Asbestos and anti-asbestos campaign: medical and economical 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 post mortem. The research is associated with bias: attributing malignancy to asbestos when fibers are found, although causality remains unproven. A history of exposure does not necessarily prove a cause-effect relationship. Asbestos bans enhance damage from fires and accidents. Potential toxicity of serpentine and amphibole asbestos is analyzed here. Many animal experiments indicate similar level 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.

Key words: Asbestos, lung cancer, mesothelioma; chrysotile, amphiboles

Since many years we have discussed bias in the epidemiological research on the biological impact of low-dose ionizing and radiofrequency radiation [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 per se is not a sufficient proof of asbestos-related etiology [6,7]. 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 [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, technological advance and 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), ionizing radiation, SV40 virus 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 in some places [19]. Furthermore, bronchoscopy is sometimes used in patients with supposedly asbestos-related conditions, which might have transferred infectious agents such as SV40 [23-25]. 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 may be difficult to distinguish 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 PMs are sometimes classified as other cancers [34].

Asbestos research in Russia and the Soviet Union

In most countries of the former Soviet Union, the prohibition of use concerns amphibole asbestos and does not apply to chrysotile, which has been supported by research [42-55]. Asbestos-related diseases have been broadly studied in Russia. It has been repeatedly suggested that chrysotile is less harmful than amphiboles. Although carcinogenic potency of chrysotile was detected in some, predominantly experimental, studies [45-47], the prevailing opinion is that modern chrysotile production and processing industries are 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]. Admittedly, the latest study did confirm an elevated risk of PM and lung carcinoma in chrysotile workers [47]. 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 turned out to be lower than among the entire population [43,44]. 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 published statements are questionable: “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]. Results of 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 and conclusion

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. There is an opinion that “grassroots organizations intimidated governments into approving more restrictive regulations” [103]. 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. 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.

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 increase damage from fires and accidents. Strictly observed realistic safety regulations will bring more benefit for the public health than excessive restrictions. 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 and selection bias will to the appearance of new reports on elevated risks, which would not prove cause-effect relationships. Reliable results can be obtained in lifelong bioassays.

Conflict of interest

The author declares no conflict of interest.

References

1. Jargin SV. Overestimation of medical consequences of increased radiation background. Medical Radiology and radiation safety 2008;53(3):17-22.

2. Jargin SV. On the biological effects of radiofrequency electromagnetic fields. Siberian Scientific Medical Journal. 2019;39(5):52-61.

3. Paustenbach D, Brew D, Ligas S, Heywood J. A critical review of the 2020 EPA risk assessment for chrysotile and its many shortcomings. Crit Rev Toxicol. 2021;51:509-539.

4. Noonan CW. Environmental asbestos exposure and risk of mesothelioma. Ann Transl Med. 2017.5(11):234.

5. Ilgren E, Van Orden DR, Lee RJ, Kamiya YM, Hoskins JA. Further studies of Bolivian crocidolite - Part IV: Fibre width, fibre drift and their relation to mesothelioma Induction: Preliminary Findings. Epidemiology Biostatistics and Public Health. 2015;12:e11167-11161.

6. Casali M, Carugno M, Cattaneo A, Consonni D, Mensi C, et al. Asbestos lung burden in necroscopic samples from the general population of Milan, Italy. Ann Occup Hyg. 2015;59:909-921.

7. Bayram M, Bakan ND. Environmental exposure to asbestos: from geology to mesothelioma. Curr Opin Pulm Med. 2014;20:301-307.

8. Yang H, Testa JR, Carbone M. Mesothelioma epidemiology, carcinogenesis, and pathogenesis. Curr Treat Options Oncol. 2008;9:147-157.

9. Wolff H, Vehmas T, Oksa P, Rantanen J, Vainio H. Asbestos, asbestosis, and cancer, the Helsinki Criteria for Diagnosis and Attribution 2014: Recommendations. Scand J Work Environ Health. 2015;41:5-15.

10. Tran T, Egilman D, Rigler M, Emory T. A critique of Helsinki Criteria for using lung fiber levels to determine causation in mesothelioma cases. Ann Glob Health. 2021;87(1):73.

