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Strangulation of Asbestos Industry: Cui Prodest?

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Abstract

Evaluation of asbestos-related health risks has been partly based on the experience when the content of asbestos fibers in the workplace air was higher. Fibers are present in the environment because of erosion of surface deposits and human activities unrelated to the asbestos industry. If searched purposefully, fibers are often found at autopsies. Results of many studies are biased. When fibers are detected, mesothelioma or lung cancer is sometimes classified as asbestos-related, although the causal relationship remains unproven. Some studies rely on unverified history of professional or domestic contact with asbestos. Reliable data can be obtained in experiments by recording the average life duration of animals. There are industrial interests behind chrysotile. Different types of asbestos have their technical advantages and preferred applications. Certain amphiboles are acid-resistant, thermally stable and durable. It can be reasonably assumed that non-use of asbestos-containing brake linings, fire-resistant and insulating materials will lead to increased damage from road accidents, fires and armed conflicts. Certain environmentalists should be aware that their activities may be used to disadvantage their countries. Strictly observed realistic safety rules would be more useful for the public healthcare than excessive restrictions that would be neglected in some parts of the world, bringing to the trespassers economic and strategic advantages.

Keywords: Asbestos; Chrysotile; Amphiboles, Mesothelioma; Lung Cancer.

Introduction

The assessment of asbestos-related health risks is based on historical experience from the time when airborne fiber concentrations in workplaces were higher than they are today. The linear no-threshold hypothesis - like that in the field of radiation protection - was used, even though its applicability to low doses remain unproven and contested about both pleural tumors and lung cancer [1,2]. Most of the high-dose occupational exposures to asbestos, in the United States ended approximately 40 years ago. Exposures to asbestos from new products, if any, are regarded to be negligible [2]. Asbestos fibers are present in the natural environment because of the erosion of surface deposits [3,4]. Natural emissions contribute to the dispersal of chrysotile and amphibole fibers, in some places exceeding the anthropogenic contribution to atmospheric fiber dispersion [3,5]. Human activities unrelated to the asbestos industry, such as road construction, tunneling, mining, and residential development on asbestos-bearing land amplify the dispersal. Agricultural activities can cause asbestos particles in the soil to decompose and become airborne. Fibers can be transported by air and water, spreading in the environment [6-9]. In a study conducted in Milan, asbestos fibers were detected in 35 out of 55 (63.6%) routine autopsies [10]. In another study from Italy, asbestos fibers were detected in 22.2% of cases with

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no documented history of occupational or environmental exposure [11]. During autopsies of individuals with a history of asbestos exposure, a greater number of lung and pleural tissue samples are collected for histological analysis, the examination is performed meticulously using specialized methods. Consequently, fibers are detected more frequently than in routine autopsies. The detection of fibers does not prove occupational asbestos exposure or a causal role for asbestos in the etiology of the diseases. The inhalation and clearance of fibers from the respiratory tract are normal processes maintained in a dynamic balance [10,12]. By analogy with other environmental factors, one can hypothesize the existence of a harmless (threshold) concentration of fibers in the ambient air. Experimental data support the existence of thresholds for cancer and other diseases [13,14]. Screening and medical check-ups have obviously contributed to an increased detection rate of mesothelioma and lung cancer among individuals exposed to asbestos. Many studies lack sufficient objectivity. For instance, lung and pleural tumors are sometimes classified as asbestos-related upon the detection of fibers, even though a causal link remains unproven. Some studies rely on questionable medical history data regarding occupational or domestic asbestos exposure and on interviews with the relatives of deceased individuals [15].

