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PLAINTIFF'S EXHIBIT GP-152

UNITED STATES GOVERNMENT

Memorandum

U.S. CONSUMER PRODUCT SAFETY COMMISSION
WASHINGTON, D.C. 20207

TO : Don Clay, AED for Health Sciences DATE: June 3, 1977
THRU : Robert Hehir, Deputy AED for Health Sciences
Joe McLaughlin, Director, Division of Toxicology and Medicine
FROM : Rita Orzel, Ph.D., Division of Toxicology and Medicine
C. T. Desmond, M.D., Division of Toxicology and Medicine

SUBJECT: Toxicological/Medical Input for proposed Federal Register Notice on Asbestos

The Toxicological/Medical input on asbestos is presented in the attached document as a suggested CPSC Federal Register Notice for Asbestos.

We recognize that this is not the usual format for presenting such material. The time constraints were such that, for the sake of expediency, utilization of research background material previously prepared by OSHA, Federal Register Notice, Occupation Exposure to Asbestos, Vol 40, No. 197, page 47652-665 was used as the basis for this document.

This was determined to be the best method for accomplishing our assignment.

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I. BACKGROUND

A. General

Asbestos is a generic term used to describe a number of naturally occuring fibrous, hydrated mineral silicates that differ in chemical composition. These may be divided into two mineral groups; (1) Serpentines, which include chrysotile ($3MgO \cdot 2SiO_2 \cdot 2H_2O$), the type most widely used in U.S. industry; and (2) amphiboles, including amosite $(FeMg)\ SiO_3$), crocidolite ($NaFe(SiO_3)_2 \cdot FeSiO_3 \cdot H_2O$), tremolite ($Ca_2Mg_5Si_8O(OH_{22})_2$), anthophyllite $(MgFe)_7Si_8O_{22}(OH)_2$, and actinolite $(CaO \cdot 3MgFe)O \cdot 4SiO_2$). Asbestos fibers are generally characterized by high tensile strength, flexibility, heat and chemical resistance, and favorable frictional properties. Certain grades of asbestos can be carded, spun, and woven, while others can be laid and pressed to form paper, or used for structural reinforcement of materials such as cement, plastic, and asphalt.

Chrysotile (white asbestos) is the fibrous form of the mineral serpentine. It is the most common variety of asbestos, widely distributed geographically, with the largest deposits being in Canada, Russia, and Rhodesia. It accounts for over 90 percent of world consumption. Chrysotile can be readily crushed or fiberized into fine, white, silky fibers which may be processed into numerous products. The fibers have good heat resistance, but are destroyed by acids. Crocidolite (blue asbestos) is another important, although more specialized, form of asbestos.

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It is the fibrous form of riebeckite, and has fine, resilient fibers of a characteristic blue color. It is mined in South Africa and Australia, and to a lesser extent, in Bolivia. Crocidolite is a strong, fast filtering fiber used especially in the manufacture of asbestos cement sheets and pressure pipes. It is also characterized by its high resistance to acids.

Amosite is the fibrous variety of the mineral grunerite, a ferrous magnesium silicate mined only in South Africa. Amosite can be readily broken down into long, somewhat harsh fibers, with a brownish-yellow to almost white color, depending upon the quality. It is used largely in the production of asbestos cement and heat-insulating products. It is characterized by a good resistance to acids and other chemicals.

Anthophyllite is a magnesium silicate of somewhat variable composition which has rather fragile, brownish or off-white fibers. It is rarer than other types described, but significant quantities have been mined in Finland, Kenya, and other countries. It is used primarily as an inexpensive filler, and for some specialized applications for which good heat or chemical resistance is required.

Tremolite, a calcium magnesium silicate, may be a component of industrial and commercial talc. It is mined in various parts of the United States including New York, Vermont and Montana.

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Actinolite, a calcium magnesium iron silicate, is rarely used in industry. It is found in various parts of the world, but its low fiber strength makes it less desirable for industrial use.

Nearly one million tons of asbestos are consumed in the United States annually. According to the Bureau of Mines Mineral Yearbook, 1973,approximately 77 percent of asbestos products consumed in 1972 were used in the construction industries (624,000 short tons) while 23 percent are used in non-construction industries (186,000 short tons). Approximately 92 percent of the asbestos used in construction is firmly bonded, i.e., the asbestos is "locked in" in such products as floor tiles, asbestos cements, and roofing felts and shingles; while the remaining 8 percent is friable or in powder form present in insulation materials, asbestos cement powders, and acoustical products. These latter products generate more airborne fibers than the firmly bonded products. The 186,000 short tons of asbestos used in the non-construction industries in 1972 were utilized in such products as textiles, friction materials including brake linings and clutch facings, paper, paints, plactics, roof coatings, floor tiles, and miscellaneous other products.