11. Janosikova M, Nakladalova M, Stepanek L. Current causes of mesothelioma: how has the asbestos ban changed the perspective? Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2023;167(2):99-108.

12. Febres-Aldana CA, Fanaroff R, Offin M, Zauderer MG, Sauter JL, Yang SR, Ladanyi M. Diffuse pleural mesothelioma: advances in molecular pathogenesis, diagnosis, and treatment. Annu Rev Pathol. 2024;19:11-42.

13. Røe OD, Stella GM. Malignant pleural mesothelioma: history, controversy and future of a manmade epidemic. Eur Respir Rev. 2015;24:115-131.

14. Berry TA, Belluso E, Vigliaturo R, Gieré R, Emmett EA, Testa JR, et al. Asbestos and other hazardous fibrous minerals: potential exposure pathways and associated health risks. Int J Environ Res Public Health. 2022;19(7):4031.

15. Carbone M, Adusumilli PS, Alexander HR Jr, Baas P, Bardelli F, et al. Mesothelioma: Scientific clues for prevention, diagnosis, and therapy. CA Cancer J Clin. 2019;69:402-429.

16. Kane AB, Hurt RH, Gao H. The asbestos-carbon nanotube analogy: An update. Toxicol Appl Pharmacol 2018;361:68-80.

17. Numano T, Higuchi H, Alexander DB, Alexander WT, Abdelgied M, El-Gazzar AM, et al. MWCNT-7 administered to the lung by intratracheal instillation induces development of pleural mesothelioma in F344 rats. Cancer Sci. 2019;110(8):2485-2492.

18. Nel A. Carbon nanotube pathogenicity conforms to a unified theory for mesothelioma causation by elongate materials and fibers. Environ Res. 2023;230:114580.

19. Carbone M, Gazdar A, Butel JS. SV40 and human mesothelioma. Transl Lung Cancer Res. 2020;9(Suppl 1):S47-S59.

20. Testa JR, Carbone M, Hirvonen A, Khalili K, Krynska B, Linnainmaa K, et al. A multi-institutional study confirms the presence and expression of simian virus 40 in human malignant mesotheliomas. Cancer Res. 1998;58(20):4505-4509.

21. Garcea RL, Imperiale MJ. Simian virus 40 infection of humans. J Virol. 2003;77:5039-5045.

22. Cicala C, Pompetti F, Carbone M. SV40 induces mesotheliomas in hamsters. Am J Pathol. 1993;142:1524-1533.

23. Likhacheva EI, Iarina AL, Vagina ER, Klimina MS, Obukhova TIu, et al. Clinical features of pulmonary diseases caused by chrysotile asbestos dust. Med Tr Prom Ekol. 2000;(11):30-33.

24. Milishnikova VV, Loshchilov IU, Gladkova EV, Aksenova AO, Turkina LA. Endoscopic and morphological characteristics of the bronchi and lungs in asbestosis and dust-induced bronchitis in asbestostextile industry workers. Gig Tr Prof Zabol. 1990;(7):19-22.

25. Jargin SV. Bronchoscopy with questionable indications. J Med Clin Stud. 2025;8(3):230.

26. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Arsenic, metals, fibres, and dusts. IARC Monogr Eval Carcinog Risks Hum. 2012;100(Pt C):11-465.

27. Carbone M, Yang H. Mesothelioma: recent highlights. Ann Transl Med. 2017;5(11):238.

28. Kerger BD, James RC, Galbraith DA. Tumors that mimic asbestos-related mesothelioma: Time to consider a genetics-based tumor registry? Front Genet. 2014;5:151.

29. Panou V, Vyberg M, Weinreich UM, Meristoudis C, Falkmer UG, Røe OD. The established and future biomarkers of malignant pleural mesothelioma. Cancer Treat Rev. 2015;41:486-495.

30. Chen Z, Gaudino G, Pass HI, Carbone M, Yang H. Diagnostic and prognostic biomarkers for malignant mesothelioma: an update. Transl Lung Cancer Res. 2017;6:259-269.

