exposure is refined.

The comparatively low risk increases for lung cancer ought to be the result of a rather low-graded average exposure. The diagnosed illnesses of the respiratory organs, where chronical obstructive lung diseases are subtracted, ought to reflect the risk for other asbestos related lung diseases apart from lung cancer. As is shown in tables 2-5 it seems that

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with increased exposure a slight increase in the fatality in the diagnosis takes place, which hardly would have been the case if the exposure to asbestos had been continuously low graded. Furthermore, the predominant pleuraplaque in a selected group of voluntary participants in a health control totalled approx. 50%.

Amphib^{ole}~~ious~~ and serpentine asbestos

For data technical reasons the importance of various types of fibre to the risk of lung cancer has not been evaluated properly.

However, the highest risk was ascertained for persons who at some time had worked with repairs to steam locomotives and who had been employed for minimum 30 years. Repairs to steam locomotives entail regular exposure to amphib^{ole}~~ious~~ sorts of asbestos, which certainly are more carcinogenic than ordinary ~~chrysotile~~ ^{crystalline}. The interpretation was complicated by the fact that the repairs to steam locomotives also were highly dusty, wherefore the high dust contents in itself also can be an explanation for the high risk of lung cancer within this subsample. Five cases of mesothelioma have been found, which is a low figure compared with other samples exposed to different types of asbestos fibre. Three of the five cases had been exposed to amphib^{ole}~~ious~~ dust from overhauls of steam locomotives, but in the other two cases no sure exposure of this kind could be demonstrated.

Sundry information:

A slight increase in the number of deaths from pancreas cancer has been observed amongst 15-30 year old employees. It is possible that a few cases of peritoneal mesothelium can be hidden under this diagnosis. The increased fatality in pancreas cancer is however not related to the length of asbestos exposure, and this lack of dose response connection means that the explanation to the increased fatality in pancreas cancer conceivably depends on other factors, or is haphazard. Within the group employed during 1930-1935 a lot of former shoe workers were found. In spite of possible exposure to petrol, no increased risk of leukemia could be observed within this group.

