BD

Asbestos Information Association/North America

22 East 40th Street
New York, N. Y. 10016
212-689-3378

14561A

PLAINTIFF'S EXHIBIT
JM-1921

December 27, 1972

Gentlemen:

Attached for your information are two recent medical papers of considerable importance with regard to asbestos-health.

The first, the "Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer," presents in question and answer format the joint professional opinions of nearly three dozen of the world's top asbestos-health experts on various aspects of this problem. This report is secheduled to be released shortly. Until then, however, despite the wealth of valuable information contained therein, we have been asked to keep it "semi-confidential," which means that it should not be given wide distribution even within your own companies, and certainly should not be distributed to those outside the industry.

The second paper is entitled "Mortality in Relation to Occupational Exposure in the Asbestos Industry." It is a study of 1,400 retired United States asbestos manufacturing workers, and while the results indicate an excess of asbestos related disease among the cohort, the rates are by no means as high as those reported by Selikoff in his various studies.

Sincerely,

Matthew M. Swetonic
Executive Secretary

Enclosures (2)

CC:

A. E. Alpine	- Certain-teed Products Corporation
G. M. Armstrong	- American Asbestos Textile Corporation
James Armstrong	- Bendix Corporation
A. A. Cross	- Cape Asbestos Company, Ltd.
Hugh Dawson-Walker	- Hill & Knowlton (UK) Limited
J. Hall	- GAF Corporation
S. Holmes	- Turner Brothers Asbestos
Wilfred Howard	- Asbestos Information Committee (England)
Michael F. Howe	- Turner & Newell
H. M. Jackson	- Johns-Manville Corporation
Art Neilson	- Fireman's Fund Insurance Company
S. Monoky	- Certain-teed Products
P. V. Pelnar, M. D.	- Institute of Occupational & Environmental Health
F. L. Pundsack	- Johns-Manville Corporation
Ivan Sabourin	- Johns-Manville Legal Counsel
Hans Weill, M. D.	- Tulane University
G. W. Wright, M. D.	- St. Luke's Hospital
I. C. Campbell	- Q.A.M.A.
J. A. Gossip	- Q.A.M.A.
Martin Sendecki	- Congoleum Industries Inc.
George P. Vogel	- Ertel Engineering
Mike Donovan	- Johns-Manville Corporation
C. Nelson Codding	- Jim Walter Research Corporation
R. C. Carter	- Johns-Manville Corporation
Lyman Field	- Rogers, Field, Gentry, Benjamin & Robertson

EFM
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To send to Avy
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AIA/NA MEMBER COMPANIES

E. C. Bratt	- H. K. Porter
George Barge	- Atlas Asbestos Company
H. Randolph Brown	- Uvalde Rock Asphalt Company
J. D. Christian	- Cassiar Asbestos Corporation, Ltd.
A. H. Fay	- National Gypsum Company
J. K. Whittaker	- Nicolet Industries, Inc.
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A. R. Hooker	- The Flintkote Company
J. H. Marsh	- Raybestos-Manhattan
C. G. Morgan	- North American Asbestos Corporation
C. A. Neumann	- Kentile Floors Inc.
J. L. Rainey	- American Asbestos Textile Corporation
J. W. Rawlings	- Union Carbide Corporation
Kurt Schwarz	- Supradur Manufacturing Corporation
F. J. Solon, Jr.	- Johns-Manville Corporation
Wes Sauerland	- Jim Walter Corporation
S. D. Weaver	- Cement Asbestos Products Company
Paul Weiner	- GAF Corporation
J. R. Stetson	- Congoleum Industries, Inc.

AIA/NA ENVIRONMENTAL CONTROL SUB-COMMITTEE

E. M. Fenner	- Johns-Manville Corporation
Ike Weaver	- Raybestos-Manhattan
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AIA/NA LEGAL COUNSEL

Bradley Walls	- Burns, VanKirk, Jube & Kafer

AIA/NA PUBLIC RELATIONS COUNSEL

Jack Steinberg	- Cunningham & Walsh
Mike Isser	- " "
Tony Federico	- " "
C. L. Forbes	- " "

AIA/NA ENVIRONMENTAL CONTROL CONSULTANT

C. L. Sheckler

RECEIVED JAN 8 1973

MRC
Medical Research Council

Pneumoconiosis Research Unit
Llandough Hospital
Penarth, Glamorgan

telephone Penarth 708701

reference JCW/Y 212-8

4th December, 1972

Dear George,

I enclose the Report and Recommendations of the Advisory Committee as submitted to Dr. J. Higginson.

Yours sincerely,

Chris
J. C. WAGNER
Secretary of the Advisory Committee

Dr. G. Wright,
Division of Medicine,
St. Luke's Hospital,
11311 Shaker Boulevard,
Cleveland, Ohio 44101, USA

REPORT OF THE ADVISORY COMMITTEE ON ASBESTOS CANCERS TO THE

DIRECTOR OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

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The meeting was held at the International Agency for Research on Cancer, Lyon, France on 5th and 6th October, 1972. The Committee consisted of three panels - Epidemiology, Pathology, Physics and Chemistry.

Epidemiology Panel

Dr. M. Becklake	McGill University, Department of Epidemiology & Heal 3775 University Street, Montreal 110, Quebec, Canad
Dr. H. Bohlig	Chefarzt der Strahlenabteilung des Stadtischen, Krenkenhauses Ludenscheid, 588 Ludenscheid, Germany
Dr. N. Day	Unit of Epidemiology and Biostatistics, Internation Agency for Research on Cancer, 150 Cours Albert Tho 69008, Lyon, France.
Prof. P. C. Elmes	Department of Therapeutics and Pharmacology, Queen' University of Belfast, Institute of Clinical Scienc Grosvenor Road, Belfast, BT12 6BJ, N. Ireland, UK.
Dr. J. C. Gilson	Medical Research Council, Pneumoconiosis Unit, Llan Hospital, Penarth, Glamorgan, Wales, UK. (Chairman)
Dr. J. Lepoutre	Medical Department, N.V. Eternit, Kapelle-op-den-Bo Belgium.
Prof. J. C. McDonald	McGill University, Department of Epidemiology & Hea 3775 University Street, Montreal 110, Quebec, Canad
Mr. C. E. Rossiter	Medical Research Council, Pneumoconiosis Unit, Llan Hospital, Penarth, Glamorgan, Wales, UK.
Dr. H. Sakabe	Department of Industrial Physiology, Ministry of La National Institute of Industrial Health, 2051 Kizukisumioyoshi-Cho, Kawasaki, Japan.
Dr. I. J. Selikoff	Department of Medicine, Mount Sinai School of Medic Fifth Avenue and 100th Street, New York, N.Y. 100[illegible]
Dr. G.K. Sluis-Cremer	South African Medical Research Council, National R Institute for Occupational Diseases, P.O. Box 47[illegible] Johannesburg, S. Africa.
Dr. W. Smither	Cape Asbestos Co. Ltd., 114 Park Street, London W[illegible] England, UK.
Dr. G. Wright	Division of Medicine, St. Luke's Hospital, 11311 S Boulevard, Cleveland, Ohio 44101, USA.