31. Goldberg M, Imbernon E, Rolland P, Gilg Soit Ilg A, Savès M, et al. The French national mesothelioma surveillance program. Occup Environ Med. 2006;63:390-395.

32. Lorenzini E, Ciarrocchi A, Torricelli F. Molecular fingerprints of malignant pleural mesothelioma: not just a matter of genetic alterations. J Clin Med. 2021;10(11):2470.

33. Sorino C, Mondoni M, Marchetti G, Agati S, Inchingolo R, Mei F, et al. Pleural Mesothelioma: Advances in Blood and Pleural Biomarkers. J Clin Med. 2023;12(22):7006.

34. Lu Z, Zhang W, Huang K, Zhu M, Gu X, Wei D, et al. Systematic review, meta-analysis and bioinformatic analysis of biomarkers for prognosis of malignant pleural mesothelioma. Diagnostics. (Basel). 2022;12(9):2210.

35. Harris EJA, Musk A, de Klerk N, Reid A, Franklin P, Brims FJH. Diagnosis of asbestos-related lung diseases. Expert Rev Respir Med. 2019;13(3):241-249.

36. Gee GV, Koestler DC, Christensen BC, Sugarbaker DJ, Ugolini D, et al. Downregulated microRNAs in the differential diagnosis of malignant pleural mesothelioma. Int J Cancer. 2010;127:2859-2869.

37. Reid G. MicroRNAs in mesothelioma: from tumour suppressors and biomarkers to therapeutic targets. J Thorac Dis. 2015;7:1031-1040.

38. Zahiu T, Mihu CM, Bosca BA, Mărginean M, Mocan LP, Ștefan RA, et al. Molecular insights into pleural mesothelioma: unveiling pathogenic mechanisms and therapeutic opportunities. Diagnostics (Basel). 2025;15(11):1323.

39. Rossi G, Davoli F, Poletti V, Cavazza A, Lococo F. When the diagnosis of mesothelioma challenges textbooks and guidelines. J Clin Med. 2021;10(11):2434.

40. Cersosimo F, Barbarino M, Lonardi S, Vermi W, Giordano A, Bellan C, Giurisato E. Mesothelioma malignancy and the microenvironment: molecular mechanisms. Cancers (Basel). 2021;13(22):5664.

41. Blyth KG, Murphy DJ. Progress and challenges in mesothelioma: From bench to bedside. Respir Med. 2018;134:31-41.

42. Protas SV. Hygienic evaluation of air pollution with asbestos dust in subway parkings. Med Tr Prom Ekol. 2014;(1):23-6.

43. Varivonchik DV. Epidemiologic situation in Ukraine, concerning malignant mesothelioma prevalence. Med Tr Prom Ekol. 2014;(1):18-22.

44. Nagornaia AM, Varivonchik DV, Kundiev IuI, Fedorenko ZP, Gorokh EL, Gulak LO, et al. Cancer morbidity risks among workers of asbestos-cement productions. Med Tr Prom Ekol. 2008;(3):27-33.

45. Pylev DN, Smirnova OV, Vasil'eva LA, Khrustalev SA, Vezentsev AI, et al. Experimental rationale for carcinogenic risk of asbestos cement industry and its products. Gig Sanit. 2010;(6):61-65.

46. Troitskaia NA. A comparative study of cytotoxicity of dust of carbon fibers and other fibrous materials. Gig Sanit. 1993;(3):28-30.

47. Schüz J, Kovalevskiy E, Olsson A, Moissonnier M, Ostroumova E, Ferro G, et al. Cancer mortality in chrysotile miners and millers, Russian Federation: main results (Asbest Chrysotile Cohort-Study). J Natl Cancer Inst. 2024;116(6):866-875.

48. Kogan FM, Kashanskii SV, Plotko EG, Berzin SA, Bogdanov GB. Effect of low concentration of asbestos-containing dust. Med Tr Prom Ekol. 1993;(5-6):6-10.

49. Shtol' AV, Plotko EG, Seliankina KP. Children's health and environmental air pollution with dust containing asbestos. Med Tr Prom Ekol. 2000;(11):10-13.

