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Review

Asbestos-Related Diseases and Policies: an Update

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Abstract

Asbestos-related risks have been estimated on the basis of data from the past, when professional exposures were higher. Fibers are present in the environment due to erosion of surface deposits and human activities unrelated to asbestos industry. If searched for, asbestos fibers are frequently found at autopsies. Bias can be encountered in asbestos research e.g. attributing of mesothelioma and lung cancer to asbestos when fibers are present, although cause-effect relationships remain unproven. Some studies rely on work or residence histories of questionable reliability. Asbestos is banned in some countries while others are increasing production and exports. Asbestos is a low-cost material and an excellent reinforcing fiber. Different asbestos types have their technical advantages and preferred application areas. The traffic is safer with asbestos-containing brake linings. Asbestos cement constructions are sturdy and inexpensive. The fireproofing properties of asbestos are well known. It can be reasonably assumed that the non-use of asbestos-containing brakes, fireproofing and insulation lagging has increased the damage and numbers of victims of traffic accidents, fires and armed conflicts. Nowadays, when a probability of conflicts seems to be enhanced, the attitude to asbestos should be changed. Most importantly, asbestos-related science must be separated from economical and political interests. Reliable information can be obtained in lifelong bioassays.

Keywords: asbestos; chrysotile; mesothelioma; lung cancer

Introduction

This review is an update and continuation of the previously published [1,2]. Health risks from asbestos have been evaluated on the basis of data from the past, when workers' exposures were higher than today. The linear no-threshold model, known from the radiation protection, has been applied to asbestos-related risks although its relevance is unproven and remains arguable both for pleural and lung tumors [3,4]. There is an opinion that the vast majority of exposure to chrysotile asbestos ended (in developed countries) nearly 40 years ago and that exposure to asbestos in new products practically does not occur [4]. Asbestos fibers are present in the natural environment due to erosion of surface deposits. Naturally occurring asbestos has been commonly found in populated areas [5]. The natural emission contributes to a global dispersion of chrysotile and amphibole asbestos fibers. Presumably, natural releases dwarf anthropogenic contributions to the atmospheric

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dispersion of both fiber types [5,6]. Air, soils and waters may be contaminated by asbestos and other potentially harmful fibers due to human activities unrelated to asbestos industries e.g. land excavation, slopes reprofiling and tunneling [7,8]. In a study from Milan, asbestos fibers were found in 35 of 55 (63.6%) autopsy cases from the general population [9]. Of note, in exposed people the pulmonary and pleural tissues are sampled post mortem more abundantly and examined more thoroughly than at routine autopsies, hence the higher probability to find fibers. The presence of fibers per se proves neither a professional exposure nor asbestos-related disease. Inhalation and discharge of fibers occur normally being in a dynamic balance [9,10]. By analogy with other substances in the natural environment, it can be assumed that there is a harmless (threshold) fiber concentration in the ambient air. The concept "one fiber can kill" may have as little relevance to reality as it is for environmental levels of numerous substances and physical factors that would be toxic at higher doses. The screening has probably contributed to the enhanced detection rate of mesothelioma and lung cancer in asbestos-exposed populations. Bias is not infrequent in asbestos research, e.g. attributing to asbestos of mesothelioma and lung cancer cases with the presence of fibers, although a cause-effect relationship remains unproven. Some studies rely on work or residence histories and interviews with relatives of questionable reliability [11].

Malignant pleural mesothelioma (MPM)

The stable or increasing incidence of MPM in developed countries despite asbestos bans is caused, at least in part, by increasing awareness, improvements of diagnostic equipment, screening effect in asbestos-exposed populations, and some overdiagnosis in view of the unclear demarcation of MPM as an entity. Apart from asbestos, potential etiologic factors of MPM include various mineral and artificial fibers, virus SV40, ionizing radiation, chronic inflammation (empyema, tuberculosis) and genetic predisposition [12-21]. For example, erionite is regarded to be a more potent carcinogen than asbestos. Human activities result in dispersal of erionite and other potentially carcinogenic fibers into populated areas [8,20]. Certain types of carbon nanotubes have been classified as possible human carcinogens [22]. Furthermore, there are indications that SV40 has contributed to the worldwide incidence increase of mesothelioma in recent decades despite asbestos bans [23]. SV40-like DNA sequences have been regularly found in MPMs [24]. After a laser microdissection, SV40 was demonstrated in MPM cells but not in nearby stromal cells [23]. When SV40 was injected via the intracardiac or intraperitoneal routes, ≥50% hamsters developed mesothelial tumors; 100% of hamsters injected into the pleural space developed mesotheliomas [25]. Systemic injections caused mesothelioma in ~60% of hamsters [20]. The incidence increase of MPM in the 1960s coincided with human exposure to SV40 in the period 1955-1963 when poliovaccines were contaminated with viable SV40 [23]. It can be assumed that invasive manipulations e.g. bronchoscopy used above-average in people exposed to asbestos contributed to dissemination of SV40, resulting in additional MPM cases. In the former Soviet Union (fSU), bronchoscopy

