FILE NAME: Flooring Defense Exhibits (FLDX)

DATE: 1969

DOC#: FLDX003

DOCUMENT DESCRIPTION: Pneumoconiosis - Proceedings of the International Conference in Johannesburg

TITLE
Pneumoconiosis; proceedings of the international conference, Johannesburg, 1969.
AUTHOR
International Conference on Pneumoconiosis, ; Shapiro, H. A.
VOLUME
ISSUE
ARTICLE AUTHOR

ARTICLE TITLE

DATE
PAGES
ISBN
ISSN
PATRON NAME Jillian Brekeen
PATRON PHONE
PATRON EMAIL jbrekeen@mgmlaw.com
PICKUP LOCATION Main
DUE DATE 11/8/2016

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NOTES

PNEUMOCONIOSIS

Proceedings of the International Conference Johannesburg 1969

EDITED BY

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CAPE TOWN
OXFORD UNIVERSITY PRESS
LONDON NEW YORK TORONTO
1970

Oxford University Press, Ely House, London W. 1

GLASGOW NEW YORK TORONTO MELBOURNE WELLINGTON
CAPE TOWN SALISBURY IBADAN NAIROBI DAR ES SALAAM LUSAKA ADDIS ABABA
BOMBAY CALCUTTA MADRAS KARACHI LAHORE DACCA
KUALA LUMPUR SINGAPORE HONG KONG TOKYO

Oxford University Press, Thibault House, Cape Town

PRINTED BY CAPE & TRANSVAAL PRINTERS LTD.

Preface

This Conference (convened in Johannesburg from 24 April to 2 May 1969) was the third International Conference on Pneumoconiosis to be held in South Africa. The first one took place in Johannesburg in 1930 and the second (also in Johannesburg) in 1959.

The Third Conference was attended by over 250 participants, including some 60 overseas participants among whom were included well-known scientists in industrial medicine from the United Kingdom, the United States of America, Canada, France, Italy, Belgium, Switzerland, Finland, West Germany, Australia, Japan, Rhodesia, etc. The South African participants included scientists, engineers and administrative officials actively engaged in research and the administration of compensation legislation, etc.

Pneumoconiosis is an important industrial problem which requires the application of control and preventive measures, intensive research in the medical and engineering fields and a constant review and reassessment of legislative measures. The purpose of the present Conference was, therefore, to bring together recognised experts in the various disciplines in order to pool, on an international basis, knowledge, experience and ideas; to review the present state of research and preventive and control measures; and to determine the further research necessary to deal with the problem effectively.

Publication of the *Proceedings* of this important international survey has been made possible by the Department of Mines.

We are indebted to the participants for their co-operation in approving their contributions for publication. The excellent summaries of the various discussions have been included as a result of the work of the very efficient band of rapporteurs. We also wish to thank Mrs. E. C. Shute for the preparation of the Subject and the Author Index.

To allow the *Proceedings* to reflect the international character of the authors, a rigid conformity of presentation has not been imposed on their contributions. The lists of References at the ends of articles should often be regarded as bibliographies as well as footnotes applicable to the text.

H. A. Shapiro,
PH.D., M.B., CH.B., F.R.S.S.Af.,
Editor.

Johannesburg, 1970.

International Conference on Pneumoconiosis, Johannesburg 1969

Message from the Minister of Mines of the Republic of South Africa

It is a great pleasure for me to introduce to you this book, The Proceedings of the International Pneumoconiosis Conference held in Johannesburg from 23 April to May 2, 1969, under the aegis of my Department.

As is well known, the mining industry of this country is large and consists of both mining and processing of a great variety of minerals. Although each of these is associated with specific health hazards, such hazards may vary because of the circumstances in which the mining takes place.

The last Pneumoconiosis Conference held in Johannesburg, was in 1959 and it was considered opportune that, after a decade, this subject (so important to my country) should be reviewed by experts from the Western world in conclave with our own South African scientists.

Approximately 70 overseas scientists were present at the discussions which covered a wide field including both the medical and the engineering aspects. Lines of research were suggested which could be carried out in our country and which could benefit those who work in dusty occupations in many different countries throughout the world.

I am confident that this book, which records the papers and the discussions, will be of interest to a wide field of readers whose work brings them into contact with the problem of pneumoconiosis.

Minister of Mines, of Planning and of Health.

Pretoria,
September 1969.

Message from the President of the 1969 International Conference on Pneumoconiosis

When I was asked by the Minister of Mines, Dr. the Honourable Carel de Wet, M.P., to lead the Organizing Committee and to act as President of another international conference on pneumoconiosis, my thoughts turned to the last conference which was held in Johannesburg in 1959.

At that time the Pneumoconiosis Research Unit had been in existence for only 3 years and scientists from various countries were invited mainly to advise our young but enthusiastic research team on the best evolution of their research projects.

Based on the recommendations of the 1959 Conference, the foundation laid by scientists of previous generations such as Pitchford, Irving, Mavrogordato, Simson and Strachan and the activities of the present scientists, the 1969 Conference has paved the way for continued progress and for continued intellectual curiosity.

With my colleagues of the Organizing Committee I considered that every effort should be made to tackle many of the still unresolved problems inherent in exposure to dust.

As will be appreciated, we have at our disposal unique facilities and material for purposes of research and we are anxious to repay, through close collaboration, some of the scientific debts we owe to other countries.

At the conclusion of the Conference our Organizing Committee and the Vice-Presidents agreed on the best methods by which we in this country can continue to play an active part in assisting to solve problems of universal importance. It is sincerely hoped that the final decisions, which include recommendations about the extension of the terms of reference of the Pneumoconiosis Research Unit and the provision of adequate funds for such purposes, will be speedily put into effect in the interests of all concerned.

I regard intellectual contact at the international level as an enriching experience, both to the giver and the receiver; the deliberations of the present conference are ample justification of this philosophy.

Grateful thanks are extended by myself and the Organizing Committee to all who have made contributions to the success of the Conference.

S F Oosthuizen.

Professor S. F. Oosthuizen,
President.

Officers of the Conference

Patron: The Hon. Dr. Carel de Wet, *Minister of Mines of the Republic of South Africa.*

President: Prof. S. F. Oosthuizen, *Professor of Radiology, University of Pretoria, President of the South African Medical and Dental Council and Medical Adviser to the Minister of Mines of the Republic of South Africa.*

Secretary-General: Dr. G. K. Sluis-Cremer, *Director of the Miners' Medical Bureau of the Republic of South Africa* and *Director of the Clinical Sciences Division of the Pneumoconiosis Research Unit of the C.S.I.R.*

Assistant Secretaries-General:

Dr. I. Webster, *Director of the Pathology Division of the Pneumoconiosis Research Unit of the C.S.I.R.*

Mr. J. F. Brauckmann, *Administrative Deputy Director of the Miners' Medical Bureau.*

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Prof. P. H. Rossier: *France.*

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Mr. T. L. Gibbs: *South Africa.*

Rapporteurs

Pathology: Dr. B. Goldstein, Dr. I. Prinsloo, Dr. S. D. Berson.

Clinical Sciences: Dr. F. J. Wiles, Dr. H. v. Doorn.

Dust Physics: Dr. R. S. J. du Toit, Mr. L. W. Isserow, Mr. A. F. H. Lombard, Mr. P. H. Kitto.

Radiation Hazards: Dr. J. K. Basson.

Editorial Committee

Dr. G. K. Sluis-Cremer, Dr. I. Webster and Prof. H. A. Shapiro (*Editor*).

Sponsors

The Conference was held under patronage of the Ministry of Mines of the Republic of South Africa and the Hon. the Minister of Mines, Dr. Carel de Wet, the Patron of the Conference.

The Conference was sponsored financially by the Department of Mines of the Republic of South Africa, the Asbestos Mining Industry of South Africa, the Chamber of Mines of South Africa and the Mining Trade Unions. Accommodation for the Conference and other conference facilities were provided by the South African Institute for Medical Research.

Organizing Committee

Chairman: Prof. S. F. Oosthuizen.

Members: Dr. G. K. Sluis-Cremer, Dr. I. Webster, Dr. J. K. Basson, Prof. J. H. S. Gear, Mr. F. A. Snyman, Dr. R. S. J. du Toit, Mr. J. F. Brauckmann.

Ladies Committee

Convener: Mrs. J. Webster.

Members: Mrs. E. Oosthuizen, Mrs. J. E. V. Sluis-Cremer, Mrs. J. C. Snyman, Mrs. H. Basson, Mrs. J. Gear, Mrs. N. Brauckmann, Mrs. G. E. F. du Toit.

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Wednesday, 23 *April* 1969

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Chairman: I. J. Selikoff, *Environmental Sciences Laboratory, Mount Sinai School of Medicine, United States of America*

Chairman: J. C. McDonald, *Department of Epidemiology and Health, McGill University, Canada*

Thursday, 24 *April* 1969

Symposium on Asbestosis

Chairman: S. F. Oosthuizen, *Adviser to the Minister of Mines of the Republic of South Africa*

Friday, 25 April 1969

Symposium on Asbestosis (Continued)

Chairman: S. Forssman, *National Institute of Occupational Health, Sweden.*

Symposium on the Radiology of Asbestosis

Chairman: E. P. Pendergrass, *Department of Radiology, University of Pennsylvania, United States of America*

Symposium on the Radiology of Pneumoconiosis

Chairman: G. Worth, *Krankenhauses Bethanien, Germany*

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Chairman: F. M. Engelbrecht, *Department of Physiology, University of Stellenbosch, South Africa*

Chairman: P. H. Rossier, *Medical Clinic, University of Zurich, Switzerland*

Chairman: J. H. S. Gear, *South African Institute for Medical Research, Johannesburg, South Africa*

Symposium on Chronic Bronchitis and Emphysema

Chairman: G. Wright, *Department of Medical Research, Saint Luke's Hospital, United States of America*

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Chairman: M. Gelfand, *University College of Rhodesia and the Pneumoconiosis Medical Bureau, Rhodesia*

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Chairman: J. R. Rüttner, *Histopathologischen Institut, Universitat, Zurich, Switzerland*

Chairman: A. G. Heppleston, *Department of Pathology, University of Newcastle Upon Tyne*

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Chairman: F. G. Hill, *Rand Mines Limited, South Africa*

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Chairman: A. J. A. Roux, *Atomic Energy Board, South Africa*

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Monday, 28 April 1969

Chairman: P. H. Kitto, *Chamber of Mines of South Africa*

Chairman: V. Timbrell, *Medical Research Council, Pneumoconiosis Unit, United Kingdom*

Tuesday, 29 April 1969

Chairman: W. H. Walton, *Institute of Occupational Medicine, United Kingdom*

Chairman: C. T. Hardy, *Government Mining Engineer's Division, Department of Mines, South Africa*

Symposium on Asbestos

TABLE 13: d SPACINGS (Å) FROM SELECTED AREA DIFFRACTION PATTERNS

Amosite	*Anthophyllite*	*Crocidolite*	*Rhodesian Chrysotile*	*Canadian Chrysotile*
3·88	4·58 M	3·41	7·27	7·60
3·45	2·65 S	2·57 S	4·58	4·58
3·00	2·27	2·24	3·67 S	3·67 S
2·64 S	1·75 S	2·13	2·61	2·55
1·74	1·55 S	1·78	2·14	2·09
1·61	1·33 M	1·70	1·76 M	1·73 M
1·55	1·28	1·61	1·55 S	1·54 S
1·32 S	1·23	1·49	1·34 S	1·33 S
1·05	1·14	1·30 S	1·29	1·28
0·89 S	1·06	1·27	1·20	1·20
0·88	1·005	1·06	1·065	1·05
0·755	0·93	0·985	1·00 M	1·00 M
	0·895	0·89	0·89 S	0·895 S

S = strong rings. M = medium strength.

