

Asbestos has been responsible for a massive epidemic of disease and death since its commercial exploitation primarily beginning at the turn of this century. As we enter the new millennium do we intend to promote the myth that only the amphibole types of asbestos were responsible for the disease and death associated with asbestos usage? Asbestos, a natural mineral fiber, has been used for a variety of purposes with disastrous consequences to human health. The fact that Austria, Belgium, England, The Czech Republic, Denmark, Finland, France, Germany, Italy, the Netherlands, New Zealand, Poland, Saudi Arabia, Sweden, and Switzerland have all banned asbestos, leads us to recognize that these countries feel the safe use of asbestos is not attainable and that alternative materials posing less risk to public health are desirable.

Further substantiation that asbestos cannot be used safely comes from the most recent International Programme for Chemical Safety Environmental Health Criteria 203-Chrysotile Asbestos (IPCS, 1998). The document concluded "Exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose dependent manner. No threshold has been identified for carcinogenic risks." It further warns that "Some asbestos- containing products pose particular concern and chrysotile use in these circumstances is not recommended." "Construction materials are of particular concern for several reasons. The construction industry workforce is large and measures to control asbestos are difficult to institute. In- place building materials may also pose risks to those carrying out alterations, maintenance and demolition. Minerals in place have the potential to deteriorate and create exposures."

The conclusions of the IPCS are very consistent with the evaluation of 'the amphibole hypothesis carded out by Stayner, Dankovic and myself in 1996 (Stayner et al., 1996). However, there are still today others that claim chrysotile asbestos is not as harmful as the amphiboles and can be used safely and should not be banned (McDonald, 2000). We are at a point in the history of asbestos usage when chrysotile is the predominant type of asbestos produced and consumed in the world today; it constituted about 98.5% of US consumption in 1992 (Pigg, 1994). While it is true that asbestos consumption had declined in both the US and Europe, sales to other countries (e.g., Southeast Asia, South America, and Eastern Europe) has increased based on its usage in construction materials, the very materials that IPCS has warned against using (Lemen and Bingham, 1994). Our review of the lung burden, epidemiologic, toxicologic, and mechanistic studies, which provide the basis for the amphibole hypothesis lead us to conclude that chrysotile asbestos exposure carries an increased risk of both lung cancer and mesothelioma and that the hypothesis that these observations may be attributable to trace amounts of tremolite contamination may seem to be primarily of academic interest, because chrysotile exposures in workers and the public are also contaminated with tremolite (Stayner et al., 1996).

The primary evidence for the amphibole hypothesis comes from pathologic studies in which lung burdens were measured. However, interpretation of these studies is hampered by the fact that chrysotile lung burdens are a poor reflection of integrated exposures and

the fact that chrysotile exposure is highly correlated with lung burden of the amphiboles (e.g., tremolite). In addition, that pattern of asbestos fiber deposition in the lung does not appear to be consistent with the pattern of deposition in the target tissue (i.e., pleura). Suzuki et al. in a review of 92 consecutive cases of mesothelioma found that even while only 28.3% of the asbestos fiber type in the lung was chrysotile, it was the major fiber type identified in the mesothelial tissue (Suzuki et al., 1998). These findings further suggest that lung burden analysis for determining fiber type in mesothelioma etiology may not be appropriate and that determining predominate fiber type in the mesothelial tissue is the more rational determinant.

Therefore, with this knowledge at hand, is it not our moral and ethical responsibility, as scientists, to use this knowledge to call for a ban on the usage of asbestos in order to prevent a prolonging of an epidemic that could have been prevented, should have been prevented and that has already taken a disastrous toll? Many countries, such as those now importing asbestos are at the very early stages of receiving new technologies, including construction materials made of asbestos. Many of these countries are still trying to improve their basic public health infrastructures by improving sanitation, reducing infant mortality, improving health care delivery, etc. For these countries, the introduction of such new technologies, as asbestos-containing-construction products, can represent a very critical, and off times overwhelming adjustment.