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and bronchial biopsy were performed and recommended in patients with asbestos-related bronchitis [26,27]; more details are in [28]. Due to the ageing population and because some people are predisposed to MPS given the presence of various mutations and carcinogens, the majority of mesotheliomas in future are expected to be spontaneous and unrelated to asbestos [4].

MPM is not clearly demarcated from other cancers; it had no diagnostic category within the ICD system until the 10th edition [29]. Histologically, MPM can resemble different cancers while the lack of specific biomarkers makes the diagnosis difficult. Tumors can undergo de-differentiation, becoming histologically similar to MPM. The differential diagnosis varies depending on the MPM subtype. Spindle cell tumors of pleura are particularly difficult to diagnose while immunohistochemistry may be of limited help [18,30,31] The differential diagnosis of MPM is a known problem; revisions of histological archives regularly found misclassified cases [31,32]. In one study, the initial diagnosis was confirmed in 67% of cases, ruled out in 13%, and remained uncertain in the rest [33]. Another expert panel changed the diagnosis of 14% from 5258 mesotheliomas [20]. According to an estimate, ~10% of MPMs in the United States have been misdiagnosed [32]. Among reasons is insufficient experience due to the rarity of MPM in the general population [31,32]. On the contrary, in asbestos-exposed people specialized experts perform well-aimed search for MPM. Accordingly, more MPMs are found, questionable or borderline cases being sometimes diagnosed as MPM. Litigation might also contribute to misattribution of cases to asbestos [11].

Lack of accurate biomarkers makes the diagnosis of MPM challenging [23]. Mesothelin has been discussed as one of the most promising markers [34]. However, it is overexpressed in several cancers including lung adenocarcinoma [35]. Mesothelin is not sufficiently sensitive [21,23,34,36]; it is often negative in sarcomatoid and epithelioid MPM [31,37,38]. Osteopontin has been viewed as a promising marker of MPM but results have been inconsistent. Similar to mesothelin, the clinical utility of osteopontin and fibulin-3 is limited by low sensitivity [39]. The microRNA down-regulation in MPM compared to lung cancer was regarded to be a promising marker [40,41]; but diagnostic accuracy is moderate as microRNA are deregulated also in some other malignancies [41-44]. Chromosomal and genomic aberrations of MPM are heterogenous [15,45,46]. Available information about the molecular basis of MPM is insufficient [47]. According to the Helsinki Criteria, established for attribution of mesothelioma to asbestos, no specific recommendations can be given for the use of markers in the screening for MPM [48,49]. Moreover, MPM may exhibit various molecular setups in different areas i.e. intra-tumoral heterogeneity and subclonality [50]. Contrary to other malignancies, driver mutations have not been clearly determined in MPM. There are no strong genetic markers [51,52]. Diagnosis of MPM on cytomorphological grounds is challenging, especially when reactive atypical mesothelial cells are present [53,54]. Notwithstanding the plethora of markers, none has been sufficiently

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specific [48,55]. A tumor diagnosed as MPM using algorithms and panels is not necessarily different from other cancers. The above explains enhanced diagnostic yield in exposed populations.

