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Asbestos and anti-asbestos activism: medical and economical aspects

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Asbestos and anti-asbestos activism: medical and economical aspects

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 found, although cause-effect relationships remain unproven. Some studies rely on work or residence histories of questionable reliability. 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. Chrysotile 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 short communication is aimed to discuss medical and economical aspects of asbestos and anti-asbestos propaganda in some countries. 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 (Hodgson and Darnton 2000; Jargin 2017, 2018; Paustenbach et al. 2021). 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 (Paustenbach et al. 2021). Asbestos fibers are present in the natural environment due to erosion of surface deposits. Naturally occurring asbestos has been commonly found in populated areas (Noonan 2017). The natural emission contributes to a dispersion of chrysotile and amphibole asbestos fibers. Presumably, natural releases dwarf anthropogenic contributions to the atmospheric dispersion of above-named fiber types (Ilgren et al. 2015; Noonan 2017). 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 (Berry et al. 2022; Malinconico et al. 2022). In a study from Milan, asbestos fibers were found in 35 of 55 (63.6%) autopsy cases from the general population (Casali et al. 2015). At autopsies of exposed people the pulmonary and pleural tissues are sampled more abundantly and examined more thoroughly, hence the higher probability to find fibers. The presence of fibers proves by itself neither professional exposure nor asbestos-related disease. Inhalation and discharge of fibers occur normally being in a dynamic balance (Bayram and Bakan 2014; Casali et al. 2015). 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 cancer cases with the presence of fibers, although a cause-effect relationship remained unproven. Some studies rely on work or residence histories and interviews with relatives of questionable reliability (Yang et al. 2008).

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 (Carbone et al. 2019; Dipper et al. 2021; Donaldson et al. 2013; Greim et al. 2014; Jasani and Gibbs A. 2012; Panou et al. 2015; Røe and Stella GM. 2015; Røe et al. 2009; Rossini et al. 2018; Tomasetti et al. 2009). 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 (Berry et al. 2022; Carbone et al. 2019). Certain types of carbon nanotubes have been classified as possible human carcinogens (Kane et al. 2018). Furthermore, there are indications that SV40 has contributed to the worldwide incidence increase of mesothelioma in recent decades despite asbestos bans (Carbone et al. 2020). SV40-like DNA sequences have been regularly found in MPMs (Testa et al. 1998). After a laser microdissection, SV40 was demonstrated in MPM cells but not in nearby stromal cells (Carbone et al. 2020). 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 (Cicala et al. 1993). Systemic injections caused mesothelioma in ~60% of hamsters (Carbone et al. 2019). The incidence increase of MPM in the 1960s coincided with human exposure to the virus in the period 1955-1963 when poliovaccines were contaminated with viable SV40 (Carbone et al. 2020). 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 (SU), bronchoscopy and bronchial biopsy were performed and recommended in patients with asbestos-related bronchitis (Likhacheva et al. 2000; Milishnikova et al. 1990). Due to the ageing population and because some people are predisposed to MPM, given various mutations and carcinogens, the majority of mesotheliomas in future are expected to be spontaneous and unrelated to asbestos (Paustenbach et al. 2021).

MPM is not clearly demarcated from other cancers. 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 especially difficult to diagnose while immunohistochemistry may be of limited help (Carbone and Yang 2017; Kerger et al. 2014; Panou et al. 2015) The differential diagnosis of MPM is a known problem; revisions of histological archives regularly found misclassified cases (Carbone and Yang 2017; Chen et al. 2017). In one study, the initial diagnosis was confirmed in 67% of cases, ruled out in 13%, and remained uncertain in the rest (Goldberg et al. 2006). Another expert panel changed the diagnosis in 14% from 5258 mesotheliomas (Carbone et al. 2019). According to an estimate, ~10% of MPMs in the United States have been misdiagnosed (Chen et al. 2017). Among reasons is insufficient experience due to the rarity of MPM in the general population (Carbone and Yang 2017; Chen et al. 2017). On the contrary to the general population, in asbestos-exposed people specialized experts perform the search for MPM. Accordingly, more MPMs are found, questionable or borderline cases being sometimes classified as MPM. Litigation might also contribute to misattribution of cases to asbestos (Yang et al. 2008).

Lack of reliable biomarkers makes the diagnosis of MPM challenging (Carbone et al. 2020). Mesothelin has been discussed as one of the most promising markers (Creaney al. 2015). However, it is overexpressed in several cancers including lung adenocarcinoma (Ho et al. 2007). Mesothelin is not sufficiently sensitive (Bibby et al. 2016; Carbone et al. 2019; Creaney al. 2015; Dipper et al. 2021); it is often negative in sarcomatoid and epithelioid MPM (Carbone and Yang 2017; Grigoriu et al. 2009; Pantazopoulos et al. 2013). Osteopontin has been a promising marker

