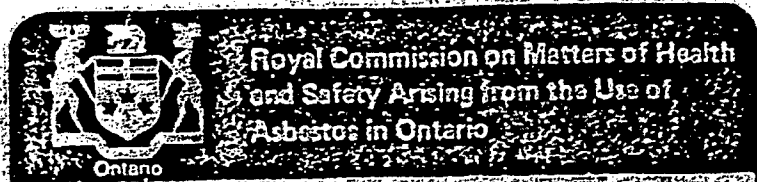


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Royal Commission on Matters of Health
and Safety Arising from the Use of
Asbestos in Ontario

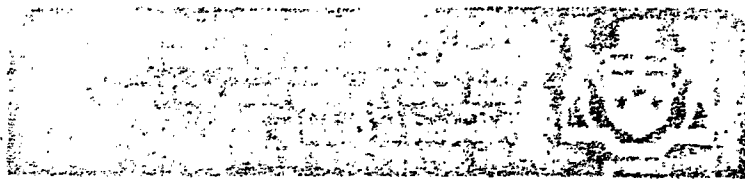
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**A REVIEW OF FOUR MAJOR REPORTS
ON THE HEALTH HAZARDS OF ASBESTOS**

A Background Paper Prepared By:

The Canadian Centre for Occupational Health and Safety





**A REVIEW OF FOUR MAJOR REPORTS
ON THE HEALTH HAZARDS OF ASBESTOS**

REPORT OF THE ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

A Background Paper Prepared By:

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100 King Street West, Suite 1000, Toronto, Ontario M5X 1C5

for

The Royal Commission on Matters of Health and Safety

Arising from the Use of Asbestos in Ontario

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This background paper was commissioned by the Royal Commission on Asbestos, but the views expressed herein are those of the author and do not necessarily reflect the views of the members of the Commission or its staff.

July 1981

006086

A Review of Four Major Reports on the Health

Hazards of Asbestos

Prepared at the Request of

The ROYAL COMMISSION on ASBESTOS in ONTARIO

By the

**CANADIAN CENTRE for OCCUPATIONAL HEALTH and SAFETY
120 Main Street West
Hamilton, Ontario**

July 1981

This background paper has been prepared by the Canadian Centre for Occupational Health and Safety for the Royal Commission on Asbestos to survey and summarize four studies of the health effects of asbestos fibre exposure. The paper does not report on the hearings on the health effects of asbestos held during the summer of 1981. The views expressed herein are those of the Canadian Centre for Occupational Health and Safety and not necessarily those of the Royal Commission or its staff.

006087

EDITOR'S NOTE

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Introduction

The Royal Commission on Asbestos invited the Canadian Centre for Occupational Health and Safety to undertake a review of four major studies of the health effects of asbestos. This was carried out in the form of answers to nine questions submitted by the Royal Commission.

- (1) What adverse health effects in humans are caused by exposure to asbestos?
- (2) What is the rate of occurrence of these health effects in those who work with asbestos and in the general public?
- (3) Do the major types of asbestos differ in their toxicity? If so, how?
- (4) What is the shape of the dose-response relationship? Is it linear, or some other shape?
- (5) Is there a safe threshold for exposure to asbestos? (By disease, by type of asbestos?) If so, what is it?
- (6) How does the health risk relate to the age of the exposed person? How is it affected by other factors such as smoking?
- (7) What is the magnitude of the health risk from asbestos for asbestos workers? (In general — not in any particular plant, firm or industry.)
- (8) What is the magnitude of the health risk from asbestos for the general population (excluding asbestos workers and those who live in the immediate vicinity of asbestos mines or mills)?
- (9) What was the chronology of the understanding of health risks from asbestos exposure by the health researchers?

The studies reviewed were:

- (1) Advisory Committee on Asbestos, Final Report, Report to the Health and Safety Commission, 1979, HMSO London. (referred to as the HSC Report)
- (2) NIOSH-OSHA Asbestos Work Group, Workplace Exposure to Asbestos: Review and Recommendations, April, 1980, U.S. Dept. of Health, Education and Welfare, U.S. Dept. of Labor. (referred to as the NIOSH-OSHA Report)*
- (3) National Research Council of Canada, Associate Committee on Scientific Criteria for Environmental Quality, Effects of Asbestos in the Canadian Environment by Stephen Snugar, 1979, NRCC 16452. (referred to as the NRCC Report)
- (4) Report of a Working Group of Experts Prepared for the Commission of the European Communities, Public Health Risks of the Exposure to Asbestos edited by Prof. R.L. Zaelhuis, EUR5653e 1977, Pergamon Press. (referred to as the CEC Report)

* A later version of this Report was published in November 1980 as DHHS (NIOSH) Publication No. 81-103. Although there is no textual difference between the versions, page references will differ.

We observed that the Reports differed from each other in purpose, style, and layout. Therefore, the conclusions were not always easy to bring together in one summary. We found that the NIOSH-OSHA and HSC Reports were clearly intended as policy documents with recommendations for decision-makers. The NHCC Report seemed intended solely as a historical resource to aid in the development of scientific guidelines, having no policy recommendations. Yet, it has the greatest number of citations from the literature. The CEC Report was prepared by a group of Dutch specialists as a reference document on which a report to the Council of Ministers (of the Commission of the European Communities) would be prepared. The NIOSH-OSHA Report cited scientific studies from 1976 onwards, presumably because this Report represents a revision of previous documents particularly the NIOSH revised recommended standard on asbestos published in 1977.

In spite of these inconsistencies we endeavoured to make the CCOHS review as objective as possible. Seven of the nine questions are answered with tables in our attempt to be as accurate as possible of the cited Reports' similarities and differences. The evidence in the Reports relative to Question 3 is plainly contradictory. Therefore, we called on an outside expert, Dr David Muir, to provide an answer which might better help clarify the issue for the Royal Commission. In answering Question 4 we felt that the Reports did not address an important issue which we have therefore detailed.

We found that, for the purposes of our review, the HSC Report came closest to providing answers to all the questions of the Royal Commission on Asbestos.

Canadian Centre for Occupational Health and Safety (CCOHS) personnel who contributed to the preparation of this survey of studies were: Dr Gordon Atherley, president; Barry Spinner, project scientist/asbestos project manager; Wendy King, research officer; David Cohen, staff writer/press officer; Lynn Lurie and Rita Steinmeyer, word processors; and Dr Robert McFadden, project scientist.

Section I: Diseases Caused by Asbestos

Question 1 WHAT ADVERSE HEALTH EFFECTS IN HUMANS ARE CAUSED BY EXPOSURE TO ASBESTOS?

There are five diseases which, in the general agreement of the Reports, are associated with exposure to asbestos: asbestosis, mesothelioma, and cancers of the lung, larynx and gastro-intestinal tract. (For the purposes of the CCOHS review, only these diseases are examined in detail and are described in Table I.)

Exposure to asbestos has also been implicated as the causative agent in cases of cancer of the lip, tongue, salivary gland, and mouth. And, according to the NRCC Report, circumstantial evidence exists connecting asbestos exposure and other cancers: primary leiomyosarcoma of the diaphragm, adenocarcinoma of the testis, ovarian cancer and cancer of the hematopoietic system.

The descriptions in Table I come from the Reports. The descriptions are technical summaries and medical descriptions of the five diseases. CCOHS has added brief commentary because, at times, the medical summaries and descriptions may not be comprehensible to persons without medical training.

Some of the manifestations of asbestos-related disease are characteristic. Examples include asbestos warts and asbestos bodies. Other manifestations of asbestos-related disease, such as the symptoms of coughing and shortness of breath, are common features of respiratory disease generally and, therefore, their existence does not necessarily indicate asbestos exposure. Generally, the process of diagnosing asbestos-related disease depends upon the physician's ability to relate various pieces of evidence. Often, evidence characteristic of asbestos exposure is hard to come by, leaving considerable room for uncertainty. Generally, the diagnosis of occupational disease, and not just asbestos-related disease, is a matter of presumption rather than certainty; often, the diagnosis turns on the occupational history given to the physician in response to his questioning of the patient. And many people have genuine difficulty in recalling all the pertinent details of a working lifetime, sometimes extending over forty or more years.

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The following detailed list of manifestations and symptoms is found in the NHCC Report. Most are respiratory system symptoms and abnormalities which typically appear in victims of asbestosis who have had long-term exposure to the dusts:

- fibrosis of the lung tissue
- the presence of 'asbestos bodies'** in the sputum and lung
- gas exchange restriction in the lung
- detrimental changes in lung function, especially vital capacity (VC)
- restriction of lung expansion
- blockage of bronchial airways
- the presence of pleural plaques***
- pleural effusions
- dyspnea (shortness of breath)
- loss of weight
- coughing
- cyanosis (low oxygen content of blood)
- crepitations (rales) and crackles (dry crackling sounds at the base of the lung, detected by auscultation)
- clubbing of the fingers
- asbestos warts on the hands.

Generally speaking, scientific literature assumes that readers are familiar with the relevant medical and scientific terminology. To some extent, the Reports make this assumption, although the HSC Report does attempt to explain some of the medical and technical terminology. In the opinion of OCOHS, however, the explanations are inadequate in that they fail to convey adequately what asbestos-related diseases really are in terms of their effects on human beings. Much of the substance of the Reports will be difficult going for persons without the necessary specialist training, we feel.

** Asbestos bodies are small (20-25 micrometre) particles, more correctly called ferruginous bodies. In and of themselves they do not indicate disease, but rather exposure to asbestos.

*** Pleural plaques are soft or collagenous tissues which form on the pleura (the membrane lining the walls of the thoracic cavity). The plaques have been described as being porcelain-like, whitish in colour, and opaque, in contrast to the healthy pleura which is pink, transparent and thin.

Table 1: Descriptions of Diseases Associated with Asbestos

Descriptions from the Reports

Asbestosis

Asbestosis is a progressive disabling disease which is not reversible. ... It is characterized by diffuse fibrosis of the lungs, reduction in pulmonary gas exchange, dyspnea, pleural thickening, and pain. These abnormalities occur more frequently among those occupationally exposed to asbestos than among non-exposed controls. (NHCC, p-403)

Mesothelioma

Mesothelioma, a neoplasm primarily affecting the pleura and peritoneum, is accompanied by a series of unpleasant and ultimately fatal symptoms (sic) such as weight loss, chest pain, cough and anorexia. Once diagnosis has been achieved, there is little that can be done in terms of successful treatment. (NHCC, p-412)

Cancer of the Lung
No descriptions given.

Cancer of the Larynx
No descriptions given.

Cancer of the Gastro-intestinal Tract
No descriptions given.

Explanatory Comments by UCOHS

Asbestosis is a progressive disabling disease, fibrosis of the lung, which is not reversible. Fibrosis of the lung is a condition of extreme scarring of the fibrous lung tissue. The damage progresses even after withdrawal from exposure. It is characterized by fibrosis of the lung tissues, shortness of breath, and pain. There may or may not be changes in the x-ray of the lung. The symptoms are non-specific in that people of their points directly to the disease. Diagnosis of asbestosis must involve an occupational (or para-occupational) history of exposure. Asbestosis may cause death, or lead to complications causing death, or only be disabling.

Mesothelioma is a malignant tumour affecting the linings of the lungs and of the abdominal cavity. It is accompanied by distressing symptoms such as chest pain, weight loss, and cough. It is rapidly fatal.

Lung cancer includes malignant cancers of the trachea, bronchus and the lung itself. The tumour related to asbestosis has no special diagnostic features. It is usually fatal. Lung cancer is considered a fatal disease, although surgical or medical treatment is sometimes successful in containing the disease or even eradicating it.

Gastro-intestinal tract cancer includes malignant tumours of the stomach, colon, rectum and the esophagus.

COMMENT BY COONS ON DISEASES CAUSED BY ASBESTOS

The Terms "Asbestos-Related" and "Asbestos-Induced"

By "asbestos-related" diseases we mean those which, it is generally agreed, are at least partially caused by exposures to asbestos. By "asbestos-induced" diseases we mean those which it is generally agreed have asbestos as a predominant cause. We can therefore arrange the diseases as follows:

Asbestos-Induced Disease

asbestosis
mesothelioma

Asbestos-Related Disease

cancer of the lung
cancer of larynx
cancer of the gastro-
intestinal (GI) tract

This distinction, we think, is useful because it points up the fact that mesothelioma and asbestosis are both typical occupational diseases. Both result from work with asbestos (in the case of mesothelioma contact with an asbestos worker or proximity to an asbestos fabrication facility can also cause the disease).

The Inadequacy of Medical Descriptions of Mesothelioma

Human pain and suffering represent factors not always given their due in official reports. Asbestosis is a debilitating disease; mesothelioma is a devastating one. Mesothelioma usually attacks suddenly, and then over a relatively short period (weeks or months) invades the body as a weed invades a garden. Typically, death occurs within a matter of months after the first diagnosis. This cancer of the chest or abdominal linings causes painful accumulations of fluid, making the stomach (if the malignancy is located there) rock hard. There are prolonged constipations, swelling of the feet and ankles, great weight loss and, perhaps most disheartening of all, dramatic and sudden changes in appearance. There is no cure for mesothelioma. Nor for asbestosis.

Mesothelioma is spoken about at length in each of the four Reports but without, as indicated above, an adequate impression of its seriousness. We assume that physicians, at least, are familiar with mesothelioma and its effects on the victim and the victim's family. But it is a rare disease, and few physicians will ever have actually encountered a case, let alone been involved sufficiently to appreciate all its implications.

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Section 2: Occurrence of Disease

Question 2 WHAT IS THE RATE OF OCCURRENCE OF THESE HEALTH EFFECTS IN THOSE WHO WORK WITH ASBESTOS AND IN THE GENERAL PUBLIC?

The diseases of major concern associated with asbestos exposure are: asbestosis, lung cancer, mesothelioma, cancer of the larynx, and gastro-intestinal cancers.

Table 2 describes a categorization of four types of exposures to asbestos used in the CEC Report. CCOHS uses this categorization in Appendix C.

For groups with occupational exposures it has been possible to rank the various diseases which cause death. The HSC Report reviewed the experience of various industrial populations which worked with asbestos, insulators, shipyard workers, asbestos miners, asbestos production workers, etc.:

"...the excess mortality found in these studies is accounted for in order of importance by lung cancer (associated or not associated with asbestosis), asbestosis, mesothelioma, and gastro-intestinal cancer." (HSC, page 49)

The data on cancer of the larynx indicate that the risk is much smaller than for the other diseases listed.

Tables 4 through 8 indicate the comments on this topic by the Reports.

In the subsections below each of the four diseases implicated in excess mortality (incidence rate of mortality above that expected for the studied industrial populations) is detailed.

Asbestosis

The Reports generally indicate that asbestosis has been rarely found in the general population and is not considered a public health risk.

Asbestosis is found only in groups of individuals with long-term exposure to asbestos dust. Specifically, occupational and para-occupational exposures have caused asbestosis.

The rates of incidence and prevalence are defined as follows:

Incidence rate is the proportion of persons developing a disease in a given population over a given unit of time, such as the rate of new cases of asbestosis occurring in a factory in a year.

Prevalance rate is the proportion of persons with a disease in a given population at a given time.

Table 2: Types of Exposure to Asbestos

(adapted from the CEC Report)

I Occupational exposure

- (a) direct:
- (1) mining & milling and transporting
 - (2) processing fibre
 - (3) installing, adapting and working with new products
 - (4) repairing and servicing existing products
 - (5) demolition
 - (6) dumping
- (b) indirect industrial
- (c) in agriculture

II Para-occupational exposure

- (a) domestic
- (b) hobbies

III Neighbourhood exposure

IV General environmental exposure

- (a) water
- (b) beverages
- (c) food and drugs
- (d) air

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Various studies of industrial groups have examined the percentage of all deaths in those groups caused by asbestosis and other causes of death. There are problems in comparing the various studies and volume 2 of the HSC Report elaborates these problems. The HSC Report summarizes the data of various studies for a number of diseases causing death in a table reproduced here as Table 3 (Table 13, HSC, Volume 1, page 50). The various studies examined are listed separately.

