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# OUTLINE -- ASBESTOS AND HUMAN HEALTH

I. Asbestos is generic term for a group of fibrous minerals

   A. Silicates of Mg, Fe, Na and/ or Ca.
   B. Filamentous
   C. Resistant to heat
   D. Resistant to chemical action - [illegible] - can be degraded by strong alkalies

II. Major types of asbestos

   A. Chrysotile(95% of world production
   B. Crocidolite (e.g. "Cape Blue")
   C. Amosite
   D. Anthophyllite
   E. Tremolite

III. World production of asbestos(tons)

| | |
|---|---|
| 1880 | 500 |
| 1900 | 35,000 |
| 1920 | 200,000 |
| 1930 | 450,000 |
| 1950 | 1,300,000 |
| 1960 | 2,300,000 |
| 1965 | 4,000,000 |

IV. Major uses of asbestos

   Cement products(pipes, sheets, shingles)
   Asphalt and vinyl asbestos floor tile
   Asbestos papers
   Millboards
   Roofing felts
   Brake linings
   Caulking compounds
   Insulating materials(pipe and boiler coverings,etc.)
   Spray asbestos
   Filters

V. Suspected or proven effects on health

   A. Asbestosis (asbestos pneumoconiosis)
   B. Bronchogenic carcinoma
   C. Mesothelial tumors of pleura and peritoneum
   D. Tumors of the gastrointestinal tract

Outline -- Asbestos and Human Health (continued)

VI. Asbestosis

A. Chronology

1900 -- First case described by H.Montague Murray
1907 -- Above case reported
1924 -- Other cases reported by W.E. Cooke
1930 -- Merewether found 81% of workers 30 years or more in British asbestos products industry had asbestosis(x-ray)
1938 -- U.S.Public Health Service studies by Dreessen et al set unofficial limits for dust exposure in asbestos industries not changed until 1968.
1963 -- Leathart in U.K., Selikoff in USA report high incidence of asbestosis in insulation workers; same confirmed in Bay Area 1964-1965.

B. Salient features

1. Pathologic changes: fibrosis around respiratory bronchioles and alveolar ducts; fibrosis of pleura; calcification of pleura; "asbestos" bodies;

2. Functional: reduced compliance, diminished VC and impaired diffusing capacity; increased pulmonary artery pressure, cor pulmonale

3. Time to develop: Heavy exposures 5-10 years; present levels of exposure: 20-30 years.

C. Outstanding current problems

1. Relationship between dose and response: what is safe level?
2. Different effects of different types of abbestos
3. Importance of pulmonary arterial effects
4. Frequency of delayed effects from early heavy exposures.

VII. Association of asbestos and lung cancer

A. Chronology

1935 -- Lynch & Smith reported single case in South Carolina.
1947 -- Merewether in UK stated 31 of 235(13.2% of certified cases of asbestosis 1924-46 died of cancer of lung; contrasted with 1.3% of 6,484 cases of silicosis.
1955 -- Doll studied 105 men who had worked 20 or more years with asbestos, concluded lung cancer risk 10 x that of general population
1958 -- Braun & Truan studied Quebec asbestos miners and mill workers; foudd no significant excess.
1959 -- McCullough updated UK study 63/365 asbestosis cases had died of lung cancer
1963 -- Mancuso studied cohort of 1,495 workers in asbestos products plant in 1938-39 using Social Security records; Thru 1960, lung cancer deaths were 3 x expected.
1963 -- Selikoff ffound lung cancer deaths in NY and NJ insulators in trade 20 years of more 6.6 times expected.
1964 -- Dunn found lung cancer deaths 3 x expected in California insulators.
1968 -- Cooper et al found 8 x expected in Bay Area insulators.

VII. (continued)

B. Salient features

1. Lower lobe predominance
2. Cell types not different from expected.
3. Apparent synergism with cigarette smoking

C. Unanswered questions

1. Importance of different types of asbestos
2. Role of cofactors, e.g. metals and hydrocarbons--is asbestos merely a carrier?
3. Dose and response relationships.

