LECTURE NOTES

ASBESTOS:
EXPEDIENCE, EXPOSURE, AND HUMAN EXPERIENCE

I. Asbestos: an introduction

A. Statistics (HEW)

1. 13 to 18 percent of all cancers in U.S. related to asbestos exposure

2. 21 to 38 percent of all cancer cases might be occupationally related

3. eight to eleven million U.S. workers have been exposed to asbestos

4. 4 million workers heavily exposed while working in naval shipyards during World War II

a. 20 to 25 percent die of lung cancer
b. 7 to 10 percent die of pleural or peritoneal mesotheliomas
c. 8 to 9 percent die of G.I. cancers

5. 67,000 cancers per year attributed to asbestos are expected in next 30-35 years. This would represent 17 percent of all cancers detected annually in the U.S.

6. non-occupational exposures:

a. asbestos workers' families
b. construction-site spectators
c. people living in asbestos factory neighborhoods
d. other accidental exposure:

1. school children
2. office staff
3. people drinking contaminated water

II. Asbestos: a substance defined

A. Definition

1. hydrated silicates combined with magnesium, iron, calcium, aluminum, sodium, other trace elements

B. Classes

1. serpentine (contains chrysotile - which accounts for 93 percent of the world's use)

2. amphiboles

a. crocidolite
b. amosite
c. anthophyllite
d. tremolite
e. actinolite

C. Properties

1. high tensile strength

2. acid and temperature resistance

3. flexibility

4. non-biodegradable and environmentally cumulative

III. Production, consumption, and population at risk

A. Production

1. 1974 - world's annual production - in excess of 5 million tons

B. Consumption

1. one million tons annually in U.S.

C. Industrial Uses

1. cement

2. pipe

3. sheet

4. flooring products

5. friction products

6. roofing products

7. paper

8. coatings and compounds

9. Packing materials and gaskets

10. textiles

11. plastics

12. thermal insulation

13. electrical insulation

D. Defining Populations at Risk

1. occupational exposures

a. mining and milling: < 1,000 workers
b. primary manufacturing: > 37,000 workers
c. secondary manufacturing: 300,000
d. tertiary occupational exposure: millions of workers

1. automotive sales, service, and repair: 2 million
2. shipyard workers: 185,000

2. para-occupational exposure

a. workers in the vicinity of asbestos fibers

3. non-occupational exposure

a. emission from natural sources

4. domestic and environmental exposure

a. family members exposed to dust from workclothes: mesothelioma and pleural plaques
b. neighborhood exposure from asbestos mine: mesothelioma
c. non-asbestos workers in asbestos region: pleural calcification
d. family members of asbestos-exposed workers: pulmonary fibrosis

IV. Asbestos in Historical Perspective

A. first uses

1. in clay pots in Finland 4,000 years ago
2. for cloth in Greece, 430 B.C.
3. for insulation due to steam engine use

B. first recorded asbestos-related diseases

1. 1st century: lung sickness in slaves who wove asbestos into cloth

2. 1900: pulmonary fibrosis in asbestos worker

3. 1919: Pancoast's review of 15 asbestos worker's x-rays (abnormal)

4. 1927: Cooke developed description and coined word "asbestosis"

5. further references in the thirties

a. Lynch & Smith (bronchogenic carcinoma)
b. Gloyne (carcinoma of the lungs)

C. other significant studies linking cancer and asbestos

1. 1949: Hamilton and Hardy

2. 1949: Merewether

3. 1950: Passmore and Swanston

4. 1955: Doll

5. 1965: Buchanan

6. 1960: Wagner

7. 1970's: Newhouse

V. Toxicology - animal

A. Laboratory studies with rats

1. respiratory exposure resulted in asbestosis

2. oral administration resulted in carcinomas of lung, kidney, and liver

3. exposure to various asbestos types resulted in pulmonary fibrosis in rats and other laboratory animals

4. pleural and peritoneal cavity injections caused fibrosis, granulomas, adhesions, and pulmonary and mediasinal abscesses

B. Other laboratory studies

1. tissue culture reactions to asbestos

a. anaerobic metabolism stimulation
b. cell membrane damage
c. chromosome damage
d. lysosomal enzyme leakage
e. autolysis of the cell
f. fibrogenesis

VI. Causation

A. Criteria of causality (Selikoff and Lee) - general postulates

1. statistically significant association between exposures and syndrome development

2. dose-response relationship

3. tissue concentration

4. animal toxicology

5. attending variables/synergism

B. Major categories of asbestos-induced disease

1. parenchymal asbestosis or pulmonary fibrosis

2. pleural asbestosis

3. mesothelioma

4. carcinoma

C. Other relevant factors

1. exposure to carcinogens (particularly cigarettes)

2. intensity of exposure

3. individual susceptibility

4. duration of exposure

5. elapsed time since initial exposure

VII. Pathophysiology

A. Routes of entry

1. inhalation

a. asbestos fibers in the five to ten micron range penetrate to respiratory regions of lungs

1. penetration of uncoated fibrils'

a. travel directly to pleural and peritoneal spaces
b. fibrosis usually occurs first in base of lungs
c. pleural effects usually confined to lower two-thirds of the thorax

1. pleural effusions
2. pleural fibrosis
3. pleural calcification
4. neoplasia

2. ingestion

a. some excreted with feces
b. some penetrate G.I. tract
c. extra-pulmonary penetration

1. tonsils
2. thoracic and abdominal lymph nodes
3. the pleura
4. the peritoneum
5. liver
6. spleen
7. kidneys
8. adrenals
9. small intestine

VIII. Asbestosis

A. Definition

1. pulmonary fibrosis due to inhalation and deposition of asbestos fibers in the lung

2. manifestations appear twenty to forty years after exposure

3. one year of exposure sufficient

B. Pathology

1. early stages

a. normal lung size
b. mild fibrosis in sub-pleural regions of lung periphery

2. progression of disease

a. spreads from base to apex of lung

1. to sub-pleural depth of one centimeter
2. from lower lobes to middle lobes and then to upper lobes

b. cut surface of lung reveals

1. fine, gray-colored, patchy fibrosis adjacent to visceral pleura

3. advanced disease

a. lung small, pale, firm, and rubbery
b. honeycombed cysts frequent in fibrotic areas
c. impalpable black nodules
d. necrobiotic nodules in fibrotic tissue occasionally found
e. centrilobular and bullous emphysema
f. pulmonary massive fibrosis uncommon
g. asbestoma: a solitary fibrotic nodule sometimes found

C. Microscopic findings

1. human biopsy material

a. early stage - resembles any other type of interstitial pneumonia with characteristics of:

1. mixed leukocyte infiltration of alveolar walls
2. phagocytes in alveoli

b. fibrotic stage

1. occurrence around respiratory bronchioles
2. alveolar walls covered with reticulin fibers, macrophages, and dust
3. ferruginous bodies

c. late stage

1. evolution to neighboring alveoli

2. diffuse alveolar wall thickening with peribronchiolar and perivascular fibrosis
3. diffuse interstitial fibrosis

2. factors contributing to functional impairment

a. loss of respiratory surface area with destruction of alveoli
b. membrane thickening causing a barrier to diffusion and gas exchange
c. perivascular fibrosis

D. Pathogenesis

1. exposure to asbestos most significant factor

a. heavy for a short time, or
b. low for a longer time

2. factors indicating immunologic and possibly autoimmune involvement in progression of disease include:

a. elevated levels of ANA
b. rheumatoid factor
c. decrease in lymphocyte count

IX. Clinical features

(See slide-tape presentation, videotape, and clinical application section of this text.)