HISTORY

The use of asbestos dates back thousands of years when asbestos fibers were being incorporated into pottery as early as 2500 B.C. (Agricola, 1556; Noro, 1968). The modern industry dates from about 1880, when asbestos was used to make heat and acid resistant fabrics (Hendry, 1965; and Hueper, 1965). By the late 1800's and early 1900's the use of asbestos was being widely advertised. Johns-Manville ran full-page advertisements in several publications, like the January 13, 1906 issue of *The Saturday Evening Post* saying "Serves More People in More Ways than any Institution of its kind in the World."

Highlights in the production history of asbestos include its use as heat insulation as early as 1866 (Bowles, 1937); asbestos cement used as a boiler covering in 1870 (_____, 1903); opening of the first asbestos factory in Great Britain in 1871 (_____, 1953); commercial production of asbestos insulation materials in 1874 (_____, 1903); the first processing of Canadian asbestos into textiles in the U.S. in 1890 (Berger, 1963); the asbestos cement pipe industry had its origins in Italy at the turn of the twentieth century (_____, 1973); asbestos cement production in the U.S. began in 1903 (Berger, 1963); flat asbestos cement board was produced in the U.S. in 1904 (_____, 1958); asbestos was first used as a brake lining in 1906 (_____, 1953); the first pipe making machines were imported into the U.S. in 1928 (Berger, 1963); asbestos spraying first began in tunnels in 1932 (Lancet, 1932) and, asbestos spraying of deckheads and bulkheads began in British Navy Ships in 1944 (Harries, 1968; and Harries, 1971). In 1963 the spraying of British Navy ships with asbestos was discontinued (Harries, 1968; and Harries, 1971).

LUNG DISEASE

The first recorded case of asbestosis was reported, in London, by a Charing Cross Hospital physician Dr. Montague Murray in 1906 (Murray, 1907). It is interesting to note that Adelaide Anderson, Lady inspector of Factories included asbestos among the dusts known to cause injury to man, in a 1902 publication on dangerous industries in England (Anderson, 1902). In 1918, American and Canadian insurance companies would not insure asbestos workers due to the un-healthy conditions in the industry (Hoffman, 1918). In 1912 the American Association for Labor Legislation mentioned asbestos related disease in their *Industrial Diseases* (_____, 1912), as did the government of Canada Department of Labour (1912). Pancoast et al. (1917) first commented in the medical literature on x-ray changes in 15 individuals exposed to asbestos. Case reports of asbestosis were reported by Fahr (1914), Cooke (1924) and Seiler (1928). The first complete description of asbestosis, including the naming of the disease and a description of “curious bodies”, observed in lung tissue, appeared in 1924 and 1927 (Cooke, 1924; McDonald, 1927; Cooke, 1927; Cooke and Hill, 1927; and Oliver, 1927). Mills (1930) reported the first case of asbestosis in the United States and in the same year Lynch and Smith (1930) reported on “asbestos bodies” found in the sputum of asbestos exposed workers. Other early studies led many investigators to conclude that people exposed to asbestos dust developed the disease “asbestosis” (Merewether and Price, 1930; Merewether, 1930, 1933; Wood and Gloyne, 1930; Wood and Page, 1930; Soper, 1930; Lynch and Smith, 1930; Sparks, 1931; Russell, 1932; Ellman, 1933; Wood and Gloyne, 1934; Donnelly, 1934; Egbert, 1935; Fulton et al., 1935; Lanza et al., 1935; Donnelly, 1936; Egbert & Geiger, 1936 and Dreessen et al., 1938). Merewether and Price (1930) in their report found 26 percent of the asbestos textile workers examined with pulmonary fibrosis and for those with greater than 20 years exposure, 80% had x-ray abnormalities. In 1930, 1931 and 1933, Ellman reported asbestosis cases among production workers and described the slow development of the disease with the patient often free of symptoms for several years. The Ellman (1933) report even carried the case of asbestosis in a 10 year old rough-haired terrier dog used as a ratter in an asbestos factory which had been discussed earlier in a paper by Schuster (1931). Other studies followed describing the latent period for disease development. Also, these studies demonstrated the longer the duration of exposure the greater the risk of developing disease. They also demonstrated disease occurrence after short periods of exposure, with radiographic changes occurring in workers exposed less than one year (Stewart et al., 1931; Wood & Gloyne, 1934). It is of interest to note that in the study by Lanza et al. (1935) of asbestos textile workers, 53 percent were found to have had fibrosis (lung scarring); 58 percent of workers with 10-15 years exposure and 87 percent of workers with over 15 years exposure had x-ray changes. Lanza (1936) also mentioned that the first official claim for asbestosis, in the United States, was in 1927. McPheeters (1936) described continued exposure to asbestos could increase the fibrosis in existing asbestotics and reported some evidence that asbestosis develops more rapidly in younger persons.