50. Tsurikova GV, Spitsyn VA, Gladkova EV, Minaeva OP. Biodemographic parameters as indicators of genetic adaptation to harmful occupational factors (e.g. asbestos). Gig Tr Prof Zabol. 1992;(6):28-30.

51. Kaptsov VA, Kashanskii SV, Domnin SG, Tikhova TS, Trofimova EV, Novoselova TA, Bogdanov GB. Railway use of asbestos-containing rubble: environmental hygienic aspects. Gig Sanit. 2003;(5):11-15.

52. Kashanskii SV, Domnin SG, Plotko EG, Kuz'min SV, Seliankina SV, Likhacheva EI. Contemporary problems of asbestos and prospective research directions. Med Tr Prom Ekol. 2004;(9):16-19.

53. Kovalevskii EV. Hygienic evaluation of asbestos-containing friction goods application. Med Tr Prom Ekol. 2009;(7):1-6.

54. Izmerov NF, Elovskaia LT, Milishnikova VV, Burmistrova TB, Kovalevskii EV. Chrysotile asbestos in Russia: certain results and promising research directions. Med Tr Prom Ekol. 1998;(10):1-7.

55. Kashanskii SV, Zhetpisbaev BA, Il'derbaev OZ, Ermenbai OT. Mesothelioma in the Republic of Kazakhstan: a review. Gig Sanit. 2008;(5):13-7.

56. Pylev LN. The role of modifying factors in the carcinogenic effect of asbestos and asbestos-containing dusts. Eksp Onkol. 1987;9(5):14-17.

57. Bernstein DM. The health risk of chrysotile asbestos. Curr Opin Pulm Med. 2014;20:366-370.

58. Coin PG, Roggli VL, Brody AR. Persistence of long, thin chrysotile asbestos fibers in the lungs of rats. Environ Health Perspective. 1994;102:197-199.

59. Kohyama N, Suzuki Y. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. Ann N Y Acad Sci. 1991;643:27-52.

60. Nicholson WJ. Comparative dose-response relationships of asbestos fiber types: magnitudes and uncertainties. Ann N Y Acad Sci. 1991;643:74-84.

61. Stayner LT, Dankovic DA, Lemen RA. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. Am J Public Health. 1996;86:179-186.

62. Suzuki Y, Yuen SR. Asbestos fibers contributing to the induction of human malignant mesothelioma. Ann N Y Acad Sci. 2002;982:160-176.

63. Bernstein D, Dunnigan J, Hesterberg T, Brown R, Velasco JA, et al. Health risk of chrysotile revisited. Crit Rev Toxicol. 2013;43:154-183.

64. Pezerat H. Chrysotile biopersistence: the misuse of biased studies. Int J Occup Environ Health. 2009;15:102-106.

65. Larsen G. Experimental data on in vitro fibre solubility. IARC Sci Publ. 1989;(90):134-139.

66. Asgharian B, Owen TP, Kuempel ED, Jarabek AM. Dosimetry of inhaled elongate mineral particles in the respiratory tract: The impact of shape factor. Toxicol Appl Pharmacol. 2018;361:27-35.

67. Currie GP, Watt SJ, Maskell NA. An overview of how asbestos exposure affects the lung. BMJ. 2009;339:b3209.

68. Smith AH, Wright CC. Chrysotile asbestos is the main cause of pleural mesothelioma. Am J Ind Med. 1996;30:252-266.

69. Ramada Rodilla JM, Calvo Cerrada B, Serra Pujadas C, Delclos GL, Benavides FG. Fiber burden and asbestos-related diseases: an umbrella review. Gaceta Sanitaria. 2022;36(2):173-183.

70. Finkelstein MM. Letter to the Editor re Bernstein et al: Health risk of chrysotile revisited. Crit Rev Toxicol 2013;43(2):154-183. Crit Rev Toxicol. 2013;43(8):707-708.

71. Yu CP, Asgharian B, Pinkerton KE. Intrapulmonary deposition and retention modeling of chrysotile asbestos fibers in rats. Journal of Aerosol Science. 1991;22:757-763.