01050

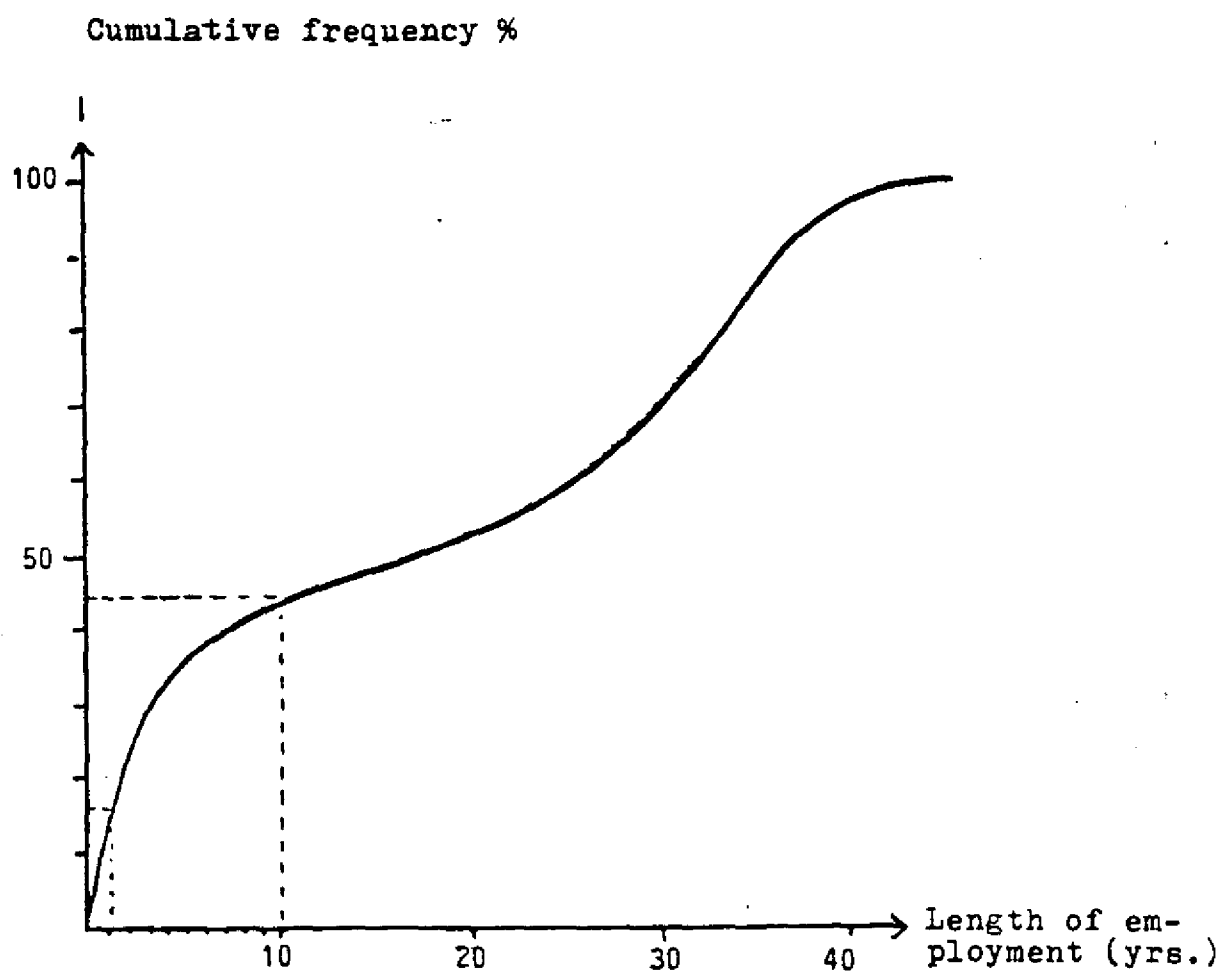
Conclusion

This sample investigation of 3297 workshop employees exposed to asbestos shows a comparatively low graded increase in risk of lung cancer in comparison with the national average. The risk can however be calculated as being approx. trebled for the highest exposed, when it is taken into consideration that the starting risk of lung cancer for the sample could have been considerably lower than the average for the country's male population. Relative risks by exposure to different types of fibre could not be calculated, but the results indicate that several decades of mixed exposure (steam locomotive repairs) have resulted in an approx. threefold increase in risk, whereas shorter periods of employment without contact with the highest dust intensities have not resulted in a significant increase in the risk of lung cancer. Employment in workshop activities without contact with steam locomotive repairs has since the second world war not resulted in any significant increase in the risk of lung cancer.

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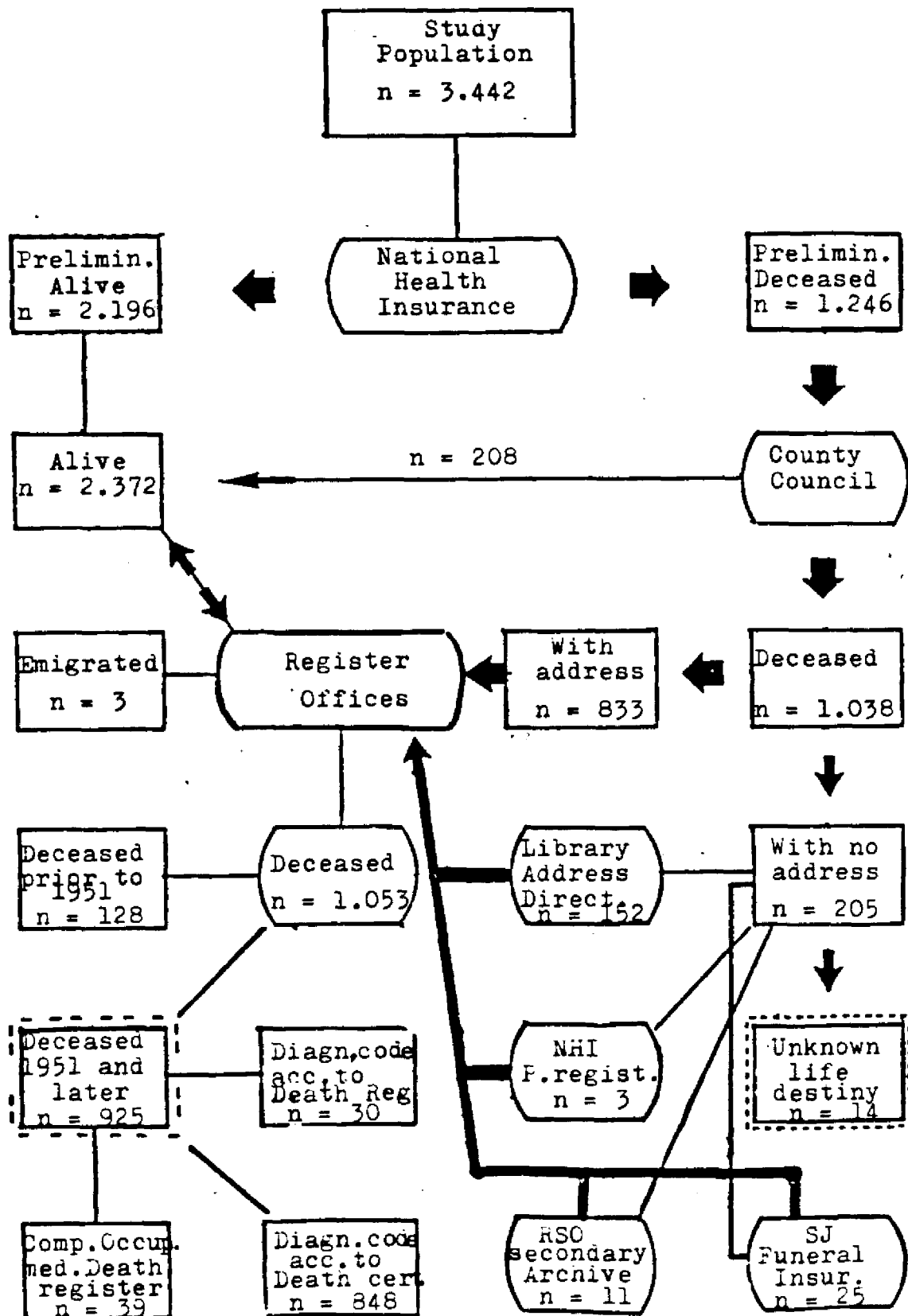
Fig. 1. Distribution of length of employment
within the CV-cohort