Malignant pleural mesothelioma (mpm)

The stable incidence of MPM in some developed countries, despite asbestos bans [16-18], is partly attributable to improvements in diagnostic equipment, the effects of screening, and overdiagnosis resulting from the lack of clear delineation of MPM as a distinct disease entity. In addition to asbestos, etiological factors for MPM include various mineral and man-made fibers, the SV40 virus, ionizing radiation, chronic inflammation (empyema, tuberculosis), genetic predisposition, and spontaneous mutations [19-22]. According to the Helsinki Criteria, intended to assess the causal link between mesothelioma and asbestos, even a brief or low-level exposure should be considered sufficient for mesothelioma to be classified as occupation related [23]. This approach leads to misclassification of cases due to other causes as asbestos related [24]. Several fibrous silicates and synthetic materials exhibit mesothelioma-inducing potential. For example, erionite is considered a more potent carcinogen than asbestos. Human activities lead to the spread of erionite and other potentially carcinogenic fibers in inhabited areas [7-25]. Concerns also extend to man-made fibers, carbon nanotubes and other nanomaterials that share physical properties with asbestos, in particular, high biopersistence. Replacing asbestos with man-made fibers will not necessarily eliminate health risks [6-21]. Nanoparticles can induce structural changes in membrane proteins and activate the synthesis of inflammatory mediators, thereby disrupting normal cellular metabolic mechanisms [26]. Experiments have demonstrated the carcinogenic effect of nanotubes [22,27]. Certain types of carbon nanotubes have been classified as possible human carcinogens [28]. There is evidence that the SV40 virus has contributed to the rising incidence of mesothelioma in recent decades. SV40-like DNA sequences are frequently detected in MPM. Following laser microdissection, SV40 was found in MPM cells but not in surrounding tissues [29]. Following SV40 inoculation, ≥50% of hamsters developed mesothelial tumors; after injections into the pleural cavity, mesotheliomas developed 100% of the hamsters [19]. The rise in MPM incidence in the 1960s coincided with exposure to the virus during the 1955-1963 period (and later in some countries), when polio vaccines contained viable SV40 [29]. Antibodies to SV40 were detected in the serum from 34% of MPM patients, compared with 20% of healthy individuals ($p < 0.05$). This indicates the role of SV40 in the etiology of MPM and suggests that the virus circulates within the population [30]. Invasive procedures - particularly bronchoscopy, which is more frequently performed in high-risk groups - have contributed to the spread of SV40 and occurrence of additional MPM cases. Bronchoscopy with biopsy has been recommended in Russia for suspected cases of asbestos-related bronchitis, pneumoconiosis and pneumonia [31-33]. Endoscopy has been used in Russia for practical and scientific purposes sometimes with questionable indications [34]. Despite bans on asbestos use, a further increase in MPM morbidity and mortality is expected. Given the diversity of carcinogens and spontaneous mutations, it is anticipated that most future mesothelioma cases will not be attributable to asbestos [2]. The rarity of MPM, nonspecific clinical and radiologic presentation, lack of validated screening strategies and of pathognomonic markers, complicate the diagnostics [35]. Histologically, MPM can resemble various types of cancer. Other tumors may undergo anaplasia and come to resemble MPM. The differential diagnosis depends on the MPM subtype. A diagnostic challenge is distinguishing pleural involvement by sarcomatoid tumors, such as sarcomas and pleomorphic carcinoma of the lung, from sarcomatoid MPM [35,36]. Reviews of histological archives have regularly revealed cases of misdiagnosis [36,37]. In one study, the initial diagnosis was confirmed in 67% of cases, revised in 13%, being indeterminate in the remaining cases [38]. Another group of experts revised the diagnosis in 14% of 5,258 mesothelioma cases [19]. It is estimated that approximately 10% of MPM cases in the United States were misdiagnosed [37]. Unlike the general population, screening for MPM in the high-risk groups is conducted by specialized experts. Consequently, a higher number of mesothelioma cases are detected. No single marker has been identified as reliable and sufficient on its own [29,35]. Staining patterns among the histologic MPM subtypes can differ, while the sensitivity and specificity

