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Asbestos and Anti-Asbestos Activism: Medical, Economical and Political Aspects

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Abstract

Potential health damage due to asbestos exposure has been evaluated using information from the mid-20th century and earlier, when exposures of workers and residents were greater. A linear no-threshold model has been applied, although its relevance is unproven. The fibers get into the air due to erosion of surface deposits and industries unrelated to asbestos. If looked for, the fibers are often found postmortem. The research is associated with bias: attributing malignancy to asbestos when fibers are found, although causality remains unproven. A history of professional exposure does not necessarily prove a cause-effect relationship. Asbestos bans weaken defenses, enhance damage from fires, terrorism and armed conflicts. Potential toxicity of serpentine and amphibole asbestos is analyzed here. Many animal experiments indicate similar levels of toxicity, while epidemiological studies of humans witness in favor of higher toxicity of amphiboles. This can be partly attributed to a bias in the latter study type. Epidemiological research in humans will not provide much information on low-dose impact. Reliable results can be obtained in large-scale lifelong bioassays with inhalation of fibers comparable to professional exposures.

Keywords: Asbestos; Lung cancer; Mesothelioma; Chrysotile; Amphiboles.

Introduction

It is essential in our time of international instability that researchers retain impartial. Since many years we have tried to show that some scientific writers, exaggerating harm from low-dose ionizing radiation and nuclear facilities, are acting in agreement with the interests of fossil fuel producers [1, 2]. The same phenomenon exists in regard to asbestos. Undoubtedly, asbestos is an etiological factor of mesothelioma, lung cancer, asbestosis, and other pathological conditions. Pleural mesothelioma (PM) is an infrequent malignancy; and the fibers are not the only etiological factor. The risks were extrapolated from the past, when professional exposures were much higher than today. The linear model with the risk extrapolation down to minimal doses has been applied, although this model is not supported by evidence [3]. Asbestos fibers find their way to the environment through erosion of natural deposits. Air and water often contain asbestos due to human activities such as land excavation and tunnel construction [4, 5]. In a research from Italy, asbestos was detected in ~64 % of random post mortem examinations [6]. At autopsies of individuals at risk, respiratory organs are sampled abundantly so that a chance to find a lesion is higher than in the general population. The finding of fibers is not is not enough to prove asbestos-related etiology [6, 7].

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Some research relies on professional and residential histories of doubtful reliability [8]. The inhaling and discharge of fibers occurs permanently; both processes are in equilibrium [7]. Screening effect has contributed to the increased registered incidence of malignant PM and lung cancer in exposed people [8]. According to the Helsinki Criteria for Diagnosis and Attribution, "even a brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related" [9]. Such approach was doubted as it leads to misclassification of spontaneous cases as asbestos-related ones [10].

Pleural Mesothelioma

Asbestos has been banned in more than 50 countries [11]. Nonetheless the incidence of PM remains largely unchanged, among others, due to the screening effect, increased public awareness, advance of technology as well as some percentage of incorrect diagnoses due to unclear differentiation of PM from other malignancies. Potential etiologic factors of PM include various fibers (erionite, some artificial fibers, nanotubes, and others), ionizing radiation, SV40 viral particles and chronic inflammation [12, 13]. Erionite is at least as potent carcinogen as asbestos. Similarly to asbestos, erionite finds its way into environment due to the land excavation and tunneling [14, 15]. Some varieties of carbon nanotubes have been classified as potential carcinogens [16-18]. Moreover, the SV40 virus contributed to the incidence elevation of PM [19]. DNA sequences corresponding to SV40 are often detected in PM [20]. SV40 was found in PM cells but not in the surrounding interstitium [19]. SV40-related DNA sequences are found in PM more often than in other malignancies [21]. SV40 can persist in mesothelium that remains infected releasing viruses. Inoculation of SV40 induces mesothelioma in a high percentage of laboratory animals [15, 22]. One of the causes of enhanced PM incidence was a contamination of polio vaccines with SV40 in the 1960s and thereafter [19]. Furthermore, bronchoscopy is relatively frequent among patients with supposedly asbestos-related conditions, which might have transferred infectious agents such as SV40 [23, 24]. The bronchoscopy was applied in Russia for the diagnosis of dust diseases and other pulmonary conditions, whereas indications were sometimes questionable [25]. Finally, hereditary factors play a role in the causation of PM [3, 13]. PM had not been designated as a separate entity by the ICD until the 10th Edition [26]. Histologically, PM may be structured similarly to different cancers. Other malignancies can de-differentiate, becoming undistinguishable from PM. In particular, spindle cell tumors of pleura are hard to differentiate from other tumors even using special methods [27-29].