TABLE 14: PRINCIPAL LATTICE SPACINGS Cu Kα RADIATION $\lambda = 1{\cdot}45$ Å

Asbestos Type	*Peak No.*	2θ	d	I/I_1
Amosite	1.	10·7°	8·26 Å	100·0
	2.	27·3°	3·27 Å	36·4
	3.	29·1°	3·07 Å	52·3
	4.	32·3°	2·77 Å	43·2
Anthophyllite	1.	9·3°	9·50 Å	100·0
	2.	10·6°	8·40 Å	36·1
	3.	19·4°	4·58 Å	55·5
	4.	27·5°	3·25 Å	47·2
	5.	28·5°	3·13 Å	58·3
	6.	29·2°	3·06 Å	63·9
Crocidolite	1.	10·5°	8·43 Å	100·0
	2.	19·7°	4·51 Å	25·7
	3.	26·0°	3·43 Å	25·7
	4.	28·7°	3·11 Å	41·4
	5.	32·9°	2·72 Å	50·0
	6.	34·4°	2·61 Å	20·0
	7.	35·3°	2·54 Å	35·7
Rhodesian Chrysotile	1.	12·0°	7·38 Å	100·0
	2.	19·5°	4·55 Å	28·8
	3.	24·3°	3·66 Å	50·8
	4.	36·5°	2·46 Å	27·1
	5.	60·2°	1·54 Å	20·3
Canadian Chrysotile	1.	12·0°	7·38 Å	100·0
	2.	19·5°	4·55 Å	27·0
	3.	24·3°	3·66 Å	57·1
	4.	36·5°	2·46 Å	23·8
	5.	60·2°	1·54 Å	20·6

The electron diffraction studies have indicated that the grinding involved in the preparation of the reference samples did not affect the crystalline structure of the fibres.

ELECTRON DIFFRACTION

The X-ray diffraction patterns of the samples have been prepared and from these patterns the principal lattice spacings determined. These lattice spacings are listed in Table 14. By comparing these figures with equivalent figures obtained from material extracted from lungs it would be possible to detect changes in the crystal structure due to biological activity or other causes.

PUBLICATIONS

Data sheets are distributed periodically to recipients of the reference samples giving brief information on their characteristics, and details of the analysis methods are being published in the literature.

I wish to acknowledge on behalf of the UICC the assistance given by all those who have contributed to this work.

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DUST PROBLEMS IN THE USE OF ASBESTOS PRODUCTS

ERNST WALTHER

Former Member of the Research Advisory Committee (Engineering)
Asbestosis Research Project of the Pneumoconiosis Research Unit,
Council for Scientific and Industrial Research

1. *Introduction.* According to the preamble in the invitation to this conference, its purpose has been defined as the pooling of knowledge, experience and ideas, to reviewing the present state of research and preventive or control measures, with a view to determining further research which is necessary to deal with the problem effectively.

1.2. Many statements have been made in recent years stressing the hazards associated with mining, milling and handling of asbestos (ores and fibres) and the uses of asbestos (occupationally) and asbestos products by the general public. Some of these have been sensationally exploited by the press and other news media, often taking facts out of context and creating an atmosphere of fear, not only in occupational circles but also amongst the general public, out of proportion to any possible real hazard.

1.3. After years of extensive research and deliberation by world authorities on Asbestosis, Mesothelioma and Talcosis, we shall, in the course of the next few days, be brought up to date by their contributions on these subjects, and the objective analysis of the facts gathered so far, should allow industries and government bodies to plan future research programmes and campaigns to eliminate health hazards, where such should exist and are attributable to the products manufactured from asbestos and like fibres.

2. When discussing the dust problems attributed to asbestos, there are 2 main considerations:

2.1. The first has to do with the precise nature of exposure to asbestos fibre by workers actually handling the products, or *occupational exposure,* which has been studied extensively, and on which legislation has been passed in many countries.[1, 2] There is little doubt that excessive quantities of asbestos dust, or any dust for that matter, are harmful to health.

Dust prevention in plants, factories and building sites, introduced in recent years, has reduced and eliminated health hazards and in most cases led to fairly satisfactory conditions. It can be expected that such improvements would also reflect in a greatly reduced aerial pollution in the immediate surroundings of the operations, thus removing hazards to the general public from this source.

2.2. The second aspect falls within the definition of this paper, namely the exposure to asbestos dust, which might be *incurred by the general public* using asbestos products, where exposure will be of an infinitely lower intensity.

2.3. Asbestos products could conceivably release asbestos dust into the ambient air as a result of environmental influences upon the products. Unfortunately, there is a paucity of factual information pertinent to this question.

3. Before commenting on the specific dust hazards, I wish to refer briefly to the main diseases attributed to exposure of asbestos in recent years.

3.1. *Asbestosis and Bronchogenic Cancer.* These have been strongly linked with occupational exposure to asbestos fibre where the exposure to fibre was intense, over lengthy periods, and the number of fibres in the ambient air, very great.

3.2. L. J. Cralley *et al.*[3] pointed out, that 'all coated fibres resembling golden yellow fibrous bodies found in the lungs of asbestos workers, had been termed "asbestos bodies", even though the nature of these fibres had never been identified'. These scientists attribute the presence of these bodies, comprising fibres coated with protein and iron pigment, subsequently termed 'ferruginous' bodies, to a variety of respirable fibres. Besides asbestos, rock wool, gypsum, talc, magnesite, mica, etc., as well as certain synthetic products such as ceramic fibres, glass fibres, slag fibres and metal whiskers, etc., are listed as respirable fibres, together with those of vegetable and animal origin.

3.3. *Mesothelioma* has been linked in a similar way, but is thought to be associated with exposure of a lower order of intensity. Despite all the attention focused upon it in recent years, this rare tumour still constitutes a problem because of the difficulties of

diagnosis. The matter is further complicated as 'these tumours have occurred in people on whom no evidence of exposure to asbestos dust was established'.[4] On the occasion of the Annual General Meeting in August 1968, Dr. I. Webster, Chief Pathologist of the Pneumoconiosis Research Unit of the C.S.I.R. South Africa, stated that the 'explosion of mesothelioma feared some years ago, had not taken place'. Much of the campaign against Crocidolite could therefore, no doubt, be attributed to a 'statistical explosion', extrapolation of clinical observations in earlier years, or, that other unidentified causative agents were at work.

To place the problem of mesothelioma in its proper perspective, it has been pointed out that the total number of cases known at present in the entire world is less than 1,000:[5] according to some authorities, possibly even less than 500:[6] and this, after 10 years of research and focusing of attention upon it. It constitutes an insignificant problem compared with the death rate of about 29,000 in 1967 in the U.K. alone, from various forms of lung cancer.

3.4. *Talcosis*: Another occupational disease with symptoms similar to asbestosis should be mentioned in this context.

In their work presented to the American Industrial Hygiene Conference in May 1968, L. J. Cralley *et al.*[7] reported 'that talcum powder contained a significant percentage of respirable fibres, and was a potential *source of "ferruginous" bodies* observed in the lungs of humans'.

3.5. These diseases are considered to be *dose-related* in respect of asbestos fibre (or talc). It is, however, unlikely (even under adverse conditions) that exposure of a general nature in the normal use of asbestos products could, in any way, reach the critical levels of intensity, determined or accepted in the occupational environment.

Such exposure will be of a very low order, but we lack knowledge on the tolerable limits. We cannot transfer experience in one kind of exposure, namely industry, into the very different exposure sphere affecting the general public.

3.6. To reach clarity in these matters, a number of questions must be answered by research where we lack factual knowledge:

i. We need to know accurately, the exposure levels in terms of dust concentrations, frequency and duration of such exposure to start or induce the alleged disease processes.

ii. To which degree or concentration do asbestos fibres actually exist in the ambient air of our city streets?

iii. We need factual information concerning the kinds and numbers of all types of fibres (not only asbestos fibres) in rooms where asbestos products could conceivably release some fibres either from walls, ceilings, insulation linings, air ducts, or from floors.

iv. To which degree might asbestos cement products weather and release fibres into the ambient air directly or indirectly by subsequent re-dispersion?

v. Is asbestos fibre as indestructible as it is made out to be, or to what degree, and will it retain its identity in the process of destruction?

vi. How much real asbestos fibre is disseminated during the use of brake linings, clutch linings, in the normal operation of the automobile in built-up areas?

vii. We need to obtain a factual picture of the general pattern of living of suspected cases, in investigating their exposure levels, not only to asbestos fibres, but to other fibrous bodies in the ambient air; their individual ways and habits of living (smoking, drinking, drug addiction, nutrition), the overall pathological picture (tuberculosis), nervous tension associated with modern living, factors of general air pollution from industry and transport in combination with the weather pattern, radiation etc., all of which might lead, in their combined effect, towards a possible predisposition to trigger off the disease by reducing the individual's protective mechanism.

3.7. Most of the controversy of recent years, no doubt, stems from lack of factual knowledge on these points or from the extrapolation of observation from one field to another. There are many essential materials and processes in modern industry which involve an element of risk. This should be recognised, understood, and steps need be taken to reduce it to a tolerable level. In this connection the progress made in the asbestos mining fields in South Africa, is worth mentioning, as we shall hear in detail from the next speaker. By studying problems confronting the industry a few years ago, in co-operation with the Government Mining Engineer, the Department of Mines and the C.S.I.R., a step by step programme of improvement was implemented, so reducing

exposures and, in many cases, complete elimination of hazards was achieved.

In the Annual Report 1967/68,[9] C.S.I.R. Department of Mines, we read:

'Considerable progress continues to be made in dust-control, but as research in the medical field is, by its very nature, a long term undertaking, only limited progress has been achieved so far in this field.'