Asbestosis is not specific to humans and has occurred in animals other than under experimental situations. Besides the terrier described above by Ellman (1933) and

Schuster (1931), Webster (1963) described asbestosis in donkeys hauling asbestos ore. Environmentally induced asbestosis has also been found in field rats living in and around an asbestos mill and also in baboons living near an asbestos mill (Webster, 1963).

CANCER OF THE LUNG & MESOTHELIOMA

Lynch and Smith (1935) in the United States and Gloyne (1935) in the United Kingdom both associated occupational asbestos exposure with lung cancer. German physicians began calling lung cancer an occupational disease of asbestos workers (Nordmann, 1938). Reports appeared in the medical literature of lung cancers occurring in asbestos exposed workers (Egbert and Geiger, 1936; Gloyne, 1936; Middleton, 1936; Hornig, 1938; Koelsch, 1938; Hueper, 1942; JAMA, 1944; Merewether, 1949; JAMA, 1949; and Wyers, 1946). In 1947, the Chief Inspector of Factories in Great Britain reported on all of the known fatal cases involving asbestosis [235], in Great Britain, where 13% were due to lung cancer when only 1% of all deaths in the general population were due to this cause. Among women known to have asbestosis, 8% had lung cancer; at that time very few women smoked and female lung cancer was quite rare (Merewether, 1949). Epidemiological evidence from Doll in 1955, showed a ten-fold excess of lung cancers in those United Kingdom asbestos textile workers who had been employed before 1930 (Doll, 1955). This study was pivotal for confirming the causal association of asbestos exposure and lung cancer.

Between 1943 - 1946 reports of pleural (chest) and peritoneal (abdominal) tumors (mesotheliomas) associated with asbestos exposures appeared (Wedler, 1943 a, b and Wyers, 1946). Mesothelioma of the pleura with no fibrosis of the lung was found on pathologic examination (Mallory et al., 1947). In 1949, further reports of mesothelioma and its association with asbestos exposure were reported (Doig, 1949; Merewether, 1949; & Wyers, 1949). By 1952 other cases of pleural mesothelioma were reported in Canadian chrysotile mine employees without asbestosis (Smith, 1952 and Cartier, 1952). In 1960 a major study by Wagner et al. of miners, millers, and transporters of asbestos and of non-mining residents looked at 47 cases, occurring between 1956 and 1960 and found primarily mesothelioma of the pleura occurring in one part of South Africa, the northwestern portion of the Cape Province, known to have many asbestos mines. Their study confirmed epidemiologically an association between exposure to asbestos and mesothelioma. The fact that environmental exposures were also occurring demonstrated the fact that low-level, non-occupational exposures to asbestos could be hazardous (Wagner et al., 1960). The first studies in the United States, to report mesothelioma with asbestos exposure were by Mancuso and Coulter, of factory workers, in 1963 and by Selikoff et al., in 1964, of insulation workers.

