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Review

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Asbestos and the anti-asbestos campaign: a review

Assessment of health risks associated with asbestos is based on practical experience, when the asbestos fiber content in the air was artificially increased. Asbestos fibers enter the environment as a result of the erosion of asbestos materials and other human activities not related to the asbestos industry. When specifically searched for, fibers are often detected during autopsies. The results of many studies are more or less 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 anamnesis on professional or domestic contact with asbestos. Reliable data can be obtained in experiments recording the average lifespan of animals. Different types of asbestos have specific technical characteristics and are used in various fields. For example, asbestos is used in the manufacture of brake pads. Construction materials based on a mixture of cement and asbestos are distinguished by low cost and long service life. Asbestos products are also widely used as fire-resistant materials. On the one hand, the refusal to use asbestos has a positive effect on the environment, but on the other hand, refusing to use it will lead to increased damage from transport and fires, and will increase the cost of construction.

Keywords: asbestos, chrysotile, asbestos-related diseases, mesothelioma, lung cancer, amphiboles.

Introduction

Health risk assessment for asbestos was previously based on high concentrations of asbestos fibers. Risks were assessed using the no-threshold hypothesis, as in the case of radiation hazards, and the effect of low doses of asbestos fibers on the formation of pleural tumors and lung cancer had not been reliably proven [1–3]. There is a view that the harmful effects of asbestos in the workplace and in everyday life effectively ceased in developed countries about 40 years ago, and that modern industrial products and materials do not release any dangerous amounts of fibers [3]. Asbestos fibers are naturally present in the environment, formed as a result of erosion of surface deposits [4-5]. Natural emissions help disperse chrysotile and amphibole asbestos fibers; in areas of natural deposits, asbestos concentrations can significantly exceed those caused by human activity [4, 6]. Any environment, including air, water, soil, and living organisms, can be subject to anthropogenic contamination, including contamination not related to asbestos production and mining. Examples include tunnel digging in asbestos-contaminated soil and other earthworks [7-8]. Research results in Milan showed that asbestos fibers were detected in 35 out of 55 (63.6 %) cases of routine autopsy [9]. During autopsies of deceased individuals who had contact with asbestos, more pieces of lung and pleural tissue are taken for histology, and the examination is performed thoroughly, using special methods. Therefore, asbestos fibers are found more often than in normal autopsies. The detection of fibers alone does not prove either occupational exposure to asbestos or the role of asbestos in the etiology of diseases. It is worth noting that the removal of fibers from the respiratory system is a normal physiological process [9-10].

Compared with other natural sources of pollution, it can be assumed that there is a safe (threshold) concentration of asbestos fibers in various environments, including air. There is experimental data in favor of the existence of thresholds for oncological and other diseases [11]. Apparently, screening and medical examinations have contributed to an increase in the detection of mesothelioma and lung cancer among people who have been exposed to asbestos. Many studies are not sufficiently objective. For example, lung and pleural tumors are sometimes classified as asbestos-related when fibers are detected, although the causal relationship remains unproven. Some studies rely on questionable medical history data on occupational or domestic exposure to asbestos and on interviews with relatives of deceased patients [12].

Experimental

To compile this review, we analyzed literature sources in databases (e-library, Scopus, PubMed, Google Scholar) for the period 1990–2024. Additional information was obtained from dissertations, conference and meeting materials, and specialized literature on the physiology of asbestos-related diseases. The following keywords were used in the search queries: asbestos, lung cancer, mesothelioma, asbestos-related diseases, epidemiological studies.

Review

Malignant pleural mesothelioma (MPM)

The stable incidence of MPM in some developed countries, despite asbestos bans [13–15], is partly due to improvements in diagnostic equipment, the effect of screening, and overdiagnosis due to the unclear definition of MPM as a nosological entity. In addition to natural asbestos, the etiological factors of MPM include artificial and mineral fibers, ionizing radiation, the SV40 virus, chronic lung inflammation such as inflammation, empyema, and tuberculosis, genetic predisposition, and the results of mutagenesis [15–25]. For example, scientists have classified erionite as a stronger carcinogen than asbestos fiber. In general, human activity contributes to the spread of potentially dangerous substances with carcinogenic properties [7, 16]. Even nanomaterials made from traditional components can exhibit carcinogenic properties.