Re-evaluations of specimens regularly detected misdiagnosed cases [27, 30]. The absence of specific markers renders the differentiation problematic; immunochemistry and molecular pathology are not always helpful. Approximately 10% of malignant PMs in the United States have been misclassified [30]. After a review of specimens, the morphological diagnosis of PM remained unmodified in 67%, changed in 13 % of cases, being equivocal in the rest [31]. The molecular-pathologic image of PM is rather vaguely defined [32]. Proposed criteria are not sufficiently specific. Mesothelin is frequently mentioned although it is expressed also in other cancers [33]. Fibulin-3 has diagnostic value [34], but it can be found in different malignancies [33]. Osteopontin was encouraging although the data are variable [35]. The differential-diagnostic usefulness of the altered microRNA expression is not very high [36, 37]. FISH testing detected the loss of p16/CDKN2A from 9p21 deletion, reportedly showing 100% specificity for malignant mesothelial cell proliferation. However, its sensitivity for pleural mesothelioma ranged from 48% to 88% [38]. Moreover, PM often shows heterogeneity and sub-clonality [39]. Driver mutations were not determined convincingly [40]. Conclusiveness of pleura biopsy including cytological methods has not been high [41]. A tumor classified as PM is not always biologically different from other neoplasia. The unclear demarcation from other cancers elevates the screening effect in exposed people: PM is sometimes diagnosed on doubtful specimens. On the other hand, in the general population PM is often classified as other cancers [34].

Studies in Post-Soviet Countries

In most of post-Soviet countries the prohibition of use concerns amphibole asbestos and does not apply to chrysotile [42]. During the period 1992-2011, only 3 cases of malignant mesothelioma were detected in asbestos workers in Ukraine [43]. Previously, no cases of malignant PM had been identified among those who worked with asbestos. The cancer incidence rates of workers in the asbestos cement industry of Ukraine turned out to be lower than among the entire population [43, 44]. Asbestos-related diseases have been studied in Russia. It has been repeatedly suggested that chrysotile is less harmful than amphiboles. However, carcinogenic potency of chrysotile was detected in experimental and epidemiological studies [45-47]. The prevailing opinion is that modern chrysotile production and processing industries are sufficiently safe if adequate precautions are observed; whereas prohibitions imposed by some nations are superfluous. No risk elevation was found in people residing around factories working with asbestos. Cancers in patients exposed to asbestos are morphologically indistinguishable from sporadic ones. Results of epidemiological research are compatible with existence of a threshold [48, 49]. Evolutionary adaptation to environmental concentrations of fibers is supposed to exist [50].

Corrugated asbestos is applied in the construction industry. Concentrations of asbestos fibers in the indoor air are negligible. Asbestos-cement water pipes are safe as no harm from oral intake has been demonstrated. Analogously to asbestos-cement, risks from asbestos board are low or inexistent due to aggregation with cellulose. Asbestos-containing broken stone was used for railroad embankments. Asbestos-containing brake linings cause no remarkable air pollution. Building materials with asbestos are broadly used. Repair of devices with asbestos-containing parts (insulation, gaskets) is regarded to be safe [51-53]. Elevation of the registered incidence of PM has been found neither in employees nor in inhabitants in the vicinity of asbestos factories [54]. However, the latest study did confirm an elevated risk of PM and lung carcinoma in chrysotile workers [47]. 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 [55].