II. TOXICOLOGICAL CONSIDERATIONS OF EXPOSURE TO ASBESTOS

A. EFFECTS OF ASBESTOS EXPOSURE

Asbestos, in its several commercial forms, has been shown in very recent times to be associated with the production

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of a variety of disease entities. These include:

1. Asbestosis: a diffuse, interstitial, nonmalignant, scarring of the lungs;

2. Bronchogenic carcinoma: a malignancy of the interior of the lung;

3. Mesothelioma: a diffuse malignancy of the lining of the chest cavity (pleural mesothelioma), or of the lining of the abdomen (peritoneal mesothelioma);

4. Cancer of the stomach, colon, and rectum.

Asbestos may be evident, in its advanced stages, by characteristic manifestaions on x-ray films, by restrictive pulmonary function, or by clinical signs, of finger clubbing or rales (dry, cracking sounds within the lung). Its most important symptom is dyspnea, or undue shortness of breath. The disease is progressive, even in the absence of further exposure, as those inhaled fibers which have been trapped within the lung continue their biological action. In its severe forms, death results from the inability of the body to obtain requisite oxygen or from the heart's failure to pump blood through the scarred lungs.

Mesothelioma tumors are diffuse and spread rapidly throughou the cavity of origin. They have yet to be successfully cured by any type of treatment including chemotherapy, radiation, or surgery. Death usually results within a year of diagnosis. It may account for one death in several thousand in the absence of an environmental or occupational

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asbestos exposure. In some groups of asbestos workers, it may account for one death in ten.

Once established, the other asbestos associated cancers differ little from those occuring in the general population, although there may be variations in the location of the primary site. Appropriate treatment and prognosis follow for the particular tumor. There is very limited long term survival from lung cancer therapy; and only somewhat better from treated cancer of the colon or rectum.

Asbestosis and asbestos cancer whether it be lung cancer, pleural mesothelioma, peritoneal mesothelioma, cancer of the stomach, colon, rectum-usually do not become clinically evident until more than 20 years have passed from onset of exposure. This time-interval is now widely recognized.

While some such cancers may appear during the second decade following onset of occupational exposure, peak incidence is often not noted until the 30-years-from-onset point, or later. This is true both with regular, long-term, brief or intermittent exposures. While variations in the time of occurrence may depend upon intensity and duration of exposure, with heavier exposure often being associated with shorter latency periods, variations among individual cases make it impossible to predict the latency period for the risk of any particular person.

B. RECENT DOCUMENTARY EVIDENCE OF OCCUPATIONAL EXPOSURE

(1) Asbestosis. Subsequent to the hearings on the

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current Occupational Safety and Health Administration (OSHA) standard, uncertainty has arisen as to whether the existing British asbestos standard and the mandated 2 fiber/ml U.S. standard provide effective protection even against asbestosis. The data from Great Britain obtained in 1966 indicated that little clinical disease, including x-ray evidence of asbestosis, had occurred among workers first employed in that factory at some time after 1933, when important improvements in work practices had been achieved. In 1972, results of evaluation of new x-rays that had been taken in 1970, of the work force then employed in the same factory, were reported as showing that many now had abnormal findings either in the lung or in the coverings of the lung pleurae (Lewinsohn, 1972). There was thus a difference between the prevalence of abnormal x-ray findings among workers x-rayed in 1966 as reported to the British Occupational Hygiene Society, and evaluation of other x-rays of workers in the same factory four years later.

Additionally, clinical data are becoming available concerning asbestos lung scarring in individuals exposed at levels much lower than those of occupational circumstances. Among 210 family contacts of former asbestos factory workers, 38% have been reported to have x-ray changes characteristic of asbestos exposure (Anderson, Selikoff, Lilis and Daum, 1975).

(2) Cancer. In December 1972, important new information on the spectrum of asbestos cancers was presented

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at the Conference on the Biological Effects of Asbestos, sponsored by the International Agency for Research on Cancer of the World Health Organization. At this conference, and subsequently, data on large groups of asbestos workers became available (Selikoff, Hammond and Seidman, 1973; Enterline, de Coufle and Henderson, 1972). As expected, the high risk of bronchogenic carcinoma and mesothelioma persisted among factory employees and insulators. Moreover, these later studies confirmed the excess gastrointestinal cancer that had been suggested earlier, and extended the spectrum of asbestos related cancers.