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can vary. Common markers for mesothelial lineage include calretinin, cytokeratin 5/6, Wilms tumor-1 (WT1), HEG1, and podoplanin (D2-40), whereas markers used to exclude other cancers include TTF-1, Napsin A, MOC-31, polyclonal CEA, Ber-EP4, and claudin-4 [35]. Podoplanin, calretinin, WT1, and HEG1 are useful but lack sufficient specificity [39]. Mesothelin has been discussed as a promising marker; however, its expression is observed also in other tumors. The sensitivity of mesothelin detection is insufficient [19,40]. It is frequently absent in sarcomatoid and epithelioid MPM [36-42]. Osteopontin was considered a promising marker, yet the data remains inconsistent. Like mesothelin, the utility of osteopontin and fibulin-3 is limited by low sensitivity [43]. Furthermore, the heterogeneity of chromosomal aberrations in MPM has been notived [44-46]. Reliable genetic markers are lacking. Certain genotype-phenotype associations are unclear or speculative [47,48]. FISH testing enables the detection of p16/CDKN2A gene loss resulting from 9p21 deletion, a change specific to the neoplastic proliferation of mesothelial cells. However, the sensitivity for MPM ranges from 48% to 88% [49]. Homozygous deletion of p16/CDKN2A shows 100% specificity but moderate sensitivity (59-69%) for MPM, being absent in benign conditions [35]. The above-mentioned Helsinki Criteria do not provide clear recommendations regarding the use of biomarkers for MPM screening [23,50]. Moreover, MPM can exhibit intratumoral heterogeneity and subclonality [51]. Despite the abundance of markers, none has proven sufficiently specific [50,52]. A tumor diagnosed as MPM using algorithms and panels is not always bona fide distinguishable from other cancers. The foregoing explains the increased detection rate of MPM in high-risk groups.

Russian asbestos science

Asbestos-related diseases have been extensively studied in Russia. The prevailing view is that, provided necessary precautions are observed, modern asbestos production and processing technologies are safe, and the bans implemented in some countries are excessive [31-55]. Health risks associated with low fiber concentrations have not been proven. No elevated risks have been identified among individuals residing near modern asbestos industry facilities. Results of epidemiological studies indicate the existence of a safe (threshold) concentration of fibers in the air [56,57]. Adaptation to a certain level of asbestos fiber inhalation is considered possible [58]. Asbestos-cement sheets (slate) are widely used for roofing. Fiber release from roofing materials during construction is negligible. Indoor air fiber concentrations are an order of magnitude below the permissible level [59]. Asbestos-cement pipes are used for drinking water distribution and are considered safe; the risk associated with the oral ingestion of asbestos fibers has not been proven, particularly given that the fibers are modified through aggregation with the cement [60,61]. Studies have shown that asbestos-cement pipes do not affect drinking water quality; their use has been approved by the Ministry of Health [62]. Consuming water containing 7-10 million fibers per liter does not lead to an increased risk of stomach cancer [63]. Asbestos-containing crushed stone - a byproduct of chrysotile processing - was used for railway embankments, with elevated fiber concentrations in the air observed both on trains and in nearby settlements [64]. Like asbestos-cement, the carcinogenicity of asbestos-board fibers is reduced due to their aggregation with cellulose [65]. Chrysotile fibers extracted from chrysotile-cement exhibit lower carcinogenic potential than commercial chrysotile. The chrysotile-cement industry is considered a source of carcinogenic risk, albeit a significantly lower one than the asbestos industry [66]. No toxic effects have been identified for asbestos-containing brake pads compared to asbestos-free ones, and no significant air pollution from automotive brakes has been observed [67,68]. During the braking process, asbestos transform into virtually harmless forsterite [69,70]. Asbestos-containing materials (cardboard, paper, clothing, gaskets) remain in widespread use. The installation and repair of asbestos-containing components without processing them are considered safe [68]. Among 69 patients with MPM in Kazakhstan, asbestos exposure was not identified in a single case, and no geographical link to asbestos mining or the manufacturing industry was observed [71]. In the period 1992-2011, only 3 cases of malignant mesothelioma were diagnosed in asbestos workers of Ukraine. The cancer incidence among Ukrainian asbestos workers was found to be lower than in the general population [72,73]. No increase in the incidence of mesothelioma has been observed among workers at asbestos facilities or residents of areas with a developed asbestos industry in Russia [74]. Based on a study of 3,576 cases of mesothelioma, it was concluded that asbestos is neither a primary nor an obligate causative factor [75]. Compliance with Russian maximum permissible concentration levels ensures safe working conditions for virtually all workers - that is, free from an increased risk of asbestosis or cancer [63]. The prevailing opinion is that serpentine asbestos (chrysotile) is less toxic than the amphiboles. However, there are contradictions between the data from epidemiological and experimental studies. In Russia almost exclusively chrysotile is produced; and there are industrial interests in continued production and sales. However, some experts doubted the supposition that inhaling amphibole fibers is far more dangerous than chrysotile [76]. The carcinogenic, fibrogenic, mutagenic, and cytotoxic effects of chrysotile have been confirmed in experimental and epidemiological studies [77-79]. Comparative studies demonstrated that anthophyllite is less fibrogenic than chrysotile [80]. In experiments, chrysotile exhibited toxicity by inducing a granulomatous tissue reaction [81], while its carcinogenic effect did not differ significantly from that of amphiboles [82].