Chrysotile vs. Amphiboles

It is generally accepted that amphibole asbestos (crocidolite, amosite, anthophyllite, tremolite) is more toxic than chrysotile. However, there is discrepancy between epidemiological and experimental data. In many experiments, exemplified below, the carcinogenicity of amphiboles did not differ significantly from that of chrysotile [56]. Certain research produced conclusions similar to Russian publications: "Following short-term exposure the longer chrysotile fibers rapidly clear from the lung and are not observed in the pleural cavity" [57]. However, it is known that chrysotile fibers can split longitudinally and migrate to pleura [58-62]; therefore, asbestos burden cannot be comprehensively evaluated by counting fibers only in the lungs. Suppositions of short retention time of chrysotile in living tissues were endorsed by references to experimental works [57, 63]. Of note, results these studies were explained by processing of fibers by acids: "Study protocol induces a very short fiber half-life… findings contradict results obtained by independent scientists… 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" [64]. Induced fragility in the acidic environment is not the same as solubility in living tissues. Various fibers were tested in the Gamble solution (simulated lung fluid): both chrysotile and amphiboles demonstrated minimal solubility [65]. This latter study was not discussed in a review despite figuring in the reference list [63]. As mentioned above, the earlier disappearance of chrysotile from pulmonary tissues can be partly explained by splitting into thinner fibrils that are more difficult to visualize. The total quantity of fibers would thus increase [58, 59, 62, 66-71]. Chrysotile fibers predominate in the pleural tissues including pleural plaques [61, 72, 73]. The concept of chrysotile fibers translocation to the pleura is in agreement with the predominant location of the primary affect of PM in the parietal pleura [74]. The PM incidence is increased after exposures to pure chrysotile [75, 76]. The comparatively frequent PM in persons working with amphiboles was attributed to more intense exposures in the past [77]. The evidence for a difference in pathogenicity of amphiboles vs. chrysotile was characterized as weak or inexistent [78]. In some bioassays, the carcinogenicity of chrysotile vs. amphiboles was approximately the same for PM [68, 79-81] and lung carcinoma [82, 83]: "There was no evidence of either less carcinogenicity or less asbestosis in the groups exposed to chrysotile than those exposed to the amphiboles" [81].

Chrysotile possessed a higher potency compared to the amphiboles in one research with the following conclusion: "There was no evidence of either less carcinogenicity or less asbestosis in the groups exposed to chrysotile than those exposed to the amphiboles" [81]. In another rat experiment, chrysotile fibers produced more pulmonary sclerosis and neoplasia than amphiboles [84]. Furthermore, chrysotile produced precancerous changes in cell cultures [79, 85]. Based on human epidemiology, the risk difference for lung carcinoma between chrysotile vs. amosite and crocidolite was estimated between 1:10 and 1:50. The same value for PM was, correspondingly 1:100:500 [86], quoted in reviews [31, 87]. In a later paper, another proportion was presented: 1:5:10 [88]. The same authors stressed that, considering that various asbestos types demonstrated a similar carcinogenic potency in animal bioassays, it is hard to explain the discrepancy with epidemiological data in humans [86].

Experiments including primates and other species could help to clarification. As discussed above, the chrysotile clearance mechanism includes cleavage of the fibers with a subsequent relocation to pleural tissues. Considering the research in exposed human populations, some results have been influenced by the overdiagnosis of PM in questionable cases among asbestos-exposed people due to unclear demarcation of PM from other malignancies and by unreliable exposure histories. The seminal review concluded that animal bioassays indicate a nearly equal risk from asbestos of all 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 " [61]. Furthermore, "Bernstein and colleagues completely ignored the human lung burden studies that refute their conclusion about the short biopersistence of chrysotile" [70]. In his response to this latter expert, the authors depreciated the criticism commenting that the research [89,90] "appears to

support the concepts put forward by Bernstein" [91]. Many articles cited above, disagreeing with the concept, have not been referenced in reviews [57, 63]. Moreover, Bernstein et al. [63] picked up a quote from the paper "Mesothelioma from chrysotile asbestos" that chrysotile is an "overwhelming fiber exposure" [92] but disregarded the main conclusion: "Chrysotile asbestos, along with all other types of asbestos, has caused mesothelioma" [92]. It was pointed out that by not citing dissonant papers, Bernstein has not performed a veritable assessment but, apparently, compiled an argument in favor of chrysotile producers [64, 70].