Pathology Panel

Dr. A. C. Allison	Clinical Research Centre, Watford Road, Harrow, Middlesex, HA1 3UJ, UK.
Mr. G. Berry	Medical Research Council, Pneumoconiosis Unit, Lland Hospital, Penarth, Glamorgan, Wales, UK.
Dr. P. Bogovski	Unit of Environmental Carcinogens, International Age for Research on Cancer, 150 Cours Albert Thomas, 690 Lyon, France.
Dr. M. Kannerstein	Pathology Department, Barnert Memorial Hospital Cent 680 Broadway, Paterson, N.J. 07514, USA
Prof. W.T.E.McCaughey	School of Pathology, Trinity College, University of Dublin, Dublin 2, Eire.
Prof. D. Magner	Canadian Tumour Reference Centre, Department of Pathology, University of Ottawa, Ottawa, Ontario, KIN 6N5, Canada.
Prof. H. Otto	Direcktor des Pathologischen Instituts der Stadt Krankenanstalten Beurhausstrase 40, 46 Dortmund, W. Germany.
Dr. H. T. Planteydt	Stichting Streeklaboratorium"Zeeland", Noordpoortple Middelburg, Netherlands.
Dr. M. Stanton	Department of Health, Education and Welfare, Nationa Institute of Health, Bethesda, MD 20014, USA.
Dr. J. C. Wagner	Medical Research Council, Pneumoconiosis Unit, Llan Hospital, Penarth, Glamorgan, Wales, UK. (Chairman
Prof. S. Watanabe	Department of Pathology, National Cancer Centre, Re Institute, Tsukiji, 5-1-1, Chuoku, Tokyo, Japan.
Prof. I. Webster	Medical Research Council South Africa, National Res Institute for Occupational Diseases, P.O. Box 4788, Johannesburg, South Africa.

Physics and Chemistry Panel

Dr. L. Le Bouffant	Laboratoire du Centre D'Etudes et Recherches des Charbonnages de France, Verneuill-en-Hallatre, BP N 60 - Creil, France.
Mr. G. W. Gibbs	McGill University, Department of Epidemiology & Hea 3775 University Street, Montreal 110, Quebec, Canad
Dr. S. Holmes	Asbestosis Research Council, c/o Turner Bros. Asbe Co. Ltd. P.O. Box 40, Rochdale, England, UK.
Mr. A. Morgan	Atomic Energy Research Establishment, Health Physic Medical Division, Building 364, Harwell, Berks., England, UK.

Physics and Chemistry Panel (Continued)

Dr. F. D. Pooley	Department of Mineral Exploitation, University Colle of S. Wales and Monmouthshire, Newport Road, Cardiff. Wales, UK.
Dr. S. Speil	Johns Manville Research and Engineering Centre, P.O. 159, Manville, New Jersey, USA.
Dr. V. Timbrell	Medical Research Council, Pneumoconiosis Unit, Lland Hospital, Penarth, Glamorgan, Wales, UK.
Dr. R. du Toit	Government Mining Engineer's Division, Department of Mines, P.O. Box 1132, Johannesburg, South Africa.
Mr. W. H. Walton	Institute of Occupational Medicine, Roxburgh Place, Edinburgh EH 8 9SU, Scotland, UK. (Chairman)

The panels met in a separate session and at the final session (Chairman Dr. Gilson) prepared this report to the Director of the International Agency for Rese on Cancer.

TERMS OF REFERENCE

1. The Committee was to report on the present evidence relating exposure to asbestos dust to cancers, especially that obtained since the meeting of the UICC Working Group on Asbestos Cancers in 1964.

2. The Committee was to make recommendations for further research and indicat priorities for work of immediate and long-term value.

CO-ORDINATION OF INTERNATIONAL CO-OPERATION

Following the meeting of the UICC Working Group on Asbestos and Cancers in 1964, a Sub-Committee of the UICC Commission on Geographical Pathology and Environmental Carcinogens (Chairman Dr. J. Higginson) was formed to co-ordinate work required to achieve the recommendations.

In April 1970 agreements between the UICC and the IARC led to the winding up of the UICC Sub-Committee and the IARC taking on responsibility for the Sub-Committee's work and extending it by supporting certain projects on asbestos cancers in several countries. The Agency has done this as part of their wider programme of investigating environmental carcinogens. Common memberships betwe the UICC Sub-Committee and the Committee advising the IARC ensured continuity of policy.

In October 1972 the IARC held an international Conference with 137 participants from 20 countries to review all the evidence relating asbestos with cancers. Subsequently the Advisory Committee prepared its report. This is divided into two sections. First, a general review in the form of answers to a number of important general questions about the relation of asbestos to cance of different sites and, secondly, recommendations for further research.

GENERAL REVIEW

1. Are all major commercial types of asbestos able to cause lung carcinoma?

Yes. Since 1964 the evidence of a causal relationship has been increased by epidemiological studies showing exposure-response relations for the incidenc of lung carcinomas. The production of lung carcinomas in certain animals by al types of asbestos supports this conclusion. The epidemiological evidence in ma however, shows that there are clear differences in risk with type of fibre and nature of exposure.

2. Is there evidence of an increased risk of lung carcinoma at low levels of exposure to asbestos, such as have been encountered by the general population urban areas?

The evidence of an exposure-response relationship based in part on past d measurements and in part on the type of job within the industry suggests that excess lung carcinoma risk is not detectable when the occupational exposure ha been low. These low occupational exposures have almost certainly been much greater than that to the public from general air pollution.

3. Since 1964 has the evidence relating past exposure to asbestos and mesotheliomas changed?

The evidence has been greatly strengthened by further prospective and retrospective mortality studies in many countries of populations exposed to a There is evidence that all commercial types of asbestos except anthophyllite responsible. Evidence for an important difference in risk in different occupations and with the type of asbestos has increased. The risk is greatest with crocidolite, less with amosite and apparently less with chrysotile. With amosite and chrysotile there appears to be a higher risk in manufacturing than in mining and milling. There is also evidence from population studies that a proportion of cases of mesothelioma have no known association with exposure to asbestos.

4. Is there evidence of an increased risk of mesothelial cancers at low levels of exposure to asbestos, such as have been encountered by the general population in urban areas?

There is evidence of an association of mesothelial tumours with air pollution in the neighbourhood of crocidolite mines and of factories using mixtures of asbestos fibre types. The evidence relates to conditions many years ago. There is evidence of no excess risk of mesotheliomas from asbestos air pollution which has existed in the neighbourhood of chrysotile and amosite mines. There are reported differences on incidence of mesothelioma between urban and rural areas, the causes of which have not been established. There is no evidence of a risk to the general public at present.

5. Since 1964 has the evidence changed on the importance of other factors such as cigarette smoking, waxes, oils and trace elements as contributory facto to the cancer risks?

The evidence has accumulated indicating:

(1) Cigarette smoking is an important factor enhancing the lung carcinoma risk in asbestos-exposed workers, in both men and women. Asbestos workers have specially strong grounds for giving up smoking to protect their health. No association has been demonstrated between cigarette smoking and mesotheliomas.

(2) Animal experiments designed thus far to test the importance of waxes and oils as contributory factors in the production of

mesothelioma have shown these contaminants are unlikely to be relevant.

(3) From animal experiments there are no good clues suggesting that trace elements are likely to be a major factor in the production of asbestos cancers.