IX. Association of asbestos with mesothelioma of pleura and peritoneum

A. Chronology

1943 -- Wyers reported mesothelial tumor in thesis
1954 -- Leicher reported peritoneal mesothelioma
1955 -- Denoer reported 4 tumors in 72 cases of asbestosis
1960 -- Neal reviewed 15 deaths in London inwomen with asbestosis and found that 9 had had ovarian or peritoneal cancer
1960 -- Wagner, Sleggs and Marchand reported 33 diffuse mesotheliomata in So.Africa(NW Cape) in crocidolite areas.
1962 -- McCaughey et al found 15 in Belfast, in 12 of whom asbestos bodies were foundin sections and 9 had worked in shipyards.
1962 -- Maneuso reported 3 peritoneal mesotheliomata in a cohort of asbestos products workers.
1963 -- Selikoff reported 3 pleural mesotheliomata in NY insulators serie
1964 -- Enticknap and Smith reported 11 diffuse abdominal tumors associated with asbestos exposures
1965 -- Elmes and Wade expanded series(of McCaughey) to 45; 32 with history of light or heavy exposure to asbestos
1965 -- Jacob & Anspach reported 6 pleural tumors in Dresden asbestos workers
1965 -- Owen reviewed 17 pleural mesothelioma in Liverpool;12 had been industrially exposed to asbestos
1965 -- Selikoff et al. reviewed 2,500 autopsies; found 26 cases of asbestosis, & in these 4 pleural and 3 peritoneal mesothelioma found; reviewing AFIP files, 45 mesotheliomata. In 307 asbestos worker deaths(over 20 yrs in trade), 10 mesothelioma. In 31,682 deaths in ACS study, 3 pleural mesotheliomata.
1966 -- NewHouse and Thompson did matched case study in London; 51 of 76 mesotheliomata had some history of asbestos exposure.
1967 -- Bleban and Vistawka report 42 cases in Pennsylvania.

B. Salient features

1. Diagnosis difficult
2. Latent periods very long(30-40-years)
3. Usual questions regarding role of co-factors and dose-response relationships.

X. Association of asbestos with tumors of the GI tract

A. Chronology

1934 -- Selikoff et al reported 29 deaths from cancer of stomach, colon or rectum in a cohort of insulating workers at least 20 years in trade, compared with expected of 9.4; 3 x expected. Gastric cancer obs/exp was 12/4.3; colon or rectum 17/5.2
1963 -- Mancuso found slight excess of stomach, intestinal and rectal Ca
1965 -- Enterline found slight excess in asbestos textile workers.
1969 -- Selikoff's series now shows 37 compared to 11.2 expected.

B. Salient features

1. Insufficient data to begin defining role of asbestos type co-factors, or dosage relationships.

XI. Epidemiologic significance of the "asbestos body".

A. Chronology

1906 -- Coated fibers described by Marchand in France
1927 -- Cooke and Hill called coated fibers "curious bodies"
1929 -- Stewart and Haddow suggested term "asbestosis body"
1929 -- Cooke stated could be formed by other fibrous foreign bodies.
1951 -- Gloyne recommended "pneumoconiosis body"
1955 -- Gough suggested "mineral-fibre body"
1966 -- Cross suggested "ferruginous body"

1951 -- Simson and Strachan reported in 48 of 50 asbestos workers' sputum. regarded as index of exposure, not of disease.

1963 -- Thomson et al found 26.4% routine autopsy lung smears positive in Cape Town, only 4.4% had more than rare or few.
1965 -- Thomson and Graves found 27.2%+ in Miami, only 3.6% had many
1965 -- Cauna et al found 41% + in Pittsburgh, only 5% had many.
1965 -- Webster reported 39.2% + in Johannesburg.
1966 -- Meurman reported 57.6% of 264 + in Finland; 6.4% had many.
1966 -- Anilvel reported 48.6% + in Montreal, 4% had many.
1966 -- Cooper et al found 42% + in San Francisco.
1966 -- Chemni et al reported 54% of men and 44% of women in Milan series
1968 -- Ueidjian, Gross & deTreville reported 97% + in Pittsburgh, using digestion technique.
1969 -- Selikoff et al report little increase since 1934, more found in blue-collar than white collar workers.

B. Salient features

1. Proof of asbestos as central fiber difficult, conflicting reports from Pittsburgh and New York studies
2. Most positive cases have very few ferruginous bodies
3. Sources and significance unclear.