(a) Lung cancer. The most important cancer afflicting asbestos workers is cancer of the lung, although mesothelioma has attracted considerable attention because of the high frequency among asbestos workers and infrequent occurrence in the population as a whole.

In many groups of asbestos workers, approximately 20% of all deaths are caused by lung neoplasms. This has been true both among asbestos product factory workers (Selikoff, Hammond and Churg, 1972; and Nicholsor 1976) and among users of these products (Selikoff, Hammond and Seidman, 1973). The exact percentage varies with circumstances of exposure, age of the workers, duration of the workers' exposure and, and perhaps most of all, according to the duration from the onset of their asbestos work history. In addition, the last several years have seen the discovery of another critical variable affecting the incidence

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of lung cancer among asbestos workers. In 1968, Selikoff, Churg and Hammond reported that lung cancer was not significantly increased in incidence among asbestos workers with no history of cigarette smoking, although when such history was present, the incidence of lung cancer increased markedly over what would be expected among other cigarette smokers, in the absence of asbestos exposure. Thus, these scientists calculated that an asbestos worker who smoked cigarettes had 92 times the risk of dying of lung cancer, as compared with like individuals without cigarette smoking or asbestos work. This finding has been confirmed by larger studies (Hammond and Selikoff, 1973) where, again, it was found that non-smoking asbestos workers had few lung cancers while those who smoked had much more lung cancer than would have been expected had they not been asbestos workers. Calculations suggest that cigarette-smoking asbestos workers have approximately eight times the risk of developing lung cancer compared to other smokers.

(b) Pleural and peritoneal mesothelioma. In 1960, Wagner, Sleggs and Marchand demonstrated an important association between asbestos exposure and pleural mesothelioma. This cancer, which appears to be unrelated to smoking, had previously been considered to be a very rare tumor. Numerous reports have confirmed the finding of Wagner and his colleagues that mesothelior can be commonly associated with asbestos exposure. A subsequent report by Enticknap and Smither, 1964, concerning workers in a British asbestos factory demonstrated that the same tumor could be commonly

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found in the abdomen (peritoneal mesothelioma), as well as in the chest.

The exact risk of death of these invariably fatal neoplasms has not been as well defined as has lung cancer, although recording of cases from hospitals near one large asbestos factory has indicated that it must be very common indeed (Borow, Conston, Livornese and Schalet, 1967). Information available from the experience of asbestos insulation workers suggests that approximately five to seven percent of deaths may be due to this neoplasm (Hammond, Selikoff and Churg, 1965; Selikoff, Hammond and Seidman, 1973). More recently, it has been suggested that this estimate is too low, on the basis of the experience of workers in a British asbestos factory where calculations predicted that between 10 and 11 percent of deaths would be due to mesothelioma (Newhouse and Berry, 1975).

(c) Gastro-intestinal cancer. Gastro-intestinal cancers (cancer of the stomach, colon and rectum) are also increased in incidence among asbestos workers, A number of studies now indicate that the increase is on the order of two or three times the number of expected tumors (Selikoff, Hammond and Seidman, 1973; Elmes and Simpson, 1971). Although this increased risk is relatively limited, especially when compared with lung cancer and mesothelioma, it is nevertheless of considerable importance since a two - or three-fold increase in such common tumors becomes an important cause of death for the workers involved.

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It has been suggested that other tumors are also increased in incidence among asbestos workers, particular cancers of the larynx (Stell and McGill, 1973; Newhouse and Berry, 1973), and of the esophagus (Selikoff, Hammond, and Seidman, 1973). However, data concerning these neoplasms are less extensive than for lung cancer, mesothelioma and gastro-intestinal cancer and further experiences are awaited. In any case, they are not very common tumors in general and any increase does not weigh heavily on the overall cancer risk of asbestos workers.

Considering all neoplasms, among some groups of asbestos workers, employed either in asbestos factory work or in the use of asbestos products, as much as 40 to 45 percent of all deaths have been due to one or another type of cancer, an approximately three-fold or four-fold increase.