6. What other types of cancer are related to exposure to asbestos?

Prospective surveys of occupational groups exposed to asbestos have in general shown a small excess risk of some other types of cancers (in addition to bronchial and mesothelial), especially those of the gastro-intestinal tract. The excess of these tumours is relatively small compared with that for bronchial cancer. Evidence for an association with ovarian tumours has not been supported by the first large mortality survey of women previously exposed to asbestos.

7. Is there evidence of an increased risk of cancer resulting from asbestos fibres present in water, beverages, food or in the fluids used for the administration of drugs?

Such evidence as there is does not indicate any risk.

8. Is there evidence of a risk of lung fibrosis from low levels of exposure to asbestos such as have been encountered by the general population in urban areas?

There is at present no evidence of lung damage by asbestos to the general public. The amount of asbestos in the lungs of members of the general public is very small, compared to those occupationally exposed. It is greatest where asbestos is mined or worked and lowest in rural areas.

9. Has the relationship between asbestos exposure and the development of pleural plaques been established?

Pleural plaques have been associated with past exposure to all commercial types of asbestos. But additional factors, other than asbestos itself, are involved. The plaques may remain fibrous or become calcified. Not all pleural plaques are associated with asbestos.

Projects which the Panels rated high in priority are marked *; those which will require close co-operation between the Panels are marked ✓.

EPIDEMIOLOGY

The Panel agreed that asbestos related cancers occur in several sites in the body. The incidence of the different cancers varies with a number of definable factors and for other reasons, such as competing causes of death. Epidemiological studies will usually provide information on more than one type of cancer. Research directed at only a single type may, on occasions, be useful but in general the inevitable uncertainties, in some cases in the differential diagnosis of, for example, peripheral lung carcinomas and pleural mesotheliomas, and between peritoneal mesotheliomas and other intra-abdominal cancers will require that more than one type is studied at the same time.

The Panel recognised that some of the epidemiological projects could only be pursued if there was close co-operation between epidemiologists, pathologists, physicists and chemists, and others, because their success will depend upon the development of improved techniques, some of which are referred to in the recommendations of the other two Panels.

PROJECTS

(1) Further development of objective methods for early detection and surveilla[nce] of effects caused by asbestos. Topics for particular study include :

(a) Immunological techniques for screening for fibrosis and neoplasia.

(b) Functional tests of changes in the peripheral airways.

(c) Detection of pleural thickening.

(d) Assessment of the specificity of small irregular opacities in the chest radiograph as defined in the ILO U/C Classification (1971).

(e) Tests of the usefulness of different techniques of chest radiography, including the use of 100 mm films.

(f) Development of statistical procedures for analysis and presentation of serial observations.

(2) Evaluation of the usefulness of early detection in the prevention of progressive fibrosis and asbestos cancers, also in the identification of hazardous conditions. Routine health surveillance of industrial populations should be designed to assist epidemiological studies and should include measurement and recording of environmental dust levels. Surveillance of new entrants could be particularly valuable. Arrangements should be made to register workers so that their morbidity and mortality experience can be studied even after cessation of exposure to asbestos.

(3) Assessment of excess cancer risks following exposure to only one type of fibre.

(a) Chrysotile: The much higher cancer risk reported for chrysotile textile workers compared with mine and mill workers requires explanation. How much is explicable by differences in size of airborne fibres and past dustiness? There is need to make more use of past dust records for relating to indices of disease.

(b) Amosite: The excess lung carcinoma and mesothelioma risk is apparently much greater in the manufacturing and application sections of the industry than in the mining and milling of this type of fibre. What are the important factors in this reported difference?

(c) Crocidolite: Further studies are required in occupational groups exposed only to crocidolite or amosite or chrysotile in the manufacturing and application parts of the industry to establish more clearly differences in risks due to different fibres.

* / (4) Studies of the amount and type of asbestos in the lungs of cases of mesotheliomas (if possible by cell type) in (a) national survey of mesotheliomas, (b) representative samples of cases arising in groups with a definable past exposure.

* (5) Studies of secular changes in incidence of pleural and peritoneal mesotheliomas nationally and internationally.

* (6) Epidemiological studies to investigate the association between past exposure to asbestos and cancer of sites other than lung, pleura, and peritoneum.

(7) Studies of secular trends in the asbestos content of the lungs in the general population.

* / (8) Studies to relate amount and type of asbestos in the lung and estimates of past dust exposure and interval since last exposure.

(9) Experimental and epidemiological studies to investigate possible differences of effect of continuous low and intermittent high exposure to asbestos.

(10) Opportunities afforded by intercurrent deaths should be used to interrelate radiographic appearances, lung pathology, respiratory function, dust content, and type in asbestos workers. Standardised techniques and classification recommended by the Panels should be used.

(11) Investigate the prognostic significance and aetiological factors in the development of calcified and uncalcified pleural plaques in different environments.

(12) Investigate talc-exposed groups in mining and manufacturing to establish any differences in morbidity or mortality which might be related to the amount and shape of the fine respirable particles.

(13) Development of cost/benefit analyses to study the health, safety, social and economic interrelations of the use of asbestos.

PATHOLOGY AND EXPERIMENTAL PATHOLOGY

The Panel reviewed the progress made on the 1964 UICC recommendations. It was agreed that considerable progress had been made on the majority of the recommendations. Some require further study, or modification of previous methods of investigation; these are included in the list of recommendations that follows. The recommendations are divided into three categories, morbid anatomy and histology, clinical research, and experimental studies.

PROJECTS

Morbid Anatomy and Histology

1. Asbestosis

(1) Further consideration should be given to methods for determining the amounts, types and structural features of asbestos in tissue. A Sub-Committee should be established with members of the Physics and Chemistry Panel, and others to accelerate work on this problem.

(2) The methods for assessing the severity of asbestosis (see paper 9) should be tested for consistency by different observers.

2. Carcinoma

(1) An investigation of whether reduction of asbestos exposure to levels below those producing asbestosis also abolishes excess risk of carcinoma was considered important.

(2) A comparison of lung carcinomas in persons occupationally exposed to asbestos and those not so exposed, including both cigarette smokers and non-smokers, in respect of sites of origin and cytology of tumours and presence or absence of asbestosis, would be of value.

3. Mesotheliomas

(1) The International Panel of Pathologists[1] and National Panels established following the 1964 meeting[2] have served a useful purpose. It is recommended that panels be established in other countries and membership of the International Panel be extended. The main purposes of these Panels is to ensure uniformity of diagnostic criteria and recording of histological types of diffuse mesotheliomas. Collaborative study of histology slides in National Panels is recommended. The diagnosis of mesothelioma can be made by exfoliative cytology of the pleural fluid. If the cytological diagnosis is made by a competent cytologist, biopsy may be unnecessary.

(2) To improve consistency of diagnosis there is an urgent need for a Comprehensive Atlas on mesotheliomas, or alternatively, for inclusion of an enlarged section on mesotheliomas in the new edition of the WHO Monograph on Tumours of the Lung. Criteria for diagnosis by exfoliative cytology and a description of the fine structure of mesotheliomas should be included.

Clinical Research

(1) Monitoring by immunological methods of populations exposed to asbestos should be investigated to ascertain whether it is possible to recognize those who are developing, or will develop, tumours.

(2) The use of chromatographic methods for the study of mucopolysaccha[rides] and other tumour-associated substances in pleural fluids should be explored. Sensitive methods might be developed and applied to identify secretory products of mesotheliomas in blood and urine.