The table shows that asbestosis caused between 1.3% and 11.0% of all deaths among workers exposed to asbestos in those studies. If calculated as a percentage of excess mortality, the values would be even higher.

In its review of the data the NRCC Report describes the range of mortality due to asbestosis as 1.03% to 10.8% and states that the rates are probably underestimates. (NRCC, page 103).

The incidence rate of asbestosis is difficult to ascertain, since the clinical and other signs on which diagnosis is based are non-specific and subject to wide variations in interpretation. As well, comparatively little is known about the progression of the disease (HSC, page 54).

A study was done of a British textile factory in Rochdale where mostly chrysotile was used. (Annals of Occup. Hygiene, page 47, 11, 1968). This study, upon which the British Occupational Hygiene Society based the 1968 standard, used three conditions to measure morbidity: (a) certified asbestosis, (b) possible asbestosis, and (c) crepitations. The incidence rates observed for (b) and (c) are higher than for certified asbestosis. An incidence rate for certified asbestosis of 0.5% occurs at the level of exposure supposedly equivalent to a lifetime's exposure at 2 fibres/mL.*

An update of this study was done and later published (Berry et al, Br. J. Ind. Medicine, page 98, 36, 1979). It concludes that the incidence rate for certified asbestosis of 0.5% per year occurred in persons with a cumulative dose of less than 10 fibre-years per cc. This is said to correspond to a level of exposure of 2 fibres per cc for a lifetime's exposure of 50 years.* Using the category of 'possible asbestosis', a detectable rate of incidence occurs at exposures less than 50 fibre-years per cc. And if the development of crepitations is considered, the average incidence is 1.3% per year for cumulative exposures of less than 50 fibre-years per cc.

According to the HSC Report the only study giving a relatively unbiased assessment of the rate of impairment of lung function (asbestosis) is the updated British study referred to above.

* fibres/cc or fibres/mL or 1/mL is a measure of airborne concentration, number of asbestos fibres in one millilitre of air. A fibre is defined as being longer than 5 micrometres and with a length to diameter ratio greater than three.

Lung Cancer

Unlike the data on asbestosis from the various studies compared in Table 3, the information on lung cancer as a cause of death is less clear.

Asbestosis is believed to occur only in those exposed to asbestos dust, lung cancer occurs in all populations. Hence the portion of observed lung cancer deaths due to asbestos dust alone cannot be accurately measured. Further, the synergistic interaction of tobacco smoke and asbestos complicates any analysis of the lung cancer deaths. (See Section 6 for a discussion of the Reports' views on this synergy.)

The usual approach to dealing with such difficulties is to calculate the mortality ratio. This has been done for the data in Table 3. The number of observed (actual) deaths in the industrial group due to any cause is labelled 'O'; the number of expected deaths in some group comparable to the industrial group due to the same cause is labelled 'E'. The ratio O/E is the mortality ratio. For example for lung cancer deaths in study 1 of Table 3, O equals 58 and E equals 22 and the mortality ratio O/E, expressed as a relative risk, is 2.7. This indicates that lung cancer is a more likely cause of death for this group than for some comparable group not exposed to asbestos dust.

The data of Table 3 show mortality ratios for lung cancer in a range 1.2 to 15.9 for the various studies. Interpretation of such comparisons is extremely difficult for at least two reasons: the choice of which deaths to include in the number of deaths; and the basis of calculating the expected number of deaths of some comparable group. The first reason is demonstrated directly in Table 3 where, in study 9, cancers of the larynx are included for both observed and expected numbers of deaths.

Mesothelioma

The Reports generally agree that mesothelioma is extremely rare in the general population in the absence of exposure to asbestos dust.

Even though the disease is extremely rare, mesothelioma has been observed where neighbourhood exposure occurs. The incidence rate is in the range 1 to 5.6 per million per year.

The HSC Report ranks mesothelioma third as the cause of excess mortality in industrial populations.

Table 3 shows that mesothelioma causes between 0 and 7.1% of all deaths among the workers exposed to asbestos in the various studies summarized.

Gastro-Intestinal Cancers

The Reports make no comments about the occurrence of gastro-intestinal tract cancers in the general population; instead they focus on the association of gastro-intestinal cancers and exposure of workers to asbestos.

The Reports generally agree that an increased incidence of death due to gastro-intestinal cancers occurs in industrial groups exposed to asbestos. They note that there is no general scientific agreement on the degree of association of asbestos exposure and gastro-intestinal cancers. Table 3 shows the mortality ratio is in a range 0.5 to 3.2.

006101

Section 3: Toxicity of Different Types of Asbestos

Question 3 DO THE MAJOR TYPES OF ASBESTOS DIFFER IN THEIR TOXICITY? IF SO, HOW?

This topic is controversial. In understanding the contradictory evidence, it is necessary to understand that there are at least four different commercially important fibre types: chrysotile (white asbestos) and the amphiboles, amosite (brown asbestos), crocidolite (blue asbestos), and anthophyllite (an asbestos not of commercial concern within Canada). Applications often involve mixtures of different fibres. Also these fibres are produced in different fibre sizes and geometries depending on the production process. Hence, differences in toxicity may occur due to fibre type, size or geometry. Ordinary air monitoring does not differentiate among these features.

The contradictions arise from evidence collected from different sources, either animal experiments or human epidemiological studies. Human experience supports the argument that there are differences in toxicity. Animal experiments show the inhalation of samples of chrysotile produces at least as many malignant lung tumours and mesotheliomas as the amphiboles. Apparently this evidence is not applicable to the human situation. The extremes of the controversy are clearly demonstrated by the following quotations from the HSC and NIOSH-OSHA reports.

(1) "As far as mesothelioma is concerned, evidence from miners; from process workers exposed to a single fibre type; from the distribution of neighbourhood and domestic cases; and from the geography of mesothelioma, when combined, presents a powerful case from four different sources that crocidolite has been more dangerous than chrysotile and anthophyllite. The position of amosite may be intermediate between crocidolite and chrysotile. There is no doubt that crocidolite was used at Roynedale, but the significance of this as a cause of the mesothelioma cases there is not certain. It can be concluded that exposure to chrysotile alone has rarely been shown to cause mesothelioma." (HSC, paragraph 80, page 53)

(2) "...presently available evidence is insufficient to determine the role of specific fibre types (regarding cancer excesses). On the basis of available information, ... concluded that there is no scientific basis for differentiating between asbestos fibre types for regulatory purposes." (NIOSH-OSHA, page 3)

Tables 9 through 11 indicate the breadth of comment on this topic by the Reports.

Given the contradictory evidence and opposing positions taken by the Reports, CCOHS asked Dr David Muir of McMaster University to provide an opinion on this topic. His letter is attached as Appendix B.

The analysis by Dr Muir clearly shows how it is possible for animal experiments demonstrating no basic differences in toxicity to be reconciled with the epidemiologic data. Quite rightly the focus for analysis should remain on the hazard to humans inhaling asbestos fibres and information about controlled toxicity experiments should be viewed in light of their relevance to the hazards of humans.

He states that it is necessary to distinguish clearly between differential risks associated with the use of various types of fibre in contrast with the risks according to the measured airborne concentrations. Stating that he believes animal experiments are of remarkably little help in sorting out the confusing situation, he analyses the pertinent literature and the reports to determine whether they had a clear understanding of the difficulties in making the comparisons between fibre types and whether they have drawn sensible final conclusions.

His conclusion is "I have little doubt that exposure to crocidolite is associated with an increased risk of mesothelioma."

Table 2 Differences in Toxicity - ASBESTOSIS

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 3
Differences in Toxicity for Asbestosis

1 HSC Advisory Committee on Asbestos
Final Report
pg. 53

-Evidence for a difference in risk of asbestosis
with fibre type is slight.

2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 3

-No scientific basis for differentiating between
asbestos fibre types for reg.atory purposes.

3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...

NO COMMENT MADE

4 CEC Working Group of Experts:
Public Health Risks of
Exposure to Asbestos
pg. 65

-Asbestosis may develop from exposure to all
four important types of asbestos, regardless
of the dimensions of the inhaled fibres; the
longer ones, however, seem to be more pathogenic.

Table in Differences in Toxicity - LUNG CANCER

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 3
Differences in Toxicity for Lung Cancer

006105

- 1 HSC Advisory Committee on Asbestos
Final Report
pg. 52, 53

-A firm judgment on the question whether amosite has been more dangerous than chrysotile...is not possible.
-Relationship of fibre type to lung cancer is inconclusive, but where it exists it indicates that exposure to amphiboles (crocidolite and amosite) has been more dangerous than to chrysotile.
-The weight to be attached to this conclusion is limited.

- 2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 3

-No scientific basis for differentiating between asbestos fibre types for regulatory purposes.

- 3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 116

-One study stated that exposure to chrysotile and crocidolite resulted in a 5.3 fold increase in the rate of respiratory cancer; exposure to chrysotile alone resulted in a 2.4 fold increase.

- 4 CEC Working Group of Experts
Public Health Risks of
Exposure to Asbestos
pg. 65

-The incidence of bronchial (lung) cancer...does not depend upon the type of asbestos, but mainly on the dose level.

Table 1b Differences in Toxicity - MESOTHELIOMA

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 3

Differences in Toxicity for Mesothelioma

- 1 HSC Advisory Committee on Asbestos:
Final Report
Pg. 53, 62

-Exposure to chrysotile alone has rarely been shown to cause mesothelioma.

-Evidence from miners, from process workers, ... from the geography of mesothelioma when combined, presents a powerful case, that crocidolite has been more dangerous than chrysotile.

-The position of amosite may be intermediate.

-On the other hand, it is essential to restate the proven capacity of chrysotile to produce mesotheliomas in animals if the fibre is of the appropriate configuration.

- 2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
Pg. 3

-No scientific basis for differentiating between asbestos fibre types for regulatory purposes.

- 3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...

-Some (epidemiological) studies appear to indicate that different types of asbestos have different toxicities.

- 4 CFC Working Group of Experts:
Public Health Risks of
Exposure to Asbestos
Pg. 90

-There is general agreement that the risk of mesothelioma is fibre related in the order: crocidolite greater than amosite greater than chrysotile, but the magnitude of difference...is not well established.

006106

Section 4: Shape of the Dose-Response Relation

**Question 4 WHAT IS THE SHAPE OF THE DOSE-RESPONSE
RELATION? IS IT LINEAR OR OF SOME OTHER
SHAPE?**

Preamble - Questions 4 and 5 relate to each other very closely because they are both exploring the same area of scientific knowledge, but from slightly different points of view. Because the two questions have so much in common, it is useful to begin with a preamble which defines all the technical terms used in the two questions. The following definitions are presented with acknowledgement to the Oxford English Dictionary.

The technical terms are: threshold
 safe threshold
 dose
 response
 dose-response relation
 linear relation
 other shapes
 Threshold Limit Value

There is an important possibility for confusion when using a technical term with a name which may be familiar to readers and which contain the word "threshold". That term is "Threshold Limit Value" (TLV), which is a registered trademark of the American Conference of Governmental Industrial Hygienists. In some respects "Threshold Limit Value" is relevant to the matters addressed by questions 4 and 5, and this is why confusion might arise. A definition of "Threshold Limit Value" has been given so as to avoid all possibility of confusion between "safe threshold" and "Threshold Limit Value".

Threshold The everyday meaning is the best guide: the entrance to a house or building.

In science, the term was first used by psychologists to describe what appeared to be boundaries in human sensation. For example: (a) "there is a certain limit below which our ... sensibilities are unable to discriminate. This boundary ... (is) ... the "threshold". (b) we do not distinguish ... separately ... presentations of less than a certain assignable intensity. On attaining this intensity presentations are said to pass over the threshold of consciousness ...

A threshold for exposure to asbestos would be a boundary or limit which marks a clear separation between two different states of asbestos-related disease — see safe threshold, below.

Safe Threshold A threshold of asbestos exposure which separates the absence of asbestos-related disease from the presence of asbestos-related disease.

If a safe threshold existed it would imply that some degree of exposure to asbestos could be endured without the development of any disease. If no safe threshold existed, then one would have to assume that any degree of exposure to asbestos, no matter how small, would be capable of producing some disease in some persons.

Dose A definite quantity or amount (of asbestos) regarded as analogous in some respects to a medical prescription or to medicine in use or effect.

Response Scientific use differs wholly from the everyday use; relative to asbestos, response means that proportion of a population exposed to asbestos which experiences a specific disease following exposure of the total population to asbestos exposure of a specified degree. (This definition was derived from that for "dose-response relationships" arrived at by the Task Group on Metal Accumulation (1973), cited by G. F. Nordberg (ed.) "Effects and Dose-response Relationships of Toxic Metals", Elsevier (1976) Amsterdam.)

Dose-Response Relation The correlation, normally expressed as a graph, between the percentage of the population affected by the disease in question and the dose of asbestos.

Linear Dose-Response Relation Where the graph relating dose and response is a straight line, necessarily beginning at zero (see figure 1b).

Other shapes for dose-response relation The graph relating dose to response may be S-shaped (see figure 1a), but at least in theory the graph could possess any conceivable shape.

Threshold Limit Value Refers to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect. Because of wide variations in individual susceptibility, however, a small percentage of workers may experience discomfort from substances at concentrations at or below the threshold limit; a smaller percentage may be affected more seriously by aggravation of a pre-existing condition or by development of an occupational illness.