Available publications have found that even viruses, such as the SV40 virus, may contribute to the recent increase in diagnosed cases of mesothelioma [26]. DNA sequences similar to SV40 are regularly found in MPM [27]. After laser microdissection, SV40 was found in MPM cells but not in surrounding cells [26]. When SV40 was introduced, ≥50 % of hamsters developed mesothelial tumors; after injections into the pleural cavity, mesotheliomas occurred in 100 % of hamsters [16, 28]. Thus, an increase in the incidence of MPM was noted in the 1960s, which is associated with the widespread distribution of this virus in 1955–1963, when anti-mumps vaccines contained live SV40 [26]. Antibodies to the SV40 virus were detected in the blood serum of 34 % of MPM patients, compared to 20 % of healthy individuals. The results obtained reliably indicate the involvement of the SV40 virus in the etiology of MPM, as it circulates widely among the populations of different countries [29]. Frequently used invasive procedures, such as bronchoscopy, directly contributed to the spread of the SV40 virus and an increase in additional cases of MPM. Doctors used bronchoscopy and biopsy to diagnose bronchitis associated with asbestos fibers, as well as to identify other dust-related diseases and pneumonia [30–33]. Therefore, even despite the ban on the use of asbestos products, there has been a steady increase in the incidence and mortality rates of MPM [34]. Given the significant presence of carcinogens in the environment, it is expected that most mesothelioma cases will not be diagnosed as asbestos-related [3].

Diagnosing MPM is often difficult. Histologically, MPM can resemble various types of cancer. Tumors can undergo anaplasia and become similar to MPM. The differential diagnosis depends on the subtype of MPM. Diagnosis of the sarcomatoid variant of MPM is particularly difficult; in this case, the usefulness of immunohistochemistry is limited [22, 35]. Reviewing histological archives allows for the identification of misdiagnoses [35-36]. Thus, some studies have shown that the initial diagnosis was confirmed in 67 % of cases, but after review, it was changed to 13 %, with the diagnosis not being accurately established in the remaining cases [37]. Other studies have led to a change in diagnosis for 14 % (of 5,258 cases) of previously diagnosed mesothelioma [16]. According to estimates, about 10 % of MPM cases in the United States were misdiagnosed [36]. The main reason is considered to be the lack of experience of doctors due to the rarity of MPM detection by general practitioners [35, 36]. Whereas in risk groups, MPM is identified by specialized doctors, which leads to a higher detection rate.

Unfortunately, there are no available and reliable biomarkers that could improve the diagnosis of MPM [26]. Immunohistochemical markers such as calretinin, WT1, podoplanin, and HEG1 play a role in diagnosis but do not have sufficient specificity [38]. Previously, mesothelin was considered a reliable marker [39], but its expression is also found in other lung tumors, such as adenocarcinoma [40]. The sensitivity of mesothelin as a marker is insufficient [16, 17, 39, 41]; its expression is often absent in sarcomatoid and epithelioid MPM [35, 42-43]. Osteopontin was considered a promising marker, but the data remain contradictory. Similar to mesothelin, the use of osteopontin and fibulin-3 is limited due to low sensitivity [44]. Information on changes at the molecular level is insufficient [34]. Heterogeneity of chromosomal aberrations in MPM has been noted [24, 45-46]. There are no reliable genetic markers [47-48]. The FISH test can detect the loss of the p16/CDKN2A gene due to 9p21 deletion, which is specific for neoplastic proliferation of

mesothelial cells. However, its sensitivity for MPM is 48–88 % [49]. The authors show an exaggeration of the role of biological immunohistochemical and other molecular markers [39]. The Helsinki Criteria, which were designed to identify a causal link between asbestos and the development of mesothelioma, do not include clear recommendations on the use of biomarkers in screening for MPM diseases [50-51]. Moreover, MPM diseases can show intratumoral variability and subclonality [52]. In other words, markers specific enough for the diagnosis of MPM have not yet been reliably established [50, 53]. Also, malignant tumors diagnosed as MPM did not always differ from other types of cancer. The above explains the increased frequency of MPM detection in risk groups.