X. Workers' Compensation

A. Difficulties in the system

1. most asbestos-related diseases not compensated; workers responsible for medical bills

2. attempts are being made to sue employers for negligence

B. Longshoremen's and Harbor Workers' Compensation Act

1. asbestos-related diseases compensated for longshore and harbor workers

XI. Regulation of occupational asbestos exposure

A. Background

1. 1918 - U.S. and Canadian insurance companies stopped selling life insurance to asbestos workers

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/8

2. 1970's - asbestos accepted as a carcinogen

3. 1972 - OSHA standard adopted

   a. two fibers per cubic centimeter of air as average exposure in eight-hour period

      1. fiber length of five micrometers
      2. length to width ratio of three to one

   b. converts to 16 million fibers greater than five micra in length per workday

      1. number of fibrils resulting from this exposure could be 100 times greater and suggests that

         a. daily intake of 1.6 billion fibrils is tolerated under OSHA standard

   c. not clear that standard successfully protects against asbestosis, let alone cancer

NARRATIVE

ASBESTOS SLIDE-TAPE PRESENTATION

This is provided so the instructor can personally narrate the presentation if he/she so desires.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/10

ASBESTOS SLIDE TAPE

1

2

3 Mr. Jacob Christman died of asbestos-induced mesothelioma in 1979. It was his belief that we knew little about asbestos-induced disease in 1942 when he was working in the shipyards. But some facts were clear by that time.

4 In 1900, Montague Murray described the first reported case of asbestosis in England. In 1918, U.S. and Canadian insurance companies stopped selling life insurance policies to asbestos workers. In 1927, Cooke named and gave the first complete description of asbestosis, and, in 1930, the first U.S. case of asbestosis was described.

5 During the 30's, several reports were released including:

- Merriweather's description of a dose-response relationship between long exposure to asbestos and the appearance of asbestosis;
- reports on the suspected links between cancer and asbestosis; and
- Gloyne's description of a method of diagnosing asbestosis.

6 In 1949, Hamilton and Hardy published a textbook citing a case study in which 6% of the asbestos workers developed bronchogenic carcinoma. Yet, it was not until the 1970's that a serious effort was made in the United States to legislate maximum tolerable levels of exposure for asbestos workers. The results of the lack of definitive action in the United States prior to 1972 will continue to be seen well into the 21st century.

7 In fact, the National Cancer Institute and the National Institute for Environmental Health Sciences recently estimated that the number of newly diagnosed cancers, attributable to past asbestos exposure, will average 67,000 per year. This rate will continue for 30-35 years even if no further exposures occur.

8 It has been estimated that 8 - 11 million workers in the United States have been exposed to asbestos both during and since the second world war. Of these, 4 million shipyard workers are believed to have sustained very heavy exposures.

On April 25, 1978, the Surgeon General and Assistant Secretary for Health issued a physician's advisory stating that 20-25% of heavily exposed workers die of lung cancer 20 or more years after initial exposure.

Seven to ten percent die of pleural or peritoneal mesothelioma; and . . .

. . . other reports suggest an additional 8-9% die of G.I. tumors.

Thus, the total percentage of heavily exposed workers dying from cancer is between 35% and 44%.

This means that, from 1979 until 2010, 1.6 million of the 4 million heavily exposed shipyard workers are expected to die of asbestos-related cancers.

But these figures do not take into account workers in other professions who were equally exposed to asbestos, or

the spouses and children of asbestos workers who brought home asbestos-laden workclothes, or

sidewalk superintendants watching building demolition, or

people who live and play in the vicinity of asbestos industries and their waste, or

school children in California who molded donated asbestos into school projects, or

people around the country whose rose bushes came from Tyler, Texas, wrapped in discarded burlap bags that came

from a local asbestos plant in which the worker death toll from lung cancer is expected to exceed 50%.

22 Beginning in 1948, Selikoff studied a group of 632 insulation workers in New York and New Jersey in an attempt to quantify the problem.

23 During the 20 year period from 1943 to 1962, 196 deaths were expected based on the size of the population.

24 But 253 workers died, revealing an excess of 57 deaths.

25 Looking at cancer related deaths, 31 were expected, and 95 occurred; 3 times more than predicted.

26 Looking specifically at lung cancer, 6 deaths would normally be expected in the test population, but 42 persons died of lung cancer; 7 times the predicted number.

27 Based on this study, and other reports, it is predicted that as many as 45% of all asbestos workers will die from cancer. With the knowledge that most of these cancer deaths were preventable and with the conviction that the primary care physician can play a strategic role in preventing the continuation of what must be considered a pandemic, we offer a review of our knowledge of asbestos and its interaction with human health.

28 The first part of this program provides an overview of the problem. We will identify the four major asbestos fiber types; try to define the population at risk; and show some examples of exposure sources.

29 In the second part of the program, we'll look at the health problems in more detail, by defining the problems, examining their clinical manifestations and, where applicable, describing the pathophysiology and suggested therapy.

30 Asbestos is the generic name for several naturally occurring fibers. Those fibers can be divided into two groups: the serpentine group, which contains magnesium silicates and the amphibole group, which contains aluminum silicates.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/13

31 There is only one fiber in the serpentine group: chrysotile, however it constitutes 93% of the asbestos mined in the world and is the major fiber type mined and used in the United States. It is found in many different grades with minor differences in chemical structure.

32 In the amphibole group, there are several types of fibers. Anthophyllite, amosite and crocidolite are the 3 most common.

33 The population most likely to be exposed to asbestos fibers includes those persons who have been exposed occupationally, both directly and secondarily, and those persons who have been exposed non-occupationally.

34 Direct occupational exposures take place in several ways;

- miners of asbestos are obviously in direct contact with asbestos fibers. Currently, there are fewer than 1,000 persons mining asbestos in this country.
- persons who participate in the manufacturing of goods that use unmodified asbestos, such as asbestos pipemakers, can also be directly exposed to the fibers. There are more than 37,000 such individuals in the United States.

35 Additional millions have undergone secondary exposure on their jobs including:

- over 300,000 persons who process asbestos-manufactured products to make other products
- millions of construction workers who are frequently exposed to asbestos insulation; and
- millions more who have worked in the asbestos-laden environment of the shipyards.

36 In the non-occupationally exposed sector of the population, there is no way to monitor or estimate the severity of the exposure since it does not take place in a controlled environment; however, people living near asbestos plants have developed mesothelioma and, in a survey of the families of 3500 shipyard workers, Selikoff found that 35% of the spouses and children had developed asbestos-induced changes on their x-rays.