Of significant importance, new data have recently been made available concerning the cancer risk of workers at the textile mill reviewed for the British standard, including those workers first emloyed after 1933 (Howard, Kinlen, Lewinsohn, Peto and Doll, 1975). It was found that there was excess mortality from lung cancer among those workers who entered scheduled areas after 1 January 1933. There was "clear evidence of some excess of lung cancer and respiratory deaths among those first exposed between 1933 and 1950." Equally important was the finding that "there still

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appears to be an excess of deaths due to lung cancer after 15 or more years' exposure" even among those first exposed in 1951 and subsequently. Indeed, it is known that mesothelioma deaths have occurred among the specific group of 290 workers whose experience prior to 1966 had led to the development of the current standard as detailed above (Brody, 1974).

C. NON-OCCUPATIONAL, INDIRECT OCCUPATIONAL EXPOSURE OR INTERMITTENT OR BRIEF EXPOSURE

Recent data indicate the occurrence of asbestos cancer among individuals exposed to low levels of asbestos, as in environmental circumstances, or to brief or intermittent exposures to higher levels.

Analysis of the history of asbestos exposure among individuals in a large series of cases of mesothelioma in Great Britain and South Africa have provided evidence that brief or intermittent exposure to asbestos may, after the passage of decades, result in mesothelior (Greenberg and Davies, 1974; Webster, 1973). In such circumstances, it appears that the lifetime exposure was less than 100 fiber-years/ml. The same discrepancy between present projected exposures and the risk of asbestos cancer exists when considering cases of mesothelioma resulting from household contact to asbestos among members of families of asbestos workers (Lillington, 1974) or among residents living in the vicinity of asbestos plants.

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Of considerable industrial importance, has been the recent description of asbestos disease among shipbuilding and ship repair workers, few of whom actually work with asbestos, but many of whom were, in the past, inadvertently exposed to the asbestos dust resulting from the use of asbestos products by a relatively few of their work mates. In 1968, Harries of the Royal Navy reported cases of mesothelioma among shipyard workers at the Royal Navy dockyard in Devonport, in trades which did not directly involve worker exposure to asbestos, but in which there had been occasional opportunity for exposure merely by virtue of working in the same areas. This original finding has been widely confirmed and numerous cases of mesothelioma have since been reported in former shipyard workers (Whitwell and Rawcliffe, 1970; McEwen et al, 1970; Stumphius, 1971; Greenberg and Davies, 1974). Studies of populations of current shipyard workers have shown much radiological evidence of asbestos abnormalities among workers in trades only indirectly exposed to asbestos in the yards (Sheers and Templeton, 1968; Fletcher, 1972).

Gillam et al (1975), studying the mortality and reviewir the chest x-rays of 439 underground metal miners exposed to an asbestiform mineral, found three times the risk of malignant respiratory disease than expected. The fiber concentrations averaged 0.24 fibers/r

Further, evidence has indicated that asbestos also acts as a lung carcinogen at levels much below those

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which will produce asbestosis. Two surveys of shipyard workers who had x-ray evidence of pleural plaques, but generally not of pulmonary fibrosis, showed a 2.5-fold excess risk of death from lung cancer and high risk of mesothelioma. (Fletcher, 1972; Edge, 1975). In a study of the mortality experience of a large U.S. asbestos products manufacturing facility, it was found that workers in low-dust areas, with a minimum risk of death from asbestosis, had the same high risk of death from various cancers as workers in dustier areas (Nicholson, 1975).

Reports of the hazard, or potential for hazard, from exposure to asbestos resulting from use of asbestos-containing products are very limited. To determine the type and extent of non-occupational exposure, the investigator must generally rely on relatives or persons other than the exposed patient for exposure history.

Furthermore, it is extremely difficult, if not impossible, to document brief exposures reported to have occurred 20 or more years before the onset of symptoms. The only known quantitative study of asbestos levels in products regulated by this Commission was reported by Rohl, et al (1975) of asbestos fiber concentrations measured during the use of consumer spackling, patching and taping compounds. This study indicated that airborne fiber concentrations, exceeding the interim OSHA allowable excursion exposure level, were detected during application and cleanup operations. Fibers were detected in adjacent rooms during mixing

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operations and it was reported that "... significant concentrations of asbestos remained suspended and could pervade living quarters for a considerable duration of time..." The authors suggest that the use of spackling and other patching compounds (in mixing, sanding and cleanup operations) may expose the user and other members of the household to "... significant concentrations of asbestos".

III. CERTAIN CONSIDERATIONS CONCERNING CARCINOCENICITY

In the case of asbestos, we are dealing with a substance that poses a range of health risks. These include the threat of cancer, as well as asbestosis. In considering the issue of carcinogenicity the data cited above should be considered along with leading scientific principles and opinions believed to reflect the research conclusions of international cancer experts.