Median length of employment = 16 years

01052

Fig.2. Flow chart for the tracing of the life destiny of the study population's 3.442 persons.



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Table 1.

Estimate of the relationship between dust points
and fibre contents in the air

		<u>Fibres/ml</u>	
<u>Dust points</u>		<u>Type value</u>	<u>Spread</u>
0	-	0	0
1	-	0.5	0.5 - 1
2	-	3	1 - 5
3	-	7	5 - 10
4	-	15	10 - 20

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Table 2

Mortality/causes of death for the entire CV-cohort without latent time or age limit and sub-cohort with 20 yrs.latent time to 79 yrs.

Cause of death	Total cohort without latent time			Sub-cohort demanding 20 yrs. latent time ≤ 79 years of age		
	obs.	exp.	SMR	obs.	exp.	SMR
Total mortality	925	1132,6	0,82	586	727	0,81
All cancer	199	234,2	0,85	144	166	0,87
Lung cancer	37	31,8	1,16	27	25,7	1,05
Pancreas cancer	17	15,3	1,11	13	11,5	1,13
Gastro-intestin- al cancer	41	71,6	0,57	30	51,1	0,59
Illness of circ.organs	489	538,9	0,91	310	339,9	0,91
Sciatical heart disease	317	355,1	0,89	210	232,4	0,90
Chron. obstruct. lung diseases	6	16,8	0,36	5	11,3	0,44
Respir.organ diseases (excl. chron.obstr.dis.)	48	45,9	1,05	18	22,8	0,79
Violent death or poisoning	62	89,1	0,70	34	51,0	0,67

01055

Table 3

Mortality for various causes of death before 80 years of age by various lengths of employment and demand for 20 years latent time (if the length of employment has been shorter than 20 years).

Exposure category															
Causes of death	< 2 år		2 - < 5 år		5 - < 15 år		15 - < 30 år		30 - år						
	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR					
Total mortality	42	83,5	0,50	20	26	0,77	43	59,1	0,73	166	148,0	1,12	315	410,4	0,77
Lung cancer	0	3,7	-	1	1,2	0,83	0	2,47	-	6	5,36	1,12	20	13,0	1,54
Gastro-intestinal cancer	0	4,94	-	1	1,56	0,64	2	3,7	0,54	10	10,47	0,96	17	30,4	0,56
Chron. obstruct. lung diseases	0	1,52	-	2	0,53	3,77	0	1,0	-	1	2,36	0,42	2	5,89	0,34
Respir. organ diseases (excl. chron.obstr. diseases)	0	1,98	-	1	0,57	1,75	0	1,52	-	6	4,57	1,31	11	14,2	0,77