37 The effect of these exposures is that many people are at risk for asbestos related health problems including: shipbuilders

38 shipbreakers

39 construction workers who work with drywall

40 persons who work with asbestos/concrete pipe

41 factory workers who make brake shoes

42 insulation workers

43 high steel riggers

44 painters, tapers and plasterers

45 auto mechanics who work with brake shoes and brake drums and many more.

46 Even office workers are potentially at risk since they daily breath air blown past asbestos insulated girders

47 as do children sitting in classrooms liberally insulated with asbestos.

48 And, in spite of all we know about asbestos, in December 1979 it was brought to the attention of the public that in Globe, Arizona a residential development had been constructed on the waste of an abandoned asbestos mill, 200 yards from an active milling operation.

49 Summarizing the problem, the HEW pamphlet, Asbestos: An Information Resource, states that "as a consequence of man's utilization of asbestos, coupled with natural occurrence of the mineral, asbestos fibers are found in the air we breathe, the food we eat and the water we drink."

50 The OSHA standard of 1972 states that exposure to asbestos cannot exceed a time-weighted average of 2 fibers per cubic centimeter of air in the breathing zone over an eight hour workday, based on a count of fibers longer than five micra with a length to width ratio of at least 3 to 1 as detected by light microscopy. No peak concentrations can be in excess of 10 fibers as determined by sampling over a 15 minute period. But these standards do not apply to persons involved in mining of asbestos or to persons secondarily or non-occupationally exposed to the fibers.

51 These measurements are most commonly made using a midget impinger placed on workers' clothing or using a stationary device in the work area.

52 In essence, the OSHA standards have reduced the legally acceptable exposure limits to small amounts; however, there are 2 opposing views of the standard. One group asserts that the standards are acceptable in terms of disease prevention and are as low as we can afford.

53 The second group points to evidence that the standard may not adequately prevent asbestosis, let alone cancer, and believes that the statistical models used to determine tolerable levels are too imprecise to effectively judge dose-response relationships for carcinogens. Therefore, they recommend that the standard be lowered to 0 until we can be sure of the levels that can be tolerated by humans.

54 The asbestos-induced health problems which we will examine include the non-pathologic problems of asbestos bodies and asbestos corns and warts, as well as the pathologic problems of asbestosis, carcinomas (primarily pulmonary) and mesothelioma.

55 In a 1977 study, Andrew Churg and Martha Warnock reported finding asbestos bodies in the lungs of 96% of the population tested.

56 Originally referred to as 'asbestos bodies' these structures are now more commonly called ferruginous bodies since the fibers become coated with an

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/16

iron-containing pigment and because other substances such as fiberglass, cotton, talc and diatomaceous earth can make up the central core. However, the core fiber can be determined using electronmicroscopy.

57 The presence of asbestos bodies in the human lung or sputum is not indicative of disease but is reflective of some previous exposure to asbestos.

58 When ferruginous bodies are reported in a pathological specimen, you should request further analysis of the specimen to determine if the central core is asbestos and be sure to acquire an extensive history, checking for possible asbestos exposure.

59 Asbestos corns and warts imply exposure to Amosite. These calouses occur on the dorsal and palmar surfaces of the hand, on the knuckles, fingertips, and forearms. They are seen infrequently now since asbestos fibers are rarely handled by individuals without some protection.

60 Asbestosis is a diffuse interstitial fibrosis of the lungs, first appearing in lower lobes of the lung and progressing to upper lobes. Early references to asbestosis can be found as far back as Montague Murray's 1900 description, but it was not until 1927 that the word ASBESTOSIS was coined.

61 There appears to be a dose-response relationship between the amount of exposure and the appearance of the disease. Both heavy exposure for a few years and light exposures for many years are likely to result in asbestosis, with severity of the fibrosis partially host-dependent.

62 The workers most at risk include those who mill or pulverize the asbestos ore and those working with asbestos textiles, thermal and acoustic insulation or in any job in the shipyards.

63 Selikoff, drawing upon Wagner's work, has suggested the sequence of asbestosis development:
The initial lesion occurs with fibers lodging in alveoli.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/17

64 Macrophages and red cells around the fibers are soon enmeshed in a network of reticulin fibers.

65 Cellular degeneration takes place and the reticulin fibers are gradually replaced by collagen with obliteration of the alveoli.

66 During the next phase, fibroblasts around the periphery of the lesion carry collagen fibers into alveolar ducts and atria, with possible leakage to mesothelial layers.

67 In the process, asbestos bodies are usually formed and may well be the cause of the resulting interstitial fibrosis.

68 Although you will most frequently see parenchymal and pleural changes together, we will examine them separately in an attempt to demonstrate the subtleties of the two components.

69 There are 5 main symptoms of parenchymal asbestosis: breathlessness on exertion, possibly some chest pain or tightness, cough, shallow rapid breathing and loss of appetite.

70 Shortness of breath is the most prominent symptom. It generally occurs approximately 10 years after exposure and may precede any clinical signs. This symptom initially takes place only upon exertion, but often progresses to dyspnea at rest, with resultant disability.

71 There is usually only a tightness of the chest in the early phases of the disease. If there is pain, it is usually in the lower part of the chest. Pain may increase as the disease advances.

72 The cough is initially non-productive and often only present in the morning. Unless the patient smokes, the cough may not be a complaint until later stages of the disease.

73 With advancing disease, breathing becomes progressively more rapid and shallow as the lungs become less compliant.
The patient may also experience a loss in appetite.
There are 6 clinical signs indicative of asbestosis.

74 The most prominent sign is the presence of fine dry rales having a crisp, clean quality occurring late in inspiration, almost always over the lower to mid-lung fields. They are not common at the apices as in tuberculosis. Wheezing is also uncommon.

75 Respiratory excursion gradually decreases and thus serves as an index of disease progression. Asbestotic lungs tend to be small and are usually not distended as in emphysema.

76 Clubbing of the fingers may be present. Smoking may play a part in the development of this sign.

77 There are also changes in pulmonary function test results as the disease progresses. These changes are important because they usually precede radiographic changes, often by several years. Although there is sometimes an obstructive component to the disease, most often you will see results indicative of a restrictive process.

78 These include decreases in vital capacity, total lung capacity and flow rates, with residual volume remaining near normal or slightly increased.

79 Parenchymal changes detectable in the lung fields on x-ray result from the progressive fibrosis and begin as a diffuse, barely visible reticulation over the lower two thirds of the lung. When accompanied by some pleural change, it may have the appearance of ground glass. The fine lines can progress until they completely obliterate the normal lung markings. The fibrosis may be either unilateral or bilateral.

80 Another common radiographic abnormality is an elevation and flattening of the diaphragm resulting from loss of lung volume. This rise of the diaphragm tends to narrow the costophrenic angle.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/19

81 Pleural Asbestosis may or may not be accompanied by parenchymal asbestosis. Pleural changes include pleural thickening, pleural effusion, hyaline plaques and calcified plaques.