A. LATENT EFFECTS

In humans, the latency period for chemical carcinogens may well extend between 20 to 40 or more years. Analogou periods exist for test animals. This means that the disease may undergo a long period of development before a tumor is actually detected. Prudence would seem to dictate that every reasonable measure should be taken to eliminate human exposure to chemical compoun as soon as their carcinogenic nature is identified.

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B. INDIVIDUAL VARIATION

Cancer development may be influenced by such factors as the differing susceptibility of various body organs. In animal studies it has been found that individual variability in response to carcinogens is great depending upon factors such as age, sex, hormonal status, diet, and genetic factors. Thus, individuals biologically compromised, may be more susceptible than other groups.

C. "THRESHOLD" LIMIT

Because of the variability of individual response to carcinogens and other factors, the concept of a "no effect" or "threshold level" may have little real significance on the basis of existing knowledge. While some level, below which exposure to a carcinogen does not cause cancer, may conceivably exist for any one individual, other individuals may have cancer induced by doses so low as to be effectively zero. This is not to say that researchers will never find a threshold level for a carcinogenic substance, but it does mean that the threshold concept for carcinogens is, at present, more a matter of responsible regulatory policy than a precise, scientific determination.

These theoretical concepts have a bearing on the asbestos issue, particularly as the question of the existence, or nonexistence, of a threshold level of carcinogenic effect. A "no effect" level theoretically may exist, but it has not been demonstrated. Therefore, there is no known threshold level below which exposure to asbestos would be considered safe.

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The use of asbestos as simulated ash in artificial fireplace logs and in consumer patching compounds poses an unreasonable risk of inhalation of potentially hazardous asbestiform fibers and are exposures which are avoidable.

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Atmospheric Env. 7:481-483 (1973)
Air sampling done in a number of cities from Iceland to S. Africa. Asbestos fibers found in minute amounts in all. Suggest a potential risk in the presence of higher concentrations because lung mechanisms cannot handle & coat large number of fibers.

23. Hueper, W.C. Occupational & non-occupational exposures to asbestos

NY Acad of Sci 132:184-195 (Dec 1975)
For an assessment of the potential scope of an environmental health hazard and occupational & non-occupational implications of asbestos exposure, it is necessary to determine the distribution pattern of the agent from the site of its original occurrence and production (also production figures are included).

24. Huff, J.E. Asbestos: An Overview

Env. Chemicals - Human & Animal Health 3rd Annual Conference Proceedings (1974)
A review of the literature on the occupational & environmental hazards of asbestos.

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25. IARC Working Group on the Evaluation of the Carcinogenic Risk of Chemicals to Man: IARC Monographs on the Evaluation of the Carcinogen Risk of Chemicals to Man: Asbestos Intl. Agency for Research on Cancer. 1977.

A review of asbestos and associated health hazards. This includes experimental studies, sources of exposure and evaluation of the carcinogenic risk to man.

26. Kleinfield, M. Biologic Response to kind & amount of asbestos.

Joccup Med. 15(3): 296-300 (Mar 73)
Discusses various factors established & theoretical in the production of asbestosis and asbestos related cancers.

27. Kleinfeld, M et al Mortality among talc miners & millers in New York State

Arch. Env. Health 14:663-667 (May 1967)
Malignancies 4 times greater in this group of 220 with 15 years exposure than expected. No mesotheliomas. Significant increase occurred 20 yrs later (60-79 yrs) than in asbestos workers (40-59 yrs)

28. Kleinfeld, M et al A study of workers exposed to asbestiform minerals in commercial manufacture

Env. Res. 6:132-143 (1973)
In a total of 74 workers exposed to talc containing tremolite & anthophyllite fibers over a mean time of about 6 years, no malignancie are reported. Two different studies 2 exposure levels.

29. Langer, et al Identification of asbestos in human tissues

J. of Occ. Med 15(3): 287-295 (Mar 1973)
Discusses methodologies used in asbestos identification.

30. Langer, et al (Selikoff) Inorganic fibers including chrysotile in lungs at autopsy: preliminary report

Inhaled Part. Vap. 2:683:694 (1970)
Chrysotile fibers break down chemically and separate into 30-40 nm wide fibers in vivo. The amphiboles have not been observed to do this.