Russian science on the dangers of asbestos

Diseases associated with asbestos and its effects were also widespread in the Russian Federation (and the former USSR). Many researchers believed that global bans on the processing and use of asbestos materials were overly strict and that compliance with safety regulations did not lead to contamination and an increase in diseases among the population [30, 54-55]. Thus, there were no studies that proved the risk of developing asbestos-related diseases from low concentrations of asbestos in the environment. No high risks were identified for populations living near various enterprises involved in the production of asbestos fiber. Extensive epidemiological analyses show the presence of safe concentrations of asbestos (asbestos fibers) in the atmosphere [56-57]. It is believed that humans can adapt to certain levels of asbestos fiber concentration [58]. For example, asbestos slate is widely used in construction for roofing. At the same time, the release of asbestos fibers from slate sheets into the environment is very insignificant. It has been established that the average concentration of asbestos fibers in rooms is significantly lower than the maximum permissible level [59]. Asbestos-cement pipes are considered safe for drinking water delivery. The risk of asbestos fibers entering the digestive system has not been proven. It is worth noting that asbestos fibers are almost not separated from the mixture after modification with cement [60-61]. Some studies have assessed the safety of using asbestos-cement pipes for transporting drinking water, and their use has been approved by the Ministry of Health [62]. Consuming water containing 7–10 million fibers per liter does not increase the risk of stomach cancer [63]. Asbestos-containing crushed stone was used in the construction of railway embankments. Its relatively high concentration in the atmosphere has been noted, both at stations and in trains themselves [64]. Asbestos cardboard was widely used, and its carcinogenicity was reduced by aggregation with cellulose fibers [65]. Chrysotile fibers isolated from chrysotile cement have a lower carcinogenic potential than commercial chrysotile. The chrysotile cement industry is considered a source of carcinogenic hazard, but significantly less than asbestos [66]. The toxicity of brake pads containing asbestos fibers has also not been reliably established; no reliable air pollution from such brakes on cars has been recorded, but their effectiveness in road traffic has been noted [67–70]. This fact is related to the fact that materials (cardboard, paper, clothing, gaskets) containing asbestos fibers are still used in various industries [68]. Numerous studies have not found an increase in the incidence of mesothelioma among workers at asbestos plants or among the population living in the vicinity of asbestos plants [71]. An analysis of causal relationships in 3,576 cases of diagnosed mesothelioma showed that asbestos is neither the leading nor the obligatory causal factor [72]. Thus, in Kazakhstan, an analysis of the course of the disease among 69 patients with MPM did not allow a reliable link to be established with the extraction, processing, or use of asbestos [73]. Compliance with Russian MPCs ensures safe working conditions for virtually all workers, i.e., without an increased risk of asbestosis and cancer [63].

Chrysotile and amphibole asbestos

The prevailing opinion is that serpentine asbestos (chrysotile) is less toxic than amphibole asbestos (actinolite, tremolite, amosite, crocidolite, anthophyllite, etc.), but there are contradictions between the data from epidemiological and experimental studies. In Russia, almost exclusively chrysotile is produced. Some experts believed that the opinion about the danger of certain forms of asbestos was not sufficiently substantiated [74]. Thus, the cytotoxic, carcinogenic, mutagenic, and fibrogenic effects of chrysotile have been reliably confirmed by a number of epidemiological studies, as well as in experiments [75–77]. Comparative experiments have determined that anthophyllite is less dangerous than chrysotile in terms of its fibrogenic effect [78]. In laboratory experiments, chrysotile was sufficiently toxic to cause a noticeable granulomatous tissue reaction [79]. However, its carcinogenic effect did not differ significantly from other types of asbestos [80]. The studies comment: “After short-term exposure, longer chrysotile fibers are quickly removed from the lungs” [81]. Since chrysotile fibers can migrate from lung tissue to the pleura [82–87], it is very difficult to assess biopersistence solely by counting asbestos fibers in the lungs. Bernstein’s research protocol [81] is the reason for the very long half-life of the fibers. Therefore, the carcinogenicity of chrysotile is