TYPES OF DOSE — RESPONSE RELATIONS

Figure 1a: Linear Relation

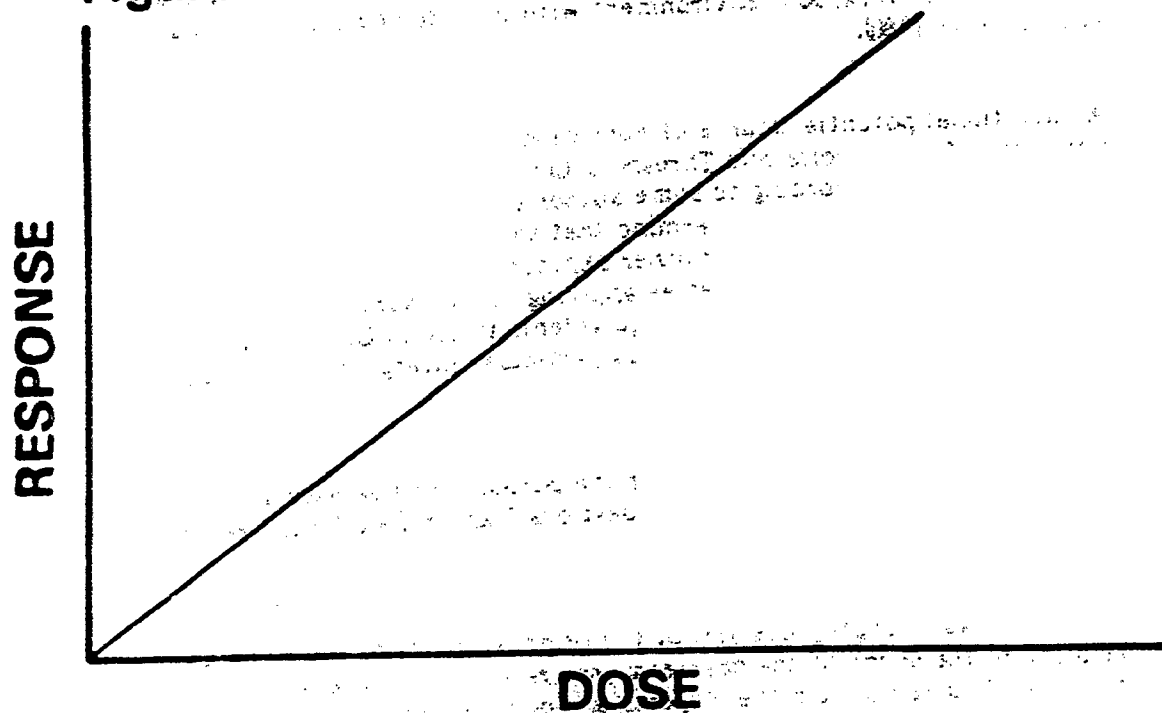
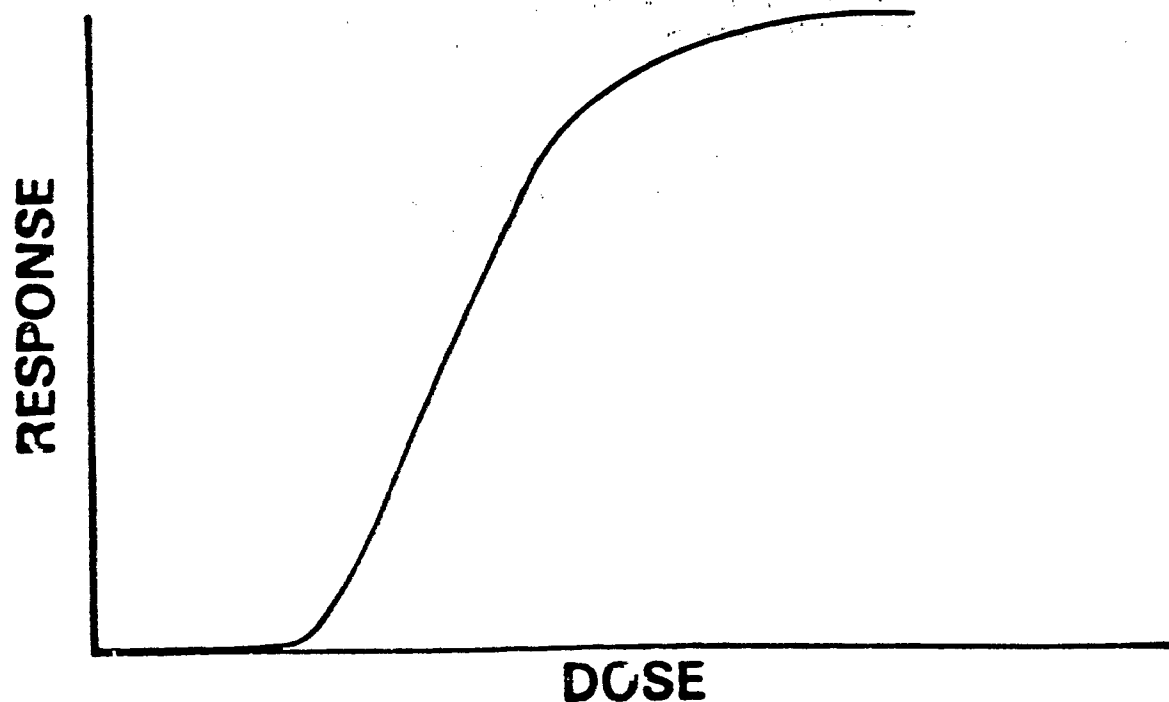


Figure 1b: S-shaped Relation



As previously stated, the term "Threshold Limit Value" is a registered trademark belonging to the American Conference of Governmental Industrial Hygienists, and the definition just quoted is taken from their booklet "Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1980" (Cincinnati: 1980).

An additional potential source of confusion lies in the connection between safe thresholds and Threshold Limit Values and other forms of exposure limit. According to some authorities, dose-response relations should be used in order to determine that threshold which is to be used as the threshold limit value or other exposure limit. Accordingly, Question 5 could be interpreted as applying to exposure limits such as Threshold Limit Values and their equivalents in use in Ontario. We know from the Royal Commission that this particular interpretation is not intended.

Dose-response relations are used for the purposes of risk assessments; for within our terms of reference Questions 7 and 8 specifically refer to matters of risk.

There follows a lengthy analysis of the principal parts of the Reports relating to the shape of the dose-response relations. The analysis consists of extracts from the Reports and commentary. The gist is that for practical purposes such as risk assessment the Reports judge that the dose-response relations are generally linear. As stated in the answer to Question 4, once the dose-response relations are designated linear with origins at zero, there comes automatically an answer to Question 5: by definition, with such linear dose-response relations there can be no thresholds, safe or otherwise.

The analysis is quite detailed because we have detected a fundamental question to which we would like to draw attention; that question is presented in the comments by CCOHS which follow the analysis.

The NIOSH-OSHA Report

The following are extracts from the NIOSH-OSHA Report, indicative of the approach to the question of the linearity or other shape of the dose-response relation. It can be seen that there is acknowledgement of some uncertainty about the shape of the dose-response relation but the tenor of the Report is an acceptance of the linear nature of the dose-response relation, with no doubt expressed about the importance of dose-response relation relative to decision-making on asbestos.

Extract 1

"Evidence available to date indicates that a large dose of asbestos will produce a bigger health hazard than a small dose. Seidman et al. (1979), using the length of time worked in an amosite asbestos factory as a measure of the dosage of asbestos, reported an increased risk of dying from lung cancer with increasing duration of employment.

Henderson and Enterline (1979), using cumulative dust exposure as an estimate of dose, reported that the dose-response relationship for lung cancer is more likely linear. They predicted the relationship to be $SMR = 10 + 0.658 \text{ times cumulative dust exposure (mppcf-yrs)}$. Liddell et al. (1977) also reported a similar relationship, i.e., a tendency for the mortality for lung cancer to increase with the dose." (p. 51)

Extract 2

"Newhouse and Berry (1973) suggested that the risk of dying from mesothelioma increases with increasing dose. Jones et al. (1979) reported a linear relationship between mesothelioma rate and length of exposure. In a study of the women workers in a wartime gas mask factory, they found women having a long employment period had a higher proportion of death due to mesothelioma than those who had a short period of employment." (p. 52)

Extract 3

"Although there appears to be little dispute that a larger dose of asbestos will pose a greater health risk, the exact nature of the dose-response relationships may be subject to considerable debate. This is so primarily because of problems of exposure estimation. Methods of measuring dust have changed over time with respect to sampling instrument (thermal precipitation, vs. midget impinger vs. membrane filter); location of sampling (personal vs. area), dust counting (particles vs. actual fibres) and/or evaluation techniques (whole fields vs. eyepiece graticule). As a result, conversion of dust levels obtained by one method to levels comparable to another method is far from simple, and is subject to considerable error. Another factor which may lead to differences of opinion on the exact shape of the dose-response curve is the measure of the dose. The commonly used measures of exposure are the cumulative dose and the duration of employment. Since using cumulative dose as a measure of exposure gives equal weight to the concentrations of dust experienced in each year of exposure, exposure of many years ago is considered as important as recent exposure. This practice is unrealistic for the chronic diseases having a long-latency period. Duration of employment has also been used as a measure of exposure under the assumption that increasing the work time approximates increasing the dose. This procedure has the same problem as using the cumulative dose. Furthermore, in the absence of reliable past exposure data, the duration of employment may not equal the total dose of asbestos." (p.52-53)

Extract 4

"With regard to the linear hypothesis, the British Advisory Committee on Asbestos, in 1979, stated as follows:

Our reasons for preferring a linear hypothesis are:

(1) It fits the data for occupational exposures, (2) it is the simplest hypothesis and the one most readily used for extrapolation to the probable effects of low doses; (3) it is likely to lead to an overestimate rather than underestimate of risks at very low doses. (HSC, Vol. 2, p. 14).

Data available to date provide no evidence for the existence of a threshold level. Virtually all levels of asbestos exposure studied to date demonstrated an excess of asbestos-related disease." (NIOSH-OSHA, p. 53)

The HSC Report

The following extract from the HSC Report (Vol. 2) covers dose-response relation in rather more general terms than the NIOSH-OSHA Report. The clear assumption is that dose-response relation is a reality not yet fully understood but the linear form is to be preferred, for the reasons given in the quotation embodied in the NIOSH-OSHA Report.

"169 This section discusses the quantitative evidence about the relationship between the dose of asbestos inhaled by members of a population and the frequency of three principal biological responses - asbestosis, lung cancer and mesothelioma. No data have been found which permit any quantitative comment to be made about the relationship between dose and response for cancer of the larynx. That related to cancer of the alimentary tract is limited to two studies. The importance of establishing, if possible, the nature of the relationship between dose and biological response is that it may assist in framing policy about an acceptable level of exposure. For example if it could be shown that a level of exposure exists below which no risk of sickness or death is incurred (a so-called threshold) at least such a level would be likely to be acceptable from the medical point of view." (p. 34)

"170 Some of the possible relationships between dose and response in connection with the effects of asbestos...are (a) a threshold relationship whereby below a certain limiting value of the dose there is no associated excess sickness or mortality; (b) a (recti)linear relationship whereby the response increases proportionately to the dose; (c) a quadratic relationship whereby the response increases proportionally to the square of the dose; (d) a sublinear relationship where the response increases proportionally to a power of the dose, the power being less than unity; (e) a cumulative normal relationship whereby the response increases as if the distribution of susceptibility levels to asbestos was normally distributed..." (p. 34)

COMMENT BY CCUHS ON SCIENTIFIC NATURE OF THE DOSE-RESPONSE RELATION

In our opinion, there are no scientific data in the Reports, nor in any studies relied upon by them, from which any firm conclusions can be drawn about the shape of the dose-response relation. Indeed, the scientific data are so vague that we must ask whether scientific understanding of the dose-response relation is a practical reality and not an unattainable goal of the scientific community engaged in certain fields of research. We are collecting data in answer to that question, and some of our preliminary observations are presented below. Meanwhile, we would like to bring up for discussion the question: Is dose-response relation a non-measurable concept?

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From the definition of dose-response relation we can see that it implies a correlation between dose and the proportion of the population affected by whichever asbestos-related disease we are considering. But dose also implies a measurable quantity -- of asbestos inhaled or otherwise taken in by the people exposed to it. If we accept dose-response relation as a measured concept we are accepting, in effect, that the correlation between dose of asbestos and the asbestos-related disease in question is sufficiently strong for whatever purposes we choose to use dose-response relation for.

But the dose of asbestos for human beings at work or in the community at large has never been measured directly. Instead, dose is inferred from the exposure known or presumed for the exposed population. Thus for practical purposes such as risk estimation in connection with asbestos exposure, two reasonably strong correlations have to be presumed between: (a) dose and response, and (b) exposure and dose. If one or both correlations are weak then the risk assessments are likely to be so uncertain as to be misleading. Our opinion is that the Reports do not deal adequately with the issue of the correlations and, therefore, we would like the Commission to consider our question, just proposed. There follows our preliminary observations on dose-response relation; these are presented as a background to our question.

Our preliminary conclusions about dose-response relation

We believe that dose-response relation should be seen as an idea which possessed considerable promise when it was first mooted thirty or so years ago. Since then, extensive research has been carried out, apparently, dose-response relation has no better scientific basis now than it had at first. Indeed, surprisingly little research has been aimed at producing a secure scientific foundation for it; by far the major effort has gone into research which presumed its reality, hardly ever questioned its validity, and almost never reported adequate evidence of its existence. To make matters worse (and this is surprising), dose-response relation has never been defined in terms which everyone can agree to, and neither has it been defined in terms which adequately reflect the scientific complexity of "dose", or "response", both of which are examined in detail in the HSC Report. A confusion appears to have arisen leading to a widespread and fundamental misunderstanding about dose-response relation.

This misunderstanding came about and is perpetuated because:

- (1) Researchers report on what they call "dose-response relations". The idea then grows that "dose-response relation" represents a scientific reality. The research is not false: the researchers have successfully demonstrated the existence of something which they term "dose-response relation".
- (2) Individuals and organizations concerned with risk assessment, or other practical matters, take the researchers' conclusions as valid, and proceed on the basis that what is needed can be deduced from something the individuals and organizations regard as "dose-response relation".
- (3) No one has established that the two "somethings" coincide scientifically.

The meaning of the dose-response relation in studies reporting its existence.

Dose-response relations, sometimes referred to as exposure-response relations, belong to one of two categories: (a) years of exposure, and (b) years of exposure combined with asbestos levels.

(a) Years of Exposure - In this category, dose is measured solely in terms of the years of exposure and no quantitative information is given about the nature, level, or pattern of the group's exposure to asbestos. Thus, the correlations labelled "dose-response relations," are between the proportion of the population experiencing the stated disease and the years of employment or the exposure (if there is a distinction drawn between the two).

For some purposes, these correlations produce useful conclusions. However, the usefulness is limited because years of employment correlate with years of age and, as is well known, the frequency of occurrence of many diseases also correlates with age, at least within certain age ranges. For example, lung cancer is relatively uncommon (though it does occur) before the age of forty; it occurs much more frequently after that age. Asbestos-related cancer and asbestos-related pneumoconiosis display a relation with age and, consequently, in some designs of experiment it may not be possible to distinguish age-related effects from asbestos-related effects.

That difficulty becomes clearer when we take as an example the latent period of mesothelioma, that is to say, the time elapsing between the first recorded exposure to asbestos and the confident diagnosis of the mesothelioma. There appears to be evidence of latent periods of thirty years or even more.

An example (from studies mentioned in the Reports) indicative of the long latent period is the female workers in Canada and Britain during the Second World War, who manufactured gas masks. Blue asbestos is said to have been used for parts of some or all of the gas masks; the female workers were employed on work which included putting the blue asbestos into the gas masks. Manufacture ceased with the end of the Second World War; instances of mesothelioma began to be noticed in the 1960s, and continued to occur through the 1970s. On the presumption that the gas mask blue asbestos was the only blue asbestos to which the workers were exposed, these instances are clear evidence of long latent periods for mesothelioma associated with blue asbestos.

For workers in processes which were continuous over a period of many years, such as asbestos cement pipe manufacturing in Ontario, there would be no such clear end point to the exposure. If these workers also were to develop mesothelioma after twenty to thirty years exposure, there would be no way of knowing for sure whether all of that twenty to thirty years exposure was needed to induce the disease, or whether the first few years, few months, few weeks, or even few days were sufficient to lay an unchangeable foundation for the mesothelioma which would then develop in its own time.

Manifestly, for any diseases with long latent periods, including asbestos-related pneumoconiosis, correlations between years of employment and frequency of disease may be misleading where the workers' diseases have developed during their period of employment. There may be no way of knowing whether the same workers would have developed the same diseases if exposure had ceased within days, weeks, months, or years of the start of exposure.

(b) Years of exposure combined with asbestos levels - This category is represented by those studies in which the data are presented as some combination of asbestos levels and years of exposure.

Many such studies rely on a measure of dose derived from a measure of asbestos levels multiplied by the total years of employment of the people exposed to those levels. A common example of this measure is million particles per cubic foot x years of exposure abbreviated, often, to mppcf-yr. Equivalent to this measure is fibres per cubic centimetre x years, abbreviated to (f x y)/v.

These dose-response relations may be termed "cumulation-response relations", because the product of the level of asbestos contamination and years of exposure/employment corresponds to a measure of cumulation.

In some studies, the cumulation is calculated from the product of years of exposure and a single "average" level of asbestos during the exposure years. But, this is not really a cumulation-response relation; mathematically, it is the same as the years-of-exposure/employment-response relation, that is to say category (a). Cumulation-response relation applies only where there is some variation over the years in the level taken to typify the asbestos contamination.

Nearly all the studies which present cumulation-response relations are dependent upon asbestos measurements averaged over year-long periods. So far, we have identified not one study with data relating to averages taken over less than eight hours.

For diseases with relatively long latent periods, cumulation-response relations possess the same weaknesses as years-of-exposure/employment-response relation. The Reports acknowledge problems in the interpretation of cumulation-response relations, and critical consideration of these factors is evident in some of the more recent studies cited by the Reports. So far, however, we have traced no studies which have adequately dealt with the question of fluctuation of asbestos levels from one year to another. And there appear to exist no studies whatever which adequately deal with the effects of fluctuations in the course of a working day.

As stated previously, the data on asbestos levels reported in most of the studies are typically averaged over a period of a year. This averaging implicitly assumes that there are no long-term effects due to short period fluctuations in asbestos levels. However, such effects are in principle present, and can be calculated from various mathematical models of the development of disease over time. But the magnitude of effects cannot be determined from the models alone. For that purpose, data would be required about the short-period fluctuations in level and, so far as we have been able to find, no such data are available.