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Serpentine versus amphibole

It has been claimed that longer chrysotile fibers are rapidly cleared from the lungs [83]. Given the potential of chrysotile fibers to migrate from lung tissues to the pleura [84-87], the biopersistence of asbestos cannot be assessed solely by measuring fiber content in the lungs. In particular, the research protocol by D.M. Bernstein [81] resulted in a very short fiber half-life, leading him to conclude that chrysotile has low carcinogenic potential. Bernstein's findings are partly at variance with those by independent researchers. His results can be explained by aggressive pre-treatment of the fibers as well as by conflicts of interest in favor of chrysotile [53,88,89]. Acid dissolution does not prove solubility in tissues of a living organism. At that, Bernstein's observations are seminal. Obviously unfounded claims have been made with references to his works: "It has been shown that chrysotile is rapidly cleared from the lungs of experimental animals following inhalation" and moreover: "Chrysotile, which rapidly degrades in the lungs, behaves more like non-fibrous mineral dust [90]." In this way, biased information is spreading to support the chrysotile production. Fiber solubility was tested in Gamble's solution, which simulates lung interstitial fluid. The values ranged from a few nanograms of dissolved silicon per square centimeter of fiber surface (chrysotile and crocidolite) to thousands of ng/cm² (glass fiber). Aramid and carbon fibers proved to be virtually insoluble [91]. Experiments with Gamble's solution have shown that a relatively large amount of magnesium dissolves from chrysotile. The silicate structure is based on silicon and oxygen atoms arranged in Si-O-Si chains. Fiber strength is primarily determined by the bonds between these atoms. Electrostatic forces act between the chains, arising from the interaction between negatively charged oxygen atoms (bonded to silicon) and cations, including magnesium in the first place [84,92-94]. The leaching of magnesium from the fiber surface may contribute to their longitudinal splitting. As a result, the total number of fine fibrils increases [84-97]. It has been hypothesized that the thinner the fiber (within certain limits), the higher its carcinogenic potential, as it penetrates tissues more effectively [97]. This topic requires further research. To sum up, the reportedly accelerated clearance of chrysotile from lung tissue is partly due to the splitting of fibers into thin fibrils that are more difficult to detect. Asbestos fibers are found in the pleura postmortem, with chrysotile being the predominant fiber type in the pleura and pleural plaques [86-99]. The concept of fiber migration to the pleura is consistent with the observation that, in individuals with a history of asbestos exposure, the primary site of mesothelioma is more frequently located in the parietal rather than the visceral pleura [100]. Several studies have confirmed the biopersistence of chrysotile in the human lung [101]. In experiments on rats, chrysotile induced inflammation and, subsequently, malignant tumors within a relatively short period, whereas crocidolite exerted a carcinogenic effect over a longer time frame [102]. The incidence of mesothelioma increases following exposure to pure chrysotile [103,104]. The relatively high frequency of mesothelioma among workers exposed to amphiboles has been attributed to higher average doses [105]. As previously mentioned, there are discrepancies between the results of animal experiments and epidemiological data. It was noted that evidence of differences between chrysotile and amphiboles regarding lung cancer is weak at best [106]. In some experiments, the carcinogenic activity of amphiboles and chrysotile was nearly identical with respect to mesothelioma [94-109] and lung cancer [110,111]. In one study, chrysotile proved to be more carcinogenic than amphiboles, and it was noted that no evidence was found of lower carcinogenicity or less pronounced asbestosis in groups exposed to chrysotile compared to those exposed to amphiboles [109]. In a study on rats, chrysotile caused more pulmonary fibrosis and tumors than amphiboles. This has been attributed to the high content of fibers longer than 20 mcm in the chrysotile specimens used [112]. Chrysotile induced chromosomal aberrations and pre-neoplastic cell transformations in vitro [107,113]. Fiber morphology and biopersistence have been linked to their potential to cause fibrosis, lung cancer, and MPM [114]. The toxicity of asbestos and other fibers depends on the dose, dimensions and bio persistence [20,115-118]. The latter being equal, differences in carcinogenicity are linked to fiber length and thickness [119]. Notably the target gene of the chrysotile-induced mesothelioma is the same as for amphiboles, confirming that the pathogenesis is quite similar for all asbestos-induced mesothelial carcinogenesis [115]. It has been noticed that long chrysotile fibers exhibit relatively high toxicity because they are less efficiently engulfed and cleared by macrophages [120,121]. According to another study, short, thin chrysotile fibers predominated in the lungs and pleura among patients with MPM [122]. Furthermore, the presence of tremolite as admixture to chrysotile can enhance the carcinogenic effect [123]. However, an epidemiological study found that the difference in MPM risk between pure chrysotile and mixtures of chrysotile with amphiboles was negligible [124]. The toxicity of various asbestos types was compared in a meta-analysis of epidemiological studies, which assessed the impact of study quality on the dose-response relationship for lung cancer. As expected, the difference between chrysotile and amphiboles was smaller when the meta-analysis was restricted to high-quality studies [125,126]. After adjusting for quality, the difference between chrysotile and amphiboles proved to be negligible [125,127], illustrated by the graphs in the article [125]. According to a systematic review, pooled lung cancer risk estimates were higher following exposure to amphiboles - 1.74 (95% confidence interval: 1.18-2.57) - than to chrysotile - 0.99 (0.78-1.25). Typically, the difference was greater in studies of