considered insignificant. However, some of Bernstein's conclusions contradict the results obtained by independent researchers. Perhaps these results are related to aggressive sample preparation when determining asbestos fibers in the lungs [88]. The decomposition of asbestos by acids does not prove its solubility in tissues in vivo. With references to the named author, unfounded statements are made: "It has been shown that chrysotile is rapidly removed from the lungs of experimental animals after inhalation"; "chrysotile, which rapidly decomposes in the lungs, behaves more like non-fibrous mineral dust" [89].

Experiments were conducted on the dissolution of asbestos fibers in Gamble's solution, which mimics the interstitial fluid of the lungs. Solubility ranged from a few nanograms of dissolved silicon per square centimeter of fiber surface (chrysotile and crocidolite) to thousands $^{of\ ng/cm^2}$ (glass fiber). However, aramid and carbon fibers proved to be practically insoluble [90]. Experiments with Gamble's solution showed that a relatively large amount of magnesium dissolves from chrysotile. Silicates are based on silicon and oxygen atoms in Si-O-Si chains. The strength of the fibers is mainly determined by the bonds between these atoms. Electrostatic forces act between the chains due to negatively charged oxygen atoms bound to silicon atoms and cations, including magnesium [82, 91–93]. The leaching of magnesium from the surface of the fibers can contribute to their longitudinal splitting. As a result, the total number of thin asbestos fibers can increase significantly [82, 83, 87, 92–97], causing an increase in the carcinogenic effect. The authors suggested that an increase in fiber thickness leads to an increase in the carcinogenic effect due to better penetration into human and animal tissues [97]. Further research in this area is needed.

Thus, the rapid removal of chrysotile from lung tissue can be explained by the breakdown of fibers into thin fragments that are difficult to identify. Asbestos fibers are usually found in the pleura postmortem, with chrysotile being the predominant fiber in the pleura and pleural plaques [85, 86, 98, 99]. The idea of fiber migration from the lungs to the pleura is consistent with the proven fact that the primary focus of mesothelioma in individuals who have been exposed to asbestos is more often located in the parietal pleura than in the visceral pleura [100]. A number of studies have confirmed the biopersistence of chrysotile in the human lungs [101]. However, it cannot be ruled out that chrysotile dissolves more quickly in the acidic environment of lysosomes. In experiments on rats, chrysotile caused inflammation in a relatively short period of time, followed by malignant tumors, while crocidolite had a carcinogenic effect at a later stage [102].

It has been noted that the incidence of mesothelioma increases significantly when exposed to pure chrysotile [103-104]. Thus, the significantly higher incidence of mesothelioma among workers who had contact with amphiboles was explained by higher doses of this substance [105]. As mentioned above, there are discrepancies between the results of animal experiments and epidemiological data. It has been noted that the evidence for differences between chrysotile and amphiboles in lung cancer is "weak at best" [106]. Some experiments have demonstrated virtually identical carcinogenic activity of amphiboles and chrysotile in relation to mesothelioma [93, 107–109] and lung cancer [110-111]. However, some studies show a higher level of carcinogenic effect of chrysotile compared to amphibole. Thus, it was noted: "No evidence of lower carcinogenicity or less severe asbestosis was found in groups exposed to chrysotile compared to groups exposed to amphiboles" [109]. In experiments on laboratory animals (rats), chrysotile caused a greater number of tumors and pulmonary fibrosis compared to amphibole. This is explained by the higher concentration of fibers longer than 20 μm in the chrysotile used in the experiment [112]. An increase in chrysotile concentration contributed to the occurrence of chromosomal breaks, which led to pre-tumor transformation of cells in vitro [107, 113].