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[1] The International Panel consists of: Dr. M. Kannerstein (USA), Prof. D. Magner (Canada), Dr. L. Meurman (Finland), Prof. W.T.E. McCaughey (Eire), Prof. H. Otto (FRG), Dr. H. T. Planteydt (Netherlands), Dr. E. Roitzsch, (GDR), Prof. L. Santi (Italy), Prof. I. Webster (South Africa), and Dr. J.C.Wagner (UK) as Secretary.

[2] Great Britain, South Africa, United States, Canada, Netherlands.

Further Experimental Studies

(1) Information is required about the role of fine particles, especially influence of fibre size, in the induction of tumours. These studies should be extended to include fibres other than asbestos. A Sub-Committee should be established to review the need for, and arrange the distribution of standard samples of asbestos and other fibres in addition to the UICC Reference Samples.

(2) The fate of inhaled particles of various sizes, shapes and chemical compositions, should be studied to determine more precisely the quantities and sites of initial deposition, change within the body and later retention. The feasibility of increasing fibre elimination by various methods should be explored. Studies should be made of means of reducing the fibrogenicity and carcinogenicity of fibres already retained in the lungs.

(3) The use of cell and organ culture, including mesothelial tissue from man and other species, should be further investigated with a view to developing methods of screening dusts for fibrogenic and carcinogenic properties.

(4) Further studies should be carried out to determine the nature of the combined effect on tumour induction when animals are exposed to asbestos dust and cigarette smoke, metals or other chemical carcinogens, including those which act systemically such as nitrosamines.

(5) Inhalation experiments should be extended to test various types of fibre; of special interest are forms of chrysotile and crocidolite including the finer grade materials.

(6) It was felt that studies of the pathological effects of asbestos on species other than rodents would be of value.

(7) The effect of long-term ingestion of fibres of various sizes, shapes and chemical compositions should be studied.

(8) The effects of fibres and associated metals on the metabolism of target organs should be investigated.

PHYSICS AND CHEMISTRY

The Panel reviewed the progress made on the 1964 UICC Recommendations. The proposals for the preparation and characterization of the UICC reference samples of asbestos had been satisfactorily implemented, and the Panel recommended that a list of references to papers featuring the samples should be distributed to investigators in this field. Considerable progress had been made on methods of identifying the type of fibre in tissues but a quantitative method when several types of fibres were present had yet to be developed.

The Panel discussed the further contribution that physical and chemical studies can make to research on the biological effects of asbestos and other fibrous materials. Of especial interest are the effects of fibre size and shape on the retention of material in the lungs, the site of deposition, the migration of fibres within the body, and their carcinogenic or other biological activity. The following recommendations were made:

PROJECTS

1. Materials for experimental work

a) Supplies of asbestos from relevant sources should be obtained where there is evidence of variation in geological form, trace element content or significant biological findings.

b) Small samples of various fibrous materials should be prepared for studies on the influence of fibre size and shape on carcinogenicity. For this purpose the samples should be milled to different degrees of fineness.

c) For investigations on the influence of particle shape and size, on the inhalation and subsequent fate of asbestos fibres a chrysotile and an amphibole of fibre length greater than the UICC samples should be prepared.

2. Methods

a) There is an urgent need for the quantitative assessment, size analysis and characterization of particles and fibres in the lungs and other organs. Details of available methods should be circulated, international comparisons undertaken, methods standardized and new techniques developed.

b) No methods are at present available for the preparation of fibres in narrow ranges of diameter and length in sufficient quantities for inoculation experiments. Techniques for these purposes are urgently required, especially in view of the advantages such graded samples could provide for investigating the influence of these physical factors on the carcinogenicity of fibres of different materials.

c) Since the degree of dispersion of fibres (especially chrysotile) used in inoculation studies may have a marked influence on their carcinogenicity, methods are required for quantifying dispersion.

d) Inhalation studies require precise control of the characteristics of the dust clouds. Improved methods of dispensing fibrous dusts in such investigations need to be developed.

e) Methods are available for collecting the important size fractions of dust clouds in inhalation studies when the particles are of compact shape. Similar methods must be developed for fibrous particles.

f) The present membrane filter methods of measuring the levels of airborne asbestos dust require standardization. This should be done by inter-laboratory trials on a continuing basis. Particle counting by electron microscopy should also be developed. Gravimetric assessment methods and the automation of particle counting should be explored.

3. Inhalation studies

Considerable information is now available on the deposition, retention and migration of particles of compact shape. Recently developed methods, especially radio-active tracer techniques, should be used to obtain similar knowledge for fibrous particles. This information is needed to identify the biologically important size fraction and to help interpretation of epidemiological and pathological studies.

4. Occupational and environmental studies

The use of both fibre counts and gravimetric methods for assessing asbestos dust concentrations should be encouraged. Data collected over an extended period will be particularly valuable in identifying the parameters of the dust which can be correlated with epidemiological evidence on the health hazard.

5. Physics and Chemistry Panel

It is recommended that an international Panel be established to assist in implementing these recommendations. The Panel would periodically review requirements for materials for experimental work; provide guidance on physical and chemical problems; and arrange national and international standardization trials.

Journal of Occupational Medicine
JOM
December 1972 Vol. 14 No. 12

Mortality in Relation to Occupational Exposure in the Asbestos Industry

Philip Enterline, Ph.D., Pierre DeCoufle, Sc.D., and Vivian Henderson, M.Sc.

Mortality estimates for large occupational or industrial groups can be made by relating counts in population census returns to counts based on death certificates. For a given occupation, this would consist of a count of persons in that occupation as enumerated in a national population census combined with counts of persons with the same occupation as it appears on death certificates. From time to time government publications are issued based on combining these two sources of information, and mortality rates are shown for a wide variety of occupational and industrial groupings. For example, mortality rates by occupation were published for the year 1951 for England in Wales in the Registrar General's Decennial Supplement on Occupational Mortality and for the United States for the year 1950 in a series of publications issued by the US Department of Health, Education, and Welfare.[1,2] In addition to these government publications, life insurance companies publish data on mortality for various occupation and industry groups based on their experience with individual and group policyholders.[3]

While such data point to occupations and industries whose workers have an unusual mortality experience, they are of limited use in assessing the health effects of particular occupational exposures since they do not usually permit subdivisions by type of exposure or exposure levels. Moreover, occupational mortality estimates which combine census returns with death certificates are often questionable due to differences in the way occupation is defined in preparing these two documents, while data published by life insurance companies only relates to mortality experience while life insurance policies are in force. A recent review of data from these two sources for a particular occupation provides some idea as to their value and limitations.[4]

Probably a more useful approach to studying mortality for a particular occupation is to identify groups of persons in that occupation and follow them for a long period of time, observing their mortality regardless of whether they continue in the occupation of interest or not. This has the advantage of using a single definition of an occupation and of permitting observations on deaths which occur after employment in a particular occupation has ceased but which may be related to some environmental factor in that occupation. A large number of such studies have been reported on in recent years. These include long follow-up studies of uranium miners, steelworkers, asbestos miners, asbestos insulators, asbestos products workers, gasworkers, and rubber workers.[5-11]

To be most useful this type of study should include estimates of exposures and should allow a sufficiently long follow-up period for any effects of exposure on mortality to be observed. Such studies are usually time consuming and expensive since they require tracing large numbers of individuals as they move through a variety of jobs and geographic locations and establishing the fact of death and causes of death. Nevertheless, for many health conditions where death is a likely outcome, they are a valuable means for evaluating the importance of a particular exposure and can assist in the setting of environmental standards.