The procedures used for data-gathering up to the present, at least, absolutely preclude the possibility of finding out the significance of fluctuations over hours or days, a potentially important factor affecting the long-term risk of asbestos-related disease.

The prevalence of asbestos-related disease has been shown to increase after exposure to asbestos has apparently ceased. This observation demonstrates that any simple approach based on averages, and/or years-of-exposure/employment-response relation, and/or the cumulation-response relation are inadequate as an explanation of the nature of asbestos-related disease.

Table 1a Dose Response

FINDINGS OF THE REPORTS
Agency Report

1 HSC Advisory Committee on Asbestos:
Final Report
pg. 54-56

2 NIOSH-OSHA Asbestos Work Groups:
Workplace Exposure to Asbestos
pg. 4, 51, 52

3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 138

4 CEC Working Group of Experts:
Public Health Risks of
Exposure to Asbestos
pg. 116, 119

ANALYSIS FOR QUESTION 4
Dose Response

- Asbestosis: evidence of disease is directly proportional to cumulative dose (but) irregular.
- Lung cancer: chrysotile-linear relation and amosite-sublinear.
- Mesothelioma: information about precise relationship between dose and response is lacking.
- Gastro-intestinal cancers: nothing definite can be said about the shape of the curve.
- A linear relationship appears to best describe the shape.
- Evidence to date indicates that a large dose of asbestos will produce a larger health hazard than a small dose.
- The exact nature of the dose-response relationship may be subject to considerable debate primarily due to problems of exposure estimation; another factor is the measure of dose.
- Dose response relationships exist (in animal experiments) for incidence of lung cancer and mesothelioma.
- There apparently exist qualitative dose-response relationships ...
- However, very few quantitative data exists.
- Cancer mortality is highly dependent upon intensity of exposure, but quantification is still not possible.

Section 5: Existence of a Safe Threshold of Exposure

Question 5 IS THERE A SAFE THRESHOLD FOR EXPOSURE TO ASBESTOS (BY DISEASE, BY TYPE OF ASBESTOS), AND IF SO WHAT IS IT?

Tables 13-15 summarize the salient points from each of the Reports, relative to the notion of safe threshold.

Overall, the notion of safe threshold appears to have been rejected by the Reports with the possible exception of the position of the HSC Report on asbestosis. Here, a possibility of a threshold is considered, although the Report cites evidence pointing in the contrary direction. The CEC Report raises the possibility of a threshold for lung cancer and for mesothelioma, and then it goes on to reject it in both instances. The NIOSH-OSHA Report states there is no safe threshold for asbestos-related disease without having differentiated between the diseases.

COMMENT BY CCOHS ON SAFE THRESHOLD

The term "threshold" implies a boundary between one state and another, say, the presence or absence of disease. Threshold Limit Value, on the other hand, implies a boundary not clear cut, despite the use of "limit" in the title. In discussing "safe threshold" we must make clear whether the model represents a threshold in the sense of a clear cut boundary, or whether it represents a vague border zone, as does the Threshold Limit Value of the ACGIH.

The S-shaped dose-response curve (see figure 1b) shows a kink which could be interpreted as a threshold — a threshold dividing these two: (a) asbestos exposure associated with disease in a relatively small proportion of the exposed population, and (b) disease in a relatively high and steeply increasing proportion of the exposed population.

By contrast, a linear dose-response relation give no support whatever to the idea of threshold. At any value above zero there is some asbestos-related disease; the proportion of persons affected increases proportionately to the dose of asbestos received. Here one can see why Questions 4 and 5 are so closely related: if the dose-response relation is linear for any or all of the asbestos-related diseases then, by definition, there can be no safe threshold for asbestos exposure. On the other hand, if the dose-response relation is S-shaped then there may be a threshold, a threshold which divides a relatively low frequency of disease from a relatively high frequency of the disease. Unless the S-shaped curve lies above the horizontal axis at all points apart from its origin then, on strict definitions, that threshold could not be called "safe" threshold because it does not separate the condition of no disease (the safe state, presumably) from the condition of some diseases (presumably, the unsafe state). Only if the S-shaped curve cuts the horizontal axis at some point to the right of the vertical axis can we speak of a "safe threshold".

Table 13: Safe Threshold - ASBESTOSIS

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 3
Safe Threshold for Asbestosis

- | | |
|---|---|
| <p>1 HSC Advisory Committee on Asbestos:
<u>Final Report</u>
pg. 54</p> | <p>-There appears to be a threshold at an estimated cumulative dose somewhere between zero and 100 fibre-years per millilitre.
-However, an annual incidence of 0.5% has occurred after cumulative doses of less than 100 fibre-years per mL, that is, at the level of 2 fibres per mL.</p> |
| <p>2 NIOSH-OSHA Asbestos Work Group:
<u>Workplace Exposure to Asbestos</u>
pg. 53</p> | <p>-Data available provide no evidence for the existence of a threshold level.
-Virtually all levels of asbestos exposure studied demonstrated an excess of asbestos-related disease.</p> |
| <p>3 NRCC Associate Committee on...Criteria:
<u>Effects of Asbestos...</u>
pg. 54</p> | <p>-Not possible to specify an asbestos concentration associated with no measurable effect on health.</p> |
| <p>4 CEC Working Group of Experts:
<u>Public Health Risks of Exposure to Asbestos</u>
pg. 118</p> | <p>-There is very likely a <u>practical level of exposure below which it will be impossible to detect any excess mortality or morbidity due to asbestos.</u></p> |

Table 14. Safe Threshold - LUNG CANCER

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 5
Safe Threshold for Lung Cancer

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- 1 HSC Advisory Committee on Asbestos:
Final Report
pg. 55
 - No evidence ... for a threshold of dose below which there is no evidence of risk.
- 2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 53
 - Data available provide no evidence for the existence of a threshold level.
 - Virtually all levels of asbestos exposure studied demonstrated an excess of asbestos-related disease.
 - Excessive cancer risks ...demonstrated ...at all fibre concentrations.
- 3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 54, pg. 113
 - Not possible to specify an asbestos concentration associated with no measurable effect on health.
- 4 CEC Working Group of Experts:
Public Health Risks of Exposure to Asbestos
pg. 111 (pg. 118)
 - There is suggestive evidence for a threshold limit, below which an excess risk is extremely small. Adequate data to establish such a threshold limit are not available.

Table 15: Safe Threshold - MESOTHELIOMA

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 5
Safe Threshold for Mesothelioma

1 HSC Advisory Committee on Asbestos:
Final Report
pg. 56

-The risk of this tumour increases with increasing dose. Relatively low doses may be followed by development of this cancer.

2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 53

-Data available provide no evidence for the existence of a threshold level.
-Virtually all levels of asbestos exposure studied demonstrated an excess of asbestos-related disease.

-Excessive cancer risks ...demonstrated ...at all fibre concentrations.

3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 54

-Not possible to specify an asbestos concentration associated with no measurable effect on health.

4 CEC Working Group of Experts:
Public Health Risks of Exposure to Asbestos
pg. III

-There is suggestive evidence for a threshold limit, below which an excess risk is extremely small or non-existent. Adequate data to establish such a threshold limit are not available.

-One may even seriously doubt the possibility of deriving a valid measure for external dose as an inductor of mesothelioma.

Section 6: Other Factors Affecting Risk of Disease

Question 6 HOW DOES THE HEALTH RISK RELATE TO THE AGE OF THE EXPOSED PERSON? HOW IS IT AFFECTED BY OTHER FACTORS SUCH AS SMOKING?

A variety of factors affecting the incidence and severity of asbestos-related and asbestos-induced diseases include:

- exposure period
- dose
- smoking
- latent period
- age
- pattern of exposure

The importance of the degree of exposure and the dose have been discussed in Section 4. Also the uncertainties attached to the concept of dose have been discussed in Section 4.

Other factors which have been identified include the presence of a history of tobacco smoking, the existence of periods of latency, the age of the exposed individual, and the pattern of exposure.

Smoking

Table 16 outlines the comments made by the Reports about the effect of tobacco smoking on the occurrence of cancer. Here there is a basic agreement concerning smoking:

- (1) The effects of tobacco and asbestos are known to interact synergistically (cause an effect above and beyond separate effects) in smokers exposed to asbestos and result in an increased incidence of lung cancers.
- (2) Smoking has little or no effect on the occurrence of mesothelioma.
- (3) Smoking has little or no effect on the occurrence of asbestosis, although a smoking history may lead to pulmonary complications.

However, whether smoking and asbestos exposure have an additive or multiplicative synergy in lung cancer occurrence is not agreed upon. The NIOSH - OSHA Report claims a multiplicative relation; the HSC Report claims the relation is uncertain and may be less than multiplicative.

Latent Period

Latency, a delay or a "lag period" in the appearance of disease, is an important factor relating to the health risks of asbestos exposure. All of the diseases associated with asbestos have latency periods. There is a delay between first exposure and the onset or recognition of the disease.

Asbestosis may or may not occur during the time while a worker is exposed to asbestos. It may appear long after exposure to asbestos has ended. The interval is known to be extremely variable, a number of months up to decades - apparently depending on the degree of exposure. Lung cancer has a varying latency period ranging from ten years up to decades after first exposure. Of course, smoking as a factor may or may not complicate the latency period. For mesothelioma the latency period ranges from ten to sixty years.

The implications of the existence of latent periods have been well stated in the HSC Report.

"10 ... These delays introduce the following problems: (1) cases of asbestos related disease may occur many years after the worker has left the relevant job; (2) cases of disease occurring at present relate to conditions of work which are likely to have changed in the intervening period; (3) current trends in occurrence of asbestos related disease can give little indication of recent improvements or otherwise (say within the last decade) in working conditions; (4) a period of at least 30 years may have to elapse before it is possible to give a definite answer concerning the introduction or control of an asbestos-related hazard; (5) in persons currently developing disease, data about the relevant dust levels is likely to be poor; (6) there is a serious risk among the uninformed of attributing present disease frequencies to present conditions of work." (HSC, page 22)

Pattern of Exposure

It is recognized that the pattern of exposure, specifically the intensity and time pattern, is related to the health risk. Certain cases of mesothelioma have occurred after very brief yet intense exposures to crocidolite mixtures. (HSC, page 56). As well, similar occurrences of asbestosis have been documented.

Traditionally the exposure pattern has been averaged by using the concept of "dose", where dose is the averaged product of intensity multiplied by the period(s) of exposure. As has been discussed in Section 4, this concept has theoretical and practical difficulties.

Age

The importance of the age of the individual as an independent factor is extremely difficult to analyse. Exposure to asbestos occurs at some point in time, either as a single exposure or as a protracted one. The diseases do not occur immediately thereafter. In fact, the moment when the disease is considered to exist is never known. The moment of positive diagnosis may take place years after the existence of the disease. Latency is a further problem which affects the interpretation of the factor of age.

COMMENT by CCOHS

The Royal Commission should be concerned with heightened susceptibility of children to asbestos-associated diseases. The published instances of asbestos-related disease due to the exposure to asbestos of children are rare. (HSC, page 60). However, it is known that the susceptibility to some cancers varies with age, the very young being at risk (ionizing radiation and leukemia). Whether asbestos-related malignancies are similarly related is not known. As well, since children can be expected to live longer than adults, they have more chance of being affected by cancer-causing substances with long latent periods, of which asbestos is an important example.

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Table 1a: Smoking as a Factor in Risk

FINDINGS OF THE REPORTS
Agency Report

- 1 HSC Advisory Committee on Asbestos:
Final Report
pg. 49, 52, 55

- 2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 46, 47

- 3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 115, 112

- 4 CEC Working Group of Experts:
Public Health Risks of Exposure to Asbestos
pg. 12

ANALYSIS FOR QUESTION 4
Health Risks Related to Smoking

-Exposure to asbestos dust and tobacco probably increases the risk of lung cancer synergistically but the exact relationship is uncertain. The mention of asbestos on death certificates of people dying from lung cancer who were smokers exposed to asbestos is almost certainly deficient. It may therefore be impossible to determine to what extent a bronchial lung tumour is related to asbestos. Smoking has no effect on mesothelioma.

-Both asbestos and smoking are independently capable of increasing the risk of lung cancer mortality. When exposure to both occurs, the combined effect with respect to lung cancer appears to be multiplicative rather than additive.

-There is little evidence that smoking increases the risk of mesothelioma.

-Data on non-smokers demonstrates no great increase in lung cancer incidence due to asbestos exposure among men who did not smoke cigarettes regularly. On the other hand, asbestos exposure did increase the risk of lung cancer for cigarette smokers beyond what was expected for smoking alone.
-Smoking was not implicated in the incidence of mesothelioma.

-Cigarette smoking is a "highly contributive factor, usually in combination with existing asbestosis", in the development of lung cancer in asbestos-exposed workers.

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Section 7: Risk of Disease for Workers

Question 7 WHAT IS THE MAGNITUDE OF THE HEALTH RISK FROM ASBESTOS FOR ASBESTOS WORKERS (IN GENERAL, NOT IN ANY PARTICULAR PLANT, FIRM OR INDUSTRY)?

Some of the Reports attempted to estimate the magnitude of health risks for workers.

In a review of studies of occupational populations the HSC Report ranked the major causes of excess mortality among asbestos workers where the cause of death was known and stated the proportion of deaths due to each cause:

lung cancer	40%
asbestosis	24%
mesothelioma	13%
gastro-intestinal cancer	8%
and all other causes	15%

Surprisingly, a study of British death certificates suggested that asbestos-related mesothelioma is a more frequent cause of death than asbestos-related lung cancer (HSC, Vol 2, page 23 and Table 20)

The incidence of mesothelioma in occupationally exposed populations has been estimated to be from 2 to 90 times higher than the incidence rate in the general unexposed population.

There is a need for caution in the interpretation of instances such as these because of the effects of the various factors which could affect the picture:

- latent periods
- changes in work conditions
- changes in measurement procedures
- the varying evaluations of occurrence
- (for example, diagnosis)

The NIOSH-OSHA Report made no statements concerning estimates of occurrence, but does state that the present standard (2 fibres/mL) appears to cause unacceptable levels of incidence and prevalence. It then recommends a considerable reduction below the current fibre standard.

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Table 17: Risk of Disease for Workers

FINDINGS OF THE REPORTS
Agency Report

- 1 HSC Advisory Committee on Asbestos:
Final Report
pg. 23, Vol. 2 & Table II

- 2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos
pg. 4, 6

- 3 NRCC Associate Committee on...Criteria:
Effects of Asbestos
pg. 54

- 4 CEC Working Group of Experts:
Public Health Risks of
Exposure to Asbestos

ANALYSIS FOR QUESTION 7
Risk of Disease for Workers

-Excess mortality in industrial populations working with asbestos:
40% by lung cancers
24% by asbestosis
13% by mesothelioma
8% by gastro-intestinal cancer
-British death certificates indicate mesothelioma more frequent than (other) asbestos-related cancers.

-A variety of factors demonstrate the current standard is grossly inadequate to protect workers from disease.
-Disease prevalence data...collected subsequent to 1969 suggest that this standard (2 fibres/mL) is insufficient to prevent a large incidence of asbestosis. All levels of asbestos exposure have demonstrated asbestos-related disease. Compelling evidence demonstrates that prevention of asbestos-related diseases requires that an occupational standard minimize all asbestos exposures and definitely be set far below the current (2 fibres/mL) standard.

-Not possible to specify an asbestos concentration associated with no measureable effect on health.

NO COMMENT MADE

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Section 8: Risk of Disease for the Public

Question 8 WHAT IS THE MAGNITUDE OF THE HEALTH RISK FROM ASBESTOS FOR THE GENERAL POPULATION (EXCLUDING ASBESTOS WORKERS AND THOSE WHO LIVE IN THE IMMEDIATE VICINITY OF ASBESTOS MINES OR MILLS)?

From the Reports, it is clear that the magnitude of risks of disease from asbestos for the general population is not known with any certainty. Actual instances of the diseases associated with environmental asbestos exposure are rare although there have been occurrences in situations of para-occupational and neighbourhood exposures.