Russian science on asbestos

Asbestos-related diseases have been extensively studied in fSU. The prevailing opinion is that, if necessary precautions are taken, modern technologies of asbestos production and processing are safe, while bans applied in some countries are excessive [56,57]. Health hazards from low fiber concentrations are unproven. No enhanced risks have been demonstrated in residents near modern asbestos-processing plants. Epidemiological studies indicate a threshold [58,59]. Genetic adaptation to a certain level of asbestos fiber inhalation is deemed possible [60]. In fSU, corrugated asbestos sheets have been broadly used for roofing being often sawn by hand. The fiber emission from roofing materials during construction and use of buildings is regarded to be negligible [61]. Fiber concentrations in the indoor air are an order of magnitude below the permissible level [61]. Asbestos-cement pipes have been routinely used for drinking water distribution deemed safe as no risks from oral intake of fibers have been proven, the more so as fibers in asbestos cement are modified by aggregation with cement particles [62,63]. Studies show that the use of asbestos-cement pipes does not impair the quality of drinking water and their use has been approved by the Ministry of Health [64]. Asbestos-containing broken-stone ballast – a by-product of chrysotile enrichment – has been used for the gravelling of railroad embankments while enhanced concentration of airborne fibers was noticed both in trains and in nearby townships [65]. Similarly to asbestos-cement, carcinogenicity of fibers in asbestos board is decreased due to connection with starch [66]. Toxic effects from brake linings with and without asbestos do not differ significantly; there is no considerable air pollution from car brakes, while the traffic is safer with asbestos-containing linings [67,68]. In the process of braking, asbestos is transformed to forsterite, which is practically harmless [69,70]. Asbestos-containing materials (flat sheets, millboard, paper, clothing, gaskets, etc.) are broadly used now as before. Installation and repair without processing of asbestos-containing parts is deemed safe [68]. No increase in the registered incidence of mesothelioma has been found either among asbestos workers or residents of the areas with asbestos industry [71]. It was concluded on the basis of 3576 MPM cases that asbestos is neither a leading nor obligate causative factor [72]. Among 69 cases studied in Kazakhstan, asbestos exposure was detected in no one; geographic association of mesothelioma was found neither with asbestos mining nor with processing industry [73].

Chrysotile vs. amphiboles

Asbestos produced in Russia is almost exclusively chrysotile. The low toxicity of chrysotile compared to amphiboles is often stressed in the Russian literature. However, some experts admitted that the concept of much higher toxicity of inhaled amphibole fibers compared to chrysotile has not been sufficiently founded

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[74]. Carcino-, fibro-, mutagenicity and cytotoxicity of chrysotile was confirmed both in experiments and epidemiological studies performed in Russia [75-77]. In experiments, chrysotile was reported to possess acute toxicity, inducing the granulomatous tissue reaction [78]; its carcinogenicity did not differ significantly from that of amphiboles [79]. At the same time, there are strong industrial interests behind chrysotile. Accordingly, statements in favor of chrysotile (sometimes without references) can be encountered [80,81], for example: "Chrysotile fibers are easily dissolved and discharged" [81]. However, some data do not agree with this concept [74,79]. Of note, amphiboles (crocidolite, anthophyllite and others) have advantages for some areas of industrial use: they are more acid-resistant, thermo-stabile and durable than chrysotile [82].

Some papers by David Bernstein and co-workers [83,84] sound similar to Russian publications cited above [80,81], for example: "Following short-term exposure the longer chrysotile fibers rapidly clear from the lung and are not observed in the pleural cavity" [83]. Given the possibility of a post-depositional translocation of chrysotile fibers from the lung to pleura [85-90], the rate of asbestos retention cannot be determined only by measurements of fiber contents in pulmonary tissues. Conclusions by Bernstein et al. [83,91] about low biopersistence of chrysotile fibers were supported by self-references. However, results of their experiments can be explained by a chemical pre-treatment of fibers, inducing hydration, fragility and breaking [92]. "Bernstein's study protocol induces a very short fiber half-life, from which he concludes weak chrysotile carcinogenicity. Bernstein's findings contradict results obtained by independent scientists. Bernstein's results can only be explained by an aggressive pre-treatment of fibers, inducing many faults and fragility in the fibers' structure, leading to rapid hydration and breaking of long fibers in the lungs" [92]. The decomposition by acids does not prove solubility in living tissues. Different types of fibers were tested in the Gamble's solution imitating pulmonary interstitial fluid: both chrysotile and crocidolite exhibited very low solubility [93]. The dissolution ranged from a few nanograms of dissolved silicon per cm^2 of fiber surface (chrysotile and crocidolite) to several thousands of ng/cm^2 (glass wool). Aramide and carbon fibers were practically insoluble [93]. The study [93] was referenced but not discussed by Bernstein et al. [91]. The accelerated clearance of chrysotile from the lung can be partly attributed to the longitudinal splitting of fibers into thin fibrils, which can evade detection. As a result, the total number of fibrils would increase [89,94,95] possibly together with the carcinogenic potency [87,89,90,96-99]. Presumably, the thinner a fiber, the higher would be its carcinogenicity, as it can penetrate tissues more efficiently [100]. Asbestos fibers are found in the pleura post mortem, chrysotile being the predominant fiber in pleural plaques [101] and pleural tissues in general [85,88,101,102]. The concept of fiber migration to the pleura agrees with the fact that the primary affect of asbestos-related mesothelioma is usually in the parietal rather than visceral pleura [103]. The pathogenesis of MPM is related to the inflammatory microenvironment created by the fibers in pleura [51].