In an earlier paper, we reported on the mortality experience at ages 65 and over of a group of men who completed their working lifetime as production or maintenance-service employees in the asbestos industry.[12] These were men who retired with an industry pension and who were nearly all exposed to asbestos dust in varying degrees in the course of their employment. The average duration of employment in the asbestos industry for these men was 25 years with a range from 3 to 51 years. Historic dust levels, measured with a midget impinger, were sometimes in excess of 50 million particles per cubic foot of air.

For each of these retirees it was possible, by combining personnel records with asbestos dust level estimates for each job and time period, to reconstruct their asbestos dust exposure history and, for each man, to assign a measure of his cumulative asbestos dust exposure at the time of retirement. It was found that this time-weighted measure of asbestos dust exposure was directly related to respiratory cancer, with the excess ranging from 1.7 times expected for men in the lowest exposure group to 5.6 expected for men in the highest. Separation of the effects of duration of exposure from the effects of average dust level on respiratory cancer mortality showed that the contributions of each was about the same and that a time-weighted measure of asbestos dust appeared to be an appropriate method for predicting respiratory cancer effects.

This is a report on additional data arising from the same study. The cohort upon which this study was based con-

From the Department of Biostatistics, Graduate School of Public Health, University of Pittsburgh. Dr. DeCoufle is now with the National Institute of Occupational Safety and Health.

Reprint requests to [illegible] School of Public Health, University of Pittsburgh, Pittsburgh, Pa 15213 (Dr. Enterline).

Presented at the American Industrial Hygiene Conference, San Francisco, May [illegible] 1972.

Table 1. — Vital Status as of December 31, 1969 — Males Retiring from the Asbestos Industry, 1941-1967.	
Vital Status	Numbers
All Retirees	1,464
Alive	642
Dead	822
Death certificates found	802
Death certificates not found	20

sisted of all men retiring during the years 1941 through 1967 and deaths occurring among these men through 1969. Such a study could be conducted almost entirely using industry records, except that where deaths occurred official copies of death certificates needed to be obtained. Included in this study were three kinds of retirees: those who retired normally at age 65, those who retired before age 65 for personal reasons but lived to age 65, and men who retired prior to age 65 because of a disability but who lived to be 65. The observed mortality among these men was compared with an expected mortality based upon the experience of the entire US white-male population living the time-age intervals that characterized the retired population. Inclusion of disabled retirees improves comparability with the US white-male population since the latter also includes men unable to work for health reasons. The modified life-table method was used for computing the expected number of deaths. This method is widely used and was described in an earlier report.[9]

There were a total of 1,492 men in this study who reached age 65. Of these, complete exposure and job histories were available for 1,464. Table 1 shows the vital status as of December 31, 1969, of the 1,464 men for whom complete exposure and job histories were available and the success we had in locating death certificates. As might be expected for an older age group, the mortality rate was high, with deaths occurring in well over half of the cohort. For only 20 out of the total 822 deaths could a death certificate not be located.

Table 2 shows observed and expected deaths and standardized mortality ratios (SMR's) for selected causes of death for the 1,464 men for whom complete job and exposure data could be obtained. The expected value of the SMR, where observed and expected deaths is the same, is 100 and deviations from 100 are percentage deviations from expected mortality. As noted above, expected deaths were based upon the mortality experience of the entire US white-male population living at the same ages and time periods as the cohort under study.

It is not known how much SMR's for individual causes of death might be changed had all death certificates been found. It is unlikely, however, that there would be much of an increase for any single cause and no attempt has been made to allocate the 20 unlocated death certificates to a cause of death groupings. All death certificates were coded according to the Seventh Revision of the International Classification of Diseases by a regular coder from the National Center for Health Statistics. In this type of study the coding procedure is important if comparability with US mortality data is to be maintained. Numbers in parentheses in Table 2 correspond to the International Classification of Disease rubrics.

Table 2 shows that these men who retired from the asbestos industry and reached age 65 had an overall mortality rate 14.5% higher than all US males living at the same ages and time periods. This excess is due almost entirely to cancer and diseases of the respiratory system. For cancer, the greatest excess was in cancer of the respiratory system, but with some excess also in cancer of the digestive system. For respiratory disease, the excess was due entirely to pneumonoconiosis and pulmonary fibrosis of which 18 deaths were due to asbestosis (ISC 523.2). Since the expected number of deaths in these latter two categories is extremely small, SMR's were not calculated.

Of the 1,464 retirees whose mortality experience is shown in Table 2, a total of 1,026 had worked as production workers while 438 had been classified as maintenance-service workers. The latter consisted largely of trade-related jobs such as machinist, painter, electrician, pipe fitter, millwright, etc. These maintenance-service jobs constitute a distinct grouping, and there was relatively little movement between these trade-related jobs and production jobs during the course of employment.

Table 3 shows the standardized mortality ratios for selected causes of death for these two groups of retirees. Actual numbers used in calculating these ratios appear in the Appendix. It is clear from Table 3 that the mortality experience of these two groups of men subsequent to retirement was quite different. While the same general pattern of excess appears in both groups, with high death rates for respiratory cancer and diseases of the respiratory system, cancer mortality rates are greatly exaggerated for the men who had worked in maintenance-service jobs. For all causes the excess mortality for production workers was only 10.7%, whereas for maintenance-service workers the excess was 24.3%. For respiratory cancer the excess for production workers was 73.7% while for maintenance-service workers it was 328.6%. Considerably higher rates also appear among maintenance-service workers for cancer of the digestive system and for residual cancers. For other causes rates for maintenance-

Table 2. — Observed, Expected Deaths, and SMR's for Selected Causes of Death, Males Retiring from the Asbestos Industry, 1941-1967 and Followed through 1969			
Cause of Death	Observed	Expected	SMR
All Causes	822	717.3	114.5
Cancer (140-205)	176	119.4	147.4
Digestive (150-159)	59	45.5	[illegible]
Respiratory (162-163)	59	[illegible]	[illegible]
All other cancers	58	[illegible]	116.5
Stroke (330-334)	68	[illegible]	[illegible]
Heart Disease (400-443)	353	[illegible]	[illegible]
Respiratory Disease (470-527)	74	49.2	[illegible]
Pneumoconiosis and Pulmonary Fibrosis (523-525)	33		
Asbestosis (523.2)	18		..
All Other Causes	121	[illegible]	[illegible]
Death certificates not located	20		....

Table 3. — Standardized Mortality Ratios for Selected Causes of Death, Males Retiring from the Asbestos Industry, 1941-1967 and Followed through 1969 By Type of Work

Cause of Death	Standardized Mortality Ratios	
	Production	Maintenance Service
All Causes	110.7	124.3
Cancer (140-205)	123.7	205.2
Digestive (150-159)	113.8	165.4
Respiratory (160-163)	173.7	4[illegible]3.6
All other cancers	109.2	122.9*
Strokes (330-334)	78.6	91.7
Heart Disease (400-443)	104.9	115.1
Coronary heart disease (420)	97.1	120.7
All other heart diseases	133.5	87.4
Resp. Disease (470-527)	[illegible]9.8	120.7
Excluding Pn PF	104.9	94.8
All Other Causes	96.4	79.5

service workers are not remarkable, while for the respiratory disease and other heart diseases (including cor pulmonale) rates were somewhat lower than for production workers. Regarding respiratory diseases, there were a total of 18 deaths coded as due to asbestosis, as noted in Table 2. Seventeen of these were in the group of 1,026 production workers and only 1 in the 438 maintenance-service workers. If these and other deaths from pneumonoconiosis are excluded respiratory disease death rates are not much different in the two groups.