82 Pleural changes add very little to the symptoms of parenchymal changes; however, there may be some restriction of the chest expansion if the pleura is grossly thickened.

83 With the exception of obvious radiographic changes, clinical signs may not be apparent though there may be some dullness on percussion and some dimunution of breath sounds. Occasionally, friction rub can be heard. Radiographic changes become marked as the disease progresses.

84 Pleural thickening may cause obliteration of the costophrenic, and at times, the cardiophrenic angles, even in the early stages of the disease. If pleural effusion is present, it will show up as a denser shadow that shifts with changes in the patient's position.

85 The border between the lung and pleura will usually become ill-defined,

86 and interlobar fissures may become visible.

87 Hyaline plaques are raised areas which frequently appear on both the visceral and parietal pleurae. They will often be seen as rounded, oblong somewhat denser areas on x-ray but for the most part are only revealed with oblique views.

88 Calcified plaques appear as discreet elevated gray-white lesions on the inner surface of the rib cage and on the diaphragm and are often first detected on x-ray. While unilateral pleural calcification may be secondary to empyema, tuberculosis or other pleural effusions, as well as traumatic pneumothorax, bilateral pleural calcifications, or plaque, are pathognomonic of previous exposure to asbestos. Again, calcified plaques are best seen on the oblique views.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/20

89 As with parenchymal changes, the lower two thirds of the lung fields are usually involved. The changes may be unilateral but are more commonly bilateral.

90 The International Labor Office in Geneva has popularized the widely accepted ILO-U/C classification system for interpreting chest x-rays regarding the presence and staging of pneumoconiosis, including Asbestosis. This system of interpretation, which is based on the P-A view of the chest, requires precise technique when taking and interpreting the films.

91 If one is looking for fibrotic changes, it is recommended that the film be shot at 140 kV. When looking for pleural changes, it is recommended that the film be shot between 45 and 80. Oblique views are frequently required to bring out subtle pleural changes. Special training is mandatory if you are interested in using the classification system. The American College of Radiology maintains a directory of physicians with the appropriate training.

92 While there is no known cure for asbestosis, as with other chronic pulmonary diseases, appropriate supportive therapy may significantly reduce both frequency of hospitalizations and length of stay, and result in an improved quality of life for the patient. Specifically, if the patient develops an elevated hematocrit, hypoxia or corpulmanale, early intervention with oxygen therapy may be beneficial.

93 In 1935, Lynch and Smith reported the first case of bronchogenic carcinoma in a patient with asbestosis. The Chief Factory Inspector of Britain reviewed 274 autopsies of reported cases of asbestosis from 1924 to 1947 and found 13.1% to have tumors of the lungs and pleura. In a similar series of 6000 silicotics he only found 1.3% to have tumors.

94 There are several types of carcinomas associated with asbestos exposure. Therefore, special attention must be given to early detection of suspicious signs and symptoms, such as; fatique, unexplained weight loss, loss of appetite, chronic cough, with or without hemoptysis, lymph adenopathy, and

night sweats. Specific attention should be focused on the lung, G.I. tract, and kidneys.

95 Since there appears to be an association between asbestos exposure and G.I. tumors, the self-administration of tests for blood in the stool should be taught to patients with known significant asbestos exposure. These should be done every two months, with any positive tests followed up by the patient's physician.

96 Selikoff has reported a marked synergistic effect between asbestos exposure and cigarette smoking, with the combination carrying a risk factor for bronchogenic carcinoma 92 times greater than that for the general population. However, this association has not been found regarding mesothelioma.

97 The clinical signs and symptoms, as well as the prognosis and treatment for asbestos induced carcinomas are the same as for any carcinoma. However, the physician should keep in mind that asbestosis symptoms may mask the onset of the carcinoma. Always be alert for an increase in chest pain or sense of restriction. Fibrotic changes may also mask early radiographic changes. Therefore, sputum cytology should be repeated frequently to enhance the possibility of early detection and treatment of those cancers that do result.

98 Since lag time between exposure and cancer appearance is usually 20 to 40 years, there will be many asbestos related cancers for the next 30-35 years, even if no harmful exposures have taken place since 1972.

99 The first mention of pleural cancer, following the 1947 British Study, was published by Wagner in 1960 in a report of 16 new cases of the once rare mesothelioma in South Africa. Of the 16, 6 had worked in the asbestos mines while the other 10 had merely lived in the mining area.

100 The latency period for these tumors is extremely variable, ranging from 3 to 55 years from onset of exposure, with a mean latency of 37 years. While

very heavy exposure favors a shorter latency, it is of great concern that exposures of 1 day to 1 month have been definitely associated with the developments of tumors years later.

101 Mesothelioma can involve both pleural and peritoneal surfaces. Tumors range in thickness from less than a centimeter over a limited portion of the lung, to large lobulated masses encasing the entire lung.

102 There are four distinct histological patterns to mesothelioma: Tubulo papillary, which is the type most likely to have secondary spread;

103 sarcomatous (pause 3 seconds);

104 undifferentiated polygonal cell of the epithelial type (pause 3 seconds), and

105 mixed.

106 Although mesothelioma may be insidious at the beginning, persons suffering from pleural tumors may have increased breathlessness without cough. When pain occurs, it often starts as a nagging pain, in the chest or shoulder, worsening as the disease progresses. In the later stages of the disease, the person usually suffers weight loss, lassitude and occasional night sweats

107 Clinical signs of a pleural tumor include unilateral pleural effusion and possible local tenderness on palpation with masses of tumor frequently palpable on the chest wall.

108 Radiographically, pleural mesothelioma may appear as a unilateral opacity projecting into the lung field from the costal and diaphragmatic margins. If seen early, however, it may be associated with evidence of rib erosion. Pleural plaques are fairly common in association with the tumor, but signs of asbestosis are unusual.

109 Signs and symptoms of the peritoneal tumor include diffuse swelling of the abdomen secondary to fluid, lethargy, upper and lower intestinal

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/23

obstruction, marked weight loss, a firm and distended abdomen with signs of ascites and, at times, signs of asbestosis in the lungs.

110 Metastases to hilar and abdominal lymph nodes are fairly common in cases of mesothelioma. Secondary spread is also found in the contralateral pleural lining, the liver, thyroid, adrenal glands and bone.

111 Procedures such as thoracotomy, thoracoscopy, laparotomy and aspiration tend to encourage the spread of the tumor into and outside the site of the incision causing rib erosion and invasion of the chest wall.

112 Mesothelioma is inevitably fatal, usually within 8 months of diagnosis. Radiotherapy, cytotoxic drugs and surgery are all ineffective. Treatment of the pain is the only suggestion we can offer. Keep in mind the needed emotional support of the patient and family, and encourage the patient to lead as normal and productive a life as possible. Other important points relating to asbestos are covered in depth in the text accompanying this module; but we would like to finish this program with a few words about some positive steps you can take as a primary care physician.

113 Be sure to incorporate a good occupational history while taking the general history; learn the signs and symptoms associated with asbestos-related disease.