Table 4

Mortality for various causes of death before the age of 80 and at different point-years and a demanded 20 years latent time

		Point-years category					
		< 25		25 - < 50		50 - < 75	
Causes of death		obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR
Total mortality	104	168,0	0,62	75	93,3 0,80	121	139,2 0,87
Lung cancer	2	7,48	0,27	2	3,74 0,53	6	4,48 1,34
Gastro-intestinal cancer	3	10,17	0,29	6	6,22 0,96	7	10,14 0,69
Chron. obstruct. lung diseases	2	3,04	0,66	0	1,58 -	1	1,99 0,50
Respir. organ diseases (excl. chron. obstruct. diseases)	1	3,94	0,25	1	2,55 0,39	5	4,71 1,06
						11	11,7 0,94

* p 0,05

01057

Table 5

Mortality for various causes of death before the age of 80 at different number of years in highly-exposed work (= 3 dust points) and a demanded 20 years latent time.

		Number of years in highly-exposed work					
		< 5		5 - < 10		10 - < 25	
Causes of death		obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR
Total mortality	161	221	0,73	234	260 0,90	23	23,4 0,98
Lung cancer	4	8,6	0,47	9	8,8 1,02	1	1 1,0
Gastro-intestinal cancer	8	14,6	0,55	12	18,8 0,64	2	1,5 1,3
Chron. obstruct. lung diseases	2	3,7	0,55	1	4,0 0,25	0	0,9 -
Respir. organ diseases (excl. chron. obstruct. diseases)	1	6,3	0,16	9	8,5 1,06	2	1,4 1,4
						7	7,1 0,99

* p = 0,05

01058

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* $p < 0.05$ (1,23-2,73)

Table 7

Mortality for various causes of death before the age of 80 for sub-cohort of those, who one time or another had worked with overhaul of steam locomotive boilers. Exposure categories acc.to length of employment and 20 years latent time.

Causes of death	Exposure category						Total
	< 5	5 - < 15	15 - < 30	30 -	SMR	obs. exp. SMR	
Total mortality	10 24,7 0,40	13 15,1 0,86	52 42,5 1,22	147 168,2 0,87	222 250,5 0,89		
Lung cancer	1 1,10 0,91	0 0,58 -	3 1,64 1,83	10 5,21 1,92	14 8,53 1,64		
Gastro-intestinal cancer	0 2,43 -	0 0,95 -	3 2,95 1,02	9 12,58 0,72	12 18,91 0,63		
Chron. obstruct. lung diseases	0 0,43 -	0 0,23 -	0 0,68 -	1 2,38 0,42	1 3,72 0,27		
Respir. organ diseases (excl. chron.obstr. diseases)	0 0,53 -	0 0,41 -	0 1,21 -	3 5,83 0,51	3 7,98 0,38		

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Table 8

Mortality for various cases of death before the age of 80 for sub-cohort of those, who had never worked with overhaul of steam locomotive boilers. Exposure categories according to length of employment and a demanded 20 years latent time.

Causes of death	Exposure category														
	< 5		5 - < 15		15 - < 30		30 -		Total						
	obs.	exp.	SMR	obs.	exp.	SMR	obs.	exp.	SMR	obs.	exp.	SMR			
Total mortality	52	84,8	0,61	30	44,0	0,68	114	105,5	1,08	168	242,2	0,69	364	476,5	0,76
Lung cancer	0	3,8	-	0	1,89	-	3	3,72	0,81	10	7,79	1,28	13	17,2	0,76
Gastro-intestinal cancer	1	4,07	0,25	2	2,75	0,73	7	7,52	0,93	8	17,82	0,45	18	32,16	0,56
Chron. obstruct. lung diseases	2	1,62	1,23	0	0,77	-	1	1,68	0,60	1	3,51	0,28	4	7,58	0,53
Respir. organ diseases (excl. chron.obstr. diseases)	1	2,02	0,50	0	1,11	-	6	3,36	1,79	8	8,37	0,96	15	14,86	1,01

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Table 9

The influence of different demands of latent time for the mortality amongst people employed 10 years at CV 79 years of age.