4. Turning to various asbestos products which have been under scrutiny for some time, I wish to offer the following comments:

4.1. *Asbestos cement products* absorb a large portion of the world's asbestos production, particularly Chrysotile, Crocidolite and to a lesser degree Amosite, in combination with Portland Cement. Asbestos fibre and cement, form a homogeneous union, extremely inert to the action of the elements.

4.1.1. It has been implied that small quantities of fibres are released through the weathering of asbestos cement. Under extreme climatic conditions like heavy hail, certain frost conditions, or, through aggressive chemical actions, small quantities might conceivably be released, but 'there is no evidence that such small doses are harmful'.[5] nor will this occur with normal usage. Roofings, ceilings, partitions, etc., used in dwellings and halls often receive a decorative finish, thus further eliminating any hazard of exposure.

4.1.2. The Asbestosis Research Council in the U.K.[10a] has reached the conclusion, and emphasizes the fact, that there is no hazard in the handling, working and fixing of asbestos cement products, provided certain simple precautions, outlined in their Code of Practice, are followed. There is, therefore, no reason to assume or to expect any health hazard to the general public in using such products once they are fixed and installed.

4.1.3. In support of this, I present the following information made available by Dr. Lepoutre of Belgium:[11]

'Professor Van de Voorde and Professor Meulepas of the University of Louvain, at the request of the Belgium Eternit, have done a survey (published in Acta Tuberc. et Pneumologica Belgica No. 6 Nov.-Dec. 1967) in respect of the death causes among the inhabitants of 13 communities situated in a radius of 7 k.m. around the Eternit plants of Kapelle-op-den-Bos and Tisselt and a similar survey in 13 communities in the district of Westhoek, an agricultural area devoid of industries. In comparing the frequency rate of certain affections witnessed between 1959 and 1963 in the 2 districts, the following conclusions were reached:

'The populations living within the radius of 7 k.m. of a transformative plant of asbestos, did not present for the period 1959-63 a higher degree of mortality caused by malignant tumours than the average for the agricultural area of Westhoek.'

4.1.4. If we consider that the population of the area surveyed by Professors Van de Voorde and Meulepas, is not only living within close proximity of an extensive asbestos product operation (one of the largest single units in Europe), and that a great number of asbestos products were used in the construction of roofing, ceiling, piping, partitioning and decorating of their homes, and further, that an undisclosed but presumably large percentage of this population has been employed over a number of years by the manufacturing concern, it would appear that a health hazard to the general public from the environmental exposure of a modern, well protected operation, or from the normal use of asbestos cement products, could be ruled out.

It is noteworthy that the above operation, in 1954 introduced an annual clinical and radiological examination of all its workers including clerical staff.

4.2. *Brake Linings*. It has been suggested that the braking of countless vehicles fitted with brake linings containing a large percentage of asbestos fibre (30-50%) mostly Chrysotile, in combination with resins, polymers, oxides, pigments, metals, carbon black and graphite, would produce hazardous dust.

4.2.1. Investigators have found no substance in these allegations. Their theory of what happens to the asbestos in brake linings is explained by J. R. Lynch[12] that

'Wear occurs not by abrasion of the lining by the drum, but by the production of minute areas of intense heat at the point of contact between drum and lining. Decomposition not only of the binder, but also of the asbestos occurs at these locations, as the breakdown temperature of Chrysotile asbestos of about 900°F, is exceeded. The decomposition products will thus include no free asbestos fibres, but a different mineral resulting from the thermal metamorphosis of the asbestos'.

4.2.2. J. R. Lynch reaches the following conclusions:

'Only a very small proportion of the asbestos worn from brake linings is released as free fibre, the remainder is converted into some other mineral as a result of the extreme temperature generated at small spots on the surfaces. Thus, although urban air contains a few free fibres as a result of brake lining wear, they represent a very small proportion of the total asbestos used in the manufacture of brakes, and many sources of respirable fibres not associated with asbestos products have been identified to be present in urban air and the free fibres from brake lining

wear appear to be an inconsequential health factor in urban air pollution."[3]

4.3. *Floor Tiles.* These have been mentioned as potential dust hazards. In the floor tiles, asbestos (Chrysotile) is intimately embedded in thermo-plastic resins. The wear resistance is, however, extremely high, and the liberation of asbestos fibre in normal usage must be termed infinitesimal. We have no scientific evidence of anyone having contracted a disease from exposure to floor tiles in normal use.

In most instances, such tiles, which have a high decorative value, receive a good wax covering which acts as an additional sealer.

4.4. *Insulations.* The occupational hazards in this field have been extensively investigated, occupational exposures are controlled through regulations[1] and Codes of Practices[10b] regarding industrial hygiene, both in the new equipment, maintenance, and break-up fields.

Fibre-based insulation products, having little resistance of their own to abrasion or shock, are provided with protective coverings of varying degrees of mechanical resistance, such as metal sleeves and covers, bandages, panels, or sealers, adhesives, tough decorative coatings or other hard setting renderings. There is no factual evidence of a health hazard from these products once they are installed and properly maintained.

4.5. *Asbestos Textiles.* These are used for very special and limited applications and the general public will not normally be associated with their use. The undisputed life saving properties of these products will far outweigh any imaginary health hazards.

4.5.1. There is, further, no health hazard to the general public arising from the use of asbestos based jointings and packings.

4.6. *Battery Boxes.* A field of application for considerable tonnages of Crocidolite in thermo-setting compounds, these do not constitute any health hazard in normal use.

5. Conclusion

i. We should not lose our sense of proportion when numerically evaluating available facts and indications and keep them in proper perspective *vis-à-vis* the global hazards and diseases affecting the general public.

ii. Any industrial occupation presents certain hazards, but we cannot transfer experience gained in industrial exposure into the very different exposure sphere affecting the general public.

iii. In these days, when all substances in the air, water and in food are constantly under scrutiny as to their effect on public health, there should be no surprise that asbestos is among the subjects of intense investigation.[21]

iv. On the strength of present knowledge we may conclude that the normal use of asbestos products does not give rise to abnormal health hazards.

v. There is a strong belief that health hazards previously attributed to asbestos fibre might in fact be associated with exposure to other respirable fibres or causative agents.

vi. Additional detailed research is required to answer the question in paragraph 3.6.

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It would appear that there may be an optimal range in the diameter of fibres necessary for the production of ferruginous bodies.

Summary

The morphology of asbestos bodies was studied by means of the Cambridge Stereoscan scanning electron microscope and this revealed the characteristic dumb-bell shape with segmentation of the shaft and expanded ends.

The incidence of ferruginous bodies in the lungs of deceased mine-workers was 4.4% and most of the cases occurred in asbestos workers. In addition, the incidence of different types of malignant lung neoplasms was significantly higher in the cases with ferruginous bodies.

Experimental work is in progress to attempt to produce ferruginous bodies by administering various fibrous materials of mineral, vegetable and animal origin but so far typical bodies have only been produced with the various asbestos dusts and fibre-glass, while the material of vegetable and animal origin produced foreign body granulomas. It is suggested that there may be an optimal diameter necessary for the formation of ferruginous bodies.

We would like to thank Dr. N. F. Laubscher, Head, Statistics Division, National Research Institute for Mathematical Sciences of the Council for Scientific and Industrial Research for the statistical analysis;

Mr. R. E. Greasley of the Department of Physics, University of the Witwatersrand, for the use of, and assistance with, the Stereoscan electron microscope; and

Dr. I. Webster, Head of the Division, for the helpful suggestions in the preparation of this paper.

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ASBESTOS BODIES IN THE NEW YORK CITY POPULATION IN TWO PERIODS OF TIME*

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Since the demonstration by Thomson, Kaschula and MacDonald in 1963[1] that asbestos bodies (or, particles seemingly identical with those seen in asbestos workers) were frequently found in the lungs of urban residents at autopsy, in Cape Town, similar findings have been reported from many cities.[2-11] New York City is no different—asbestos bodies are found in approximately half of those individuals now examined at autopsy.

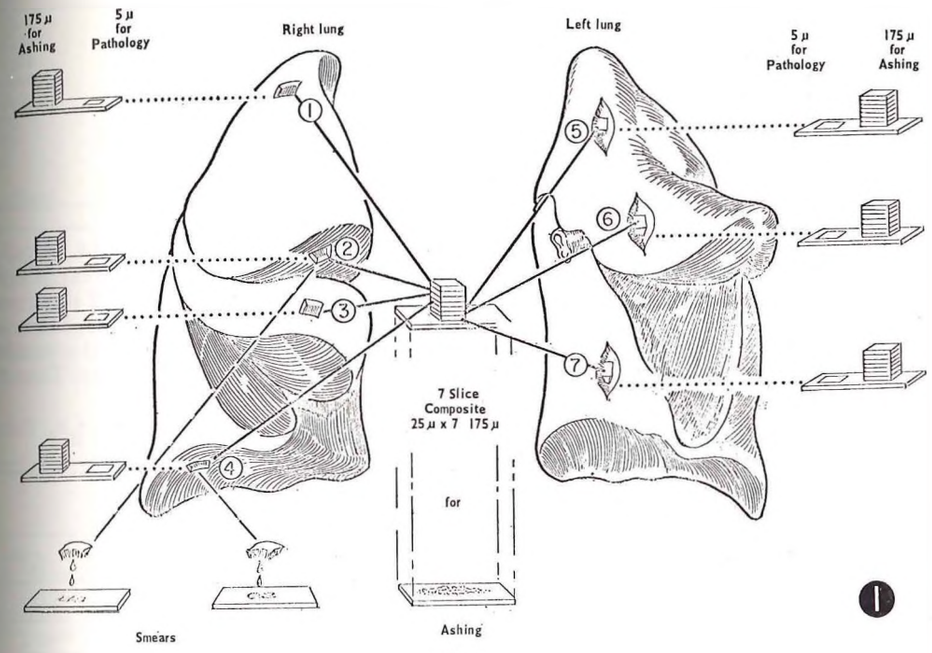

Fig. 1.

Techniques

The method used to search for asbestos bodies affects the readiness with which they are found.[12] For example, it is more difficult to see and correctly identify asbestos bodies in stained sections than in ashed sections; and the volume of tissue examined is obviously important. It is even conceivable that if enough lung tissue is processed, at least one asbestos body could be found in almost everyone.[13]

In our current studies, we use 2 techniques: basal smear and ashed sections. The latter technique may be briefly described:

Sections measuring 25 μ × 1.0 cm^2 are taken from each of the 7 sites in the lung recommended by the UICC Working Group[14] (Fig. 1). The 7 sections are stacked one upon another making a pile 175 μ thick; and this is ashed by activated oxygen.[15] The ashed material is examined by phase microscopy at 440 ×. The presence or absence of asbestos bodies (and the number if any are present) is recorded as well as presence or absence of uncoated inorganic fibers and nonfibrous mineral particles.