CHRYSOTILE ASBESTOS AND IT'S HEALTH EFFECTS - Do They Differ From Those Of The Amphiboles?

Chrysotile fibers are much more chemically and biologically reactive than amphibole fibers and because of this reactivity with the tissues, they lose their structural elements

and divide into smaller fibrils, making their recognition difficult by the usual analytical methods. In fact, many of the fibers are removed from the lung and exhaled back through the bronchi or removed by the lymphatic system to other organs of the body (Marten et al., 1989; Davis, 1979; Davis et al., 1986a; and Davis et al., 1986b). The concentration of dust in the lungs of rats exposed to Canadian chrysotile was only 1.8% - 2.2% of the dust concentration in the lungs of animals exposed to amphiboles (after 24 months of inhalation exposures). Yet the lung tumor incidence and degrees of pulmonary fibrosis were similar in all groups. These findings support the idea that chrysotile fibers cause more cellular injury, fibrosis and lung cancer, than the amphiboles, while at the same time is less readily detected in the tissue after the damage is done. Churg et al. (1989a) concludes that the failure of chrysotile to accumulate in the lung is a result of preferential chrysotile clearance during the first few days to weeks after exposure and that dissolution plays no role in the clearance and that the preferential clearance may be a result of fragmentation and rapid removal of the chrysotile fibers. Remember that I mentioned earlier that Suzuki et al. (1998) in studying 92 consecutive cases of mesothelioma observed that the major asbestos type identified in the mesothelial tissues was chrysotile when compared to the chrysotile fiber burden in the lungs of the same cases (79.0% vs. 28.3% respectively). Malorni et al. (1990) suggests that fiber penetration can rearrange the cytoskeletal apparatus of the cell and that this could indicate an interaction between the chrysotile fibers and the normal mitotic process, since giant multinucleated cells are formed. Churg et al. (1989b) further believes that the short fibers may be more fibrogenic than previous animal data suggest and deserves further study.

INTERNATIONAL SCIENTIFIC ORGANIZATIONS PLAY A GREAT ROLE IN THE NEW GLOBAL ECONOMY BY PROVIDING GUIDANCE IN PUBLIC HEALTH

Castleman & Lemen (1998) in a paper on international scientific organizations, report their vulnerability to “manipulation” by special interest groups including business, trade, and professional societies. These international scientific organizations have long been important sources of reports about toxic substances. With the globalization of trade and information, new problems have arisen from the efforts of business interests to influence bodies such as the International Labor Office (ILO) and the World Health Organization (WHO). With the emergence of the World Trade Organization as a forum where an individual nation’s occupational and environmental health regulations can be challenged, the pronouncements of such groups as ILO and WHO take on added significance. Publications of such groups, long important for providing information especially to countries with scant public health resources, may now also be used to both misinform such countries and even overturn worker and environmental protection measures in the most advanced countries.

Recent efforts to manipulate international organizations follow, which should help to better understand how best to guard against such improprieties and maintain scientific objectivity within the affected organizations. I intend to give just one example, as others at this conference will give you many more, of such manipulation, and this is one that I played a major role in exposing.

The International Program on Chemical Safety

Watterson reported serious threats to the “public credibility” of the International Program on Chemical Safety (IPCS) in 1993. IPCS is located at the World Health Organization in Geneva and is jointly sponsored by WHO, ILO, and the United Nations Environment Program. Watterson reported that manufacturers ICI, Hoechst, and DuPont wrote the first drafts of IPCS reports on chlorofluorocarbon refrigerants and the fungicide benomyl. He raised concerns over undisclosed conflicts of interest by corporate consultants on expert task groups assigned to write IPCS documents. He reported that industry “observers” usually present at IPCS task group meetings were rarely offset by representatives of non-industrial, non-governmental organizations.(1)