Causes of death	Latent								
	10 years			20 years			30 years		
	obs.	exp.	SMR	obs.	exp.	SMR	ob.	exp.	SMR
Total mortality	524	621,9	0,84	502	585,1	0,86	438	491,7	0,89
Lung cancer	27	20,5	1,32	26	19,4	1,34	25	15,7	1,59
Gastro-intestinal cancer	28	44,6	0,62	27	42,6	0,63	24	36,6	0,66
Chron. obstruct. lung diseases	3	9,08	0,33	3	8,69	0,34	2	7,3	0,27
Respir.organ diseases (excl. chron.obstr. diseases)	17	20,1	0,85	17	19,5	0,87	16	17,6	0,91

01062



ASBESTOS INTERNATIONAL ASSOCIATION

(Limited by Guarantee)

68 GLOUCESTER PLACE, LONDON W1H 3HL

MEMORANDUM

TO: Member Associations

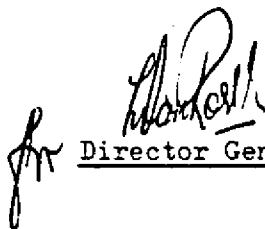
AIA/20/1/HAS

FROM: Director General

14 September 1984

Smoking and the Workplace

AIA Information Memorandum (AIM) No.2/83 dated 10 September 1984
'Smoking and the Workplace' is enclosed.


Director General

cc Medical Advisory Panel / Executive Committee

01063

SMOKING AND THE WORKPLACE

This AIM represents the views of the Medical Advisory Panel endorsed by the Executive Committee on the question of Smoking and the Workplace and is intended as a guideline for employers.

There is an adverse synergistic effect between smoking and asbestos. Therefore asbestos workers should not smoke.

A ban on smoking at home or in private cannot be enforced, but smoking in the workplace proper should be discouraged and progressively reduced until it is eliminated.

The benefits of not smoking in the workplace extend not only to the worker who normally smokes but also to his co-workers who do not smoke but who may otherwise be at risk as "passive smokers".

The example of management is of great importance and their implementation of a policy of non-smoking in the workplace is vital.

The programme of enlightenment of the worker concerning the hazards of smoking and the adverse synergistic effect between smoking and asbestos, is essential. This education needs to be given at an individual level by doctors and management, by written statements given to the worker at the time of enlistment, and by notices at the entrance to the plant and in the workplace; it should be reinforced to individuals and groups at every opportunity.

The pre-employment examination should also be used to educate the worker by personal discussion of the reasons why he should not smoke.

Table 8

Mortality for various cases of death before the age of 80 for sub-cohort of those, who had never worked with overhaul of steam locomotive boilers. Exposure categories according to length of employment and a demanded 20 years latent time.

Causes of death	Exposure category									
	< 5		5 - < 15		15 - < 30		30 -		Total	
	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR	obs.	exp. SMR
Total mortality	52	84,8 0,61	30	44,0 0,68	114	105,5 1,08	168	242,2 0,69	364	476,5 0,76
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Chron. obstruct. lung diseases	2	1,62 1,23	0	0,77 -	1	1,68 0,60	1	3,51 0,28	4	7,58 0,53
Respir. organ diseases (excl. chron.obstr. diseases)	1	2,02 0,50	0	1,11 -	6	3,36 1,79	8	8,37 0,96	15	14,86 1,01

01065

Table 9

The influence of different demands of latent time for the mortality amongst people employed 10 years at CV 79 years of age.

Causes of death	<u>Latent</u>								
	<u>10 years</u>			<u>20 years</u>			<u>30 years</u>		
	obs.	exp.	SMR	obs.	exp.	SMR	obs.	exp.	SMR
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01066

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Causes of death	Latent								
	10 years			20 years			30 years		
	obs.	exp.	SMR	obs.	exp.	SMR	obs.	exp.	SMR
Total mortality	524	621,9	0,84	502	585,1	0,86	438	491,7	0,89
Lung cancer	27	20,5	1,32	26	19,4	1,34	25	15,7	1,59
Gastro-intestinal cancer	28	44,6	0,62	27	42,6	0,63	24	36,6	0,66
Chron. obstruct. lung diseases	3	9,08	0,33	3	8,69	0,34	2	7,3	0,27
Respir. organ diseases (excl. chron.obstr. diseases)	17	20,1	0,85	17	19,5	0,87	16	17,6	0,91

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