but data are inconsistent. Similar to mesothelin, the clinical utility of osteopontin and fibulin-3 is limited due to low sensitivity (Harris et al. 2019). The microRNA down-regulation in MPM compared to lung cancer was regarded to be a promising marker (Gee et al. 2010; Reid 2015); but diagnostic accuracy is moderate as microRNA are deregulated also in some other malignancies (Han et al. 2021; Reid 2015; Sheff et al. 2012; Truini et al. 2014). Chromosomal aberrations of MPM are heterogenous (Lindholm et al. 2007; Musti et al. 2006; Røe et al. 2009). Available information about the molecular basis of MPM is insufficient (Lorenzini et al. 2021). 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 (Ferrari et al. 2020; Wolff et al. 2015). Moreover, MPM may exhibit various molecular setups in different areas i.e. intra-tumoral heterogeneity and subclonality (Rossi et al. 2021). Contrary to other malignancies, driver mutations have not been clearly determined in MPM. There are no strong genetic markers (Cersosimo et al. 2021; Vandenhoeck et al. 2021). Diagnosis of MPM on cytomorphological grounds is challenging, especially when reactive atypical mesothelial cells are present (Blyth and Murphy 2018; Eccher et al. 2021). Notwithstanding the plethora of markers, none has been sufficiently specific (Ferrari et al. 2020; Schillebeeckx et al. 2021). 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 former SU. 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 (Elovskaia 1997; Izmerov and Kovalevskii 2004; Neiman et al. 2006). 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 (Kogan et al. 1993; Shtol' et al. 2000). Genetic adaptation to a certain level of asbestos fiber inhalation is deemed possible (Tsurikova et al. 1992). In the former SU, corrugated asbestos sheets have been broadly used for roofing. The fiber emission from roofing materials during construction and use of buildings is negligible. Fiber concentrations in the indoor air are an order of magnitude below the permissible level (Kashanskii et al. 2004). Asbestos-cement pipes have been broadly used for drinking water distribution deemed safe as no risks from oral intake of fibers have been proven, the more so as fibers are modified by aggregation with cement particles (Krasovskii and Egorova 1985; Krasovskii and Mozhaev 1993). 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 (Repina et al. 2009). 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 (Kaptsov et al. 2003). Similarly to asbestos-cement, carcinogenicity of fibers in asbestos board is decreased due to aggregation with cellulose (Kashanskii and Kogan 1995). 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 (Iatsenko et al. 1994; Kovalevskii 2009). In the process of braking, asbestos is transformed to forsterite, which is practically harmless (Iatsenko and Kogan 1990; Iatsenko et al. 1991). 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 (Kovalevskii 2009). No increase in the registered incidence of mesothelioma has been found either among asbestos workers or residents of the areas with modern asbestos industry (Izmerov et al. 1998). It was concluded on the basis of 3576 MPM cases that asbestos is neither a leading nor obligate causative factor (Kashanskii 2008). 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 (Kashanskii et al. 2008).

Chrysotile vs. amphiboles

It is widely believed that serpentine (chrysotile) is less toxic than amphibole (actinolite, amosite, anthophyllite, crocidolite, tremolite) asbestos but there are discrepancies between human (epidemiological) and experimental data. Asbestos produced in Russia is almost exclusively chrysotile. The low toxicity of chrysotile compared to amphiboles is often stressed in the literature. However, some experts admitted that the concept of much higher toxicity of inhaled amphibole fibers compared to chrysotile has not been sufficiently founded (Kogan 1995). Carcino-, fibro-, mutagenicity and cytotoxicity of chrysotile was confirmed both in experiments and epidemiological studies performed in Russia (Pylev et al. 1988, 2010; Troitskaia NA. 1993). In experiments, chrysotile was reported to possess acute toxicity, inducing the granulomatous tissue reaction (Kashanskii et al. 1994); its carcinogenicity did not differ significantly from that of amphiboles (Pylev 1987). Certai conclusion by Bernstein (2014) should be commented in this connection, for example: “Following short-term exposure the longer chrysotile fibers rapidly clear from the lung.” Given the possibility of a post-depositional translocation of chrysotile fibers from the lung to pleura (Coin et al. 1994; Kohyama and Suzuki 1991; Nicholson WJ. 1991; Sebastien et al. 1980; Stayner et al. 1996; Suzuki and Yuen 2002), the rate of asbestos retention cannot be determined only by evaluation of the fiber contents in pulmonary tissues. Conclusions by Bernstein about low biopersistence of chrysotile 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 (Pezerat 2009). “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” (Pezerat 2009). 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 (Larsen 1989). 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 (Larsen 1989). The latter study was referenced but not discussed by Bernstein et al. (2013). 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 (Coin et al. 1994; Currie et al. 2009; Smith and Wright 1996) possibly together with the carcinogenic potency (Asgharian et al. 2018; Coin et al. 1994; Kohyama and Suzuki 1991; Suzuki and Yuen 2002; Yu et al. 1991). Presumably, the thinner a fiber, the higher would be its carcinogenicity, as it can penetrate tissues more efficiently (Ramada Rodilla et al. 2022). Asbestos fibers are found in the pleura post mortem, chrysotile being the predominant fiber in pleural plaques (Dodson et al. 1990) and pleural tissues in general (Gibbs et al. 1991; Sebastien et al. 1980; Stayner et al. 1996). The concept of fiber migration to the pleura agrees with the fact that a primary tumor of asbestos-related mesothelioma is more often located in the parietal rather than visceral pleura (Sekido 2013). Moreover, “Bernstein and colleagues completely ignored the human lung burden studies that refute their conclusion about the short biopersistence of chrysotile” (Finkelstein 2013). Numerous relevant publications, unsupportive of Bernstein’s conclusions, were not cited in his reviews; more details and references are in (Jargin 2017). 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 (Finkelstein 2013; Pezerat 2009)