72. Dodson RF, Williams MG Jr, Corn CJ, Brollo A, Bianchi C. Asbestos content of lung tissue, lymph nodes, and pleural plaques from former shipyard workers. Am Rev Respir Dis. 1990;142:843-847.

73. Gibbs AR, Stephens M, Griffiths DM, Blight BJ, Pooley FD. Fibre distribution in the lungs and pleura of subjects with asbestos related diffuse pleural fibrosis. Br J Ind Med. 1991;48:762-770.

74. Sekido Y. Molecular pathogenesis of malignant mesothelioma. Carcinogenesis. 2013;34:1413-1419.

75. Finkelstein MM, Meisenkothen C. Malignant mesothelioma among employees of a Connecticut factory that manufactured friction materials using chrysotile asbestos. Ann Occup Hyg. 2010;54(6):692-696.

76. Frank AL. Global use of asbestos - legitimate and illegitimate issues. J Occup Med Toxicol. 2020;15:16.

77. Stayner LT, Dankovic DA, Lemen RA. Asbestos-related cancer and the amphibole hypothesis: II. Stayner and colleagues respond. Am J Publ Health. 1997;87:688.

78. Stayner LT. Canada, chrysotile and cancer: Health Canada's Asbestos International Expert Panel report. J Occup Environ Med. 2008;50(12):1327-1328.

79. Harington JS. The carcinogenicity of chrysotile asbestos. Ann N Y Acad Sci. 1991;643:465-472.

80. Wagner JC. Proceedings: Asbestos carcinogenesis. Br J Cancer. 1975;32:258-259.

81. Wagner JC, Berry G, Skidmore JW, Timbrell V. The effects of the inhalation of asbestos in rats. Br J Cancer. 1974;29:252-269.

82. Berman DW, Crump KS, Chatfield EJ, Davis JM, Jones AD. The sizes, shapes, and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. Risk Anal. 1995;15:181-195.

83. Landrigan PJ, Nicholson WJ, Suzuki Y, Ladou J. The hazards of chrysotile asbestos: a critical review. Ind Health. 1999;37:271-280.

84. Davis JM, Beckett ST, Bolton RE, Collings P, Middleton AP. Mass and number of fibres in the pathogenesis of asbestos-related lung disease in rats. Br J Cancer. 1978;37:673-688.

85. Hesterberg TW, Barrett JC. Dependence of asbestos- and mineral dust-induced transformation of mammalian cells in culture on fiber dimension. Cancer Res. 1984;44:2170-2180.

86. Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occup Hyg. 2000;44:565-601.

87. Lenters V, Vermeulen R, Dogger S, Stayner L, Portengen L, et al. A meta-analysis of asbestos and lung cancer: is better quality exposure assessment associated with steeper slopes of the exposure-response relationships? Environ Health Perspect. 2011;119:1547-1555.

88. Hodgson JT, Darnton A. Mesothelioma risk from chrysotile. Occup Environ Med. 2010;67:432.

89. Dufresne A, Bégin R, Massé S, Dufresne CM, Loosereewanich P, et al. Retention of asbestos fibres in lungs of workers with asbestosis, asbestosis and lung cancer, and mesothelioma in Asbestos township. Occup Environ Med. 1996;53:801-807.

90. Rogers AJ, Leigh J, Berry G, Ferguson DA, Mulder HB, et al. Relationship between lung asbestos fiber type and concentration and relative risk of mesothelioma. A case-control study. Cancer. 1991;67:1912-1920.

91. Bernstein D, Dunnigan J, Hesterberg T, Brown R, Legaspi Velasco JA, et al. Response to Murray M. Finkelstein, letter to the editor re Bernstein et al: Health risk of chrysotile revisited. Crit Rev Toxicol, 2013;43(2):154-183. Crit Rev Toxicol. 2013;43(8):709-10.