114 If your patients smoke, urge them to quit. Smoking clinics and support groups are now available in most communities. Find out what industries in your area may involve exposure to asbestos and identify those of your patients who work there.

115 Urge the proprietors of asbestos-using industries to seek substitutes for asbestos, or to carefully enclose and ventilate those operations in which asbestos is determined to be absolutely essential. The proprietors should be informed of the potential health effects of asbestos, and potential liabilities should disease occur. And last, know where to refer a patient with an asbestos-related disease so that counselling regarding treatment and compensation may be secured.

RECENT PUBLICATIONS

Acheson, E.D., and Gardner, M.J., "Mesothelioma and Exposure to Mixtures of Chrysotile and Amphibole Asbestos," Archives of Environmental Health 34(4): 240-243, July - August 1979.

Becklake, M.R., "Environmental Exposure to Asbestos - A Factor in the Rising Rate of Cancer in the Industrialized World," Chest 76(3): 245-247, September 1979.

Health Hazards of Asbestos, Cancer Alert Series, OSHA 3040, U.S. Department of Labor, Occupational Safety and Health Administration, 1979.

"Health Hazards of Asbestos Exposure, A Symposium," Annals of New York Academy of Medicine 330, January 1980.

Michaels, L., and Chissick, S.S., (eds), Asbestos, Vol. 1, Properties, Applications, and Hazards, John Wiley and Sons, Ltd. (Chichester), 1979.

Samet, Jonathan M. et al, "Absence of Synergism Between Exposure to Asbestos and Cigarette Smoking and Asbestos," American Review of Respiratory Diseases 120(1): 75-82, July 1979.

Schaefer, G.L., "Asbestosis," The Western Journal of Medicine 131(1): 36-45, July 1979.

Wagner, J.C., "Diseases Associated with Exposure to Asbestos Dusts," The Practitioner 223(1333): 28-33, July 1979.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/25

SUGGESTED READINGS*

TEXT: Selikoff, I.J., and Lee, Douglas H.K., Asbestos and Disease, Academic Press, New York, 1978.

I. History of particular asbestos-related diseases

1. Cooke, W.E., "Fibrosis of the Lungs Due to Inhalation of Asbestos Dust," British Medical Journal 2: 147, 1924.

2. Lynch, Kenneth M., M.D., and Smith, W. Atmar, M.D., "Pulmonary Asbestosis III: Carcinoma of Lung in Asbesto-Silicosis," American Journal of Cancer 24(1): 56-64, 1935.

3. Merewether, E.R.A., "Asbestosis and Carcinoma of the Lung," Annual Report the Chief Inspector of Factories for the Year 1947.

4. Pancoast, M.K., et. al., "Pulmonary Asbestos: A Roentgenologic Study of the Effects of Dust Inhalation Upon the Lungs," British Journal of Medicine 2: 1024-1025, 1919.

II. General review articles

5. Becklake, Margaret R., "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," American Review of Respiratory Disease 114(1): 187-227, July 1976.

6. Haley, Thomas J., "Asbestosis: A Reassessment of the Overall Problem," Journal of Pharmaceutical Sciences 64(9): 1435-1449, September 1975.

7. Parkes, W.R., "Asbestos-Related Disorders," British Journal of Diseases of the Chest (London) 67(4): 261-300, October 1973.

8. Wright, G.W., "Asbestos and Health in 1969," American Review of Respiratory Disease 100(4): 467-479, October 1969.

---

*Entries in bold are highly recommended.

III. Government pamphlets: legislation, prevention, compensation

NIOSH publications

9. U.S. Department of Health, Education, and Welfare, "A Guide to the Work-Relatedness of Disease," NIOSH Publication No. 77-123, 1976.

10. U.S. Department of Health, Education, and Welfare, Revised Recommended Asbestos Standard, NIOSH Publication No. 77-169, December 1976.

11. U.S. Department of Health, Education, and Welfare, Asbestos: An Information Resource, NIOSH Publication No. 78-1681, May 1978.

IV. Epidemiology and clinical articles

12. Doll, R., "Mortality from Lung Cancer in Asbestos Workers," British Journal of Industrial Medicine 12(2): 81-86, April 1955.

13. Hasan, Faysal, et. al., "The Significance of Asbestos Exposure in the Diagnosis of Mesothelioma: A 28-Year Experience from a Major Urban Hospital," American Review of Respiratory Disease 115(5): 761-768, May 1977.

14. Kiviluoto, R., "Pleural Calcification as a Roentgenologic Sign of Non-Occupational Endemic Anthophyllite-Asbestosis, Acta Radiologica Supplementum 194: 1-67, 1960.

15. Newhouse, Muriel L., and Thompson, H., "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," British Journal of Industrial Medicine 22: 261-269, October 1965.

16. Newhouse, Muriel L., et al, "Predictions of Mortality from Mesothelial Tumors in Asbestos Factory Workers," British Journal of Industrial Medicine 33(3): 147-151, August 1976.

17. Newhouse, Muriel L., "Asbestos in the Workplace and the Community," Annals of Occupational Hygiene 16(2): 97-102, August 1973.

18. Selikoff, Irving J., M.D., et al, "Relation Between Exposure to Asbestos and Mesothelioma," New England Journal of Medicine 272(1): 560-565, March 18, 1965.

19. Selikoff, Irving J., M.D., "Epidemiology of Gastrointestinal Cancer," Environmental Health Perspecitves 9: 299-305, 1974.

20. Selikoff, Irving J., M.D., et al, "Asbestos Exposure, Smoking, and Neoplasia," Journal of the American Medical Association 204(2): 106-112, April 8, 1968.

21. Selikoff, Irving J., M.D., et al, "Asbestos Exposure and Neoplasia," Journal of the American Medical Association 188(1): 22-26, April 1964.

22. Wagner, J.C., et al, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," British Journal of Industrial Medicine 17: 260-271, October 1960.

V. Toxicology

23. Reeves, Andrew L., Ph.D., et al, "Experimental Asbestos Carcinogenesis," Environmental Research 4(6): 496-511, December 1971.

24. Wagner, J.C., "Asbestosis in Experimental Animals," British Journal of Industrial Medicine 20(1): 1-12, January 1963.

VI. Workers' Compensation

25. "Comments: Asbestos Litigation: The Dust Has Yet to Settle," The Fordham Urban Law Journal VII(1), 1978-79.

26. Letter on Asbestos and Workers' Compensation, Monitor 6(2): 7 Sept.-Oct. 1978.

27. "Litigation: Compensation Not Asbestos Victims Sole Remedy California Court Says" Occupational Safety and Health Reporter, Bureau of National Affairs: 1738, May 10, 1979.

28. "Medicine and the Law. Death of an Asbestos Worker" Lancet, November 12, 1966.

29. "Workers' Compensation: Federal Government Must Share Costs of Asbestos Disease, Industry Says" Occupational Safety and Health Reporter: 1738, May 10, 1979.

REFERENCES

1. Allison, A.C., "Effects of Silica and Asbestos on Cells and Culture," Inhaled Particles III, Walton, W.H. (ed.), Unwin Brothers, Ltd., Surrey, England, pp. 437-445, 1971.