The incidence of mesothelioma is enhanced after exposures to pure chrysotile (Finkelstein and Meisenkothen 2010; Frank 2020). The relatively high frequency of mesothelioma among workers having contact with amphiboles was explained by averagely higher exposures (Stayner

et al. 1997). As mentioned above, 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” (Stayner 2008). In certain animal experiments, the carcinogenic potency of amphiboles and chrysotile was nearly equal for induction of both mesothelioma (Harington 1991; Smith and Wright 1996; Wagner 1975; Wagner et al. 1974) and lung cancer (Berman et al. 1995; Landrigan et al. 1999). Chrysotile was found to be even more carcinogenic than amphiboles in a study, whereas 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” (Wagner et al. 1974). Technical details of the latter study were discussed by Bernstein et al. (2013) but not this essential conclusion. In one rat study, chrysotile induced more lung fibrosis and tumors than amphiboles, which was explained by a large fraction of fibers longer than 20 μm in the used chrysotile preparation (Davis et al. 1978). Chrysotile induced chromosomal aberrations and pre-neoplastic transformations of cells in vitro (Harington 1991; Hesterberg and Barrett 1984).

In humans, the lung cancer risk difference between chrysotile vs. amosite and crocidolite was estimated in the range from 1:10 to 1:50. The risk ratio of mesothelioma was estimated, respectively, as 1:100:500 (Hodgson and Darnton 2000), cited in reviews (Goldberg et al. 2006; Lenters et al. 2011). In a later publication, another ratio (1:5:10) was suggested (Hodgson and Darnton 2010). The same researchers noticed that, in view of the fact that different asbestos types produced a similar harvest of lung tumors in animal experiments, 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)” (Hodgson and Darnton 2000). However, there are no reasons to suppose substantial interspecies differences in the fiber clearance. 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 apparently 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 asbestos industry.

The toxicity of fibers is generally determined by the three “D’s”: dose, dimension and durability (biopersistence) (Berman and Crump 2008; Donaldson et al. 2013; IARC 1996; Wang et al. 2017). The biopersistence being equal, differences in carcinogenicity are associated with the length and thickness of fibers (Mossman et al. 2011). Long fibers of chrysotile were found to possess a relatively high toxicity as they cannot be efficiently engulfed and cleared by macrophages (Gaudino et al. 2020; Hillerdal and Henderson 1997). 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 (Suzuki et al. 2005). In addition, tremolite admixture in chrysotile products can potentiate carcinogenicity (Langer and Nolan 1994). A review concluded that there is no compelling evidence that the increased incidence of MPM in chrysotile workers was caused solely by tremolite (Stayner et al. 1996). In one epidemiological study, the difference in MPM risk from pure chrysotile and its mixtures with amphiboles was insignificant (Wong et al. 2020). 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. The difference between chrysotile and amphiboles was difficult to ascertain when the meta-analysis was restricted to studies with fewer exposure assessment limitations (Lenters et al. 2011) 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 (Lenters et al. 2011; Marsili et al. 2016). 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) (Kwak et al. 2022). 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. As mentioned above, it is widely believed that chrysotile is less toxic than amphiboles but 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 and industrial interests. Some anti-asbestos activists might have conflicts of interest related the manufacturing of asbestos substitutes (Neiman et al. 2006), lawyers' earnings from asbestos litigation, or interests of construction firms performing asbestos removal with exposures of abatement workers. 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 could be lifelong bioassays using also larger animals including primates (Gwinn et al. 2011). 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" (Bernstein et al. 2020) are of limited informativity. Substitution of asbestos by artificial fibers would not necessarily eliminate health risks (Donaldson et al. 2013; Greim et al. 2014; Toyokuni 2013; Van Berlo et al. 2012). 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 (Gupta et al. 2022). As mentioned above, carbon nanotubes are biopersistent and certain types of them have been classified as possible human carcinogens (Kane et al. 2018).

Conclusion

The number of publications about asbestos is growing; it is 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 continue production and exports (Brims 2009). Internationally traded chrysotile products contain admixtures of various amounts of amphiboles (Tossavainen et al. 2001). Different asbestos types have their technical advantages and preferred application areas. Amphiboles (crocidolite, anthophyllite and others) have advantages for some areas of industrial use: they are acid-resistant, thermo-stabile and durable (Shanin et al. 1983). 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 safer with asbestos-containing linings. Asbestos cement (fibrolite) 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 laggings has augmented 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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