Production and maintenance-service workers appeared to be similar in most other respects. Retirements occurred at about the same time so that differences in mortality for respiratory cancer and asbestosis do not reflect changes in diagnostic fashions associated with mortality in various periods. Of the production workers, 6.4% had retired due to a disability whereas only 4.3% of the maintenance-service workers had retired for this reason. Thus, the higher death rate for the maintenance-service group was apparently not due to greater disability at time of retirement. The mean age at death for men dying of respiratory cancer was about the same in the two groups—72.8 vs 71.3 for production and maintenance-service workers respectively.

Smoking histories were available for only 373 of the total 1,464 retirees representing 269 production workers and 104 maintenance-service workers. These were available in only the most recent retirements but probably give a fairly good picture of the smoking habits of the whole group. There was essentially no difference in the smoking habits of the production as compared with the maintenance-service workers. Of the 269 production workers, 77.3% were smokers, while of the 104 maintenance-service workers, 76.9% were smokers.

Table 4 shows the cumulative asbestos dust exposures at time of retirement for these two groups of men. These were estimated based on miget impinger counts expressed as million particles per cubic foot of air (mppcf). It was not always possible to very precisely estimate the dust level for each job, particularly for past periods, and often dust levels could only be roughly classified. Jobs were placed in one of six classes or ranges and for calculating total exposure mid-points of the range were used. Classes used were: no exposure (0), under 5 mppcf (2.5), 5 to 10 mppcf (7.5), 10 to 30 mppcf (20.0), 30 to 50 mppcf (40.0), 50 mppcf or more (62.5).

To compute cumulative dust exposure for each man the dust level at each job and time period was multiplied by years at that job and summed across all jobs. This total cumulative exposure can be thought of as mppcf-years. Thus, if a man with a 30-year work history initially worked ten years at a job with a dust level at that time of 20.0 mppcf, ten years at a dust level of 2.5 mppic, and ten years at a dust level of 7.5 mppcf, his cumulative exposure to asbestos dust would be: (10 x 20) + (10 x 2.5) + (10 x 7.5) or 300 mppcf years. His average exposure level would be 10 mppcf (300 ÷ 30).

As Table 4 shows, the distribution of the two groups of retirees by cumulative dust exposure was quite similar. Actual means, shown in Table 4, were slightly higher for maintenance-service as compared with production workers.

Table 5 shows SMR's for respiratory cancer by cumulative dust exposure at time of retirement for all retirees, for men who had worked as production workers, and for men who had worked as maintenance-service workers. Numbers used in compiling this table appear in the Appendix. For all retirees there is an almost linear relationship between asbestos dust exposure and respiratory cancer, with ratios of observed to expected deaths ranging from 1.7 in the lowest dust exposure category to 5.6 in the highest. This relationship also exists for both the production and the maintenance-service workers, except that at each level the SMR is higher for maintenance-service workers and seems to increase more sharply with cumulative asbestos dust exposure. No respiratory cancer deaths were observed among the 80 production workers in the 500 to 749 exposure category vs 1.1 expected (Appendix).

At the higher dust levels the death rate for respiratory cancer among maintenance-service workers is remarkable. Of the 57 maintenance-service workers with a cumulative exposure of 500 mppcf-years and over (Table 4), 37 died of

Table 4. — Number of Retirees by Type of Work and Estimated Cumulative Asbestos Dust Exposure — Males Retiring from the Asbestos Industry During 1941-67 and Followed through 1969

Cumulative Exposure to Asbestos Dust [illegible]	Numbers	
	Production Workers	Maintenance-Service Workers
[illegible]	[illegible]	15[illegible]
[illegible]	[illegible]	73
[illegible]	[illegible]	111
[illegible]	[illegible]	45
[illegible]	[illegible]	12
Actual mean mppcf-years	23[illegible].7	230.6

Table 5. — Standardized Mortality Ratios for Respiratory Cancer By Type of Work and Estimated Cumulative Asbestos Dust Exposure: Males Retiring from the Asbestos Industry During 1941-1967 and Followed through 1969

Cumulative Exposure to Asbestos Dust (mppcf-years)	SMR's		
	All Retirees	Production Workers	Maintenance-Service Workers
Under 125	169.2	145.7	213.8
125-249	224.5	184.2	303.6
250-499	290.3	222.2	444.4
500-749	500.0	000.0	1265.7
750 and over	555.6	428.6	1090.0

Table 6. — Kind of Exposure by Type of Work— Males Retiring from the Asbestos Industry During 1941-67 and Followed through 1969 Who had Worked at Jobs Greater Than 10 mppcf

Kind of Exposure	Production Workers	Maintenance-Service Workers
Total	410	207
Steady	327	9
Intermittent	83	198

Table 7. — Type of Asbestos by Type of Work— Males Retiring from the Asbestos Industry During 1941-67 and Followed through 1969 Who Had Worked at Jobs Greater Than 10 mppcf

Type of Asbestos	Production Workers	Maintenance-Service Workers
Total	522	223
Chrysotile only	355	69
Amosite only	44	5
Mixed exposure*	93	154
Exposed to some crocidolite	42	153

* Includes crocidolite

all causes of which 11 or 30% were due to respiratory cancer. Among maintenance-service workers, only respiratory cancer is highly related to cumulative asbestos dust exposure (Appendix). Numbers involved are small, however, so that information is not very conclusive for most disease categories.

One possible explanation for the higher respiratory cancer rate in maintenance-service workers is the way in which their cumulative dust exposure was received. In compiling data for this study, when the dust level of any job was found or estimated to be greater than 10 mppcf, a judgment was made as to whether that exposure was steady or intermittent. There were a total of 761 workers in such jobs, and a decision as to whether the exposure was intermittent or steady was possible for 617. Table 6 shows how this classification related to whether the worker was a production or a maintenance-service worker. From this, it is clear that a characteristic of the exposure of the maintenance-service workers was intermittency. Exposure data for these men was difficult to estimate and was intended to relate to average exposures over the course of a working day. Very high levels of brief asbestos dust exposure could be hidden in these data.

For the jobs where dust levels exceeded 10 mppcf, there was also recorded the type of asbestos involved. These data are shown in Table 7 for the 761 men working in such jobs. Clearly there were differences in the type of asbestos to which production workers were exposed as compared to maintenance-service workers. Only 26% of the production workers were exposed to amosite and/or crocidolite asbestos, whereas 71% of the maintenance-service workers were exposed to amosite and/or crocidolite. For crocidolite, the contrast is more striking, with only 8% of production workers exposed as compared with 64% of maintenance-service workers.