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31. Langer, Rohl Selikoff et al Inorganic particles in cigarettes and cigarette smoke (1974)

Science 174:585 (1971) Inorganic particles in cigars & cigar smoke; Hammond, Langer, et al.
Briefly present some support for hypothesis that the inorganic components of tobacco smoke play some role in the etiology of lung fibrosis.

32. Langer, Selikoff, et al Chrysotile asbestos in the lungs of persons in NYC

Arch Env. Health 22:348-361 (Mar 1971)
Data from 28 consecutive NYC autopsies: Chrysotile found in 24.

33. Libshitz, H.I. et al Asbestosis of carcinoma of the larynx

JAMA 228:1571-72 (1974)
"Three patients had carcinoma of the larynx, history of asbestos exposures and evidence of intrathoracic changes characteristic of asbestosis. Suggest relation between asbestosis and laryngeal carcinoma.

34. Leben, J. Mesothelioma & asbestos Exposure

Arch Env. Health 14:559-563 (Apr 67)
Study of extent of relationship of asbestos exposure to mesothelioma diagnosed in S.E. Penn.

35. Lillington G.A. et al Conjugal Malignant Mesothelioma

New Eng. J. Med. 291(11): 583-84 (Sept. 12, 1974)
Report of an industrial worker with malignant mesothelioma and that of his wife whose exposure resulted from washing his dirty clothes.

36. Masson, T.J. Asbestos like fibers in diluted water supply

JAMA 228(8): 1019-1020 (1974)
No carcinoginic effect was noted from patterns of cancer mortality in a period of 14 years.

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37. McCullagh S.F.: The Biological Effects of Asbestos
Med. J. Aust. 2:45-49 (1974).
In the urban atmosphere of both the U.S. and England, "The amount of asbestos is between one hundred thousandth and one millionth of that held to be safe in the workplace (according to the authors).

38. McLachlin, MSF et al Radiological diagnosis of crocidolite-induced pleural mesotheliomata in the rat.
Br. J. of Exp. Path. 55:164-168 (1974).

Thirty-six rats received crocidolite intrapleurally. Seven rats developed mesothelioma as diagnosed by X-ray. Three others were misdiagnosed.

39. Miller, Langer, Feinstein & Selikoff Non-Specific Interstitial Fibrosis.
New Eng J. of Med. 292(2): 91-93 (Jan. 9, 1975).

Report finding of chrysotile asbestos fibrils by electron microscopy in a patient described as having severe interstitial fibrosis with no known pathogenic dust exposure. Authors suggest EM examination of lung tissue in diagnoses of "idiopathic pulmonary fibrosis".

40. Mackish, D.E., et al: Exposure to Asbestos During Brake Maintenance.
Ann. Occup. Hyg. 10, pp.17-21 (1970).

Air samplings were made during blowing-out procedures of brake shoes and drums. Measurements of the dust cloud exceed the fiber standard but personal exposure measurements were below the standard.

41. Maggiori, Nicholson & Rubin Neighborhood Contamination with Asbestos. Analysis of settled dust as index of asbestos air pollution in prior Decades.

Environ. Sci. Lab. Annual Report (1974).

42. Martisching, K.J. et al Unsuspected Exposure to Asbestos and Bronchogenic Carcinoma.
Brit. Med. J. 1:746-749 (1977).

Asbestos regulations have reduced the risk of exposure to workers in scheduled industries, but asbestos-related diseases will probably be increasingly found among the many workers who have only incidental exposures to asbestos.

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43. Murphy R.L.H. et al. Effects of low concentration of asbestos

N. Eng. J. Med 285(23): 1270-1278
Shipyard pipe coverors, exposed to 5 million particles per cu. ft. for 13 yrs. showed asbestosis 11 times more commonly than the controls and a 38% prevalence after 30 yrs.

44. Murphy R.L. et al. Floor tile installation as a source of asbestos exposure

Am Rev. Resp. Dis. 100(4) Oct. 1971
Case studies of 2 long time floor tile installers with mesothelioma and pleural complications known to be associated with exposure to asbestos.

45. Newhouse, M.L. et al. Epidemiology of mesothelioma tumors in the London area

ANN of the NY Acad of Sci 132:579-588 (Dec. 65)
Elaboration of earlier case study report. Seems to be little doubt of risk of domestic and occupational exposure to asbestos.

46. Newhouse, M.L. et al. Predictions of mortality from mesothelial tumors in asbestos factory workers.

Brit J. of Ind. Med. 33:147-151 (1976)
It was projected that in the London asbestos textile factory cohort studied, 7-11% of the total mortality would be men and slightly more women would die of mesothelioma.