2. Asbestos Information Association of North America, Asbestos General Information, Washington, D.C., 1975.

3. Auribault, M., Bulletin de l'inspection du Travail, p. 126, 1906.

4. Bader, M.E., et al, "Pulmonary Function and Radiographic Changes in 598 Workers With Varying Duration of Exposure to Asbestos," Mt. Sinai Journal of Medicine (New York) 37(4): 492-500, July-August 1970.

5. Balaam, L.N., and McCullagh, S.F., "Vital Capacity and One-Second Forced Expiratory Volume in Australian Male Asbestos Cement Workers," Annals of Occupational Hygiene 18(2): 133-141, September 1975.

6. Beck, E.G., et al, "Comparison of Effects on Macrophage Cultures of Glass Fiber, Glass Powder and Chrysotile Asbestos," British Journal of Industrial Medicine 29: 280-286, July 1972.

7. Becklake, Margaret R., "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," American Review of Respiratory Disease 114(1): 187-227, July 1976.

8. Becklake, Margaret R., et al, "Lung Function in Chrysotile Asbestos Mine and Mill Workers of Quebec," Archives of Environmental Health 24(6): 401-409, June 1972.

9. Burger, B.F., and Engelbrecht, F.M., "The Biological Effects of Long and Short Fibres of Crocidolite and Chrysotile A After Intrapleural Injection into Rats," South African Medical Journal (Cape Town) 44(44): 1268-1270, November 7, 1970.

10. Berry, G., et al, "Combined Effect of Asbestos Exposure and Smoking on Mortality from Lung Cancer in Factory Workers," The Lancet 2(775): 476-478, September 2, 1972.

11. Bruch, J., "Response of Cell Cultures to Asbestos Fibres," Environmental Health Perspectives 9: 253-254, 1974.

12. Cooke, W.E. (Wigan), "Pulmonary Asbestosis," British Medical Journal 2: 1024-1025, December 3, 1927.

13. Davies, P., et al, "Asbestos Induces Selective Release of Lysosomal Enzymes from Mononuclear Phagocytes," Nature 251(5474): 423-425, October 4, 1974.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/29

14. Davis, J.M.G., and Gross, P., "Are Ferruginous Bodies an Indication of Atmospheric Pollution by Asbestos?" Biological Effects of Asbestos, Leon France, International Agency for Research on Cancer, Scientific Publication No. 8, pp. 238-242, 1973.

15. Doll, R., "Mortality from Lung Cancer in Asbestos Workers," British Journal of Industrial Medicine 12(2): 81-86, April 1955.

16. Dreessen, W.C., et al, "A Study of Asbestosis in the Asbestos Textile Industry," U.S. Treasury Department, Public Health Service, Public Health Bulletin No. 241, August 1938.

17. Enterline, P.E., and Kendrick, M.A., "Asbestos Dust Exposures at Various Levels and Mortality," Archives of Environmental Health 15: 181-186, August, 1967.

18. Enterline, P.E., and Kendrick, M.A., "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers," British Journal of Industrial Medicine 30: 162-166, April 1973.

19. Epstein, S., The Politics of Cancer, Sierra Club Books, San Francisco, California, 1978.

20. Ester, I., "Asbestosis in Non-Experimental Animals in South Africa," Nature 197(4866): 506, 1963.

21. Ferris, B.G., et al, "Prevalence of Chronic Respiratory Disease: Asbestosis and Ship Repair Workers," Archives of Environmental Health 23(3): 220-225, September 1971.

22. Finklea, J.F., "Evaluation of Data on Health Effects of Asbestos Exposure and Revised Recommended Numerical Environmental Limits," December 1976.

23. Gross, P., and Harley, R., "Asbestos-Induced Intrathoracic Tissue Reactions," Archives of Pathology 96: 245-250, 1973.

24. Gloyne, S.R., "Two Cases of Squamous Carcinoma of Lung Occurring in Asbestosis," Tubercle 17: 5-10, October 1935.

25. Greenberg, Morris, and Davies, T.A. Lloyd, "Mesothelioma Register 1967 to 68," British Journal of Industrial Medicine 31(2): 91-104, April 1974.

26. Gross, Paul, et al, "Experimental Asbestosis: The Development of Lung Cancer in Rats with Pulmonary Deposits of Chrysotile Asbestos Dust," Archives of Environmental Health 15(3): 343-355, September 1967.

27. Gross, Paul, et al, "Ingested Mineral Fibers--Do They Penetrate Tissue or Cause Cancer?" Archives of Environmental Health 29(6): 341-347, December 1974.

28. Haley, Thomas J., "Asbestosis: A reassessment of the Overall Problem," Journal of Pharmaceutical Sciences 64(9): 1435-1449, September 1975.

29. Hammond, E.C., et al, "Neoplasia Among Insulation Workers in the United States with Special Reference to Intra-Abdominal Neoplasia," Annals of New York Academy of Sciences 132(1): 519-525, December 31, 1965.

30. Harries, P.G., et al, "Radiological Survey of Men Exposed to Asbestos in Naval Dockyards," British Journal of Industrial Medicine 29(3): 274-279, July 1972.

31. Harrington, J.S., et al, "Mineral Fibers: Chemical, Physicochemical, and Biological Properties," Advances in Pharmacology and Chemotherapy 12: 291-402, 1975.

32. Hasan, Faysal, et al, "The Significance of Asbestos Exposure in the Diagnosis of Mesothelioma: A 28-Year Experience from a Major Urban Hospital," American Review of Respiratory Disease 115(5): 761-768, May 1977.

33. Hatch, T.F., "Significant Dimensions of Dose Response Relationship," Archives of Environmental Health 16: 571-578, April 1968.

34. Hendry, N.W., "The Geology, Occurrences, and Major Uses of Asbestos," Annals of New York Academy of Sciences 132(1): 12-22, December 1965.

35. Hoffman, Frederick L., "Mortality from Respiratory Disease in Dusty Trades: Inorganic Dusts," Bulletin of United States Bureau of Labor Statistics No. 231, Industrial Accidents and Hygiene Series: No. 17. Washington, D.C., 1918.

36. Holt, P.F., et al, "Experimental Asbestosis With Four Types of Fibers: Importance of Small Particles," Annals of New York Academy of Sciences 132(1): 87-97, December 31, 1965.

37. Holt, P.F., et al, "The Early Effects of Chrysotile Asbestos Dust on the Rat Lung," Journal of Pathological Bacteriology 87(1): 15-23, January 1964.

38. Jodoin, G., et al, "Early Effects of Asbestos Exposure on Lung Function," American Review of Respiratory Disease 104(4): 525-535, October 1971.

39. Jones, H.B., and Grendon, A., "Environmental Factors in the Origin of Cancer and the Estimation of the Possible Hazards to Man," Food Cosmetic Toxicology 13(2): 251-268, April 1975.

40. Kanazawa, K., et al, "Migration of Asbestos Fibres from Subcutaneous Injection Sites in Mice," British Journal of Cancer 24(1): 96-106, March 1970.