Discussion

This analysis of the mortality experience of retired workers from the asbestos industry supports findings in other studies in terms of specific health hazards associated with asbestos dust exposure. What has not been previously shown, however, is that men in maintenance-service jobs are apparently at greater risk of developing respiratory cancer than men in production jobs. Curiously, maintenance-service workers did not seem to develop asbestosis or diseases related to asbestosis as readily as production workers.

The intermittency and possible severity of their exposures may somehow account for the excess in respiratory cancer. Newhouse found that severe exposures to asbestos dust, whether short or long, produces significant excesses in cancer of the lung, whereas only long exposure produced excess deaths from respiratory disease.[11] Severe intermittent exposures might also account for the high respiratory cancer mortality found in insulation workers.[8]

It is also possible that the type of asbestos dust is involved in the differences between the mortality experiences of production as compared with maintenance-service workers. It is generally believed that the carcinogenic properties of crocidolite are higher than chrysotile, particularly as related to malignant mesotheliomas. This could account for the great excess of respiratory cancer among maintenance-service workers at every dust exposure level.

One limitation on data presented here is that only mortality at ages 65 and over is dealt with. What might have happened prior to age 65 is unknown, and it is possible that many workers left employment for health reasons related to dust exposure and were not available for study. We have been able to check on possible biases introduced with regard to respiratory cancer by examining special tabulations of data on coke oven workers in the steel industry, comparing SMR's for men 65 and over with those for men under 65. For a cohort of steelworkers from Allegheny County

Appendix Table A — Observed, Expected Deaths, and Standardized Mortality Ratios (SMR's) Through December 31, 1969 by Cause and Cumulative Asbestos Dust Exposure, 1464 Males Who Worked in Asbestos Production and Maintenance-Service and Retired During 1941-1967

Cause of Death and International List Number	< 125 Obs.	< 125 Exp.	< 125 SMR	125-249 Obs.	125-249 Exp.	125-249 SMR	250-499 Obs.	250-499 Exp.	250-499 SMR	500-749 Obs.	500-749 Exp.	500-749 SMR	750+ Obs.	750+ Exp.	750+ SMR	All Exposures Obs.	All Exposures Exp.	All Exposures SMR
	Cumulative asbestos dust exposure in million particles per cubic foot years (mppcf-years)																	
All Causes	265	241.8	105.8	162	139.8	115.9	184	156.7	117.4*	77	50.2	153.4*	34	26.2	129.8	722	[illegible]	[illegible]
Deaths Certificate Reported																		
All Cancer (140-205)	74	56.3	131.4*	23	23.6	97.5	57	28.9	197.1*	18	8.7	206.9*	9	4.5	200.0	176	119.5	147.3*
Digestive System (150-159)	27	21.9	123.3	5	8.9	56.2	19	10.0	190.0*	6	3.3	181.8	2	1.7	117.5	59	45.8	128.9
Lung, Bronchus, Trachea & Pleura (162-163)	[illegible]	10.7	[illegible]	11	4.9	224.5*	16	5.4	296.3*	9	1.8	500.0*	5	0.9	555.6	55	23.7	[illegible]
All Other Cancer	29	23.7	122.4	7	9.8	71.4	17	[illegible]	[illegible]	3	3.6	83.3	2	1.9	105.3	58	50.0	115.9
Cerebral Vascular Lesions (330-334)	31	41.4	74.3	14	16.2	86.4	15	18.2	82.4	5	5.6	89.3	3	3.0	100.0	68	84.7	80.3
All Heart Disease (410-443)	165	161.2	104.2	72	69.5	103.9	75	73.3	102.3	35	23.5	153.2*	11	12.3	89.4	352	335.8	107.8
Coronary Heart Disease (420)	129	121.8	102.4	59	52.2	113.0	55	57.7	95.2	24	13.9	127.6	8	9.9	80.8	275	253.5	104.4
All Other Heart Diseases	33	30.4	107.1	13	13.3	97.7	20	15.5	129.0	12	4.7	255.3*	2	2.4	[illegible]	87	72.3	120.3
Diseases of the Respiratory System (470-527)	25	19.0	162.5*	11	8.0	137.5	17	8.9	191.0*	11	2.8	392.8*	9	1.5	600.0*	74	40.2	184.1*
Pneumoconiosis & Pulmonary Fibrosis (523-525)	8	....	....	3	....	....	8	....	....	8	....	....	5	....	....	32	....	....
Asbestosis (523.2)	2	....	....	2	....	....	4	....	....	5	....	....	5	....	....	18	....	....
All Other Causes	59	66.6	90.1	33	26.5	124.5	24	29.9	80.3	7	9.6	72.9	2	4.9	40.8	125	137.5	91.6
Death Certificate not Located	6	....	....	3	....	....	1	....	....	0	....	....	0	....	....	15	....	....

* SMR significantly different from 100 at 5% level.

Appendix Table B — Observed, Expected Deaths, and Standardized Mortality Ratios (SMR's) Through December 31, 1969 by Cause and Cumulative Asbestos Dust Exposure, 1026 Males Who Worked in Asbestos Production and Retired During 1941-1967

Cause of Death and International List Number	< 125 Obs.	< 125 Exp.	< 125 SMR	125-249 Obs.	125-249 Exp.	125-249 SMR	250-499 Obs.	250-499 Exp.	250-499 SMR	500-749 Obs.	500-749 Exp.	500-749 SMR	750+ Obs.	750+ Exp.	750+ SMR	All Exposures Obs.	All Exposures Exp.	All Exposures SMR
	Cumulative asbestos dust exposure in million particles per cubic foot years (mppcf-years)																	
All Causes	256	249.0	102.8	125	107.2	116.6	113	105.5	107.1	46	31.2	147.4*	28	20.4	137.3	568	513.3	110.7*
Death Certificate Located																		
All Cancer (140-205)	51	40.3	126.5	15	18.1	82.9	27	17.7	152.5*	5	5.3	113.2	6	3.5	171.4	105	84.9	123.7*
Digestive System (150-159)	20	15.7	127.4	4	6.8	58.8	9	6.7	134.3	3	2.0	150.0	1	1.3	76.9	37	32.5	113.8
Lung, Bronchus, Trachea & Pleura (162-163)	11	7.5	146.7	7	3.3	212.2	8	3.6	222.2	0	1.1	0.0	3	0.7	428.6	29	16.7	173.7*
All Other Cancer	20	17.1	117.0	4	7.5	53.3	10	7.4	135.1	3	2.2	136.4	2	1.5	133.3	39	35.7	109.2
Cerebral Vascular Lesions (330-334)	21	30.4	69.1	11	12.4	88.7	10	12.4	80.6	3	3.5	85.7	3	2.4	125.0	48	61.1	78.6
All Heart Disease (410-443)	117	116.4	100.5	56	50.7	110.6	49	49.4	99.2	22	14.6	150.7	8	9.6	83.3	252	240.2	104.9
Coronary Heart Disease (420)	87	90.3	96.3	45	39.9	112.8	33	39.0	84.6	12	11.6	103.4	6	7.7	77.9	183	188.5	97.1
All Other Heart Diseases	30	26.1	114.9	11	10.3	106.8	16	10.4	153.8	10	3.0	333.3*	2	1.9	105.3	69	51.7	133.5
Diseases of the Respiratory System (470-527)	22	13.6	161.8*	9	6.1	147.5	10	6.0	166.7	10	1.7	588.2*	9	1.2	750.0*	60	28.5	209.8*
Pneumoconiosis & Pulmonary Fibrosis (523-525)	7	....	....	3	....	....	7	....	....	7	....	....	6	....	....	30	....	....
Asbestosis (523.2)	2	....	....	2	....	....	4	....	....	4	....	....	5	....	....	17	....	....
All Other Causes	44	48.3	91.1	28	137.3	155.7	16	20.0	80.0	5	6.1	82.0	2	3.7	54.1	95	98.5	96.4
Death Certificate not Located	1	....	....	6	....	....	1	....	....	0	....	....	0	....	....	8	....	....