47. Newhouse, M.L.. Asbestos in the workplace and the community

ANN Occup Hyg 16:97-107 (1973)
Summary of many reports, case studies, exposure rates, asbestos in the environment and notes on non-occupational exposure.

48. Newhouse, M.L. et al. Mesothelioma of Pleura and Peritoneum following exposure to asbestos in the London area

Brit J. Ind. Med. 22:261-269 (1965)
A case study of 83 patients from London Hospital with confirmed mesotheliomas. Of these 9 were relatives of asbestos workers.

49. Nicholson & Rohl. Occupational and community asbestos exposure from wallboard finishing compounds (73-74)

To be publ. Health Res. Council of NYU 2331
Dry wall tapers and painters breathe in the dust from spackling compounds i.e. fiber counts during mixing and sanding operations varied from the TLV to several times.

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50. Nicholson, Rohl & Weisman. Asbestos contamination of building air supply systems

From Env. Sci Lab, Dept. of Community Med., Mount Sinai School of Med of the City Univ of NY
Asbestos found in the air of those buildings previously fire proofed with asbesti form sprays. Recommendations are made for future monitoring and control.

51. Nicholson, Rohl Weisman. Asbestos contamination of the air in public buildings. Final Rpt to EPA, 1975

Same as asbestos contamination of building air supply sustems.

52. Nicholson & Rubin. Asbestos lung tissue burden following neighborhood exposure-evaluation of carbon extraction technique

To be pub support NCI-CP 43255
A vague outline of the technique is presented. It was considered inadequate for the purpose of the study.

53. Nicholson, W.J. Asbestos - the TCV approach

Annals of the NY Acad of Sci 271:157-169 (1976)
No known threshold for asbestos or other carcinogens, below which no health effect may be manifested. The TLV is risk limitation value.

54. Polednak, A.P. Latency periods in neoplastic diseases

J. Epid 100(5):354-6 (Nov 74)
Latency = period between exposure to a presumed causal agent and detection of (or death from) a neoplasm. Author suggests 2 models cumulative exposure; one-insult co-carcinogenic

55. Pooley, F.D. Asbestos bodies, their formation, composition and character

Env. Res 5:363-379 (1972)
Formation did not appear to take place or fibers less than 10 u in length and less on crocodolite.

56. Reeves, A.L. et al. Inhalation Carcinogenesis from various forms of asbestos

Env. Res 8:178-202 (1974)
Rats, rabbits, guinea pigs, gerbils and mice exposed to 3 air concentrations of chrysolite, crocodolite and amosite for 2 yrs. Only the rats showed lung or pleural carcinogenic response.

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57. Richards, R.J. et al. Ultrastructural changes in lung fibroblast cultures exposed to chrysotile asbestos

But J. of Exp. Path. 55:275-284 (1974)
EM exam showed chrysotile fibers within some cells by 4 1/2 hours with nuclear and cytoplasmic changes.

58. Richards, R.J. et al. Collagen and mucopolysaccharide production in growing lung fibroblasts exposed to chrysotile asbestos

Life Sciences 12(II):441-451 (1973)
Cultures exposed to chrysotile and glass fibers for 24 days. Fiber size not stated. Glass caused slight reversible changes. The chrysotile caused increases in collagen and muco polysaccharide levels indicative of fibrogenesis.

59. Rohl, A. Mineralogical analysis of ore from a major US vermiculite deposit:preliminary investigation

Unpublished - Supported by Health Res. Council 4-12-72
Author claims that potentially hazardous concentrations of asbestos may be found but he had not yet quantitated his analysis.

60. Rohl et al. Asbestos in strange places; children's arts and crafts materials, paper products (1973)

To be pub. Support NIEHS ES 00928
Asbestos fibers of different types, forms and amounts were found in various paper products.

61. Rohl, Langer, Selikoff et al. Consumer talcums and powders: mineral and chemical characterization

J of Tox & Env Health 2:255-284 (1976)
Twenty-one commercial consumers U.S. Talcs & powders were analyzed between 1971 and 1975. Methods were instrumental and chemical. An appreciable number showed various forms of asbestiform fibers. No mention of product names nor the dates of acquisition.

62. Rohl, Langer, Selikoff & Nicholson. Exposure to asbestos in the use of consumer spackling, patching and taping compounds.