41. Kaw, J.L., and Zaidi, S.H., "In Vitro Studies on the Cytotoxic Action of Different Varieties of Asbestos Dust on Macrophages," Acta Pharmacologica et Toxicologica 36(3): 225-235, March 1975.

42. Kiviluoto, R., "Pleural Calcification as a Roentgenologic Sign of Non-Occupational Endemic Anthophyllite-Asbestosis," Acta Radiologica Supplementum 194: 1-67, 1960.

43. Kleinfeld, M., et al, "Mortality Experience in a Group of Asbestos Workers," Archives of Environmental Health 15: 176-180, August 1967.

44. Knox, J.F., et al, "Mortality from Lung Cancer and Other Causes Among Workers in an Asbestos Textile Factory," British Journal of Industrial Medicine 25(4): 293-303, October 1968.

45. Lavappaks, Fumm, and Epstein, S.S., "Cytogenetic Studies on Chrysotile Asbestos," Environmental Research 10(2): 165-173, October 1975.

46. Lumley, K.P.S., "A Proportional Study of Cancer Registrations of Dockyard Workers," British Journal of Industrial Medicine 33(2): 108-114, May 1976.

47. Lynch, Kenneth M., M.D., and Smith, W. Atmar, M.D., "Pulmonary Asbestosis III: Carcinoma of the Lung in Asbestos-Silicosis," American Journal of Cancer 24(1): 56-64, May 1935.

48. Mancuso, Thomas F., M.D., and el-Attar, Anas A., M.B., "Mortality Pattern in a Cohort of Asbestos Workers: A Study Based on Employment Experience," Journal of Occupational Medicine 9(4): 147-162, April 1967.

49. McDonald, J. Corbett, et al, "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," Archives of Environmental Health 22(6): 677-686, June 1971.

50. McDonald, J. Corbett, et al, "Storage as a Factor in Disease Due to Mineral Dust," in Multiple Factors in the Causation of Environmentally-Induced Diseases, Lee and Kotin (eds), Academic Press, New York, p. 185, 1972.

51. McDonald, J. Corbett, et al, "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," Archives of Environmental Health 28: 61-68, February 1974.

52. McGill, T., and Stell, P.M., "Exposure to Asbestos and Laryngeal Carcinoma," Journal of Laryngology and Otology 89(5): 513-517, 1975.

53. Merewether, E.R.A., "Annual Report of the Chief Inspector of Factories for the Year 1947, London, Her Majesty's Stationery Office, 1949.

54. Merewether, E.R.A., "Asbestosis and Carcinoma of the Lung," Annual Report of the Chief Inspector of Factories for the Year 1947.

55. Merewether, E.R.A., and Price, C.W., "Effect of Asbestos Dust on the Lungs and Data Suppression in the Asbestos Industry," London, Her Majesty's Stationery Office, 1930.

56. Merewether, E.R.A., "Memorandum on Asbestosis," Tubercle 15(2): 69-81, November 1933; Tubercle 15(3): 109-118, December 1933; Tubercle 15(4): 152-159, January 1934.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/32

57. Meurman, L.O., et al, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," British Journal of Industrial Medicine 31(2): 105-112, April 1974.

58. Mineral Facts and Problems, U.S. Department of the Interior, Bureau of Mines, 1975.

59. "Minerals and Their Non-Asbestos Analogs," Electron Microscopy of Microfibers, Mineral Fibers Session, University Park, Pennsylvania State University, August 23-25, 1976.

60. Murphy, R.L.H., et al, "Effective Low Concentrations of Asbestos: Clinical, Environmental, Radiologic and Epidemiologic Observations in Shipyard Pipe Coverers and Controls," New England Journal of Medicine 285: 1271-1278, December 1971.

61. Murray, M., Report of the Departmental Comittee on Compensation for Industrial Diseases, London, Her Majesty's Stationery Office, pp. 127-128, 1907.

62. Newhouse, Muriel L., and Berry G., Asbestos and Laryngeal Carcinoma," Lancet 2:615, September 15, 1973.

63. Newhouse, Muriel L., "Asbetos in the Workplace and the Community," Annals of Occupational Hygiene 16(2): 97-102, August 1973.

64. Newhouse, Muriel L., "A Study of Mortality Workers in an Asbestos Factory," British Journal of Industrial Medicine 26(4): 294-301, October 1969.

65. Newhouse, Muriel L., et al, "A Study of the Mortality of Female Asbestos Workers," British Journal of Industrial Medicine 29(2): 134-141, April 1972.

66. Newhouse, Muriel L., and Thompson, H., "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," British Journal of Industrial Medicine 22: 261-269, October 1965.

67. Newhouse, Muriel L., et al, "Predictions of Mortality from Mesothelial Tumours in Asbestos Factory Workers," British Journal of Industrial Medicine 33(3): 147-151, August 1976.

68. Occupational Exposure to Asbestos: Criteria for a Recommended Standard, U.S. Department of Health, Education, and Welfare, NIOSH Publication No. 72-10267, 1972.

69. Pancoast, M.K., et al, "Pulmonary Asbestos: A Roentgenologic Study of Dust Inhalation Upon the Lungs," British Journal of Medicine 2: 1024-1025, 1919.

70. Parkes, W.R., "Asbestos-Related Disorders," British Journal of Diseases of the Chest 67(4): 261-300, October 1973.

71. Rahman, Q., et al, "Biochemical Changes Caused by Asbestos Dust in the Lungs of Rats," Scandinavian Journal of Work in Environmental Health 1(1): 50, March 1976.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/33

72. Reeves, Andrew L., Ph.D., et al, "Experimental Asbestos Carcinogenesis," Environmental Research 4(6): 496-511, December 1971.

73. Reeves, Andrew L., Ph.D., et al, "Inhalation Carcinogenesis from Various Forms of Asbestos," Environmental Research 8(2): 178-202, October 1974.

74. Reeves, Andrew L., Ph.D., "The Carcinogenic Effect of Inhaled Asbestos Fibers," Annals of Clinical and Laboratory Science 6(5): 459-466, September-October 1976.

75. Rossiter, Charles E., et al, "Radiographic Changes in Chrysotile Asbestos Mine and Mill Workers of Quebec," Archives of Environmental Health 24(6): 388-400, June 1972.

76. Schuster, Norah H., "Pulmonary Asbestos in a Dog," Journal of Pathology 34(2): 751-757, November 1931.

77. Seidman, H., et al, "Short-Term Asbestos Exposure and Delayed Cancer Risk," Proceedings of Third International Symposium on the Detection of Cancer, New York, 1976.

78. Selikoff, Irving J., M.D., and Douglas, H.K. Lee, Asbestos and Disease, Academic Press, New York, 1978.

79. Selikoff, Irving J., M.D., et al, "Asbestos Exposure and Neoplasia," Journal of the American Medical Association 188(1): 22-26, April 1964.

80. Selikoff, Irving J., M.D., et al, "Asbestos Exposure, Smoking, and Neoplasia," Journal of the American Medical Association 204(2): 106-112, April 8, 1968.