Toxic effects of fibers depend on their biopesistence and dimensions [8, 93-96]. Thin and long fibers of chrysotile were found to be relatively pathogenic as they are not effectively removed by phagocytes [97, 98]. Admittedly, there have been also contradicting reports [99]. More experiments are needed. Fiber dimensions might be more important that the asbestos type. In a population-based research, the difference in PM frequency between pure chrysotile and its mixtures with amphiboles was insignificant [100].

The carcinogenicity of various asbestos types was assessed in a meta-analysis of epidemiological studies evaluating the impact of research quality on exposure-response estimates for lung cancer. The difference between chrysotile and amphiboles was hard to confirm if the meta-analysis was restricted to studies with fewer limitations [87]. After accounting for research quality, there was not much difference between the exposure-response slopes for the amphiboles and chrysotile [87, 101]. As per another review, risk estimates for lung cancer were higher under the impact of amphiboles than of chrysotile. At the same time, the difference tended to be higher in middle- rather than high-quality studies (no "low quality" studies were analyzed) [102]. Substantial differences between results of higher- and lower-quality research may be indicative of bias.

Discussion

Certain population-based studies are biased because of the effect of screening with enhanced diagnostic yield in exposed populations and unreliable case histories. It is hard to distinguish between more and less reliable reports. Moreover, "grassroots organizations intimidated governments into approving more restrictive regulations" [103]. Asbestos is banned in many countries while others are elevating its production. Various fibers are mixed in the international trade [104]. Certain non-asbestos fibers and carbon nanotubes may be harmful for health. Similarly to asbestos, the carcinogenic effect depends on the fiber length, width and solubility [16, 18, 105, 106]. Reliable data can be obtained in lifelong experiments. Bioassays with inhalation of fibers comparable to industrial exposures are ethically acceptable. Of note, results of experiments with "exposure concentrations that were orders of magnitude greater than those reported for worker exposure" [107] are difficult to interpret as the results cannot be directly extrapolated onto workers of modern asbestos industry.

Conclusion

All forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, anthophyllite) have been classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans (Group 1) [26]. This classification is not questioned here. However, carcinogenicity of low doses and applicability of the no-threshold model remains unproven. Asbestos is widely used due to its well-known physical and chemical properties [108]. Various asbestos types have their preferred areas of application. The physical properties of amphiboles vary, but in general they are characterized by higher acid and thermal stability. In particular, crocidolite has high tensile strength [109, 110]. Asbestos cement constructions are robust and inexpensive at the same time. Asbestos bans weaken defenses, enhance the damage from terrorism and warfare. Probably not all writers and Green activists exaggerating health-related and ecological harm from low doses of asbestos fibers realize that they serve the interests of potential adversaries. Citizens should be aware that their best intentions may be exploited to disadvantage their own countries. Strictly observed realistic safety regulations will bring more benefit for the public health than excessive restrictions that would be neglected in the countries with prevailing disrespect for laws and mores, bringing to the trespassers economic advantages. Considering industrial interests behind chrysotile, any deviations from the All Fibers Equal [111, 112] concept should be based on reliable studies, devoid of conflict of interest. Epidemiological research in humans is necessary but will not provide much reliable information on low-dose effects. Screening effect, selection and ideological bias will bring about new information about elevated risks that will not prove cause-effect relationships. Reliable results can be obtained in lifelong bioassays.

Conflict of interest

The author declares no conflict of interest.

Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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