92. Kanarek MS. Mesothelioma from chrysotile asbestos: update. Ann Epidemiol. 2011;21:688-97.

93. IARC. Consensus report. Mechanisms of fibre carcinogenesis. IARC Sci Publ. 1996;(140):1-9.

94. Mossman BT, Lippmann M, Hesterberg TW, Kelsey KT, Barchowsky A, Bonner JC. Pulmonary endpoints (lung carcinomas and asbestosis) following inhalation exposure to asbestos. J Toxicol Environ Health B Crit Rev. 2011;14(1-4):76-121.

95. Wang J, Schlagenhauf L, Setyan A. Transformation of the released asbestos, carbon fibers and carbon nanotubes from composite materials and the changes of their potential health impacts. J Nanobiotechnology. 2017;15(1):15.

96. Berman DW, Crump KS. A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. Crit Rev Toxicol. 2008;38 Suppl 1:49-73.

97. Gaudino G, Xue J, Yang H. How asbestos and other fibers cause mesothelioma. Transl Lung Cancer Res. 2020;9(Suppl 1):S39-46.

98. Hillerdal G, Henderson DW. Asbestos, asbestosis, pleural plaques and lung cancer. Scand J Work Environ Health. 1997;23(2):93-103.

99. Suzuki Y, Yuen SR, Ashley R. Short, thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence. Int J Hyg Environ Health. 2005;208(3):201-210.

100. Wong JYY, Rice C, Blair A, Silverman DT. Mesothelioma risk among those exposed to chrysotile asbestos only and mixtures that include amphibole: a case-control study in the USA, 1975-1980. Occup Environ Med. 2020; oemed-2020-106665.

101. Marsili D, Terracini B, Santana VS, Ramos-Bonilla JP, Pasetto R, Mazzeo A, et al. Prevention of asbestos-related disease in countries currently using asbestos. Int J Environ Res Public Health. 2016;13(5):494.

102. Kwak K, Kang D, Paek D. Environmental exposure to asbestos and the risk of lung cancer: a systematic review and meta-analysis. Occup Environ Med. 2022;79(3):207-214.

103. Carson M. From common market to social Europe? Acta Universitatis Stockholmiensis. Stockholm Studies in Sociology N.S. 2004; 22.

104. Tossavainen A, Kotilainen M, Takahashi K, Pan G, Vanhala E. Amphibole fibres in Chinese chrysotile asbestos. Ann Occup Hyg. 2001;45:145-152.

105. Bergamaschi E, Garzaro G, Wilson Jones G, Buglisi M, Caniglia M, Godono A, et al. Occupational exposure to carbon nanotubes and carbon nanofibres: more than a cobweb. Nanomaterials (Basel). 2021;11(3):745.

106. Gupta SS, Singh KP, Gupta S, Dusinska M, Rahman Q. Do carbon nanotubes and asbestos fibers exhibit common toxicity mechanisms? Nanomaterials (Basel). 2022;12(10):1708.

107. Bernstein DM, Toth B, Rogers RA, Kling DE, Kunzendorf P, et al. Evaluation of the dose-response and fate in the lung and pleura of chrysotile-containing brake dust compared to TiO2,

chrysotile, crocidolite or amosite asbestos in a 90-day quantitative Inhal Toxicol study - Interim results Part 2: Histopathological examination, Confocal microscopy and collagen quantification of the lung and pleural cavity. Toxicol Appl Pharmacol. 2020;387:114847.

108. Thives LP, Ghisi E, Thives Júnior JJ, Vieira AS. Is asbestos still a problem in the world? A current review. J Environ Manage. 2022;319:115716.

109. Shanin NP, Borodulin MM, Kolbovsky YuYa, Krasovsky VN. Proizvodstvo asbestotekhnicheskikh izdeliy [Production of asbestos products]. Leningrad: Chemistry; 1983.

110. Vedernikov NN, Polyanin VS, Romanovich IF, Lisitsyn AE. Asbest. Moscow: Geoinformmark; 1999.

111. Jargin SV. Russian opinion on asbestos: All fibers equal. Environment and Ecology Research. 2013;1(2):79-83.

112. Culley MR, Zorland J, Freire K. Community responses to naturally occurring asbestos: implications for public health practice. Health Educ Res. 2010;25(5):877-891.