The incidence of mesothelioma is enhanced after exposures to pure chrysotile [104,105]. The relatively high frequency of mesothelioma among workers having contact with amphiboles was explained by averagely higher exposures [106]. There are discrepancies between animal and human data. The evidence for a difference in the potency between chrysotile and amphiboles in inducting lung cancer is "weak at best" [107]. In certain animal experiments, the carcinogenic potency of amphiboles and chrysotile was nearly equal for induction of both mesothelioma [95,108-110] and lung cancer [111,112]. Chrysotile was found to be even more carcinogenic than amphiboles in a study, where it was pointed out: "There was no evidence of either

less carcinogenicity or less asbestosis in the groups exposed to chrysotile than those exposed to the amphiboles" [109]. Technical details of the study [109] were discussed by Bernstein et al. [91] but not this essential result. In one rat study, chrysotile induced more lung fibrosis and tumors than amphiboles, which was explained by a larger fraction of fibers longer than 20 μm in the chrysotile preparation [113]. Chrysotile induced chromosomal aberrations and pre-neoplastic transformations of cells in vitro [108,114].

In humans, the lung cancer risk difference between chrysotile vs. amosite and crocidolite was estimated in the range from 1:10 to 1:50 [3]. The risk ratio of mesothelioma was estimated, respectively, as 1:100:500 [3], cited in reviews [33,115]. In a subsequent publication, the ratio 1:5:10 was suggested [116]. The same researchers [3] acknowledged that, in view of the fact that different asbestos types produced a similar harvest of lung tumors in animal experiments [88], it is problematic to reconcile animal and human data. The proposed explanation was that "in humans chrysotile (cleared in months) might have less effect than the amphibole fibers (cleared in years)" [3]. However, there are no reasons to suppose substantial interspecies differences in the fiber clearance mechanisms. As mentioned above, the chrysotile clearance from the lung may partly result from the fiber splitting and movement to the pleura. As for epidemiological studies, some of them are biased due to the screening effect with overdiagnosis in exposed populations, unclear demarcation of MPM from other cancers, imprecise exposure histories and, last but not least important, conflict of interest in researchers associated with the chrysotile industry.

The well known review [88], not cited by Bernstein et al. [83,91], concluded that animal experiments indicate an approximately equal risk associated with all asbestos types: "Even if one accepts the argument that chrysotile asbestos does not induce mesothelioma (which we do not), the risk of lung cancer (and asbestosis) cannot be dismissed, and chrysotile appears to be just as potent a lung carcinogen as the other forms of asbestos" [88]. Moreover, "Bernstein and colleagues completely ignored the human lung burden studies that refute their conclusion about the short biopersistence of chrysotile" [117]. In his reply to the latter comment [117], Bernstein left the essential arguments uncommented, dismissing them with the remark that the studies [118,119] "appear to support the concepts put forward by Bernstein et al." followed by self-references [120]. Numerous relevant publications [85-90,92,101,102,108,118,121-125], unsupportive of Bernstein's conclusions, were not cited in his reviews [83,91]. Another example: Bernstein et al. [91] cited the phrase from the review "Mesothelioma from chrysotile asbestos" that chrysotile is an "overwhelming fiber exposure" [126] but not the principal conclusion: "Chrysotile asbestos, along with all other types of asbestos, has caused mesothelioma" [126]. It was reasonably concluded that by failing to analyze or even mention contradicting data, Bernstein et al. did not provide an objective analysis, and have created impression that they published a document to support the interests of chrysotile producers [92,117].

The toxicity of fibers is generally determined by the three "D's": dose, dimension and durability (biopersistence), thin and long fibers tending to be more carcinogenic [12,127-129]. The biopersistence being equal, differences in carcinogenicity are associated with the length and thickness of fibers [91,130]. Long fibers of chrysotile were found to possess a relatively high toxicity as they cannot be efficiently engulfed and cleared by macrophages [132,133]. According to another report, thin short chrysotile fibers were found to be the prevailing fiber type detected in the lung and pleura of patients with MPM and deemed carcinogenic [134]. In addition, tremolite admixture in chrysotile products can potentiate carcinogenicity [135]. A review concluded that there is no