* SMR significantly different from 100 at 5% level.

Appendix Table C — Observed, Expected Deaths, and Standardized Mortality Ratios (SMR's) Through December 31, 1969 by Cause and Cumulative Asbestos Dust Exposure. [illegible] Males Who Worked in [illegible] Service and Retired During 1941-1967

Cumulative asbestos dust exposure in million particles per cubic foot years (mppcf-years)

Cause of Death and International List Number	125			125-249			250-499			500-749			750+			All Exposures		
	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR	Obs.	Exp.	SMR
All Causes	165	95.2	[illegible]	37	32.6	113.5	71	[illegible]	133.7*	31	19.0	163.2*	6	5.8	103.4	[illegible]	[illegible]	[illegible]
Death Certificates Located																		
All Cancer (140-205)	23	16.0	143.8	8	5.5	145.5	25	8.7	287.4*	12	3.4	352.9*	3	1.6	300.0	71	[illegible]	[illegible]
Digestive System (150-159)	7	6.2	112.9	1	2.1	47.6	10	3.3	303.0*	3	1.3	230.8	1	0.4	[illegible]	22	[illegible]	[illegible]*
Lung, Bronchus, Trachea & Pleura (162-163)	7	3.2	218.6	4	1.1	363.6	8	1.8	444.4*	9	0.7	1285.7*	2	0.2	1000.0*	[illegible]	[illegible]	[illegible]*
All Other Cancer	9	6.6	136.4	3	2.3	130.4	7	3.6	194.4	0	1.4	0.0	0	0.4	0.0	[illegible]	14.3	132.9
Central Nervous System Vascular Lesions (330-334)	10	11.3	88.5	3	3.8	78.9	5	5.9	56.2	2	2.1	95.2	0	0.6	0.0	[illegible]	[illegible]	[illegible]
All Heart Disease (400-443)	51	44.8	113.8	16	15.3	104.6	26	23.9	108.8	14	8.9	157.3	3	2.7	111.1	[illegible]	[illegible]	[illegible]
Coronary Heart Disease (420)	42	34.5	121.7	14	12.3	113.8	22	18.8	117.0	12	7.2	166.7	2	2.2	90.9	[illegible]	[illegible]	[illegible]
All Other Heart Diseases	9	10.3	87.4	2	3.0	66.7	4	5.1	78.4	2	1.7	117.6	1	0.5	200.0	18	[illegible]	[illegible]
Diseases of the Respiratory System (470-527)	4	5.4	74.1	2	1.9	105.3	7	2.9	241.4	1	1.1	90.9	0	0.3	0.0	14	[illegible]	[illegible]
Pneumoconiosis & Pulmonary Fibrosis (523-525)	1	....	....	0	....	....	1	....	....	1	....	....	0	....	....	3		
Asbestosis (523.2)	0	....	....	0	....	....	0	....	....	1	....	....	0	....	....	[illegible]		
All Other Causes	16	18.3	87.4	5	[illegible]	92.0	8	9.9	80.8	2	3.5	57.1	0	1.2	0.0	[illegible]	[illegible]	[illegible]
Death Certificates not Located	5	....	....	3	....	....	0	....	....	0	....	....	0	....	....	[illegible]		

* SMR significantly different from 100 at 5% level.

(Pittsburgh) identified in 1953 and followed through 1966, C. K. Redmond found that among coke oven workers the SMR for respiratory cancer at ages under 65 was 325.3 and at ages 65 and over 375.9 (personal communication, May 9, 1972). Thus, the study reported here of retired employees from the asbestos industry may provide a fair estimate of risks associated with respiratory cancer at younger ages.

Unfortunately, we have not as yet been able to examine in detail one of the important causes of death related to asbestos exposure—malignant mesothelioma. One reason for this is that the International Classification of Diseases makes no provision for coding malignant mesothelioma and, moreover, it may not appear on the death certificates, even if present. To study this condition requires an independent effort—contacting physicians, hospitals, and pathologists where mesothelioma is suspected and supplementing data recorded on death certificates. On only one of the 802 death certificates included in this study was a malignant mesothelioma recorded. This was a maintenance-service worker who died at the age of 69 with a pleural mesothelioma. His cumulative dust exposure was relatively low (60 mppcf- years) and his first exposure occurred 29 years prior to death. There were a number of men not included in this study who died prior to age 65 of a malignant mesothelioma, and it is our impression that mesotheliomas in this population occurred at a considerably earlier age than respiratory cancer. We hope to report more on this in a subsequent paper, particularly as it relates to the type of asbestos to which workers were exposed.

Summary

There are a number of publications which deal with mortality rates by occupation and which suggest effects of occupational exposures. Probably the most useful type of studies are those involving follow-up of the cohorts of workers over long periods of time with known exposures.

This is a report on a follow-up study of 1,464 men who completed their working lifetime as production or maintenance-service workers in the asbestos industry and retired with an industry pension during the period 1941 to 1967. These men were observed for deaths through 1969. The average length of employment in the asbestos industry for these men was 25 years and nearly all had exposures to asbestos dust. In some instances these exposures were very high and continued for many years.

Mortality for this cohort of men was 14.5% higher than for the entire population of US white males living at the same ages and time periods. This excess was due to cancer and respiratory disease. The cancer excess was chiefly due to respiratory cancer where mortality was 2.5 times the expected. The respiratory disease excess was entirely due to pneumoconiosis and pulmonary fibrosis, including asbestosis.

When these retirees were examined in terms of whether their work experience had been as production workers or as maintenance-service workers, some striking differences in their mortality experience appeared. For production workers the excess for all causes of death was only 10.7% whereas for maintenance-service workers the excess was 24.3%. For respiratory cancer differences were very great, with production workers having an excess of only 73.7% as compared to 328.6% for maintenance-service workers.

While maintenance-service workers had a high respiratory cancer mortality rate, they had a relatively low death rate from asbestosis, contributing only one out of 18 deaths from asbestosis observed in the entire study.

Differences in respiratory cancer mortality between these two groups of workers were probably not due to differences in time of retirement, disability at time of retirement, smoking histories, or cumulative asbestos dust exposure. At each dust exposure level maintenance-service workers had a higher respiratory cancer death rate than production workers; and, overall, their respiratory cancer death rate showed a sharper response to changes in dust levels. At the higher cumulative dust levels 30% of their deaths were due to respiratory cancer.

One explanation for the difference is the observation that maintenance-service workers had intermittent asbestos dust exposures—probably at a very high level—and that this produces excesses in respiratory cancer but not great excesses in asbestosis. Other studies lend some support to this notion. Another explanation lies in the type of asbestos to which these two groups of workers were exposed. Sixty-four percent of the maintenance-service workers with a high exposure had been exposed to crocidolite asbestos whereas only 8% of the production workers with a high exposure had been so exposed.

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