moderate rather than high quality (there was no low-quality group): 1.86 (1.27-2.72) versus 1.21 (0.79-1.87) [128]. Significant differences between the results of higher- and lower-quality studies indicate a bias in the latter.

Discussion and Conclusion

Formulating criteria for including studies in reviews and meta-analyses, it is important to consider their quality, potential sources of bias and conflicts of interest. Objective data can be obtained from animal experiments measuring mean lifespan. In addition to rodents, it is advisable to use larger animals, including primates [129]. Experiments using concentrations are many times higher than those found in the workplace providing limited information [130]. The mining and use of asbestos are banned in many countries, while others continue its production and marketing, thus gaining economic and strategic advantages [53,131]. Chrysotile products in international trade contain varying amounts of amphibole impurities [132]. Different types of asbestos offer specific technical advantages and preferred applications. Amphiboles (crocidolite, anthophyllite) are acid-resistant, thermally stable, and durable [133]. Considering industrial interests behind chrysotile, any deviation from the All-Fibers Equal [134,135] principle must be based on high-quality evidence. Reliable data can be obtained in lifelong animal experiments. Certain environmentalists should be aware that their activities may be used to disadvantage their nations [136]. Strictly observed realistic safety rules would be more useful for public healthcare than excessive restrictions that would be neglected in certain parts of the world, bringing to the violators economic and strategic advantages. Asbestos is an effective reinforcing material. The fire-retardant properties of asbestos are well known. The durability of brake pads depends on the reinforcing material used, while asbestos is well suitable and inexpensive. It is safe to assume that abandoning the use of asbestos-containing materials would increase the damage and the number of casualties resulting from traffic accidents, fires, and armed conflicts.

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