The same year, U.S. government scientists found that the IPCS environmental health criteria document on methylene chloride was based on material drafted by officials from ICI and other manufacturers of the chemical. Scientists from the U. S. National Institute for Occupational Safety and Health (NIOSH) criticized the IPCS’ failure to modify statements in the report to reflect opposing views within the expert panel. As a result, NIOSH decided to cease all participation in IPCS activities until IPCS established an objective process to develop criteria documents.(2)

The scientific community’s reaction broadened with the refusal of the Collegium Ramazzini to review drafts of the IPCS criteria document on chrysotile asbestos, prepared by “scientists with close ties to the asbestos industry.” By refusing to become involved at a late stage in the process, the Collegium said it intended to avoid inappropriately associating itself with the report.(3) The effort to issue a chrysotile report remained mired in controversy for years, and publication is finally expected in 1998.

At the invitation of the German government, IPCS held an “MCS Workshop” in Berlin in 1996. Led by corporate consultants and chemical industry “observers,” the panel decided, by an unrecorded vote, to rename Multiple Chemical Sensitivities (MCS) “idiopathic environmental intolerance’s.” The panel chairman (Dr. H. Kipen) and Dr. C. Miller from the U.S. were among those who objected to the name change. The corporate consultants returned from the conference, promptly representing the workshop’s anonymous and un-referenced conclusions and recommendations as WHO policy, at medical meetings, in court documents, and in media announcements. An industry-funded “research institute” then paid for the publication of the recommendations in a journal supplement, without the knowledge of IPCS, which still has not published the workshop report. A position statement was drawn up by chemical industry consultants for the American Academy of Allergy, Asthma, and Immunology, using the new chemical-free name of MCS and referring to the workshop as a WHO symposium.

A pattern of such events led 81 scientists to send a strong letter to IPCS and its United Nations sponsors decrying the corporate influence (Abrams et al., 1996). That in turn led the U.S. government agencies that had relations with IPCS to recommend, through the U.S. State Department, 12 specific changes in IPCS procedures.(Claussen, 1996). The WHO responded by drafting “declaration of interest” guidelines, including a disclosure form to be completed by participants in expert scientific panels. IPCS participated in this

process and circulated the draft guidelines for comment in October of 1997. In the meanwhile, IPCS sent a very incomplete third draft of its report on chrysotile asbestos to the expert panel charged with writing the final report. This was a more balanced and qualified group, and they extensively revised the draft, adding much material that had not been cited. The Canadian scientist selected by IPCS to chair the panel was persuaded to step down. The sole observer at the week's end, Dr. Graham Gibbs, had listed himself as representing the fibers committee of the International Commission on Occupational Health. Because the panel saw him as representing the positions of the asbestos industry, Gibbs was asked to leave the room during the writing of the concluding parts of the report.

PREVENTION OF ASBESTOS - INDUCED DISEASE – ANYTHING LESS THAN A BAN DOES NOT WORK!!!! YOU KNOW IT AND I KNOW IT!!!!!!

The earlier researchers said and I paraphrase.

It has long been known that suppression of dust was the best method to control diseases associated with exposures to dusts and was described by both Ramazzini, 1713 and Oliver, 1902. In the United Kingdom, the Chief Inspector of Factories, in London, recommended to one factory, after having experienced five deaths due to phthisis (lung scarring disease), exhaust ventilation and annual medical examinations (The Chief Inspector of Factories, 1910). Merewether and Price (1930) were among the very first to set forth very specific recommendations for dust suppression in the asbestos industry that included: 1) application of efficient localized exhaust ventilation at dust producing points; 2) substitution of enclosed mechanical methods for hand conveyance, and for dusty hand work generally; 3) effective enclosure of dust-producing machines; 4) substitution of wet methods instead of dry material handling; 5) elimination of certain dust-producing appliances; 6) abandonment of settling chambers in manufacturing processes, to the utmost extent; 7) effectual separation of processes to prevent unnecessary exposure to dust; 8) wide spacing of dust-producing machines in new factories and, as far as practicable, in existing works; 9) use of sacks of close texture material for internal work; 10) efficient cleaning systems with wide use of vacuum methods; 11) storage of asbestos and other goods outside workrooms; and 12) exclusion of young persons from especially dusty work. Safety Engineering magazine ran an article in 1931 on "The Very Least an Employer Should Know about Dust and Fume Diseases" warning that dust including asbestos could be seriously harmful and that controlling the dust was necessary (Willson, 1931). McPheeters (1936) suggested engineering dust controls methods for the prevention of asbestosis. Many others have also given methods for preventive actions from hazardous dusts, including the classic work of the United States Public Health Service (Bloomfield and Dallavalle, 1935 and Dreessen et al., 1938). For asbestos disease control Lanza et al. (1935) also described the serious hazard faced by the industry with dust and recommended studies on its control as related to disease prevention. However, not much attention was given to hygiene, as can be seen in the account by Selikoff and Greenberg (1991).