It is generally accepted that asbestosis occurs only in occupationally exposed groups, although pleural plaques similar to those associated with asbestosis have occurred where occupationally related exposure is unlikely. But these pleural changes are only symptoms, or abnormalities, and not firm evidence of disease.

The HSC Report states that extrapolation from the industrial experience for asbestosis to the public is not possible. There has been no general Reporting of asbestosis in the public.

Lung cancer risks, with the problem of the confounding risk factor of cigarette smoking, as discussed earlier, are not so easily presented. The HSC Report states that chrysotile is unlikely to have produced any increase in the risk of lung cancer in the populace. However, for the amphiboles, crocidolite and amosite, the situation is more doubtful than for chrysotile. There is a suggestion in the data that the risk of lung cancer may be greater for the amphiboles than for chrysotile, and that an excess lung cancer risk cannot be excluded in those exposed in buildings to amphiboles only.

For mesothelioma, chrysotile is believed to cause no appreciable number of cases. The amphiboles are most clearly associated with the majority of Reported mesotheliomas. This situation is similar to that for lung cancer.

The NIOSH-OSHA Report does not discuss public health risks; it only discusses occupational risks. However, because of the nature of the statements made, certain conclusions can be deduced.

"Excessive cancer risks, however, have been demonstrated at all fibre concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a 'safe' level of asbestos exposures." (NIOSH-OSHA, page 4)

It can be reasoned that if there is no "safe" level of exposure, then exposures to environmental concentrations must have some risk attached, whether or not measurements of health effects can be made. However, the NIOSH-OSHA Report does not use any extrapolations to estimate public health risks. Yet it does selectively quote the HSC Report concerning its own use of the linear extrapolations from industrial data to the non-industrial (HSC, paragraph 15, page 38).

The CEC Report uses the occupational data to evaluate possible public health risks. It concludes that:

- asbestosis is not a public health risk
- mesothelioma is not convincingly related to public exposures
- lung cancer cannot be excluded as a public health risk caused by asbestos exposure.

Table 18. Risk in the Populace - LUNG CANCER

FINDINGS OF THE REPORTS
Agency Report

1 HSC Advisory Committee on Asbestos:
Final Report
(pg. 59-60)

ANALYSES FOR QUESTION 8
Risk of Lung Cancer in the Populace

- The presence of chrysotile containing small quantities of amphiboles is unlikely to have produced any material increase in the risk of lung cancer in the general population.
- For amphibole rich mixtures...what data exists suggests that the risk of lung cancer associated with amphiboles may be greater than that for chrysotile.
- An excess of lung cancer risk cannot be completely excluded...in those who have been exposed only to amphiboles in buildings.

2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos

NO COMMENT MADE

3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...

NO COMMENT MADE

4 CRC Working Group of Experts:
Public Health Risks of
Exposure to Asbestos
pg. 12, 96

- There is not yet enough evidence to exclude the risk of environmentally related induced bronchial lung carcinoma as a public health risk.

Table 1b. Risk in the populace - MESOTHELIOMA

FINDINGS OF THE REPORTS
Agency Report

ANALYSIS FOR QUESTION 8
Risk of Mesothelioma in the Populace

1 HSC Advisory Committee on Asbestos:
Final Report
pg. 59

-The presence of chrysotile containing small quantities of amphiboles is unlikely to have produced any ... appreciable number of cases of mesothelioma.

2 NIOSH-OSHA Asbestos Work Group:
Workplace Exposure to Asbestos

NO COMMENT MADE

3 NRCC Associate Committee on...Criteria:
Effects of Asbestos...
pg. 106

-A higher incidence of mesothelioma in urban than in rural populations has been observed, possibly due to the greater likelihood that asbestos is present in city air.

4 CEC Working Group of Experts:
Public Health Risks of Exposure to Asbestos
pg. 12, 93

-There is no convincing evidence yet that "true" environmental (excluding para-occupational) exposure is a contributive factor to the occurrence of mesothelioma through respiratory exposure.

-It seems unlikely that asbestos in the general atmosphere is a cause of mesothelioma in the general public, (though) more evidence is needed

Section 9: Chronology of Knowledge

Question 9 WHAT WAS THE CHRONOLOGY OF UNDERSTANDING THE HEALTH RISKS OF ASBESTOS EXPOSURE BY THE HEALTH RESEARCHERS?

Asbestos has been used a long time. As early as the 1st century AD the Greek geographer Strabo reported that slaves who wove asbestos into prized ropes and burial garments for nobility suffered from a "breathing sickness". (1)* In 12th century Europe, Charlemagne's banquet guests were treated to amazing displays of fire-defying tricks performed with asbestos cloths. Now eight hundred years later it is harder to be innocently enchanted with the unusual powers of what was once called "the magic mineral".

Scientists and medical researchers now ponder the complexities of the pathological nature of the mineral. How does it cause disease? Who is likely to become a victim? How much dust can harm a person exposed over a lifetime of work? how little? Are all types of asbestos equally dangerous? How can we use our technological know-how to control exposure to the dust?

Asbestos has been studied intensively by medical and occupational health researchers. A recent estimate made by CCOHS staff says that one new article on asbestos now appears every day in the medical literature, on average.

For over a century asbestos has been mined and processed for hundreds of different industrial uses. When workers handling the mineral developed fatal illnesses in the early years of this century, factory inspectors and medical doctors began to make the connection between asbestos dust and disease. Asbestos factory owners were among the first to realize the danger. Henry Ward Johns, founder of what became the Johns-manville Corporation, died of asbestosis in 1898.

As early as 1907 at least one French factory inspector and one British medical doctor were convinced that asbestos was the cause of death for certain textile workers. (2,3) Twenty years later another British doctor recognized that disease produced by working in asbestos dust created a previously unknown type of lung fibrosis — he labelled it "asbestosis"(4).

In the early 1930s it was found that asbestos-related disease could strike many factory workers, not just an unfortunate few — as many as one-third of the workers in a British plant were affected.(5) At the same time doctors at an American clinic found, during autopsy, that a death apparently due to heart failure was actually due to the suffocating effects of asbestos dust (6).

* Bracketed numbers refer to items numbered in Appendix C.

In 1931 it was known that an exposure period as short as nine months could produce an asbestosis.(7) People who used asbestos products — pipe coverers, boiler maintenance men and even a clerical worker — developed asbestosis; it was not limited to factory employees only. That asbestosis and lung cancer could occur together in the same victim was known by 1934 (8); several cases were reported by British and American researchers in the mid 1930s.(9,11,13) During this decade, confusion over the diagnosis of tuberculosis in the asbestos-producing region of Quebec became an issue.(10,12,18)

By the late 1930s and early 1940s researchers were preoccupied with questions about the link between lung cancer and asbestos exposure.(13,14,15,16,17) Attempts to induce cancers experimentally in mice were not conclusive.(14) Numerous cases of asbestosis and lung cancers occurring together had been documented by the mid 1940s by American, British and German investigators.(19) A consensus on the existence of a causal relation between asbestosis and lung cancer was apparently reached in 1949 after a landmark study of British asbestosis victims who reported for compensation.(20) American researchers in the 1950s, however, found reasons to conclude that asbestos was not responsible for causing lung cancer.(21)

A major study of English textile employees in the mid 1950s was widely recognized as offering the most solid evidence to date that asbestos-exposed workers ran a high risk of being afflicted with lung cancer as well as asbestosis.(22)

Epidemiology, a quantitative analysis of disease distribution and causation, became increasingly popular as a research tool in the 1950s. Studies which did not include study populations or numbers of victims considered to be statistically large enough were discredited.(23) In fact the previously accepted connection between lung cancer and asbestos exposure was refuted in 1961 by prominent American epidemiologists.(24)

Some evidence appeared irrefutable, however. It became generally known by the mid 1960s, for example, that insulation work with asbestos was especially dangerous, and that it caused high excess death rates and gastro-intestinal cancer rates — this from studies in Denmark and the United States.(25,27) And by 1965, it was thought that people could develop early signs of lung disease from asbestos air pollution simply because they lived in the neighbourhood of an asbestos mine(26), and that both adults and children could become victims of mesothelioma from contact with occupationally-exposed family members (28,29).

A subject for investigation in the late 1960s and early 1970s was the question whether or not abnormalities on an x-ray film could signal the presence of asbestos-related disease — some researchers uncovered disease where x-ray evidence revealed none (30,31). Nevertheless, examination of x-ray abnormalities by industry personnel in a British factory formed the basis in 1968 for the later universally-applied 2 fibres per cubic centimetre of air standard of asbestos exposure.(32)

Research on asbestos in the 1970s became more intensive, knowledge about exposure measurement and the nature of asbestos-related disease more precise. Numerous important assertions were made in the medical literature of the decade — some may no longer be relevant, some have become generally agreed upon and some are still the subject of controversy. Among these assertions are:

- Even low exposures of asbestos can cause cancer. The two fibres per cubic centimetre standard adopted to protect British and American workers does not eliminate the risk of cancer.
- Lung cancer may develop independently of asbestosis, but workers with asbestosis are at an increased risk of developing lung cancer.
- Depending on the medical criteria chosen for diagnosis, the extent of disease reported can vary greatly, especially for mesothelioma.
- Gastro-intestinal cancer is associated with high dust exposures.
- The question of whether or not the different types of asbestos are equally dangerous for all related diseases is recognized as a difficult one, although a definite link is suspected between blue asbestos (crocidolite) and mesothelioma.
- Averaging asbestos dust exposure counts over many years is an unreliable indicator of how dangerous the exposure has been, since short but heavy "doses" may carry particular risk of malignancy.
- A lack of definite distinctions between possible sources of asbestos exposure means that greater reliance should be put on biological monitoring than on environmental monitoring.
- The relation between biological effect and fibre size is central to the hazard of asbestos. Therefore reporting the fibre dimension as well as concentration is necessary.
- Exact dose-response relations cannot be defined; however, there is often a connection between the duration and intensity of exposure and resulting illness.
- Exposure to both asbestos and tobacco smoke causes a greatly increased risk of lung cancer, possibly in a multiplicative fashion.
- There is no evidence that tobacco smoke is related to an increased risk of mesothelioma.
- The time from initial exposure to eventual onset of disease — the latency period — can be a surprisingly long period of time for diseases caused by asbestos; it may be anywhere from 10 to 40 years. This fact can greatly complicate diagnosis.
- Workers having a long employment period have a higher proportion of death due to mesothelioma than those with a short period.

It should be understood that the brief discussion here and the body of the chronology in Appendix C represent only a selection of what can be known about asbestos from the medical literature. It must be stated that the Appendix C Chronology of Knowledge in no way attempts to be comprehensive in scope. Within the limitations of this type of survey, we have tried to indicate some of the important areas of both consensus and controversy which have characterized this field of research. In a few instances it was necessary to include entries from sources other than the four Reports, to fill gaps in knowledge, especially for the period 190-1960. A chronology of knowledge of the hazard of asbestos as it can be found in non-medical publications, including an awareness of legal, social, and economic circumstances, has been prepared by CCOHS. It may provide a useful complement to this study.

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Section 10 Response to the Reports

Questions:

WHAT RECEPTION HAVE THE FOUR REPORTS BEEN ACCORDED IN CRITICAL REVIEWS IN THE MEDICAL JOURNALS?

HAVE THERE BEEN FORMAL RESPONSES FROM AFFECTED PARTIES SUCH AS INDUSTRY ORGANIZATIONS OR WORKERS ORGANIZATIONS?

WHAT WOULD BE A FAIR INDICATION OF THE STATUS AND WEIGHT ACCORDED TO THESE REPORTS IN THE MEDICAL COMMUNITY AND AMONG AFFECTED PARTIES?

In general there has been very little response to the four Reports of a formal nature in the medical journals, or in any other publications, as far as we have been able to determine. Most of the comment available is about the Report to the UK Health and Safety Commission (HSC). Where definite responses to the Reports were available in published form, we relied on them, but where it was not available, we have reported opinions conveyed to us by persons with some awareness of the how the Reports were received.

A book review in the British Journal of Industrial Medicine (1980:37) says the HSC Report is "well-presented, comprehensive, and thoughtful, and repays careful reading." Dr. M-L Newhouse, the reviewer, credits the HSC Report with providing "very succinct information" on asbestos in the workplace, and says it supports a recommendation on control limits with "very detailed argument". The one criticism Newhouse makes in her review is — "...it is not too clear what is meant by the 'social costs' referred to in the HSC recommendation that the Health and Safety Executive take into account the social costs of obtaining, using and disposing of asbestos...". On the whole the BJIM review presents a factual account of what the HSC Report says; it offers very little critical comment.

Both the BJIM review and an article in the journal Lancet (Oct. 1979) anticipate criticism from industry over the new restrictions for amosite (brown asbestos) recommended by the HSC Report. The Lancet article speculates that the HSC Report is likely to be criticized by environmentalists who regard prohibition as the only acceptable solution to asbestos-related disease, and it regrets that a consideration of the benefits of asbestos to health "was unfortunately not within the committee's terms of reference."

An article in the publication Health and Safety at Work (Jan. 1980) reviews the topics of discussion at a major public conference on asbestos sponsored by the Advisory Committee on Asbestos (ACA) in London in November 1979, one month after publication of the HSC report. The major theme of the conference may provide a clue to the weight attached to recommendations of the HSC Report — the theme turned out to be not the numerical value of the new control limits, but how existing atmospheric monitoring and fibre computing techniques could be used to help achieve compliance with them.

At the conference the chairman of the HSC advisory committee, Bill Simpson, "stressed the report's significance as a pointer to the way the HSC was preparing to tackle all other occupational carcinogens". He stated that three proposals should be seen as part of a general policy: that users should be obliged to consider substitutes, that the TLV concept should be replaced by control limits, and that control limits should be seen as maximum acceptable exposure levels.

Willred Penny, Director of the Asbestos Information Centre, presented to the same conference the industry's updated estimates of cost of compliance implications of the HSC Report — considerable reductions in what had originally been stated were shown.

Another Health and Safety at Work article says the HSC Report's discussion on substitutes for asbestos are "so extensive that industry is concerned that recommendations will be misinterpreted as a mandatory requirement to substitute. It also sees the burden of decision placed downstream as "unreasonable and unenforceable". The article goes on to say: "The second industry complaint is of inadequate medical evidence to support the setting of a 0.5 fibre/mL control limit for amosite... industry claims that production of the best fire protection boards will cease and two factories close following implementation of a 0.5 fibre standard." Simpson's reply, reported in the Health and Safety at Work article, was that there was a clear medical case for the amosite standard being set lower than that for chrysotile, although admittedly it was based on very limited epidemiology.

The main concern of industry, according to the same article, is the 1 fibre chrysotile standard which the Advisory Committee on Asbestos proposed should be achieved by December 1980. The asbestos industry in Britain complains of being at a competitive disadvantage compared to other European Economic Community (EEC) producers who have several years longer to adopt stricter control levels.

The British magazine New Scientist published three articles about the HSC Report soon after it was released. In one editorial, the HSC Report is described as an "historic document", not because it recommends stricter safety standards, but because of the maximum "control limit" philosophy behind its suggestions. Author L McGinty says the thinking behind the new safety standards for asbestos recommended in the HSC Report "will almost certainly dominate the setting of future standards for all cancer-causing substances in the workplace." He says this thinking is "new" and that it "closely reflects the philosophy of safety standards for radiation set out by the International Commission on Radiation Protection." McGinty criticizes the HSC Report for ignoring the question of very small asbestos fibres, which some researchers believe to be more dangerous than larger ones. He anticipates trade union dissatisfaction with the Report's recommended control limit for chrysotile, which he says would still allow 30 of the estimated 25,000 workers currently exposed to die from asbestos-caused disease.

The Trades Union Congress (TUC) had demanded in its evidence to the HSC before the Report's publication an overall standard of 0.1 fibre/mL, and a policy of "gradual compulsory substitution" of asbestos with less dangerous materials. McGinty says the HSC Report recommendations fall far short of substitution measures acceptable to the TUC.