81. Selikoff, Irving J., M.D., et al, "Asbestosis and Neoplasia," American Journal of Medicine 42(4): 487-496, April 1967.

82. Selikoff, Irving J., M.D., "Cancer Risk of Asbestos Exposure," in Origins of Human Cancer, Cold Spring Harbor, New York, Cold Spring Harbor Laboratory, pp. 1764-1784, 1977.

83. Selikoff, Irving J., M.D., "Epidemiology of Gastrointestinal Cancer," Environmental Health Perspecitves 9: 299-305, 1974.

84. Selikoff, Irving J., M.D., et al, "Relation Between Exposure to Asbestos and Mesothelioma," New England Journal of Medicine 272(1): 560-565, March 18, 1965.

85. Shabad, L.M., et al, "Experimental Studies on Asbestos Carcinogenicity," Journal of the National Cancer Institute 52(4): 1175-1187, April 1974.

86. Sheers, G., and Tempelton, A.R., "Effects of Asbestos in Dockyard Workers," British Medical Journal 3(618): 574-579, September 7, 1968.

87. Sincock, A., and Seabright, M., "Induction of Chromosome Changes in Chinese Hamster Cells by Exposure to Asbestos Fibres," Nature 257 (5521): 56-58, September 1975.

88. Singh, J., et al, "Enzymatic Changes in Lung Tissue of Asbestotic Guinea Pigs," Environmental Physiology and Biochemistry 5(4): 267-272, 1975.

89. Sluis-Cremer, G.K., "Asbestosis in South African Asbestos Miners," Environmental Research 3(4): 310-319, November 1970.

90. Smith, W.E., "Experimental Studies on Biological Effects of Termolite Talc on Hamsters," U.S. Bureau of Mines Information Circulation (IC8639), pp. 43-48, 1974.

91. Stanton, M.F., "Some Etiological Considerations of Fiber Carcinogenesis in Biological Effects of Asbestos," Leon France International Agency for Research on Cancer, Scientific Publication No. 8, pp. 289-294, 1973.

92. Stone, M.J., "Clinical Studies in Asbestosis," American Review of Tuberculosis 41(1): 12-21, January 1940.

93. Storeygard, A.R., and Brown, A.L., "Penetration of the Small Intestinal Mucosa by Asbestos Fibers," Mayo Clinic Proceedings 52(12): 809-812, December 1977.

94. Thomson, J.G., et al, "Asbestos as a Modern Urban Hazard," South African Medical Journal 37: 77-81, January 19, 1963.

95. Thomson, J.G., and Graves, W.M., Jr., "Asbestos as an Urban Air Contaminant," Archives of Pathology 81(5): 458-464, May 1966.

96. Turner-Warwick, M., and Haslam, P., "Antibodies in Some Chronic Fibrosing Lung Diseases. I. Non-Organic Specific Autoantibodies," Clinical Allergy 1: 83, 1971.

97. Turner-Warwick, M., et al, "Antibodies in Some Chronic Fibrosing Lung Diseases. II. Immuno-fluorescent Studies," Clinical Allergy 1: 209, 1971.

98. Turner-Warwick, M., and Parkes, R., "Circulating Rheumatoid and Antinuclear Factors in Asbestos Workers," British Medical Journal 3: 492-495, August 1970.

99. Asbestos - Rules and Regulations, Federal Register 39(125): 25003-25005, June 1974.

100. U.S. Department of Health, Education, and Welfare, A Guide to the Work-Relatedness of Disease, NIOSH Publication No. 77-123, 1977.

101. U.S. Department of Health, Education, and Welfare, Asbestos: An Information Resource, NIOSH Publication No. 78-1681, May 1978.

102. U.S. Department of Health, Education, and Welfare, Bibliography, Chapter 3, No. 3-4.

103. U.S. Department of Health, Education, and Welfare, Occupational Diseases. A Guide to Their Recognition, NIOSH Publication No. 77-181, Revised Edition, June 1977.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/35

104. U.S. Department of Health, Education, and Welfare, Revised Recommended Asbestos Standard, NIOSH Publication No. 77-169, December 1976.

105. Vigliani, Enrico C., "The Fibrogenic Response to Asbestos," Medicina del Lavoro 59(6): 401-410, June-July 1968.

106. Wagner, J.C., "Asbestosis in Experimental Animals," British Journal of Industrial Medicine 20(1): 1-12, January 1963.

107. Wagner, J.C., et al, "Diffuse Pleural Mesothelioma and Asbestos Exposure in Northwestern Cape Province," British Journal of Industrial Medicine 17: 260-271, October 1960.

108. Wagner, J.C., et al, "Epidemiology of Asbestos Cancers," British Medical Bulletin 27(1): 71-76, January 1971.

109. Wagner, J.C., et al, "Mesothelioma Rats After Inoculation With Asbestos and Other Materials," British Journal of Cancer 28(1): 173-185, July 1973.

110. Wagner, J.C., et al, "The Effects of Inhalation of Asbestos in Rats," British Journal of Cancer 29: 252-269, March 1974.

111. Wagoner, J.K., "Occupational Carcinogenesis: The Two Hundred Years Since Percivall Pott," Annals of the New York Academy of Sciences 271: 1-4, May 28, 1976.

112. Wedler, H.W., "Asbestose und Lungenkrebs," Deutsch Medizine Wochenschrift 69: 575, August 6, 1943.

113. Wedler, H.W., "Uber den Lungenkrebs Bei Asbestose," Deutsch Archiv fur Klinische Medizine 191: 189-203, 1943.

114. Wehner, A.P., et al, "Chronic Inhalation of Asbestos and Cigarette Smoke by Hamsters," Environmental Research 10(3): 368-383, December 1975.

115. Weill, H., et al, "Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products," Journal of Occupational Medicine 15(1): 248-252, March 1973.

116. Westlake, George E., M.D., et al, "Penetration of Colonic Mucosa by Asbestos Particles: An Electron Microscopic Study in Rats Fed Asbestos Dust," Lab Investigation 14(11): 2029-2033, November 1965.

117. Whitwell, F., et al, "Relationship Between Occupations and Asbestos-Fibre Content of the Lungs in Patients With Pleural Mesothelioma, Lung Cancer, and Other Diseases," Thorax 32(4): 377-386, August 1977.

118. Wright, G.W., "Asbestos and Health in 1969," American Review of Respiratory Disease 100(4): 467-479, October 1969.

119. Zedda, S., et al, "Lung Function in Relation to Radiographic Changes in Asbestos Workers," Respiration 30: 132-140, 1973.

120. Hamilton, Alice, and Hardy, H., Industrial Toxicology, Haeber (New York), 1949.

121. "Physician's Advisory," Surgeon General and Assistant Secretary of HEW, April 25, 1978.

122. "Litigation: Asbestos Workers Sue 13 Firms For Alleged Exposure to Health Hazards," Occupational Safety and Health Reporter, Bureau of National Affairs, November 9, 1978.

Published by AsbestosClaims.Law
asbestosclaims.law/library/25480/37