Tables 1 and 2 show what may be called the 'stability' of each of the 2 methods. After the first several hundred consecutive cases were

* This study was supported by the USPHS grant #UI 00440.

studied from each of 3 hospitals, we divided them into several sequential groups and recorded the percent of 'positive' cases in each group. There is no reason to suppose that subjects from the 3 hospitals should be exactly alike; and the size of each group within each hospital was such that the percent positive is subject to considerable sampling variation. ('Stability' depends on sampling of subjects, sampling of material and accuracy of technical procedure.) The 'stability' was not unreasonable; but the magnitude of the variation from group to group illustrates the necessity for including large numbers of subjects in studies of this type.

Table 1: 'Stability' of Smear Technique

	Group 1	*2*	*3*	*4*
Hospital 'A'	20/98 = 20%	15/98 = 15%	17/98 = 17%	15/98 = 15%
Hospital 'B'	8/91 = 9%	11/92 = 11%	12/97 = 12%	10/89 = 11%
Hospital 'C'	18/98 = 18%	15/98 = 15%	22/98 = 22%	15/94 = 15%

Hospital 'A'	67/392 = 17%
Hospital 'B'	41/369 = 11%
Hospital 'C'	70/388 = 19%

Table 2: 'Stability' of Ashed Section Technique

	Positive Sections per 100 Cases						
Hospital 'A'	53	52	49	50	59	50	45
Hospital 'B'	39	32	42	42	51	34	49
Hospital 'C'	43	46	51	47	52	53	50

Hospital 'A'	358/700 = 51%
Hospital 'B'	289/700 = 41%
Hospital 'C'	342/700 = 49%

Table 3 shows the findings in the first 1,149 subjects studied by both methods. From studying basal smears, 84.7% of the subjects would be called 'negative' for asbestos bodies and 15.3% would be called 'positive'. From studying ashed sections, 53.9% would be called 'negative' and 46.0% would be called 'positive'. Furthermore, 5 or more asbestos bodies were found in only 2.0% of the smears but in 7.9% of the ashed sections. However, 23 (2.0%) of the subjects had 'positive' smears but 'negative' ashed sections. Thus the use of both methods reveals slightly more 'positive' cases than the use of ashed sections alone.

Table 3: Distribution of First 1,149 Subjects in Respect to Number of Asbestos Bodies (A.B.) Found in Ashed Sections and in Basal Smears

Ashed Sections	*Basal Smears* 0 A.B.	1–4 A.B.	5+ A.B.	*Total Number*	%
0 A.B.	596	20	3	619	53·9
1–4 A.B.	330	105	4	439	38·2
5+ A.B.	47	28	16	91	7·9
Total Number	973	153	23	1,149	—
%	84·7	13·3	2·0	—	100·0

Note: In an additional 51 cases, basal smears were not obtained. Ashed sections in these 51 cases indicated 35 with no asbestos bodies, 13 with 1 to 4 asbestos bodies and 3 with 5+ asbestos bodies.

Asbestos Bodies in N.Y.C. 1966-1968

We are studying 3,000 consecutive autopsies (1966-1968) in 3 large N.Y.C. hospitals (Mount Sinai, Manhattan; Elmhurst, Queens; and the Veterans Administration Hospital in the Bronx). Concomitantly with the asbestos bodies studies, we are collecting much personal, clinical, pathological, residence and occupational information.

Table 4: Distribution of Asbestos Bodies in General Population—in 1,975 Cases

Male Age	Total Cases	Total +	% +	*Female* Age	Total Cases	Total +	% +
0–1	40	0	—	0–1	34	0	—
2–9	2	1	—	2–9	2	0	—
10–19	4	0	—	10–19	8	3	38%
20–29	22	4	18%	20–29	12	4	33%
30–39	50	14	28%	30–39	18	7	39%
40–49	178	83	47%	40–49	58	18	31%
50–59	280	149	53%	50–59	85	38	45%
60–69	359	205	57%	60–69	147	57	39%
70–79	321	180	56%	70–79	151	65	43%
80–89	105	62	59%	80–89	80	40	50%
90+	7	4	57%	90+	12	8	66%
Total	1,368	702	51%	*Total*	607	240	39%

We have analyzed the results in our first 1,975 cases (Table 4); asbestos bodies were found in 942 (47.7%). In 772, they were few in number (1-4); in 170, they were more numerous (5 or more).

Moreover, when housewives or white collar males were positive, they generally had few asbestos bodies; when men who had worked in the construction industry were positive, often many bodies were present (Table 6).

Table 5: Asbestos Bodies in 856 Autopsies in NYC, Correlated with Occupation

	Number Examined	*Number with Asbestos Bodies*	*Percentage with Asbestos Bodies*
Females	275	108	39%
Male 'white collar' employment	206	97	47%
Male 'blue collar' employment (no construction nor shipyard)	246	121	50%
Male—construction or shipyard employment	129	90	70%
Total	856	416	

We have so far completed the investigation of the lifetime occupational history of 856 cases. The data obtained suggests that *asbestos bodies are not randomly distributed among the general population of N.Y.C.*

In addition to variations with sex and age (Table 4), there was significant variation by occupational category. Broadly considered, 40% of housewives showed asbestos bodies, 50% of 'white collar' males (clerical and professional work), 50% of 'blue collar' males (manual work), except those 'blue collar' males who had any history, however brief, of ever having done shipyard or construction work (all trades—carpenters, electricians, plumbers, laborers, etc.)—they showed asbestos bodies in 70 of 129 cases or 70% (Table 5).

Parenthetically, there is evidence that examination by smear tends to demonstrate highly positive cases more readily; smears are negative much more frequently when few bodies are seen in the sections (Table 3).

These data warrant the following tentative conclusions:

First, that while asbestos bodies are

commonly present in the lungs of adults in N.Y.C., their occurrence is not a random event but, to an important extent, occupationally related.

Second, that the construction and ship building industries are rich potential sources of direct and indirect occupational asbestos exposure[16-20] and, especially if possible neighborhood and family contacts are considered, could be responsible for a large proportion of the asbestos bodies found in routine autopsies.

Secular Changes in Asbestos Bodies in New York City, 1934-1967: Preliminary Observations

We have investigated the incidence of asbestos bodies in the lungs of individuals examined at autopsy at the Mount Sinai Hospital in 2 years: 1934 and 1967.

Table 6: Asbestos Bodies by Occupation: Quantitative Results

	Examined	*0*	*1+*	*2+, 3+*
'White collar' males	206	109	87	10 (5%)
'Blue collar' males (no construction nor shipyard)	246	125	101	20 (8%)
Construction or shipyard	129	39	61	29 (23%)

Table 7: Estimated U.S. Asbestos Consumption construction industries vs non-construction industries (short tons)

	Construction Industry		*Non-Construction Industry*		*Total*
	Tons	%	*Tons*	%	
1920	63,600	40.8	92,400	59.2	156,000
1925	141,600	69·1	63,200	30·9	204,800
1930	136,200	66·0	70,300	34·0	206,500
1935	101,700	58·2	73,000	41·8	174,700
1940	160,200	61·1	102,000	38·9	262,200
1945	214,500	56·7	163,500	43·3	378,000
1950	354,300	48·6	374,400	51·4	728,700
1955	444,300	60·0	296,100	40·0	740,400
1960	437,400	65·3	232,100	34·7	669,500
1965	532,300	74·0	187,400	26·0	719,700

These conclusions are consistent with commercial data concerning asbestos use. *In the past 40 years from one-half to three-quarters of all asbestos consumed in the United States was used in the construction industry* (Table 7, Fig. 2).[21] In 1965, 532,300 tons (74%) of the 719,700 tons of asbestos consumed in the United States, were used in construction.

Method: 100 consecutive cases were selected from the autopsy files of our hospital in each of 2 years, 1934 and 1967. We did not use our 7-site procedure, since these were not available for 1934. Instead, from both years we prepared 175 μ × 1 cm^2 sections from the routine block taken by the prosector. The sections were ashed and examined as described.

Table 8: Secular Changes: Asbestos Bodies in Ashed Tissue Sections (NYC)

	1934			1967		
	Male	*Female*	*Total*	*Male*	*Female*	*Total*
0–9	1/4	0/4	1/8	0/0	0/0	0/0
10–29	0/4	2/7	2/11	0/1	3/6	3/7
30–59	17/29	8/11	25/40	9/16	15/25	24/41
60+	16/22	6/9	23/31	23/37	10/15	33/52
Age Unknown	2/5	1/5	3/10			
	36/64	17/36	53/100	32/54	28/46	60/100

Source identification was removed from all slides; each was given a random number. The sequence of examination ensured thorough admixture of both sets. All readings were the responsibility of one individual, using criteria previously established and tested.[12]

There was no significant difference in the percentage of cases having asbestos bodies in 1967 or 1934. While 53 of 100 were positive in 1934 compared with 60 of 100 in 1967 (Table 8), this may be attributed to sampling variation or differences in age distribution in the two groups. For individuals 30 or over, 50 of 81 (62%) were positive in 1934, as against 57 of 93 in 1967 (61%). Nor were there qualitative differences; 13 were positive, 5+ asbestos bodies in 1934, against 12 in 1967 (Table 9). Incidentally, the failure to have found asbestos bodies in the past is easily explained—it is very difficult to find these in routine 5 μ H & E sections even in retrospect. We examined such routine stained, unashed sections in the 200 cases. In only 3 of 335 slides were asbestos bodies found; none in the 100 1934 cases although we knew 53 of the 100 showed bodies in ashed 175 μ sections (Table 10).

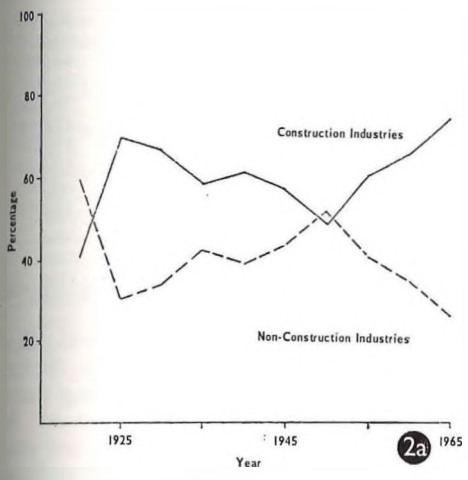

Fig. 2A. Estimated U.S. asbestos consumption.

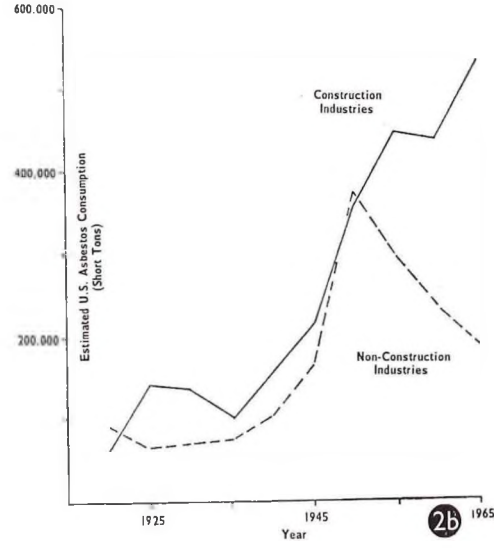

Fig. 2B. Estimated U.S. asbestos consumption.