A British newspaper reporting on safety matters to industry carried an article in the October 1979 issue, which quoted a consultant's criticism of the HSC Report; he said the recommendations were "more of a sop to the emotional environment than scientifically-determined figures", and "The figures are a compromise, with no scientific basis".

Although direct comment on the HSC Report from industry and labour representatives was not readily available, a fairly consistent sense of their respective positions was obtained.

With respect to the NIOSH-OSHA Report, it would be safe to conclude that its publication has been received as somewhat less significant by the parties concerned than was the HSC Report. No critical comment in the medical literature has appeared, as far as we can tell. However, some indications of response are available.

The Occupational Safety and Health Reporter (May 1980, p.1108) carried a news item which quotes the Asbestos Information Association (AIA) of North America as saying that the NIOSH-OSHA Report is "a short-sighted regulatory approach" that fails to acknowledge that "societally acceptable levels of risk can be established." The association charged that the Report was not based on any scientific demonstration of the healthful effects of reducing exposure to 0.1 fibres, but instead on NIOSH's assertion that the 0.1 fibre level is the lowest level accurately measureable by optical microscopy. The association also criticized OSHA and NIOSH for using the concept of dose-response relationship in the development of disease but failing to acknowledge the "correlative conclusion" that acceptable levels of risk can be set.

A prominent American epidemiologist who has published several articles in the medical literature on asbestos was contacted for his knowledge of how the NIOSH-OSHA Report was received. His response was revealing — "I vaguely remember that report." The same kind of reaction was given by one of the most influential American labour representatives, but it was clear he was pleased with the Report's call for a lowered standard of exposure to asbestos — "We've been petitioning for this for the last five years...we're all for it." One of the medical employees at NIOSH said he thought members of the medical community were generally supportive of the Report, because "they recognize that asbestos is a huge problem and they are seeing more and more evidence of disease". He said he had to agree with AIA president T.A. Dougherty that the 0.1 fibre level was chosen by NIOSH because it was the lowest level accurately measureable by optical microscopy.

US health and safety government officials announced release of the NIOSH-OSHA Report together with statements about the numbers of workers who might be afflicted with asbestos-related disease: "...restrictions on asbestos in the workplace are 'grossly inadequate' to protect 1.4 million American workers" — "an estimated 58,000 to 75,000 Americans die each year from lung cancer, asbestosis, mesothelioma and other malignancies". The statements were reported in a New York Times article the day the Report was released (April 18/80), but nothing remarkable was said about the document itself.

The Report of the National Research Council of Canada (NRCC) "seems to have been accepted at face value", according to one of the members of the NRCC Associate Committee on Scientific Criteria. Another NRCC official said the Report received "very little publicity"; "we seldom get hallelaloo afterwards", he said. He explained that since the Report went through a "multi-stage review", a question about reaction to it after publication was not applicable. The main author of the NRCC Report was not aware of any particular response to it, partly because he was no longer with the NRCC, but also because no apparent official reactions have been made strongly or openly. No comment or criticism about the NRCC Report, from either industry or labour organizations, was found.

Last, the Zielhuis report, referred to in this survey as the CEC Report. This document was designed to be an examination of the public health risks associated with asbestos. There is evidence in the Newhouse review and in status accorded to it by British researchers especially, that the CEC Report is regarded as a major study of one important aspect of the asbestos problem. Its existence is little known in North America. Unfortunately, it was not possible to get any indication of European response to the CEC Report, beyond the fact that the study of health effects in volume 2 of the HSC Report references it as one of "four recent excellent reviews".

Published Comment on the Reports

M L Newhouse, Book Reviews. British Journal of Industrial Medicine, 1980, 37: 198-20

Asbestos and Health. The Lancet, October 27, 1979, p.887

Asbestos - putting the legislation into practice. Health and Safety at Work, January 1980, p.18

HSC carcinogens policy developed in Simpson report. Health and Safety at Work, January 1980, p. 15

Tighter controls on asbestos recommended. 13 Sept. 1979, p.787

Controlling cancer - asbestos shows the way. L McGinty, 25 Oct. 1979, p.251

A safe philosophy. L McGinty, 25 Oct. 1979, p.250: New Scientist

Asbestos: Commission Plans Tough New Curd. Safety incorporating safety and Rescue, October 1979, p.1.

OSHA-NIOSH Report "Short-Sighted" New Standard Unjustified, Industry Says. Occupational Health and Safety Reporter, 1 May 1980, p.146

Asbestos Industry Finds No Justification for Plan to Cut Allowable Exposures. Job Safety and Health Reporter, May 1980

Federal Curd on Asbestos Found "Grossly Inadequate". New York Times, 18 April 1980

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APPENDICES

Appendix A Extract from the Terms of Reference

Appendix B Letter of Dr David Muir

Appendix C Chronology of Knowledge of Asbestos

APPENDIX A : Extract From Terms of Reference

A Review of Major Studies of the Health Effects of Asbestos

(A Survey of Studies)

PURPOSE

The purpose of this survey is to review the major studies of the health effects of asbestos, identifying the views taken in those four studies on a specific set of questions.

The report is not intended to be an examination of the entire problem of asbestos exposure and control, but only of the health effects at various levels of exposure.

This survey should constitute a summary that is intelligible to the general public and conveys to that public a sense of the views expressed in these major studies.

In addition, this survey should review the reaction, if any, to those studies by the scientific community, by industry, by labour, and by the general public.

Out of this should come some sense of which aspects of the conclusions of the studies have been controversial and which have been generally accepted.

If the CCOHS can contribute to the resolution of controversial issues with their own views, this would be useful.

This survey is limited to an examination of a specified set of four previous studies in order to permit its completion in the time period of three months.

It is anticipated that some of the questions will not be answered completely or satisfactorily in the specified studies. In such cases, the CCOHS should indicate that the studies do not present a satisfactory answer, and that further research, examining primary literature sources would be necessary to find an answer, if the current state of medical research admits an answer at all. At some later date, the Royal Commission on Asbestos may commission a study that would examine in detail any questions left unsolved by this survey, and any additional health questions that might arise.

The Royal Commission on Asbestos may publish the report that results from this survey and distribute it in substantial quantities to members of the general public and to interested groups concerned with public exposure to asbestos. The document may then serve to provide common background information to individuals and groups interested in the problem of public exposure to asbestos.

SCOPE OF SURVEY

The survey should review the major overview studies of the asbestos problem. This will include:

- (1) Advisory Committee on Asbestos, Final Report, Report to the Health and Safety Commission, 1979, HMSO London. (referred to as the HSC Report)
- (2) NIOSH-OSHA Asbestos Work Group, Workplace Exposure to Asbestos: Review and Recommendations, April, 1984, U.S. Dept. of Health, Education and Welfare, U.S. Dept. of Labor. (referred to as the NIOSH-OSHA Report)
- (3) National Research Council of Canada, Associate Committee on Scientific Criteria for Environmental Quality, Effects of Asbestos in the Canadian Environment by Stephen Shugar, 1979, NRCC 16452. (referred to as the NRCC Report)
- (4) Report of a Working Group of Experts Prepared for the Commission of the European Communities, Public Health Risks of the Exposure to Asbestos, ed. by Prof. K.L. Zielhuis, EUC 5653e 1977, Pergamon Press. (referred to as the CEC Report)

A number of particular questions should be addressed in reviewing the major studies. The CCOHS should summarize what the major studies say about:

- (1) What adverse health effects in humans are caused by exposure to asbestos?
- (2) What is the rate of occurrence of these health effects in those who work with asbestos and in the general public?
- (3) Do the major types of asbestos differ in their toxicity? If so, how?
- (4) What is the shape of the dose-response relationship? Is it linear, or some other shape?
- (5) Is there a safe threshold for exposure to asbestos? (By disease, by type of asbestos?) If so, what is it?
- (6) How does the health risk relate to the age of the exposed person? How is it affected by other factors such as smoking?
- (7) What is the magnitude of the health risk from asbestos for asbestos workers? (In general — not in any particular plant, firm or industry.)
- (8) What is the magnitude of the health risk from asbestos for the general population (excluding asbestos workers and those who live in the immediate vicinity of asbestos mines or mills)?
- (9) What was the chronology of the understanding of health risks from asbestos exposure by the health researchers?



APPENDIX B: COMMENTS ON DIFFERENCES IN TOXICITY BY DR. HUIR
OCCUPATIONAL HEALTH PROGRAM
McMASTER UNIVERSITY
1200 Main Street West, Hamilton, Ontario, L8S 4L9
Telephone: Area Code 416 525-9140, Ext. 2333, 2334

September 9, 1980

Dr. G.R.C. Atherley
President
Canadian Centre for Occupational
Health and Safety
1200 Main Street West
Hamilton, Ontario
L8N 3Z5

Dear Dr. Atherley:

SUBJECT: Relative hazards of different types of Asbestos

You have asked me to make some comments on the approach to this problem as it appears in the following recently published documents.

1. Effects of Asbestos in the Canadian Environment -
Associate Committee on Scientific Criteria for
Environmental Quality published by the National
Research Council of Canada.
2. Asbestos - Volume 2: final report of the Advisory
Committee published by the Health and Safety
Commission
3. Workplace Exposure to Asbestos: Review and
Recommendations published by NIOSH-OSHA, April
1980.

In approaching this problem, it is helpful to remember the experience of the coal mining industry in Britain. In 1945, it was well known that most cases of coalworkers pneumoconiosis in Britain occurred in the South Wales area. This was known to be a mining area predominantly dealing with high rank metallurgical coal. From this observation it was assumed that high rank coal was more dangerous than low rank coal. Comparison of the dustiness of the air in these pits by methods used at that time appeared to show that they were no more dusty than pits in Scotland where coalworker's pneumoconiosis was very uncommon.

This remains one of the great epidemiological blunders in the field of occupational health. We now know that two factors contributed to this state of affairs. In the first place the mining techniques used in high rank pits was such that a great deal more dust was in fact generated than was present in the low rank pits of Scotland. This has got nothing to do with the type of coal but simply with the mining

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1st Century A.D.

PLINY The Elder confirms reports by the Greek geographer, Strabo, that slaves who wove asbestos into burial garments suffered from a sickness of the lungs.

Levine, R.U. ASBESTOS: AN INFORMATION RESOURCE. Cincinnati: U.S. Dept. of Health, Education and Welfare, 1978.

[WKK-NRCC] (1)

1906

AURIBAUT, an inspector in the Department of Labour in France, links 50 deaths to exposure to asbestos dusts in a weaving mill at Caen.

Auribault, M. Note sur l'hygiene et la securite des ouvriers dans les filateurs et tissages d'amiant. Bull Insp. Trav., Paris 14, 126 (1906)

[WKK-OTH] (2)

1907

MURRAY in England reports the death of a patient from asbestos fibrosis of the lungs to a parliamentary committee on industrial disease compensation. The victim is a 33-year-old man who had worked for ten years in the carding room of an asbestos textile mill.

Murray, H.M. REPORT OF THE DEPARTMENTAL COMMITTEE ON COMPENSATION FOR INDUSTRIAL DISEASE: MINUTES OF EVIDENCE. London: Wyman and Sons, 1907.

[WKK-OTH] (3)

1917

PANCOAST et al reports x-ray appearance of pneumoconiosis of fifteen individuals exposed to asbestos.

Pancoast, H.K., [et al.] "A Roentgenologic study of the effects of dust inhalation upon the lungs." ASSOCIATION OF AMERICAN PHYSICIANS, 32 (1907): 97-108.

[WKK-OTH]

006145

1927

Dr. W.E. COOKE reports in full pathological detail the findings of a post-mortem examination on a middle-aged woman who developed asbestosis during 20 years of work in an asbestos-textile factory. "We have never seen anything parallel to this in pneumoconiosis due to other dusts..." he says. He named the disease asbestosis. Cooke, W.E. "Pulmonary asbestosis." BRITISH MEDICAL JOURNAL, (December 3, 1927): 1024-1025.

[WKK-NRCC] (4)

1930

MEREWETHER and PRICE find about one-third of a total population of asbestos factory workers suffering from pulmonary (lung) fibrosis. Merewether, E.H.A. and G.W. Price. REPORT ON EFFECTS OF ASBESTOS DUST ON THE LUNGS. London: H.M.S.O., 1930.

[WKK-UTH] (5)

1930

MILLS authors a report at the Mayo Clinic where an autopsy on a cardiac victim reveals heart failure due to asbestosis. Mills, R.G. "Pulmonary asbestosis: report of a case." MINNESOTA MEDICINE, 13 (1930).

[WKK-UTH] (6)

1930s

Lung cancer is still a relatively uncommon disease. Newhouse, M. "Asbestos in the workplace and the community." ANNALS OF OCCUPATIONAL HYGIENE, 16 (1973): 97-107.

[WKK-HSC]

1931

STEWART et al find asbestosis in factory workers including one victim with only nine months' exposure. (Arch. Path., vol. 12)

[WKK-UTH] (7)

006146

1932

Asbestosis is not limited to factory workers. Dr. Albert RUSSELL of the U.S. Public Health Service reports that a pipecoverer has developed asbestosis during maintenance work on a hospital heating plant. (Proceedings of a Conference Concerning Effects of Dusts Upon the Respiratory System, Nov. 16-17, 1932, p. 180)

[WKR-OTH]

1934

WOOD and GLOYNE report two cases of lung cancer seen at autopsy in 43 cases of asbestosis.

Wood, W. B. and S. K. Gloyne. "Pulmonary asbestosis: a review of one hundred cases." LANCET, 2 (1934): 1383.

[WKR-HSC] (8)

1935

LYNCH and SMITH report one case lung cancer in a South Carolina (USA) asbestosis victim. Lynch, K. M. and W. A. Smith. "Pulmonary asbestosis III: carcinoma of the lung in asbestos-silicosis." AMERICAN JOURNAL OF CANCER, 24 (1935): 56.

[WKR-HSC] (9)

1935

LANZA et al conclude that asbestos dust exposure does not predispose to tuberculosis. This study also describes asbestosis as a milder disease than silicosis. Lanza, A. J. "Effects of the inhalation of asbestos dust on the lungs of asbestos workers." PUBLIC HEALTH REPORTS, 50, 1 (January 4, 1935).

[WKR-OTH] (10)

1936

Two more cases of lung cancer associated with asbestosis are reported.

EGBERT and GEIGER

Egbert, D.S. and A.J. Geiger. "Pulmonary asbestos and carcinoma: report of a case with necropsy findings." **AMERICAN REVIEW OF TUBERCULOSIS AND PULMONARY DISEASES**, 34 (1936): 143.

GLOYNE

Gloyne, S.R. "A Case of oat-cell carcinoma of the lung occurring in asbestosis." **TUBERCLE**, 18 (1936): 111.

[WKR-HSC] (11)

1936

DONNELLY finds x-ray evidence of asbestosis among 34% of 151 asbestos mill workers in Quebec. No cases of active tuberculosis are found among workers with asbestosis. He concludes that there was no tendency in this type of occupation to exacerbate old tuberculosis lesions.

Donnelly, J. "Pulmonary asbestosis: incidence and prognosis." **JOURNAL OF INDUSTRIAL HYGIENE AND TOXICOLOGY**, 18 (1936): 4.

[WKR-OIH] (12)

1938

NORDMANN finds 2 cases of lung cancer in 2 cases of asbestosis and concludes a cause-effect correlation must be present.

Nordmann, M. "Der Berufskrebs der Asbestarbeiter." **ZEITSCHRIFT FUR KREBSFORSCHUNG UND KLINISCHE ONKOLOGIE**, 47 (1938): 288.

[WKR-HSC] (13)

1939

VORWALD and **KARR** report negative results in trying to show experimentally induced cancer with asbestos.

Vorwald, A.J. and J.W. Karr. "Pneumoconiosis and pulmonary carcinoma." **AMERICAN JOURNAL OF PATHOLOGY**, 14 (1938): 49-57.