The National Cancer Institute's pioneer cancer researcher Wilhem C. Hueper, as early as 1942, in his historic book **Occupational Tumors and Allied Diseases** recommended controlling asbestos by methods of wetting, closed production, ventilation or other engineering controls, as well as personnel protective devices (Hueper, 1942). Fleischer, et al, in 1946, expanded this advice and gave even more extensive guidance for dust control to end product users of asbestos containing materials, when in their study of the construction of naval vessels, recommended wetting the material, exhausting the dust where possible, employing respirator usage by the workers, isolating dusty operations in order to protect other workers not directly working with asbestos, and providing room ventilation. Fleischer et al. concluded, "There are no established figures for permissible or safe dustiness in pipe covering operations." They also describe that "During the handling, unwrapping and unrolling of the asbestos [material], considerable dust arises, but appears to settle readily. A very fine water spray should be used for wetting down the material as a high velocity spray stirs up dust." Pertaining to the use of saws, used to cut the end product, Fleischer et al. recommend that ". . . the band saw should be enclosed in a room by itself and should be equipped with adequate local exhaust ventilation both above and below the saw table." Further, Fleischer et al. point out that end product users such as ". . . asbestos pipe covering differs markedly from the asbestos textile industry where dust concentrations for an operation do not fluctuate widely and where a worker will usually remain at a specific job for some years."

Therefore, in conclusion, we not only have observed, but we can say with sound scientific evidence, that we have known the dangers of asbestos for many, many years but along with that knowledge we have also known, for many, many years, some methods to prevent the adverse health effects from exposure to asbestos. We know today that these methods do not assure that all will be protected from the awful effects of exposure to asbestos. They simply do not work. As participants of this conference, armed with the knowledge we will gain from this conference, we must now take this knowledge, as we have learned here, along with the knowledge we have known from years past to promote primary prevention in order to halt this continuing – and I might add growing tragedy of asbestos induced diseases and deaths. We know that dust suppression methods alone do not work and that even at the lowest exposure concentrations recommended, to date, such as the 0.1 fiber/cc concentration in the United States, that more than 59,000 workers will still die, in the United States, over the next thirty years, exposed at this concentration. We therefore have no choice but to **BAN** this deadly substance, **ASBESTOS**, from commercial use if we are to stop this growing epidemic of death and disease.

I will conclude by quoting the very eminent British public health statistician, Sir Bradford Hill who said in 1965 - AND I MIGHT ADD THIS STILL APPLIES TODAY:

“All scientific work is incomplete - whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone action that it appears to demand at a given time.”

We have done the research (on Asbestos) - we already know the only way to control diseases associated with asbestos is to **BAN ASBESTOS** use, not in the future - but now, as Sir Bradford Hill so apply says “... **NOT TO IGNORE THE KNOWLEDGE WE ALREADY HAVE, OR TO POSTPONE ACTION THAT IT APPEARS TO DEMAND AT A GIVEN TIME.**” **A total ban on asbestos is the only solution.**

THAT TIME IS NOW AND THE ACTION WE MUST TAKE IS CLEAR.

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