TABLE 9: SECULAR CHANGES: ASBESTOS BODIES IN ASHED TISSUE SECTIONS (NYC)

Number of Asbestos Bodies	1934	1967
0	47	40
1–4	40	48
5–14	10	10
15+	3	2
	100	100

DISCUSSION

The 1934 findings are not inconsistent with the hypothesis developed from the current (1966-1968) study: that the construction industry is a major source of occupational asbestos exposure and may be responsible for a large proportion of asbestos bodies in routine autopsies. In 1935, for example, almost 60% of asbestos used in the U.S. was used in this industry. But even more germane is the fact that building materials in which asbestos fibers were not firmly bonded, and

TABLE 10: ASBESTOS BODIES: ASHED SECTION AND ROUTINE H & E SECTIONS

	1934		1967	
Total Sections	100			100
	+	0	+	0
Ashed Section*	53	47	60	40
H & E Section**	4	0	3	0

*200 blocks of 200 cases. **335 blocks of 200 cases.

thus potentially free to contaminate the atmosphere when worked, have not changed very much in total quantity used, in the past 40 years (Table 11). The growth in asbestos consumption, 1920-1965, has been largely in products in which asbestos is 'locked-in,' as floor tiles, roofing felts, asbestos cement products. Although airborne fibers may occasionally be derived from these, this is far more unlikely than from more friable substances, as insulation materials, asbestos cement powder or acoustical products; and these, as noted, were used in the same quantities in 1935 as 1965. To summarize: if the construction industry is the major source for the asbestos fibers in asbestos bodies found in urban areas, we could expect 1934 findings to be similar to 1967, since the amount of friable asbestos products used then was the same as now.

TABLE 11: USE OF ASBESTOS IN THE U.S. CONSTRUCTION INDUSTRY (TONS) 1920–1965

	Firmly Bonded				*Friable or Powder*		
	A/C	*Tile*	*Other*	*Total*	*Insulation*	*Cement*	*Total*
1920	5,000	15,500	12,300	32,800	12,300	18,500	30,800
1925	35,300	41,300	25,800	102,400	19,200	20,000	39,200
1930	17,600	45,500	28,900	92,000	16,600	27,600	44,200
1935	14,000	38,500	24,600	77,100	7,400	17,200	24,600
1940	28,500	5,500	36,900	120,400	13,600	26,200	39,800
1945	43,800	60,000	65,700	169,500	14,800	30,200	45,000
1950	99,600	70,000	147,400	317,000	12,700	24,600	37,300
1955	139,200	121,500	150,000	410,700	12,200	21,400	33,600
1960	136,200	127,400	133,500	397,100	13,000	27,300	40,300
1965	180,800	137,500	172,000	490,300	16,100	25,900	42,000

Much more extensive data are needed to see whether the findings in these first 200 cases will be confirmed. Too, findings in other cities should be sought: New York might be unusual in some way. It would seem important to obtain this additional information since it will surely assist in efforts to define those populations at risk to asbestos associated disease. We now appreciate the significant hazard associated with direct occupational exposure and we are aware that other intimate environmental contact, as with indirect occupational,[18-20] neighbourhood[22-24] or family exposure,[23,24] may also be associated with risk, although the extent of this risk awaits more exact characterization. What is not known is whether that level of exposure which results in scant asbestos bodies in the lungs of individuals other than those in the above categories—'asbestos bodies in the lungs of the general population'—is also associated with risk. If it will be confirmed that urban populations 35-45 years ago had asbestos bodies in their lungs as often as such populations do now, and if appropriate pathological studies will demonstrate that mesothelioma was indeed uncommon until recently,[25] there will perhaps be less reason to expect that the current finding of asbestos bodies in the lungs of the general population is a harbinger of future disaster, that we face a widespread epidemic of mesothelioma.

Such definition of the problem will allow appropriate focus of public health control measures. Our data suggest that among these, construction and shipyard work must be prominently considered. In doing so, not only will hazardous direct and indirect occupational exposure be limited, but a major source of the asbestos bodies in the general population will be simultaneously minimized.

Thomson, in 1963,[1] posed an important problem. Close contact with asbestos (of varying intimacy and duration, as with direct or indirect occupational, neighbourhood or family exposure) could be associated with significant neoplasia risk. But was there also a risk associated with general community dissemination of the fibers, presumably in scant numbers and marked by his frequent discovery of occasional asbestos bodies at routine autopsies? Because of the long lapsed period between initial exposure and evidence of disease,[26] future risk attached to the current presence of asbestos bodies could not be denied, although such risk was not evident at this time.[19]

Our observations, preliminary and still to be extended and confirmed, suggest that asbestos bodies were as frequently present 35 years ago as now. If this is true, then scant asbestos bodies may not be associated with future asbestos neoplasia risk in the general population. The risk to more intimately exposed populations will remain for definition and control. This is question enough, but the problem will be a manageable one and within the scope and resources which could be available.

We further suggest that specific asbestos products and uses in the construction and shipbuilding industries have an important relation to occupational and community dissemination of asbestos; focus of control measures here are strongly indicated.

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MORTALITY EXPERIENCES OF ASBESTOS INSULATION WORKERS 1943 - 1968*

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On December 31, 1942, there were 632 members of the Asbestos Workers Union in the New York area (New York and Newark, New Jersey locals of the International Association of Heat and Frost Insulators and Asbestos Workers, I.A.H.F.I.A.W.). From January 1, 1943, to December 31, 1962, another 890 men joined the union. We have followed each of these 1,522 men prospectively to the present. Four hundred and four died by December 31, 1968. Analyses of these deaths demonstrate that serious risks have been associated with work in this trade.

MATERIALS AND METHODS

The procedures followed in this study have been described.[1, 2] Briefly, these men install insulation in the construction and (to a limited extent) in the shipbuilding industries. Approximately 10% of United States union insulation workers are in the New York-New Jersey locals and since our own observations and those of others[3-5] indicate that trade practices are essentially uniform throughout the country, the data which follow may be taken to represent mortality experience in this trade for the United States and Canada; direct study of all United States union insulation worker deaths, 1967-1968, supports this.[6]

Exposure: There is scattered information recorded concerning variation in insulation materials used in the United States, 1870-1969[1, 7]; it would appear that some materials (as, hair-felt, cork, mineral wool) have sharply decreased while others (as, fibrous glass, plastics) have increased.[1] Table 1 lists material currently used.

TABLE 1: INSULATION MATERIALS (U.S.)–1969[8]

		Asbestos Content
1.	*Minerals Containing Asbestos*	
	Product	
	Blankets	100%–amosite
	Moulded	95%–amosite (5% filler)
	Insulation block	(*a*) 85% magnesia: 10–15% amosite + chrysotile
		(*b*) 85% cal. silicate: 10–15% amosite
		(*c*) 85% cal. silicate: 10–15% amosite + chrysotile
		(*d*) 85% calcined diatomaceous silica 10–15% amosite + chrysotile
		(*e*) 85% calcium silicate: 10–15% chrysotile
	Finishing 'cements'	(*a*) 100% chrysotile
		(*b*) 50% chrysotile, 50% cement
	Sprayed asbestos	10–15% asbestos–variable
2.	*Fibrous Glass and/or Mineral Wool*	
	Block, rolls, prefabricates, high temperature insulation, finishing cements, mineral wool and cement.	
3.	*Plastics*	
	Polystyrenes and polyurethanes: prefabricated, powdered and foamed in place.	
4.	*Other*	
	Cork, glass flakes, adhesives (epoxy and solvents; asphalt base and solvent, wheat paste, silicones, magnesia, Portland cement).	

The quantity of asbestos used annually in insulation materials has not changed very much in the past fifty years—12,300 tons in 1920 and 16,000 tons in 1965 (Table 2).[8] There are currently, however, more insulation workers than previously; pre-

* This study was supported by the Health Research Council of the City of New York.

sumably, less asbestos per workman is now the case. Asbestos exposure varies by type of installation (Table 3); about 40% of an insulation

TABLE 2: ASBESTOS USED IN INSULATION MATERIALS IN THE U.S., 1920–1965

Year	*Short Tons*
1920	12,300
1925	19,200
1930	16,600
1935	7,400
1940	13,600
1945	14,800
1950	12,700
1955	12,200
1960	13,000
1965	16,100

TABLE 3: USE OF ASBESTOS INSULATION (%) VARIATION BY CONSTRUCTION TYPE, 1969

Construction	*Fibrous Glass*	*Asbestos*	*Other*
Commercial (Schools, factories)	75	10	15
Power process (Power plants, boiler rooms)	20	60	20
Marine	20	70	10

worker's time is spent in work using asbestos materials (Table 4).

TABLE 4: USE OF ASBESTOS BY INSULATION WORKMEN IN THE U.S., 1969

Material	*Volume % of Total*	*Application % of Time*
Asbestos	35	43
Fibrous glass	45	40
Other	20	17

Exposure of insulation workers in the United States cannot necessarily be equated with that of similar workmen in other countries. For one thing, besides variations in working conditions, the type of fiber and materials used may be different. Until 1930, chrysotile was almost the only asbestos used, amosite making its appearance later and crocidolite practically not at all (see below). Second, spraying of insulation has not been, until recently, widely used in this country, in contrast to some other areas and, in so far as this affects exposure, would not be reflected in our mortality observations. Finally, because of union jurisdictional agreements, the men we have studied are restricted to insulation against temperature change; acoustical insulation and fireproofing are the work of men in other trades and variables peculiar to such work would not be demonstrated by our data.

OBSERVATIONS

632 MEMBERS OF THE UNION ON DECEMBER 31, 1942

Three hundred and eighty of these men were dead by December 31, 1968. Age, year and sex specific expected death rates were available to mid-1967;[1, 10] observed rates by cause are compared in Table 5. All deaths to December 31, 1968 are tabulated in Table 6, by cause.

TABLE 5: EXPECTED AND OBSERVED DEATHS AMONG 632 NY–NJ INSULATION WORKERS, JANUARY 1, 1943 TO APRIL 30, 1967*

	*Expected***	*Observed*
All causes	251·0	349
Lung Cancer	8·9	66
G.I. Cancer	11·2	37
Pleural Mesothelioma	***	6
Peritoneal Mesothelioma	***	14
All other Neoplasms	25·0	21
Asbestosis	***	27

* Of workmen reaching 20 years from first exposure. In addition seven men died before reaching this point.