[EXP-HSC] (14)

1941

NORDMANN and SORGE report lung cancers in 2 white mice resulting from asbestos dust inhalation.
Nordmann, M. and A. Sorge. "Lungenkrebs durch Asbeststaub im Tierversuch."
ZEITSCHRIFT FÜR KREBSFORSCHUNG UND KLINISCHE ONKOLOGIE, 51 (1941): 168.
[EXP-HSC] (15)

1943

HEUPER concludes that the accumulated literature on asbestosis definitely established a carcinogenic risk.
Heuper, W. C. "Cancer and its relation to occupation and environment." BULLETIN OF THE AMERICAN SOCIETY FOR CONTROL OF CANCER, 25 (1943): 63.
[WKK-HSC]

1943

HOMBURGER lists a total of 22 cases of lung cancer in asbestosis victims, and although he noted a high association, says there is still no definite answer as to whether pulmonary asbestosis was a causative factor in lung cancer.
Homburger, F. "The Coincidence of primary carcinoma of the lungs and pulmonary asbestosis: analysis of literature."
AMERICAN JOURNAL OF PATHOLOGY, 19 (1943): 797.
[WKK-OTH] (16)

1943-51

Studies by WELDER, MEREWETHER and GLOYNE show that an average 14.7% of workers with asbestosis also had cancer of the lung.
Welder, H. W. "Über den Lungenkrebs bei Asbestose." DEUTSCHES ARCHIV FÜR KLINISCHE MEDIZIN, 191 (1943): 189.
Merewether, E. R. A. ANNUAL REPORT OF THE CHIEF INSPECTOR OF FACTORIES FOR THE YEAR 1947. London: H.M.S.O., 1949.
Gloyne, S. R. "Pneumoconiosis: a histological survey of necropsy material in 1205 cases." LANCET, 1 (1951): 810.
[WKK-OTH] (17)

006149

1944

The Tuberculosis - Asbestosis Survey conducted by Metropolitan Life Insurance Co. under the directorship of DR. G. WHEATLEY, finds a tuberculosis mortality increase in Thetford Mines, Quebec, from the mid 1920's to the early 1940's to be more than 80%. This in contrast to a significant decline (35%) in the rate for the province as a whole. In his report WHEATLEY says: "The obvious existence of a serious tuberculosis problem in Thetford Mines has led to speculation regarding the influence of asbestos dust".
Wheatley, G. TUBERCULOSIS AND ASBESTOSIS: MORTALITY IN THE ASBESTOS MINING COMMUNITY OF THETFORD MINES, QUEBEC. New York: Metropolitan Life Insurance Co., 1944.
[WKR/P-U-OTH] (18)

1948

HEUPER writes that since 1935 a total of 23 cases of asbestos cancer of the lung have been reported by American, English and German investigators.
He reports that despite a large number of workers exposed to asbestos in the U.S. (over 4,000), the incidence of asbestosis recorded was low. This information contrasts strongly with German research by BOEHME, who found radiological evidence of asbestosis in 29% of 132 asbestos factory workers. Heuper, W.C. "Environmental and occupational cancer." PUBLIC HEALTH REPORTS, supp. 209 (1948).
[WKR-OTH] (19)

006150

1949

MEREWETHER compares lung and pleural (membrane lining of the lung) cancer in cases of asbestosis reported for compensation with lung or pleural cancer in cases of silicosis. ENTERLINE (1978) sees this as the most important single study in bringing about a consensus on the relationship between asbestosis and lung cancer in Great Britain. Merewether, E.R.A. ANNUAL REPORT OF THE CHIEF INSPECTOR OF FACTORIES FOR THE YEAR 1947. London: H.M.S.O., 1949.

Enterline, P.E. "Asbestos and cancer: the international lag." AMERICAN REVIEW OF RESPIRATORY DISEASE, 118 (1978).

[WKK-HSC] (20)

1952

SMITH reviews scientific work with asbestosis and animals, concluding that the study by Nordmann and Sorge seems to demonstrate that asbestosis did not provoke lung tumours. Smith, W.E. "Lung cancer with special reference to experimental aspects." ARCHIVES OF THE INDUSTRIAL HYGIENE AND OCCUPATIONAL MEDICINE, 5 (1952): 209.

SMITH'S views on Nordmann's work gains some acceptance outside Germany. (U.S. Public Health Service, pub. no. 152, p. 35-38 (1955))

(Arch. Ind. Health, 15, 207 (1957))

[EXP-HSC] (21)

1953

DR. P. CARTIER, in charge of the Quebec asbestos companies' medical clinic from 1940 to 1974, explains to a medical symposium in Boston: "Asbestosis remains a disease which can be tolerated for many years".

Cartier, P. "Some clinical observations of asbestosis in mine and mill workers."

ARCHIVES OF INDUSTRIAL HEALTH, 55 (1955): 204-207.

[WKK-UTH]

1954
BRESLOW's epidemiological survey cites asbestos as a cause of lung cancer and called for further "intensive investigation".
Breslow, L., [et al.]. "Occupations and cigarette smoking as factors in lung cancer." AMERICAN JOURNAL OF PUBLIC HEALTH, 44 (February 1954): 149-296.
[WKK-OTH]

1955
DOLL studies northern England textile factory workers with at least 20 years of exposure to asbestos. He finds a ten-fold increase in the expected lung cancer death rate among this group.

From 1955 on, the concept of a significantly increased risk of lung cancer in workers with asbestosis becomes more widely accepted.
Doll, R. "Mortality from lung cancer in asbestos workers." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 12 (1955): 81.
[WKK-HSC] (22)

1956
The epidemiologists look for epidemiologic evidence - for example, HAMMOND and MACHLE says with regard to a relationship between asbestos and lung cancer..."there are at present too few cases and too little epidemiologic data to establish a significant relationship".
Hammond, E.C. and W. Machle.
"Environmental and occupational factors" in Mayer, E. and H.C. Maier. PULMONARY CARCINOMA, PATHOGENESIS, DIAGNOSIS AND TREATMENT. New York : New York University, 1956. p. 50.
[WKK-HSC] (23)

1956
Of 31 Danish insulation workers with more than 20 years exposure to asbestos, 19 reveal abnormalities of the membrane covering of the lung (pleura) and 6 have essential functional changes of the lungs produced by disease.
Frost, J., [et al.]. "Asbestosis with pleural calcification among insulation workers." DANISH MEDICAL BULLETIN, 2, 7 (1956): 202-204.
[WKK-HKCC]

006152

1958

The Committee on Occupational Cancer of the AMA Council on Occupational Health concludes that individual case reports contribute little to an understanding of occupational cancers and that "specific increases in age, sex, and site-specific cancer incidence above normal... represent the only reliable evidence for the existence of an occupational cancer hazard."

Occupational cancer: methods of epidemiological study." JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 166 (1958): 2171.

[WKR-HSC]

1958-61

The first two epidemiologic studies of asbestos workers in North America do not show a significant increase in lung cancer. As recently as 1961 it is said that "there is no epidemiological evidence of an association between asbestos and lung cancer among American workers".

Braun, D.C. and T.D. Traub. "An Epidemiological study of lung cancer in asbestos miners." ARCHIVES OF INDUSTRIAL HEALTH, 17 (1958): 634.

Dunn, J.E., [et al.] "Lung cancer mortality experience of men in certain occupations in California." AMERICAN JOURNAL OF PUBLIC HEALTH, 50, 2 (1960): 1475.

Johnston, R.T. "Silicosis and cancer." JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 178 (1961): 353.

[WKR-HSC] (24)

1960

KIVILUOTO finds farmers and their wives in a rural county of Finland with the same internal chest scarring as asbestos workers—they had been exposed to asbestos air pollution from a mine nearby.

Kiviluoto, R. "Pleural calcification as a roentgenologic sign." ACTA RADIOLOGIA, 194 (1960): 1-67.

[NEI-N/O] (25)

006153

1960

WAGNER et al. finds that of 33 patients with mesothelioma 32 had had contact with blue asbestos (crocidolite). All lived in an asbestos mining region. Eleven of the victims had no occupational exposure. Wagner, J.C., [et al.] "Diffuse pleural mesothelioma and asbestos exposure in the North-West Cape Province." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 17 (1960): 250.

[WKR/NEI-CEC] (26)

1964

SELIKOFF et al find excess death rates for lung cancer among Paterson, New Jersey asbestos workers to be seven times the expected rate. In the study, the rates for gastrointestinal cancer are three times that expected; they occur 20 or more years after exposure and is unrelated to smoking. Overall, a 50% higher than average mortality rate is found among this group.

Selikoff, E.J. "Asbestos exposure and neoplasia." JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION, 188 (1964): 22.

[WKR-OTH] (27)

1965

SELIKOFF et al find that 40% of New York asbestos workers died of asbestosis and occupation-related cancer.

Selikoff, E.J. "The Occurrence of asbestosis among insulation workers in the United States." ANNALS OF THE NEW YORK ACADEMY OF SCIENCE, 132 (1965): 139-155.

[WKR-NRCC]

1965

THOMSON'S 1963 study of Cape Town residents finds an average of 25% (30% of male and of female) of 50 consecutive autopsies have asbestos fibres embedded in their lungs.

A parallel investigation carried out in Miami, U.S.A. found closely matching results. Thomson, J.G. "Asbestos and the urban dweller." ANNALS OF THE NEW YORK ACADEMY OF SCIENCE, 132 (1965): 196-214.

[NEI-NRCC]

006154

- 1965 NEWHOUSE and THOMPSON find, among a London hospital series of 76 cases, 45 mesothelioma victims who had had no occupational exposure to asbestos, but who lived within half a mile of an asbestos factory or in the house of an asbestos worker. Exposure in these cases occurs more than thirty years before death. This study confirms the close association between "casual" asbestos exposure and mesothelioma. Newhouse, M.L. and H. Thompson. "Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 22 (1965): 261. [NEI and P/O-HSC] (28)
- 1965 Of 20 papers presented at a 1964 conference in New York City, no opinions dissent from the view that asbestos is a cause of cancer. Selikoff, I.J. and J. Churg. "Biologic effects of asbestos." ANNALS OF THE NEW YORK ACADEMY OF SCIENCE, 132, 1 (December 31, 1965): 1-766. [WKR-HSC]
- 1965-74 SELIKOFF reports on the prevalence of abnormal lung x-rays in relation to years from initial asbestos exposure in insulation workers. Selikoff, I.J. "The Occurrence of pleural calcification among asbestos insulation workers." ANNALS OF THE NEW YORK ACADEMY OF SCIENCE, 132 (1965): 351-367. WALLACE & LANGLANDS report the frequency of x-ray abnormalities among asbestos workers "to be 20 times greater than among non-workers". Wallace, W.F.M. and J.H.M. Langlands. "Insulation workers in Belfast." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 28 (1971): 211-216. [WKR-NBCC] (30)
- 1965-79 x-ray abnormalities are found not to be a prerequisite for diagnosing asbestos-related disease.

BADLER et al notes a reduction in the vital capacity (a measure of breathing efficiency) of asbestos factory workers without x-ray evidence of disease progression.

Bader, M.E., et al. "Pulmonary function in asbestosis." ANNALS OF THE NEW YORK ACADEMY OF SCIENCE, 137 (1965): 391-405.

JODOIN et al find obstruction and restriction when x-rays were normal
Jodoin, G. "Early effects of asbestos exposure on lung function." AMERICAN REVIEW OF RESPIRATORY DISEASES, 104 (1971): 525-535.

GREGOIRE finds that autopsy data on asbestos workers reveal that in nearly 30% of the cases with at least minimal asbestosis, x-rays before death revealed no lung fibrosis.
Gregoire, F. 1979. Clinique Lavoister, Inc., Montreal. Letter dated Feb. 27, 1979, reporting on paper presented by Dr. A. Trepanier to the Association of Pneumologists of the Province of Quebec, Jan. 30, 1979.

[WKK-NRCC] (31)

1967-72

Cases of mesothelioma developing in infancy or childhood, probably as a result of contact with asbestos dust - contaminated fathers, are reported by LIEBEN (1967) and GRUNDY (1972).

Lieben, J. "Mesothelioma and asbestos exposure." ARCHIVES OF ENVIRONMENTAL HEALTH, 14 (1967): 559.

Grundy, G.W. "Malignant mesothelioma in childhood." CANCER, 30 (1972): 1216.

[P/O-HSC] (29)

1968

The British Occupational Hygiene Society recommends the implementation of the 2-fibre/cm³ standard for asbestos exposure following examination of x-ray abnormalities in 290 asbestos factory workers by industry personnel. Comparing the x-ray abnormalities to the recorded dust conditions, the industrial hygienists concluded that a worker had a 25% chance of contracting asbestosis at the 2-fibre level.

(B.O.H.S. - Subcommittee on Asbestosis Hygiene Standards for Chrysotile Asbestos Dust, Annals of Occup. Hygiene, vol. 11 (1968) p. 47-49)

[WKK-HSC] (32)

1969

BYROM et al find that emissions of asbestos from insulation board and sprayed asbestos are greater than those found in buildings with asbestos cement sheeting and other products. Where dry-sprayed asbestos fibre is exposed to the abrasion of air movements, higher levels are measured.

Byrom, J.C., (et al.) "A dust survey carried out in buildings incorporating asbestos-based materials in their construction." ANNALS OF OCCUPATIONAL HYGIENE, 12 (1969): 141-145.

[ENV-HSC]

1971

LANGER et al confirm that chrysotile fibres or fibrils can be identified in nearly all the lungs of adult urban dwellers, if the newer techniques of electron microscopy are used.

Langer, A.M., (et al.) INHALED PARTICLES. v. 2. Old Woking, Surrey: Unwin, 1971.

[ENV-HSC]

006157

1971

CUNNINGHAM and PONTEFRACT publish the first data on asbestos particles in ground, surface and drinking water.

One to 11 million chrysotile asbestos fibres/litre are detected in city drinking water, soft drinks, liquor, and beer. River water contains more asbestos fibres than water passed through a city filtration system. The highest count comes from unfiltered tap water at Thetford mines, Quebec.

Cunningham, R.M. and R.D. Pontefract. "Asbestos fibres in beverages and drinking water." NATURE, 232 (1971): 332-333.

[ENV-CEC]

1972

ENTERLINE provides evidence that intermittent exposure to high concentrations of asbestos might induce cancer mortality but not excess asbestosis mortality. Enterline, P.E. "Mortality in relation to occupational exposure in the asbestos industry." JOURNAL OF OCCUPATIONAL MEDICINE, 14 (1972): 897.

[WKR-CEC]

1972

SELIKOFF finds measurable but minute amounts of asbestos in air near building sites in New York, where asbestos spraying is being done. High volume samplers and electron microscopy are used.

Selikoff, L.J. "Asbestos air pollution." ARCHIVES OF ENVIRONMENTAL HEALTH, 25 (1972): 1-15.

[NEI-NRCC]

006158

1972/1979

There is little or no evidence that cigarette smoking is related to increased risk of mesothelioma, according to two studies by HAMMOND et al.
Hammond, E.C., (et al.): "Relation of cigarette smoking to risk of death from asbestos-associated disease among insulation workers in the U.S." in Bogovski, P., (et al.) BIOLOGICAL EFFECTS OF ASBESTOS: PROCEEDINGS OF THE WORKING CONFERENCE OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, LYON, FRANCE, OCTOBER 2-6, 1972. IARC publication, no. 8. Geneva: World Health Organization, 1973.
Selikoff, I.J. and E. Hammond. HEALTH HAZARDS OF ASBESTOS EXPOSURE. New York: New York Academy of Science, 1979.
[WKK-N/O]

1973

Mortality study by NEWHOUSE finds, in a cohort of asbestos factory workers, excess mortality from cancer of the lung and other cancers, even with low to moderate exposure.
Newhouse, M.: "Asbestos in the workplace and the community." ANNALS OF OCCUPATIONAL HYGIENE, 16 (1973): 47-47.
[WKK-HSC]

1973

BOHLIG and HAIN mention that the distribution of mesotheliomas in Hamburg in the vicinity of an asbestos factory is dependent upon the prevailing wind; a visible snowfall-like air pollution had been emitted from the factory.
Bohlig, H. and E. Hain. "Cancer in relation to environmental exposure" in Bogovski, P., (et al.) BIOLOGICAL EFFECTS OF ASBESTOS: PROCEEDINGS OF THE WORKING CONFERENCE OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, LYON, FRANCE, OCTOBER 2-6, 1972. IARC publication, no. 8. Geneva: World Health Organization, 1973.