In humans, the difference in the risk of developing lung cancer between chrysotile and amphiboles (amosite and crocidolite) has been determined to be between 1:10 and 1:50. For mesothelioma, the risk ratio from exposure to these types of asbestos was estimated to be 1:100:500, respectively [2]. The latter risk ratio was described in sources [37, 114]. In a later report, this risk was estimated at a ratio of 1:5:10 [115]. The authors noted that in experiments with laboratory animals, all types of asbestos provoke almost the same number of lung tumors, which shows a contradiction between epidemiological and experimental studies. The following explanation was proposed for this situation: "In humans, chrysotile fibers (excreted over months) may have less effect than amphibole fibers (excreted over years)" [2]. However, no different mechanisms for the removal of fibers from the tissues of the respiratory system have been proposed. That is, a decrease in the concentration of chrysotile in the lungs may be caused by the breakdown of asbestos fibers and their movement into the pleural tissue.

The toxicity of asbestos and other types of fibers largely depends on the three "Ds"—Dose, Dimensions, Durability [18, 116–118]. If different types of asbestos fibers have the same biopersistence indicators, then the varying degrees of carcinogenicity depend on the thickness and length of the fibers [119]. Thus,

long chrysotile fibers showed significantly higher toxicity, as they are less readily absorbed by macrophages [120–121]. According to another study, short and thin chrysotile fibers predominated in MPM in the lungs and pleura [122]. It has been noted that tremolite impurities in chrysotile products contribute to an increased carcinogenic effect [123]. In an epidemiological study, the difference in the risk of MPM from pure chrysotile and its mixtures with amphiboles was found to be insignificant [124].

The toxicity of different types of asbestos was compared in a meta-analysis of 19 epidemiological studies, which assessed the impact of study quality on the dose-response relationship for lung cancer. The difference between amphiboles and chrysotile was significantly lower when the meta-analysis was limited to high-quality studies [114, 125]. After standardization for quality, the difference between the two types of fibers was not significant [114, 126].

The overall estimates of the risk of lung cancer were higher after exposure to amphiboles — 1.74 (95 % confidence interval 1.18-2.57), and slightly lower after exposure to chrysotile — 0.99 (0.78-1.25) [127]. The significant differences between the results of high- and low-quality studies indicate that the latter lack objectivity. As mentioned above, the prevailing opinion is that chrysotile is less toxic than amphiboles. This difference should be quantitatively assessed in independent studies.

Discussion

Asbestos bans were partly based on studies that were influenced by economic interests. When determining the criteria for including studies in reviews and meta-analyses, their quality and possible systematic errors should be taken into account. Objective information can be obtained from laboratory animal experiments with determination of average life expectancy. That is, it is desirable to use large animals; the best results for humans can be obtained by testing asbestos on primates [128]. Experiments involving the inhalation of fibers in doses comparable to those in the asbestos industry are ethically acceptable, as they can be carried out without the use of invasive procedures. Experiments and studies using "concentrations many times higher than those found in the workplace" [129] have limited reliability. For example, replacing asbestos with artificial fibers is unlikely to eliminate the risks to health and the development of lung diseases [18, 19, 130-131]. Thus, at present, carcinogenic effects are already being detected in materials used as substitutes for asbestos fiber, such as carbon nanotubes. Studies show that asbestos fibers and carbon nanotubes have toxic effects through the same mechanisms, in particular, chronic activation of macrophages, leading to inflammation [132]. Nanoparticles can cause structural changes in membrane proteins and activate the synthesis of inflammatory mediators, disrupting normal cellular metabolism mechanisms [133]. An experiment has demonstrated the carcinogenic effect of nanotubes [20, 134]. Carbon nanotubes are biostable, and some of their varieties have been classified as possible human carcinogens [135].

The extraction and use of asbestos is prohibited in a number of countries, while others continue to produce and export it [136]. Chrysotile products traded internationally contain impurities of varying amounts of amphiboles [137]. Different types of asbestos have their own technical advantages and preferred areas of application. Amphiboles (crocidolite, anthophyllite) are acid-resistant, thermally stable, and durable [138].