** Based on United States Mortality data.[1, 10]

*** United States data not available, but these are rare causes of death in general population.

TABLE 6: CAUSES OF 380 CONSECUTIVE DEATHS AMONG 632 INSULATION WORKERS, JANUARY 1, 1943 TO DECEMBER 31, 1968

	Before 20 Years from First Exposure	*After 20 Years from First Exposure*
Lung cancer	0	72
Pleural Mesothelioma	0	6
Peritoneal Mesothelioma	0	16
Gastrointestinal Cancer	0	37
Oropharynx Larynx Cancer	1	5
Cancer of Pancreas	0	3
All other Neoplasms	0	19
Asbestosis	0	30
All other causes	6	185
	7	373

LUNG CANCER

One in five deaths was caused by lung cancer. Despite the recent rise in prominence of mesothelioma, this remains the most important asbestos-associated neoplasm among insulation workers. It is of interest, therefore, that there appears to be a special relationship to

TABLE 7: PLEURAL AND PERITONEAL MESOTHELIOMA AMONG NYC ASBESTOS INSULATION WORKERS,* JANUARY 1, 1943 TO DECEMBER 31, 1968

		Number	Mesothelioma		Bronchogenic Carcinoma	Asbestosis
			Pleural 6	Peritoneal 16	72	30
Age at Onset						
(*Years*)	Mean		25·2	20·0	25·4	24
	Median		21	19	25	24·5
	S.D.		±5·4	±4·3	±6·36	±5·6
	Range		17–25	16–30	16–54	14–36
Age at Death						
(*Years*)	Mean		56·0	62·7	63·8	66·7
	Median		55	60	64	66
	S.D.		±2·4	±10·2	±7·25	±8·02
	Range		54–60	50–83	39–83	53–79
Lapsed Period						
(*Years*)	Mean		34·8	43·0	38·7	42·8
	Median		34·5	40·5	39	44
	S.D.		±2·3	±8·9	±8·5	±7·69
	Range		32–38	32–61	22–60	30–59

*In addition, we have observed 2 peritoneal mesotheliomas found at autopsy, in men who had died of bronchogenic carcinoma.

cigarette smoking among asbestos insulation workers[10] (see below). Overall lung cancer death rates were seven times expected.*

MESOTHELIOMA

Twenty-two of the 380 deaths were due to mesothelioma, 6 pleural and 16 peritoneal. Two occurred 1943-1954; 20, 1955-1968; average lapsed period from onset of exposure was longer and average age at death was greater for peritoneal than for pleural mesothelioma.

Peritoneal mesothelioma does not seem to be related to cigarette smoking—we have seen 4 such cases in men who never smoked cigarettes. In the 3 cases of pleural mesothelioma for which we personally recorded smoking histories, all had smoked cigarettes.

The preponderance of peritoneal mesothelioma among our cases, as in the series of Enticknap and Smither,[11] is not necessarily contradictory to the observations of Wagner,[12] Hourihane[13] and others in which pleural neoplasms were largely seen. It may, rather, reflect the derivation of the cases—consecutive series with regular occupational exposure on the one hand, against random cases, many with non-occupational environmental exposure, on the other. It may be that in those people who develop mesothelioma in association with asbestos exposure, the site of the mesothelioma tends to vary to some extent with the intimacy and duration of exposure—pleural with lesser exposure and peritoneal with greater.

*There are, in addition, three men found to have lung cancer during our survey, and who are now alive following therapy.

GASTROINTESTINAL CANCER

As in our first observations,[1] we have continued to see more deaths due to cancer of the stomach and colon than expected. However, the ratio is only 3 times expected and, with so few deaths, we are not yet certain of the relationship.

CANCER OF PANCREAS; OROPHARYNGEAL CANCER

The same insecurity noted with gastrointestinal cancer is associated with these as well. We are, however, concerned that the incidence of both of these neoplasms will be increased among asbestos workers. As a practical measure, we now inspect the oral cavity during all clinical examinations of asbestos workers; one cancer of the lip has been discovered and successfully excised.

It is of interest that 2 of the 3 recent deaths of tongue cancer, were in men who never smoked cigarettes.

ASBESTOSIS

Thirty deaths of the 380 were due to respiratory insufficiency with cor pulmonale.

890 MEN ENTERING UNION AFTER DECEMBER 31, 1942

Fifty-four of these men had prior insulation work experience before entering this union in 1943 or later; 836 did not. By and large, therefore, the men in this group are younger and would not be expected to have yet achieved a lapsed period from onset of exposure sufficient to have brought them to a period of serious risk of neoplastic death. This theoretical consideration has so far been confirmed by our experience (Table 8).

TABLE 8: ONSET OF EXPOSURE AND DEATHS OF NEOPLASM AMONG 890 MEN WHO JOINED INSULATION WORKERS' UNION, 1943–1962

	Year Onset of Asbestos Insulation Exposure	Number	Deaths 1943–1968	
			Total	Neoplasm
1	≤1929	8	3	3
2	1930–1942	46	2	1
3	1943–1947	164	9	4
4	1948–1952	250	5	0
5	1953–1957	186	5	0
6	1958–1962	236	0	0
		890	24	8

1: CA Lung (32), CA Lung (41), Leukemia (31).
2: CA Lung (31), Pancreatitis.
3: CA Lung (21), Asbestosis, Peptic Ulcer, CA Pancreas (20), CA Tongue (20), CA Colon (17), Pulm. embolism, Cirrhosis, Coronary.
4: Accident, Coronary, Cong. Heart Failure, Coronary, Coronary.
5: Pneumonia, Rheumatic H.D., Coronary, Cirrhosis, S.B.E.
6:
( ) = years from onset of exposure.

MULTIPLE NEOPLASMS

We have seen several instances of more than one primary neoplasm in the same individual (lung-lung; lung-larynx, etc.). These have been too infrequent to warrant more than brief recording except perhaps for two men who died of bronchogenic carcinoma and were found, at autopsy, to also have peritoneal mesothelioma.

EFFECTS OF CIGARETTE SMOKING

There appears to be an important influence of cigarette smoking on the incidence of lung cancer among asbestos insulation workers.[10] On January 1, 1963, there were 370 men still alive of our original 632 cohort (all now with long-lapsed periods from onset of exposure and very much at risk). Eighty-seven never smoked cigarettes: one died of lung cancer by December 31, 1968. Two hundred and eighty three had a history of cigarette smoking —27 have died of lung cancer (Table 9).

TABLE 9: CIGARETTE SMOKING AND LUNG CANCER IN ASBESTOS INSULATION WORKERS. EXPERIENCE OF 370 MEN FOLLOWED PROSPECTIVELY JANUARY 1, 1963 TO DECEMBER 31, 1968

	No History of Cigarette Smoking	History of Cigarette Smoking
Number of men	87	283
Expected lung cancer deaths to 4.30.67	0·18	2·98
Observed deaths to 4.30.67	0	24
Observed deaths to 12.31.68	1	27
Total cases to 12.31.68	1	30*

*3 men still alive (all cigarette smokers).

Computations suggest that an asbestos worker who smokes cigarettes has 90 times the risk of dying of lung cancer compared with a man who neither works with asbestos nor smokes cigarettes.

AGE AT ONSET OF EXPOSURE

Table 10 records the mortality experience of the 632 men, by age category at onset of exposure.

FIBER VARIETY AND MESOTHELIOMA; EVIDENCE FROM PRESENT OBSERVATIONS

Crocidolite seemed initially to be unique among asbestos varieties in having a special relationship to mesotheliomas. It has been considered desirable to obtain further evidence on this question, by study of populations exposed to other fiber varieties.[14,15] It may be useful, therefore, to evaluate the experiences of N.Y.-N.J. insulation workers in this regard.

CROCIDOLITE EXPOSURE OF INSULATION WORKERS IN THE UNITED STATES

There is no crocidolite mined in the United States: all must be imported either from South Africa, Bolivia or Australia. It has been possible, by review of import data, to accurately calculate crocidolite use.

TABLE 10: MORTALITY EXPERIENCE OF 632 NYC INSULATION WORKERS, BY AGE AT ONSET OF EXPOSURE

	Age at Onset of Exposure			Total
	≤20	21–30	30+	
Cancer of Lung	14 (5·7%)	52 (17·3%)	6 (7·1%)	72
Pleural Mesothelioma	3	3	0	6
Peritoneal Mesothelioma	10 (5·2%)	5 (2·7%)	1 (1·2%)	16
Gastrointestinal Cancer	12 (4·9%)	17 (5·7%)	8 (9·4%)	37
All other neoplasms	6	15	7	28
Asbestosis	11 (4·5%)	15 (5·0%)	4 (4·7%)	30
All other causes	45	92	54	191
Deceased 12.31.68	101	199	80	380
Alive 12.31.68	145	102	5	252
Total	246	301	85	632

TABLE 11: ESTIMATED U.S. ASBESTOS FIBER CONSUMPTION

Year	*Crocidolite*	*Amosite*	*Canadian Chrysotile*	*Other*	*Total*
1920	No data	No data	152,000	4,000	156,000
1925	No data	No data	200,700	4,100	204,800
1930	1,907		198,200	8,500	208,407
1935	501		154,200	20,500	175,201
1940	8,068		225,900	36,300	270,268
1945	8,700	4,500	355,800	9,000	378,000
1950	8,500	5,400	687,400	27,400	728,700
1955	11,700	18,000	699,100	11,600	740,400
1960	19,600	19,500	605,800	24,600	669,580
1965	17,100	21,400	661,100	20,100	719,700

*Total Amosite and Crocidolite**

1930	1,907
1931	823
1932	212
1933	233
1934	152
1935	501
1936	1,432
1937	2,928
1938	3,282
1939	6,129

*Source: U.S. Department of Commerce.

Very little crocidolite entered this country until World War II (Table 11). Asbestos used here was almost entirely chrysotile. The largest asbestos manufacturer in the United States first used crocidolite in 1929 (for sheet packing, not insulation). This company never used crocidolite for the manufacture of insulation at any time (1929-1968), employing it only in two product lines (asbestos cement pipe, and packings).[16] These data are consistent with an analysis of insulation materials used in past decades on the United States west coast, where only chrysotile and amosite were found.[5] During these same years, 1920-1940, more than 267,000 tons of chrysotile were used for insulation purposes.

This is not to say that it can be proven that crocidolite was *never* used before 1930 for insulation in the United States. Rather, if any was so utilized, it could only have been in very small amounts. Therefore, crocidolite exposure of United States insulation workers before 1930 and probably before 1940 is unlikely to have occurred to any significant extent and, as seen in Table 1, is still a very minor affair.