[NEI-CEC]

006159

1973

Particularly for mesothelioma, findings vary depending on the medical criteria chosen for diagnosis.
 McCaughey, W.T. and P.D. Oldham.
 "Diffuse mesotheliomas: observer variation in histological diagnosis" in Bogovski, P., (et al.) BIOLOGICAL EFFECTS OF ASBESTOS : PROCEEDINGS OF THE WORKING CONFERENCE OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, LYON, FRANCE, OCTOBER 2-6, 1972. IARC publication, no. 8. Geneva : World Health Organization, 1973.

[P/O-IARC]

1973-77

The Advisory Committee on Asbestos Cancer (IARC) says an increased risk of cancer resulting from asbestos fibres present in water, beverages, and food "does not indicate any risk".

The IARC report says evidence on the risk of gastrointestinal exposure is deficient and that causative factors for inducing gastrointestinal tumours are not well understood.

Advisory Committee on Asbestos. "Report to director of International Agency for Research on Cancer" in Bogovski, P., (et al.) BIOLOGICAL EFFECTS OF ASBESTOS : PROCEEDINGS OF THE WORKING CONFERENCE OF THE INTERNATIONAL AGENCY FOR RESEARCH ON CANCER, LYON, FRANCE, OCTOBER 2-6, 1972. IARC publication, no. 8. Geneva : World Health Organization, 1973.

[ENV-CEC]

1974

WAGNER et al find chrysotile as potent as crocidolite asbestos in inducing lung tumours in rats after inhalation exposure.

This study is interpreted by the NIOSH-OSHA Asbestos Work Group (1980) as demonstrating that "Chrysotile appears to be much more fibrogenic and carcinogenic than the amphiboles (includes amosite and crocidolite), since the concentration of dust in the lungs of rats exposed to Canadian chrysotile was only 1:8 - 2.2% of the dust concentration in the lungs of animals exposed to amphiboles - yet the lung tumour incidences and degrees of pulmonary fibrosis were similar in all groups".

Wagner, J.C. "Mesothelioma in rats after inoculation with asbestos and other minerals." BRITISH JOURNAL OF CANCER, 28 (1974): 123.

[EXP-N/O]

[WAG-N/O]

[WAG-N/O]

1974

In a review of 252 cases of mesothelioma with a known history of exposure, WEBSTER finds the same percentage of past occupational exposure (30-45%) as of environmental exposure; the last group had mainly lived near a mine or mill or in towns situated in an asbestos-producing area.

Webster, L. "The ingestion of asbestos fibres." ENVIRONMENTAL HEALTH PERSPECTIVES, 4 (December 1974): 199.

[WEI-CEC]

1974

LEWINSOHN presents a review of studies centred on the connection between mesothelioma and type of asbestos. There is general agreement that the risk of mesothelioma is fibre-related in the order crocidolite - amosite - chrysotile but the magnitude of the difference between each type is not well established.

Lewinsohn, H.C. "Health hazards of asbestos: a review of recent trends." JOURNAL OF THE SOCIETY OF OCCUPATIONAL MEDICINE, 24 (1974): 2.

[WLR/P-O-CEC]

1975

ROHL et al shows that when professional construction workers used materials similar to do-it-yourself products, the concentration of asbestos in air exceeds U.S. safety standards by a factor as great as 2.
Kohli, A.N. "Exposure to asbestos in the use of consumer patching and taping compounds." *SCIENCE*, 189 (1975): 551.
[P/O-CEC]

1975

NICHOLSON et al finds the level of airborne asbestos dust in the homes of workmen to be 10-500 nanograms per cubic metre.
Nicholson, W.J., [et al.] ASBESTOS CONTAMINATION OF THE AIR IN PUBLIC BUILDINGS. NTIS report, no. Po-254
[P/O-NRCC]

1975

Observations by ZIELHUIS et al suggest that there is a particular risk associated with "peak" exposure levels, making asbestos dust exposure levels averaged over many years an unreliable indicator of how dangerous the exposure has been. Among 48 mesothelioma cases with definite/probable past exposure, 40% have intensive exposure, out in 71% it is intermittent.
(Pleural mesothelioma and exposure to asbestos; a retrospective case-control study in the Netherlands. *Int. Arch. Occup. Hlth.*, 36)
[WKK-CEC]

1975

HOWARD et al evaluates the effect of a reduction in asbestos dust levels due to technical devices at a Rochdale factory in Britain. There is a decrease of mortality in workers entering 1950-1959, and more so in those entering after 1959. Dust levels had fallen in the 1953-57 period by approx. 50-80% throughout the factory.
Howard, S., [et al.] A MORTALITY STUDY AMONG WORKERS IN AN ENGLISH ASBESTOS FACTORY: 181
INTERNATIONAL CONGRESS ON OCCUPATIONAL HEALTH. Brighton: n.p., 1975.
[WKK-CEC]

006162

- 1976 A survey by SEBASTIEN et al of asbestos in the urban air of fireproofed buildings in Paris, France finds concentrations of chrysotile and mixed asbestos dusts from 0.1 to 80 nanograms/cm³ of air.
Sebastien, P., (et al.) "Les Pollutions atmosphériques urbaines par l'asbeste." REVUE FRANÇAISE DES MALADIES RESPIRATOIRES, 4, supp. 2 (1976): 51-62.
[ENV-NRCC]
- 1976 SELIKOFF suggests that the latent period is inversely related to dose, that is - the lower the dose of asbestos dust, the longer the period from initial exposure to onset of disease.
Selikoff, I.J. "Cancer risk of asbestos exposure: paper presented at Colo Spring Harbour Laboratories, New York. s.l.: n.p., 1976.
[WKR/P-O-HSC]
- 1977 MCDONALD et al show an excess of gastrointestinal cancers in men exposed to the highest doses of dust at Thériforo Mines, Quebec.
McDonald, J.C., (et al.) "Dust exposure and mortality in chrysotile mining, 1914-1975. Unpublished data, 1977.
[WKR-HSC]
- 1977 "Because of lack of clear distinctions between the various possibilities of exposure... greater reliance should be put upon biological monitoring than on environmental monitoring".

The CEC report also states: "Due to the fact that classification of the relation between fibre size and biological effect is central to the possible environmental hazard of asbestos, the reporting of fibre dimensions in addition to fibre concentration is imperative."

ZIERUIS et al conclude ... "inadequate data exist on which to base a quantitative dose-response relationship for asbestos-related malignancies.

On the other hand... there is often a clear dependency on dose, -duration of exposure, type of job (intensity of exposure) but quantification is not adequately possible."

Zielhuis, R.L. PUBLIC HEALTH RISKS OF EXPOSURE TO ASBESTOS : REPORT OF A WORKING GROUP OF EXPERTS PREPARED FOR THE COMMISSION OF THE EUROPEAN COMMUNITIES DIRECTORATE-GENERAL FOR SOCIAL AFFAIRS, HEALTH AND SAFETY/DIRECTORATE New York : Pergamon Press, 1977.

[WKR/P-O-CEC]

1977

EPLER and GAENSLEK find 23 shipyard and manufacturing plant workers of 763 total had "pleural effusions" (exudation of body fluids from vessels), which may predispose subjects to mesothelioma.

Epler, G.R. and E.A. Gaensler. "Asbestos effusion." CHEST, 72, 3 (1977): 399.

[WKR-NRCC]

1977

SARACCI concludes that although the available data do not allow a definite answer, the model of multiplicative risks of inhaling asbestos and smoking tobacco appear more plausible than the additive risk model, or one where asbestos has an effect only in the presence of smoking.

Saracci, R. "Asbestos and lung cancer : an analysis of the epidemiological evidence on the asbestos-smoking interaction." INTERNATIONAL JOURNAL OF CANCER, 20 (1977): 323.

[WKR-HSC]

1977

SELIKOFF surveys 485 current employees of a chrysotile mine at Bale Verte, Newfoundland. Over 10% have one or more X-ray abnormalities associated with asbestos exposure, despite a short period from initial exposure.

(The Mt. Sinai study team recorded the highest airborne level of asbestos dust found anywhere in the world at Bale Verte.)

SELIKOFF, L.J. CLINICAL SURVEY OF CHRYSOTILE ASBESTOS MINERS AND MILLERS IN BALE VERTE, A

NEWFOUNDLAND. REPORT OF THE NATIONAL INSTITUTE OF ENVIRONMENT HEALTH SCIENCES, DECEMBER 22, RESEARCH TRIANGLE PARK, N.C. s.l. : n.p., 1976.

(WKK-N/U)

1977

WEISS reports no unusual mortality experience over a 30 year period for a cohort of workers employed in a pulp and paper millboard plant using only chrysotile.

WEISS, W. "Mortality of a cohort exposed to chrysotile asbestos." JOURNAL OF OCCUPATIONAL MEDICINE, 19 (1977): 737.

(WKK-N/U)

1977-1978

ELMES and SIMPSON (1977) and SELIKOFF (1978) conclude that in the past when occupational asbestos exposures were high, asbestosis was the earlier cause of death - only those who did not succumb to its effects lived long enough to acquire lung cancer. With lower levels of asbestos exposure, the earlier cause of death is lung cancer followed by mesothelioma, and only the survivors of these suffer from the now more slowly developing asbestosis.

Elmes, P.C. and M.J.C. Simpson. "Insulation workers in Belfast : a further study of mortality due to asbestos exposure, 1940-1975." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 34 (1977): 174-181.
Selikoff, L.J. ASBESTOS AND DISEASE. New York : Academic Press, 1978.

(WKK-NRCC)

1977-78

PETO finds that at the 2 fibre/cm³ standard, one asbestos worker out of 14 runs the risk of dying prematurely from asbestos-related disease. He finds also that the lung cancer death rate at the Rochdale asbestos plant in England is almost double the expected rate. (A mortality study among workers in an English asbestos factory. *Brit. J. Ind. Med.* 34, 169 (1977))

Peto, J. "The Hygiene standard for chrysotile asbestos: paper submitted to the Advisory Committee on Asbestos." *Lancet*, 1 (1978): 484.

[WKK-HSC]

1978

DAVIS et al investigate the question of the relative harm produced by different types of asbestos fibre. He notes that WAGNER'S 1974 findings concern only lung cancer, not mesothelioma. He also concludes that the relative pathogenicity of the different types of asbestos may depend upon the proportion of fibres greater than 20 microns in length. Davis, J.M.G. "Mass and number of fibres in the pathogenesis of asbestos-related lung disease in rats." *BRITISH JOURNAL OF CANCER*, 37 (1978): 673-688.

[EXP-NRCC]

1979

ANDERSON et al find a high prevalence of x-ray abnormalities and mesothelioma among household contacts of former amosite insulation plant employees.

Anderson, H.A. "Household exposure to asbestos and risk of subsequent disease" in Lemen, R.A. and J.M. Dement. *DUSTS AND DISEASE*. Park Forest South, Ill.: Pathotox Pub., 1979.

[P/O-N/O]

1974

It is found by PYLEV that synthetic fibres as well as naturally occurring fibres were carcinogenic. He produced mesotheliomas in 54% of rats injected within the lung cavity with a milled synthetic fibre, and primary lung tumours in 23% of hamsters injected within the chest cavity with a synthetic chrysotile.

Pylev, L.N. "Pretumorous lesions, lung and pleural tumours induced by asbestos in rats" in "BIOLOGICAL EFFECTS OF MINERAL FIBRES. France: IARC, 1979.

[EXP-NRCC]

1979

WAGNER et al shows that a commercial grade of asbestos, mainly short fibre Canadian chrysotile, used primarily for paint and plastic tile fibres, can induce mesotheliomas when injected within the lung membrane of rats, and can induce lung tumours (neoplasms) when the animals inhale the fibre.

Wagner, J.C. "The Comparative effects of three chrysotiles by injection and inhalation in rats" in "BIOLOGICAL EFFECTS OF MINERAL FIBRES. France: IARC, 1979.

[EXP-N/U]

1979

HAMMOND'S 10-year follow-up study of 12,051 asbestos-insulation workers with 2 or more years of work experience obtains complete smoking histories for about 3/4 of the subjects.

He and SELIKOFF find the probability that workers' lung cancer is due to asbestos exposure to be about 8% in both smokers and non-smokers alike.

Selikoff, L.J. and E. Hammond. HEALTH HAZARDS OF ASBESTOS EXPOSURE. New York: New York Academy of Science, 1979.

[WKR-N/O]

1979

ACHESON and GARDNER conclude:

- a) Experimental work shows that lung cancer, asbestosis and mesothelioma may be produced in animals after inhalation of the four types of asbestos, and that some samples of chrysotile are at least as pathogenic as the amphiboles (including crocidolite and anthophyllite).
- b) Evidence in humans shows crocidolite and amosite to be more dangerous in producing lung cancers and asbestosis than chrysotile.
- c) As for mesothelioma, combined evidence from process workers, from miners and from the distribution of neighbourhood and domestic cases presents a powerful case that crocidolite has been more dangerous than chrysotile.

The HSC document concludes that chrysotile has rarely caused mesothelioma or cancer of the larynx. On the other hand, the proven capacity of chrysotile to produce mesothelioma in animals, if the fibre is of a specific configuration, and the probability that chrysotile is becoming increasingly finely divided and respirable should be kept in mind.

Acheson, E.D. and M.J. Gardner. "The ill effects of asbestos on health" in FINAL REPORT OF THE ADVISORY COMMITTEE ON ASBESTOS: V. 2. London: H.M.S.O., 1979. p.33.

(WAK/P-O-HSC)

1979

JONES et al report that women in a wartime gas mask factory having a long employment period have a higher proportion of death due to mesothelioma than those who had a short period of employment.

005168

Dose-response evidence from this study agrees with that from SELWYAN (1979), LIDDELL et al (1977), BERRY et al (1979) and NEWHOUSE and BERRY (1976).
Jones, J.B.P. "The Consequences of asbestos dust exposure in a wartime gas mask factory" in THE BIOLOGICAL EFFECTS OF MINERAL FIBRES. France: IARC. 1979.
[WKR-N/O]

1979

NICHOLSON et al finds that among a cohort of 544 men with at least 20 years of employment in chrysotile mining and milling at Thetford Mines, 16% of deaths were from lung cancer and 15% from asbestosis. The risk of death from mesothelioma and lung cancer in miners and millers is decidedly lower than for factory workers and insulators.
Nicholson, W.J. "Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec." ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, 1979. (in press)

[WKR-N/O]

1979

BERRY et al followup the 1968 BUHS study of 379 British asbestos textile factory workers at Rochdale. Of men exposed after 1954, 6.6% had possible asbestosis after an average followup of 16 years and an average of exposure of 5f/cm³. Cumulative dose based on combining dust concentrations may not be satisfactory.

(Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory. Br. J. Ind. Med. 36, 98)

(WKR-HSC)

1979-1980

MCDONALD, LIDDELL et al report follow-up of a cohort of 11,379 workers exposed to chrysotile for at least a month in the mines and mills of Thetford Mines, Quebec.

The study results include --

The major occupational hazards of chrysotile asbestos exposure are respiratory cancer and the complications of pulmonary fibrosis.

Excess mortality from lung cancer is directly related to accumulated dust exposure. Lung cancer deaths occur in non-smokers, but there is insufficient evidence to distinguish between multiplicative and additive risk models. There are no excess deaths from laryngeal cancer.

Overall mortality for men is high at Thetford Mines. The latent period for mesothelioma appears to be very long for these workers.

McDonald, J.C. and F.D.K. Giddell.

"Mortality in Canadian miners and millers exposed to chrysotile." ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, 380 (December 14, 1979):1-12.

McDonald, J.C., [et al.] "Dust exposure and mortality in chrysotile mining, 1910-1975." BRITISH JOURNAL OF INDUSTRIAL MEDICINE, 37 (1980):11-24.

(WKR-HSC)