Conclusion

Asbestos is an inexpensive material and an effective reinforcing fiber. Asbestos cement structures are durable and fire resistant. Asbestos-based products are highly durable and safe, and incorporating them into other products increases their reinforcing properties. It can be confidently assumed that the refusal to use asbestos-containing materials will increase the damage and number of victims of road accidents, fires, and armed conflicts. The independence of scientific research from economic interests is of great importance. However, the mechanism of development of many respiratory diseases from asbestos exposure has not been reliably established, which requires further research.

Conflict of Interest

Author declares no conflict of interest.

Author contribution

The manuscript was written through contributions of author. The author has given approval to the final version of the manuscript: **Jargin S.V.** — conceptualization, investigation, data collection, draft writing.

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С.В. Яргин

Асбест және асбестке қарсы науқан: шолу

Асбестпен байланысты денсаулыққа қауіп-қатерді бағалау ауадағы асбест талшықтарының құрамы жасанды түрде арттырылған практикалық тәжірибеге негізделген. Асбест талшықтары қоршаған ортаға асбест материалдарының эрозиясы және асбест өнеркәсібіне қатысы жоқ басқа да адам әрекеттері арқылы енеді. Арнайы іздеу кезінде талшықтар денелерді сою кезінде жиі кездеседі. Көптеген зерттеулердің нәтижелері белгілі бір дәрежеде біржақты болады. Талшықтар анықталған кезде мезотелиома немесе өкпе рагы кейде асбестпен байланысты деп жіктеледі, дегенмен себеп-салдарлық байланыс дәлелденбеген күйінде қалады. Кейбір зерттеулер асбестпен кәсіби немесе тұрмыстық байланыстың тексерілмеген анемнезіне сүйенеді. Сенімді деректерді жануарлардың орта-

ша өмір сүру ұзақтығын тіркейтін эксперименттерден алуға болады. Асбесттің әртүрлі түрлерінің өзіндік сипаттамалары бар және оларды әртүрлі салаларда қолдануға болады. Мысалы, құрамында асбест бар материалдар тежегіш төсемдерін өндіруде қолданылады. Цемент пен асбест қоспасынан жасалған құрылыс бұйымдары арзан және ұзақ уақыт пайдалануға болады. Асбест бұйымдары өртке төзімді материалдар ретінде кеңінен қолданылады. Бір жағынан, асбестті пайдаланудан бас тарту қоршаған ортаға оң әсер еткенімен, бірақ екінші жағынан, оны пайдаланудан бас тарту тасымалдау мен өрттен болатын залалды арттырады, сондай-ақ құрылыс шығындарын ұлғайтады.

Кілт сөздер: асбест, хризотил, асбестке байланысты аурулар, мезотелиома, өкпе рагы, амфоболия

С.В. Яргин

Асбест и антиасбестовая кампания: обзор

Оценка рисков для здоровья, связанных с асбестом, основана на практическом опыте, когда содержание асбестовых волокон в воздухе было искусственно увеличено. Асбестовые волокна попадают в окружающую среду в результате эрозии асбестовых материалов и других видов деятельности человека, не связанных с асбестовой промышленностью. При целенаправленном поиске волокна часто обнаруживаются при вскрытии тел. Результаты многих исследований в той или иной степени предвзяты. При обнаружении волокон мезотелиома или рак легких иногда классифицируются как связанные с асбестом, хотя причинно-следственная связь остается недоказанной. Некоторые исследования опираются на непроверенный анамнез профессионального или бытового контакта с асбестом. Достоверные данные можно получить в экспериментах, регистрирующих среднюю продолжительность жизни животных. Различные виды асбеста имеют свои технические характеристики и могут использоваться в разных областях. Например, асбест используется в производстве тормозных колодок из материалов, содержащих асбест. Строительные изделия на основе смеси цемента и асбеста отличаются низкой стоимостью и длительным сроком службы. Изделия из асбеста широко используются в качестве огнеупорных материалов. С одной стороны, отказ от использования асбеста положительно сказывается на окружающей среде, но с другой стороны, отказ от его использования приведет к увеличению ущерба от транспортировки и пожаров, а также увеличит стоимость строительства.

*Ключевые слова***:** асбест; хризотил; асбест-связанные заболевания, мезотелиома, рак легкого, амфоболия

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