MESOTHELIOMA AMONG UNITED STATES INSULATION WORKERS

The first mesothelioma reported in which occupational asbestos exposure was described, occurred in an insulation worker, a member of the I.A.H.F.I.A.W., in 1946.[17] It is not known whether cases occurred before then, but they have been noted since.[18] We have seen 22 cases, 1947-1968, in our group of men, as detailed above. *Every one has occurred in an insulation worker who began work before* 1930 (Table 12). The mean lapsed period from onset of exposure to death was 34.8 years for pleural mesothelioma and 43.0 years for peritoneal.

As detailed, crocidolite exposure of United States insulation workers, to the extent that it occurred, could not have been very extensive before 1940. Therefore, if a long lapsed period is required for asbestos-induced mesothelioma, crocidolite exposure alone cannot easily explain the mesotheliomas seen among insulation workers in the United States. Of course, it may be that crocidolite's virulence makes for shorter lapsed periods between onset of exposure and death. But if this is so, it is difficult to explain the *absence* of mesotheliomas among the 836 men in our study who began work in 1943 or later (Table 8).

These considerations are relevant only to conditions in the United States, among insulation workmen. They do not disprove a special role for crocidolite elsewhere, in other circumstances. They should, however, engender caution in relying on other asbestos varieties always to be significantly less likely to cause mesothelioma. The relative mesothelioma hazard for each fiber variety is still to be defined in humans. At this time our data suggest that it would be prudent to regard all fiber varieties as potentially hazardous, and to utilize appropriate industrial hygiene measures for all.

TABLE 12: LAPSED PERIOD FROM ONSET OF EXPOSURE TO DEATH OF 22 CASES OF MESOTHELIOMA, 1947–1968, AMONG 632 INSULATION WORKERS FOLLOWED PROSPECTIVELY FROM JANUARY 1, 1943

Patient	*Site*	*Year Onset Employment*	*Year of Death*	*Age at Death*	*Years from Onset*
1. A.C.	Pleural	1922	1947	60	25
2. A.M.	Pleural	1923	1956	58	33
3. A.S.	Pleural	1929	1961	54	32
4. J.B.	Pleural	1929	1963	54	34
5. F.C.	Pleural	1927	1964	53	37
6. T.C.	Pleural	1928	1966	56	38
7. E.M.	Peritoneal	1910	1949	61	39
8. F.S.	Peritoneal	1923	1956	50	33
9. M.C.	Peritoneal	1924	1956	52	32
10. H.H.	Peritoneal	1918	1959	60	41
11. J.B.	Peritoneal	1905	1963	86	58
12. H.D.	Peritoneal	1925	1963	66	38
13. W.N.	Peritoneal	1919	1964	63	45
14. A.C.	Peritoneal	1918	1964	68	46
15. J.D.	Peritoneal	1924	1965	58	41
16. J.LaG.	Peritoneal	1929	1965	53	36
17. J.O'C.	Peritoneal	1917	1966	79	49
18. G.V.	Peritoneal	1924	1967	59	43
19. H.C.	Peritoneal	1929	1967	57	38
20. J.P.	Peritoneal	1929	1968	58	39
21. R.B.	Peritoneal	1907	1968	83	61
22. H.T.	Peritoneal	1929	1968	56	39

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RESULTS OF ASBESTOS EXPOSURE IN FRANCE

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The asbestos industry in France employed about 14,000 persons in 1968 of whom there were 5,000 in manufactured asbestos, 1,000 in the production of the textile and 8,000 in asbestos cement.

Chrysotile represents 95% of the tonnage of crude asbestos consumed in France.

in the Rouen and in the Clermont Ferrand districts because the great textile plants are located near Rouen in Condé sur Noireau and in Clermont Ferrand.

In Condé sur Noireau, Ferodo S.A.F. has 2 plants. One for textiles employs about 600 persons and another for brake linings employs

TABLE 1: CONSUMPTION

	1938	1950	1957	1967
All production	17,100	32,400	73,100	120,300
Asbestos cement and floor tiles	11,700	25,300	61,800	101,900
Manufactured asbestos and others	5,400	7,100	11,300	18,400

The consumption in 1967 is almost the double of that in 1957 and 4 times that in 1950 (Table 1).

In France the law recognizes asbestos fibrosis as an occupational disease but not tuberculosis or carcinoma of the lungs or mesothelioma of the pleura.

about 1,200. At present there is an increase of fibrosis disease because 1968 is just 20 years after the production increase at the end of the last war.

Table 3 shows, year by year, between 1960 and 1968, the number of workers pensioned in the textile plant (Ferodo S.A.F.).

TABLE 2: STATISTICS OF ASBESTOSIS DISEASES
NUMBER OF COMPENSATIONS

Compensation	*Bordeaux*	*Dijon*	*Clermont Ferrand*	*Lille*	*Marseille*	*Nantes*	*Paris*	*Rouen*	*Strasbourg*	*Limoges*	*Total*
5%	–	–	–	1	–	–	–	–	–	–	1
10%	–	1	–	–	1	–	–	1	1	–	4
15%	–	–	1	–	–	1	–	2	1	–	5
20%	–	1	1	–	–	1	1	4	–	–	8
25%	–	–	–	1	–	–	1	4	–	–	6
27%	–	–	–	–	–	–	1	–	–	–	1
30%	1	–	1	1	1	2	1	3	–	–	10
35%	–	–	–	–	–	–	2	3	–	–	5
40%	–	–	1	–	–	3	1	8	–	–	13
45%	–	–	–	2	–	–	–	1	–	–	3
50%	–	–	1	–	–	1	2	12	–	–	16
55%	–	–	–	–	–	–	–	1	–	–	1
60%	–	–	1	–	–	–	–	2	–	1	4
65%	–	–	–	–	–	–	1	–	–	–	1
67%	–	–	–	–	–	–	2	3	–	–	5
70%	1	–	1	–	–	–	–	2	–	–	4
75%	–	–	–	–	–	–	–	2	–	–	2
80%	–	–	–	–	–	–	–	1	–	–	1
90%	–	–	–	–	–	–	1	–	–	–	1
100%	–	–	–	–	–	–	–	3	–	–	3
	2	2	7	5	2	8	13	52	2	1	94

Asbestosis is mainly observed in asbestos textile plants. We have much asbestosis because of the unsatisfactory working conditions in asbestos textiles after the war.

The statistics of Securité Sociale for all France in 1962 are shown in Table 2.

The great majority of pensioned persons are

The number for the whole textile factory is 89 workers pensioned in January 1969.

PLEURAL PLAQUES

Pleural plaques are observed among workers in textile plants. There is no parallelism between fibrosis and pleural plaques.

cant difference in the incidence of tumours of the lung was seen between workers with continuous and intermittent exposure to asbestos. To the workers with continuous exposure, 16 insulation workers working for other companies should be added. Among these, 5 cancers of the lung and 1 mesothelioma of the pleura occurred.

Our study of asbestosis in Italy also includes the measurement of airborne dust concentration in the working places. Up to date the results of 380 measurements are available. All counts were made with phase contrast microscopy at a magnification of 500 diameters, after dissolution of the millipore membrane filter with methylcellosolve. Table 12 gives the range of dust concentration in the different work places.

TABLE 13: RESULT OF COUNTS OF AIRBORNE ASBESTOS FIBRES (ON MILLIPORE MEMBRANE FILTER)

	No. of Counts with $\leqslant$ 12 *Fibres/cm*^{3}		*No. of Counts with* $>$ 12 *Fibres/cm*^{3}		*Total*
	No.	%	*No.*	%	
Balangero Mills	29	25·0	87	75·0	116
Textiles	63	57·3	47	46·7	110
Friction products	106	94·6	6	5·4	112
On board 'Laura C'	33	67·3	9	32·7	49

Table 13 gives the percentage of counts with less than 12 fibres/cm.^{3} air as compared with the percentage of the counts with more than 12 fibres/cm.^{3}

It is planned to carry out approximately 1,000 counts per year.

EPIDEMIOLOGY OF PRIMARY MALIGNANT MESOTHELIAL TUMOURS IN CANADA

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Evidence for an association between malignant mesothelioma and asbestos exposure rests mainly on the remarkable concentration of cases reported by Wagner *et al.* (1960) in the crocidolite mining areas of South Africa, the case-control studies of Elmes *et al.* (1965) in Belfast and Newhouse and Thompson (1965) in London, and the considerable excess mortality due to this disease observed by Selikoff *et al.* (1965) in American insulation workers. The survey described here was undertaken to obtain a more generally representative view of the problem in one of the two major chrysotile-producing countries of the world. Our aim has been to study all fatal cases known to have occurred in Canada since 1959.

Method

At the end of 1967 we wrote to the 423 members of either the Canadian Association of Pathologists or the Quebec Association of Laboratory Physicians inquiring whether or not since 1959 they had seen any cases of fatal primary malignant tumours of the pleura or peritoneum diagnosed by autopsy or biopsy. In due course we obtained replies from them all and, if this was in the affirmative, a personal visit was made by one of us to review the records and to exclude cases which did not meet the criteria mentioned above or about which the pathologist had serious doubt. In all, 165 cases were found; 113 were pleural, 45 peritoneal, 3 pleural and peritoneal and 4 pericardial. The diagnosis was supported by autopsy in three-quarters of the cases and by biopsy in the remainder. Later we plan to have material from as many as possible reviewed by a panel of pathologists.

During the visit to the pathologist 2 fatal control cases (one primary and one secondary lung cancer) were selected from the autopsy or biopsy records, matched for sex and as closely as possible for age and date of death. In 2 instances no adequately matching case of primary lung cancer could be found. The cases of secondary lung cancer are referred to hereafter as control group A and of primary lung cancer as control group B.

A field survey was then arranged in which investigators (doctors, public health nurses and public health inspectors) sought out the relatives and friends of the deceased and completed a detailed questionnaire relating to occupation, residence, indirect household exposure to dust, family history and smoking habits. No interviewer knew to which group the case under investigation belonged and the information recorded in the questionnaires has also been coded blind. Questionnaires have been completed so far for 433 (88%) of the 493 cases and controls. The follow-up is still in progress and information for perhaps half the remainder may eventually be obtained.