Science 189:551-553 (Aug. 15, 1975)
A number of consumer spackling compounds purchased in NYC were found to "contain asbestos minerals as well as other biologically active substances" ("quartz, talc and other minerals with disease potential")

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63. Seidman, Lilis & Selikoff. Short term asbestos exposure and delayed cancer risk

Abstract from Mount Sinai School of Medicine of City Univ of NY, 5th Ave & 100th St., NY, NY 10029
Occupational exposure to amosite in a cohort followed from 1941-1945 to 12-31-74. Claim their figures support "fact" that exposure of less than one year duration "results in significant excess risk of lung cancer."

64. Selikoff, et al. Asbestosis and neoplasia

Am J. Med 42(4):487-496 (Apr 67)
What this group has written in more detail elsewhere. Presentation of data - 1973.

65. Selikoff, et al. Asbestos exposure smoking and neoplasia

JAMA 204 #2:106-112 (Apr 8, 1968)
Early paper relating incidence of malignancies in smoking or non-smoking insulation workers.

66. Selikoff & Hammond. Community effects of nonoccupational environmental asbestos exposure

Am J. Pub. Health 58(9):1658-1666 (Sept. 68)
A cautious and scientific evaluation of the many factors involved in properly assessing and assigning cause.

67. Selikoff, et al. Asbestos air pollution

Arch of Env. Health 25(1):1 (1972)
Discussion of the usual i.e. asbestos air pollution level of disease hazard, epidemiological consideration industry responsibility.

68. Selikoff. Widening perspectives of occupational lung disease.

Prev. Med 2:412-437 (1973)
A review of past and present work in the various pneumoconioses and possible relationship to lung cancer.

69. Selikoff. Cancer risk of insulation workers in the U.S. (1974)

Insulation Hygiene Progress Reports Vol 6 #3 (Fall 1974)
Reprint of a Chapter of IARC volume by Selikoff et al. Primarily a summary of what was said before relating to cancer in insulation workers.

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70. Selikoff. The asbestos exposure of insulation workmen

Ins. Hyg Progress Rpts Vol 6 #1 (Spring 1975)
Discusses past data and concludes there is no significant protection to be offered workmen even if standards are lowered to levels less than 5 f/ml.

71. Selikoff, I.J. Lung cancer and mesothelioma during prospective surveillance of 1249 asbestos insulation workers 1963-1974

Annals of the NY Acad of Sci Vol 271:448-456 1976
Prospective surveillance with the likelihood of earlier diagnosis failed to appreciably improve prognosis in the disease.

72. Selikoff, et al. Carcinogenicity of Amosite Asbestos

Arch of Env. Health 25:183-186 (1972)
Statistical data is presented to support their statement that occupational exposure to amosite asbestos can be associated with serious cancer hazard.

73. Shabad, L.M. et al. Experimental Studies on Asbestos Carcinogenicity

J of NCI 52(4):1175-1187 (1974)
Carcinogenic activity of different types of asbestos of the USSR investigated. All chrysotile showed traces of benzopyrene.

74. Shin M.L. & Firminger H.I. Acute and chronic effects of interperitoneal injuction of 2 types of asbestos in rats ...

Amer J. Pathology 70(3) 291-230 (1973)
Basically a morphological study of tumor development. 50 mg 2 types of fiber injected with 40% mortality first 8 days and first mesothelioma in chrysotile group at 7 months.

75. Thiess, A.M. Dangers to man arising from toxic substances at the workplace.

Zbl.Bart Hyg I Abt Orig B
Article in German Toxic dust levels set including asbestos.

76. Thompson, J.G. Asbestos and the urban dweller

Ann of the NY Acad of Sci 271:196-214 (1976)
500 autopsy cases in both Cape Town, South Africa and Miami, Florida showed 30% males and 20% females had evidence of inhaling asbestos fibers. Inhaled fibers may accumulate at lung bases - says author.

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77. Wegman, D.H. et al. Worker sponsored survey for asbestosis. Detection of occupational lung disease sans a control group

Arch Env. Health 27:105-109 (1973)
Fifty-seven wall board manufacturing workers were examined by questionnaire, pulmonary function tests and limited physical exam. By predictive equations and regression analysis, disease was proved without a control group.

79. Wagner, J.C. et al. Mesothelioma in rats after innoculation with asbestos and other materials

Br. J. Cancer 28:173-185 (1973)
Intrapleural injection of asbestos into rats produced mesotheliomata approx. proportional to the dose and apparently the finer fibers the more carcinogenic.

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