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compelling evidence that the increased incidence of MPM in chrysotile workers was caused solely by tremolite [88]. In one epidemiological study, the difference in MPM risk between pure chrysotile and its mixtures with amphiboles was insignificant [136]. The question of relative potency of different asbestos types was examined in a meta-analysis of 19 epidemiological studies assessing the influence of research quality on exposure-response estimates for lung cancer [115]. The difference in carcinogenic potency between chrysotile and amphiboles was difficult to ascertain when the meta-analysis was restricted to studies with fewer exposure assessment limitations [115] i.e. to those of higher quality. After accounting for quality, there appeared to be little difference in the exposure-response slopes for cumulative exposure to chrysotile compared to amphiboles [115,137]. According to a systematic review, pooled risk estimates for lung cancer were higher after exposures to amphiboles - 1.74 (95% CI 1.18 to 2.57) than to chrysotile - 0.99 (95% CI 0.78 to 1.25). The overall risk tended to be higher in intermediate- rather than in high-quality studies (there was no poor-quality group): 1.86 (95% CI 1.27 to 2.72) vs. 1.21 (95% CI 0.79 to 1.87) [138]. Significant differences between results obtained in high- vs. low-quality studies are indicative of bias due to a conflict of interest, as it is obviously easier to find support for preconceived ideas in poor-quality and manipulated studies rather than in high-quality research. In conclusion, it is widely believed that serpentine (chrysotile) is less toxic than amphibole (actinolite, amosite, anthophyllite, crocidolite, tremolite) asbestos. However, this difference should be proven and quantified by research independent of industrial interests.

Discussion

Asbestos bans have been partly based on the research influenced by political interests. There was a suggestion that “grassroots organizations intimidated governments into approving more restrictive regulations” [139]. Of note, some “grassroots” and Green activists may serve other governments or companies. The same is partly true also for the anti-nuclear activism strangulating the nuclear energy thus boosting fossil fuel prices. Citizens should be aware that their best intentions may be exploited to disadvantage their own countries. Some anti-asbestos activists might have conflicts of interest related e.g. to lawyers’ earnings from asbestos litigation, or interests of construction firms performing asbestos removal with exposures of abatement workers. In particular, asbestos research has been influenced by the interests of chrysotile industry [140]. The quality of studies, potential bias and conflicts of interest should be taken into account defining inclusion criteria for studies into reviews and meta-analyses. A possible way to objective information may be lifelong bioassays using also larger animals including primates [141]. Such experiments might reveal threshold exposure levels for different fiber types. The bioassays with fiber inhalation, comparable to exposures in the asbestos industry, can be performed without invasive procedures thus being ethically acceptable. In this connection, animal experiments using “exposure concentrations that were orders of magnitude greater than those reported for worker exposure” [142] are of limited informativity.

Carcinogenic potencies depend not only on biopersistence but also on dimensions of fibers of various types [12,127-129]. This is an additional argument in favor of the "All Fibers Equal" [143] approach to asbestos and some other fibers. This concept can be used provisionally, pending reliable evidence. The All Fibers Equal basis of safety regulations is technically most plausible, being partly compatible with current knowledge contradictive as it is. Considering the strong economic interests behind chrysotile [140,144,145], and newly also some artificial fibers, any deviations from the All Fibers Equal [143] concept must be based on high-quality, independent research. Substitution of asbestos by artificial fibers would not necessarily eliminate health risks [12,13,146,147]. The carcinogenicity of asbestos substitutes e.g. carbon nanotubes comes to light these days. Studies indicate that asbestos fibers and carbon nanotubes with certain dimensions exert their toxic effects through the same mechanisms, in particular, chronic macrophage activation resulting in inflammation [148]. As mentioned above, carbon nanotubes are biopersistent and certain types of them have been classified as possible human carcinogens [22].

Conclusion

The number of publications about asbestos is growing; it is increasingly difficult to distinguish between objective and biased information. The asbestos research has been influenced by conflicts of interest. Asbestos is banned in some countries, while others augment production and exports [149]. Internationally traded chrysotile products contain various amounts of amphiboles [150]. Policies aimed at regulating asbestos should target both pure chrysotile and mixtures [136]. Different asbestos types have their technical advantages and preferred application areas. Asbestos is a low-cost material and an excellent reinforcing fiber. The brake pads' durability is influenced by the reinforcing materials used. The traffic is believed to be safer with asbestos-containing linings [67,68]. Asbestos cement (fibrolite) constructions are sturdy and inexpensive; their use increased during the World War II. The fireproofing properties of asbestos are well known. It can be reasonably assumed that the non-use of asbestos-containing brakes, fireproofing and insulation laggings has increased the damage and numbers of victims of traffic accidents, fires and armed conflicts. Nowadays, when a probability of conflicts seems to be enhanced, the attitude to asbestos should be changed. Most importantly, asbestos-related science must be separated from industrial interests.

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