Table 1: Distribution of Cases by Year of Death and Province

Year	*Ontario*	*Quebec*	*Other Provinces*	*Canada*
1960	—	6	10	16
1961	1	6	2	9
1962	4	4	5	13
1963	9	3	7	19
1964	6	8	3	17
1965	7	7	5	19
1966	8	13	10	31
1967	6	14	6	26
1968 (1st half)	5	8	2	15
	46	70	49	165
Incidence per million per year				
1960–1962	0·8	1·5	0·9	1·0

Findings

The distribution of cases by province and year of death is shown in Table 1 and by age, sex and site in Table 2. More cases were found in 1966 and 1967 than in previous years. Recent interest in these tumours may

TABLE 2: DISTRIBUTION OF MESOTHELIAL TUMOURS BY AGE, SEX AND SITE

Age (Years)	*Pleura*		*Peritoneum*		*Pleura and Peritoneum*		*Pericardium*		*Total*	
	M	F	M	F	M	F	M	F	M	F
0–	—	—	—	1	—	—	—	—	—	1
10–	2	1	—	—	—	—	—	—	2	1
20–	—	4	—	—	—	—	—	—	—	4
30–	7	4	1	1	—	—	—	1	8	6
40–	9	7	4	1	—	—	—	1	13	9
50–	18	3	11	4	1	—	1	1	31	8
60–	26	5	2	5	—	2	—	—	28	12
70–	15	5	7	6	—	—	—	—	22	11
80–	3	4	1	1	—	—	—	—	4	5
All	80	33	26	19	1	2	1	3	108	57
Total	113		45		3		4		165	

have contributed to this and, as the records in some hospitals were poorer for the earlier years, memory may also have been a factor. With these points in mind the rate of increase is difficult to assess. During the 8 complete years, 1960-67, the average annual incidence was 1.0 per million of population. The rate was appreciably higher in Quebec (1.5) than in Ontario (0.8) or in the rest of Canada (0.9) —1966 and 1967 again accounting for much of the difference. There was no difference in the sex distribution under the age of 50 but thereafter males were in considerable excess.

The case and two control series were very similar in age distribution and in the proportion of each for which the questionnaire was completed (Table 3). In this prelimary report only the more immediately important questions relating to asbestos exposure and to cigarette smoking have been examined. Table 4 summarizes the occupational exposure for males aged 15 years or more at death, excluding the 10 years preceding death. There was a substantial excess of 'definite' and 'probable' exposures over both control groups ($P < .001$) but virtually no difference in the frequency of jobs in which exposure was classified as 'possible'. In 61% of the cases, however, occupational exposure to asbestos was considered 'unlikely'. Only two women in all had any occupational exposure; one was classified as definite and she was a case.

The occupations involving definite or probable exposure to asbestos are listed in Table 5.

TABLE 3: AGE AND SEX DISTRIBUTION

Age (Years)	*Males*	*Controls*		*Females*	*Controls*	
	Cases	*Group A*	*Group B*	*Cases*	*Group A*	*Group B*
0–		1	1	1	1	1
10–	2			1		
20–		2		4	2	
30–	8	6	2	6	6	3
40–	13	14	24	9	10	14
50–	31	26	24	8	7	8
60–	27	35	35	12	15	12
70–	23	20	17	11	13	15
80–	4	4	4	5	3	3
All	108 (97)	108 (92)	108 (94)	57 (51)	57 (52)	56 (47)
Mean	58·7	59·1	59·1	55·8	57·4	58·8
SD	13·9	13·3	12·5	19·3	17·3	15·5

No. of questionnaires completed shown in parenthesis ( ). *Total:* 433/493 = 88%

Men employed in asbestos textile manufacture, the installation of brake-linings or as insulators were responsible for most of the excess of cases over controls. Mine or mill workers made little contribution to the difference and only one person, a hardware storeman, had worked with asbestos-cement products. The time between first exposure and death for the 21 cases in the definite or probable class ranged from 16 to 50 years; 5 persons who worked on the job for less than 4 years had intervals of between 18 and 33 years.

Residential histories have been analysed only as regards proximity to an asbestos mining area. Excluding 5 cases (3 mine or mill workers and 2 textile workers) and one mine and mill worker in each control group who lived in mining towns, only 3 men and 2 women had ever lived within 20 miles of an asbestos mine. One of the 5 was a case,

TABLE 4: OCCUPATIONAL EXPOSURE TO ASBESTOS

Males:	*Cases*		*Controls Group A*		*Controls Group B*	
	No.	%	*No.*	%	*No.*	%
Definite	11	*20·8*	1	*3·3*	1	*4·3*
Probable	9		2		3	
Possible	17	*17·7*	20	*22·0*	15	*16·1*
Unlikely	59	*61·4*	68	*74·7*	74	*79·6*
*All**	96	*100*	91	*100*	93	*100*
Females:						
Definite	1	*2*	—		—	
Probable	—		—		—	
Possible	—		1	*2*	—	
Unlikely	49	*98*	50	*98*	46	*100*
*All**	50	*100*	51	*100*	46	*100*

*One child in each group dying under the age of 15 has been excluded.

Males. Distribution of definite and probable groups compared with possible and unlikely: $\chi^2 = 14{\cdot}9$; 2 d.f, $P < 0{\cdot}001$.

TABLE 5: DISTRIBUTION OF OCCUPATIONS CLASSIFIED UNDER DEFINITE OR PROBABLE EXPOSURE TO ASBESTOS

	Cases	*Control Group A*	*Control Group B*
Asbestos mine or mill	3	1	1
Asbestos textile manufacture	5*	0	0
Brake-lining installation	2	0	0
Insulation	6	0	0
Other occupations involving contact with insulation materials	4	2	3
Hardware store (handling asbestos sheet and pipe)	1	0	0
Total	21	3	4

*Includes one person who worked in 'brake-lining manufacture'.

TABLE 6: AVERAGE DAILY NUMBER OF CIGARETTES SMOKED

Males:

Cigarettes per Day	*Case Group*		*Control Group A*		*Control Group B*		*Cases with Asbestos Exposure*	
Nil	20	*22*	19	*21*	4	*5*	2	*10*
10 or less	18	*19*	19	*21*	10	*11*	4	*20*
11–29	35	*38*	36	*40*	46	*50*	13	*65*
30 or more	19	*20*	16	*18*	32	*35*	1	*5*
*Total**	93	*100*	90	*100*	92	*100*	20	*100*

Females:

Cigarettes per Day	*Case Group*		*Control Group A*		*Control Group B*	
Nil	29	*58*	35	*68*	21	*46*
10 or less	9	*18*	5	*10*	8	*17*
11–29	11	*22*	8	*16*	13	*28*
30 or more	1	*2*	3	*6*	4	*9*
*Total**	50	*100*	51	*100*	46	*100*

*Excluding 6 persons under 15 years of age and 5 males whose smoking history was not recorded.

3 were in control group A and one in control group B.

The possibility of contact as a result of some other member of the family bringing home dusty clothes from an occupation involving the use of asbestos was also examined. Again excluding 6 men (4 cases and 1 in each control group) already themselves occupationally exposed (definitely or probably) there was one case in a female with 'definite' home exposure and one male in control group B with 'probable' home exposure. In addition there were 26 persons with a 'possible' home exposure: 10 cases, 9 in control group A and 7 in control group B.

An analysis of cigarette smoking habits is shown in Table 6. In both males and females, persons in the primary lung cancer group had smoked considerably more than those in either the case or secondary lung cancer groups. The latter 2 groups had almost an identical distribution of smoking habits. The 20 men with definite or probable occupational exposure to asbestos showed no great difference in smoking habits from cases without occupational exposure.

Conclusion

The findings from this national survey indicate that primary malignant mesothelial tumours are very rare in Canada and it is somewhat doubtful whether there has been any true increase in incidence since 1959. An association between these tumours and definite or probable occupational exposure to asbestos was clearly demonstrated by only a minority of male cases and only one female case had any such exposure. Lesser degrees of occupational exposure, indirect household contact and residence in asbestos mining areas were no more frequently reported for cases than controls. Probably our most noteworthy finding was that almost all the excess in occupational exposure was in the manufacture and industrial application of asbestos rather than in mining or milling. Taken in conjunction with the results of other epidemiological studies this suggests either that chrysotile is less associated with mesothelial tumours than other forms of asbestos or that something in addition to asbestos is necessary. If there is an additional factor, our findings imply that it is not cigarette smoking.

This survey was made possible by the great help given us by pathologists and members of provincial and other health departments throughout Canada. We are also much indebted to all those who did the field work and to the friends and relatives of the deceased for their co-operation. The investigation was supported by a grant from the Institute of Occupational and Environmental Health of the Quebec Asbestos Mining Association.

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ASBESTOS EXPOSURE IN AUSTRALIA

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Asbestos exposure in Australia has predictably produced asbestosis in many workers, particularly in the asbestos-cement and insulation industries, and pleural mesothelioma has been reported in every State, frequently with evidence of asbestos exposure. The pattern is familiar.

The exception to the pattern has been the effects of exposure to crocidolite at the blue asbestos mine in Wittenoom, Western Australia.

In 1939, large crocidolite deposits were discovered at Wittenoom, which is about 1,500 miles north of Perth, in latitude 22-19s. Significant production of fibre commenced in 1943, and the amount produced per annum has varied greatly, from a few hundred tons initially to (between 1958 and 1966) a maximum of 15,000 tons of fibre per annum.

The number of men employed has also varied considerably and rose to between 300 and 400 during this latter period.

There were very considerable fluctuations in the production figures, and in the number of men employed. The very high temperatures, the barrenness of the terrain, the lack of amenities, the dust itself, and a main labour force consisting almost entirely of new migrants to Australia, led to an exceedingly high labour turnover.

In 1966, poor fibre return, labour problems, transport and production costs, etc., forced the mine's closure.

Probably fewer than 20% of the men employed at the mine at any one time had been there for as long as three years, and this would include tradesmen, supervisory and office staff.

From the beginning, it was obvious that the mine and mill were too dusty and the Health Department first became concerned about the health of the men in 1948. Despite this concern, and despite earnest attempts at dust suppression and improved ventilation, the situation was always far from satisfactory. Costly attempts at dust suppression were defeated by lack of maintenance, lack of skilled tradesmen and the economic necessity of continuing production, at all costs.

From the outset, all the asbestos miners had an initial clinical examination, with a chest X-ray and annual periodical chest X-rays, supplemented after 1957 by an annual clinical examination. Pulmonary function tests were not done routinely, but only where there was other suggestive evidence of disease.

The first patient was a migrant, aged 33 years, an underground scraper driver, who developed pulmonary tuberculosis, and in 1958 had a lobectomy. An examination of the resected specimen showed asbestosis.

Subsequent enquiries have shown that there might have been two other asbestos miners diagnosed in earlier years as suffering from pneumoconiosis, but these were recorded simply as 'silicosis' and both had had extensive previous exposure to quartz in other industries.

Table 1: Analysis of Men Affected

Since June 1958, 103 men who worked at Wittenoom have developed pneumoconiosis:		
Mill Workers	41	(5)
Underground Workers	29	(4)
Mixed Underground and Mill Workers	6	(1)
Men with other significant mining	24	(2)
Men excluded because of lack of clinical detail	3	(2)
	103	(14)

Table 1 is an analysis of the men affected until December 1967. The figures in brackets are additional cases which have been diagnosed since this initial analysis. Of the original 103, 33 have been excluded from further analysis because of previous dust exposure, mixed mine and mill history or insufficient clinical data.

Table 2 shows the year the men commenced work at the mine and mill, and the year diagnosed.

It would appear that, although working conditions were poor and dusty from the beginning, these did not cause significant lung disease while the production of fibre remained low.