Misleading "New Insights into the Chrysotile Debate"

Irreführende "neue Einblicke in die Chrysotil-Debatte"

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ABSTRACT

Although there is no dispute among independent scientists about the carcinogenic and fibrogenic effects of chrysotile, the asbestos industry has been continuously and successfully acting to cast doubts on its harm. Another approach including asbestos insurance entities is to refuse compensation by raising the bar and fight criminal prosecution for asbestos-related diseases by the help of paid scientists. A recent publication on asbestos fibre burden in human lungs fits well in this context. The claim that chrysotile fibres are biopersistent in human lung is not based on the data provided by these authors, and, additionally, exhibits serious inconsistencies and obvious mismeasurements and significant methodological problems. The conclusion of the authors that fibre analysis of workers' lungs "is of high significance for differential diagnosis, risk assessment and occupational compensation" is unfounded and reprehensible. Also the available literature, the statements of the WHO, IARC, other decisive independent international organizations, and all our experience provide abundant evidence to the contrary. Note, the method is generally restricted to research only and is not recognized for diagnostic purpose and compensation in any other country. In conclusion, fibre counting in lung tissues should not be used to estimate former exposure to chrysotile comprising c. 94% of applied asbestos in Germany. The authors claim that the analyses can improve the compensation rates in Germany. However, the opposite has been the case; it significantly worsens the non-justified denial of well-substantiated compensation claims.

ZUSAMMENFASSUNG

Obwohl in der unabhängigen Wissenschaft Konsens über die karzinogene und fibrogene Wirkung von Weißasbest (Chrysotil) besteht, verbreitet die Asbestindustrie bis heute erfolgreich Zweifel an dessen gesundheitsschädigenden Wirkungen. Auch werden vonseiten der Arbeitgeber-Unfallversicherungen (Berufsgenossenschaften) nicht selten Entschädigungen von asbestbedingten Erkrankungen durch hohe Hürden im Anerkennungsverfahren verweigert. Dazu tragen auch gerichtliche Auseinandersetzungen mit Unterstützung bezahlter Wissenschaftler bei. In diesem Zusammenhang ist eine kürzlich erschienene Veröffentlichung zu sehen, die sich auf die Asbestfaserzählung in der menschlichen Lunge bezieht und ohne eine wissenschaftliche Basis vorgibt, dass Chrysotilfasern in der menschlichen Lunge biopersistent seien und deren Zahl von großer Bedeutung in der Begutachtung sei. Die hierfür angeführten Daten sind inkonsistent, basieren auf offensichtlichen Fehlmessungen oder methodischen Problemen. Sie widersprechen dem wissenschaftlichen Kenntnisstand, Statements der WHO, der IARC und anderer maßgebender unabhängiger internationaler Organisationen ebenso wie unserer gesamten diesbezüglichen Erfahrung. Derartige Faserzählungen sind allgemein beschränkt auf wissenschaftliche Fragestellungen; sie werden in keinem anderen Land für die Diagnostik und Begutachtung eingesetzt. Die Schlussfolgerung der Autoren, dass die Faseranalyse in der Asbestarbeiterlunge von großer Bedeutung für die Differenzialdiagnose, Risikoeinschätzung und Entschädigung sei, entbehrt jeder Grundlage und ist abzulehnen. Die Faseranalytik sollte infolge der nicht gegebenen Biopersistenz nicht zur Abschätzung einer früheren Exposition gegenüber Chrysotil, der mit ca. 94% hierzulande ganz im Vordergrund gestandenen Asbestart, verwendet werden. Denn solche Faseranalysen tragen in erheblichem Maße zur nicht gerechtfertigten Ablehnung von Entschädigungsansprüchen in gut begründeten Berufskrankheitsfällen bei.

INFO BOX

Asbestos is a naturally occurring mineral categorized into 2 main groups based on fibre structure: serpentine asbestos (chrysotile) and amphibole asbestos (crocidolite, amosite, anthophyllite, tremolite, actinolite). Chrysotile, which was the far dominating asbestos type in Germany till the asbestos ban in 1993, has been used in more than 2.000 applications, especially in the construction industry. Chrysotile causes all the same fibrogenic and carcinogenic effects as amphiboles. Its short fibre fractions have a drift towards the pleura. Furthermore, chrysotile fibres split into microfebrils. As a consequence chrysotile fibres have a short half-life in the lung. Their low numbers or even absence in human lung tissue after a latency period of many years does not allow any conclusion on previous exposure.

Background

Chrysotile, the only type of asbestos still mined and used at a volume of about 2 million tons annually, has been promoted vigorously in recent decades, despite asbestos bans in 55 countries. Offsetting the lost business in countries banning their product, the asbestos industry has increased asbestos export to some developing countries, such as India. Asbestos use has remained high and even increased in the 2 largest asbestosproducing countries, China and Russia (www.minerals.usgs. gov/minerals/pubs/commodity/asbestos/ [see 2018 data, access 13.2.2018]). The underlying promotion strategy relies on creating product defense science and casting doubt on the clear evidence that chrysotile asbestos causes harm to health. An example is the claim that animal studies showing relatively short biopersistence of chrysotile prevents chrysotile from causing malignancies. Another is the wide distribution by members of the asbestos industry and their customers in the construction industry of unfounded and misleading conclusions, e.g. that chrysotile can be used safely. The chrysotile asbestos industry has hired scientists for decades creating the propaganda that chrysotile asbestos is safer than amphibole asbestos types. Egilman et al. [1], by evaluating the published and unpublished studies funded by the Quebec Asbestos Mining Association including those from researchers at McGill University, identified that data were manipulated and unsound sampling and analysis techniques were used to back up the contention that chrysotile was "essentially innocuous". Affiliated researchers put forth several myths to suggest that chrysotile was harmless, and contended that the contamination of chrysotile with oils, tremolite, or crocidolite was the source of occupational health risk. Even today several asbestos industry affiliated scientists minimise or even deny cancerogenic potency of chrysotile fibres, especially its potency to cause mesothelioma, for example in mechanics servicing cars [2,3]. These publications were used to promote the marketing and sale of asbestos, and have had a substantial inhibiting effect on occupational health protection and compensation. Asbestos manufacturing companies, and in former times also the Canadian government, have continued to use them for ongoing marketing of chrysotile asbestos. Today, the International Chrysotile Association (ICA) and its predecessor the Asbestos International Association (AIA), now dominated by the Russian chrysotile industry, is promoting chrysotile especially in developing countries.

Egilman et al. [4,5] particularly addressed the issue of how the asbestos industry and asbestos insurance entities (Metropolitan Life Insurance Company; MetLife) have influenced not only science but also public policy and law through writing and implementing worker compensation laws in numerous states and concocting an arbitrary "protective" standard to monitor asbestos exposure. As stated by Egilman et al. "MetLife established itself as an authority in public and industrial health in the early part of the twentieth century, gaining the trust of the public and government. They were able to use this trust and authority to avoid financial loss, including the firing of sick workers, and avoid legal liability by organizing a network of experts to testify on their behalf in silica- and asbestos-related damage suits. They further manipulated the results of scientific findings from major research institutions, delaying important knowledge about the asbestos-cancer relationship" [4].

Another broad approach by the asbestos industry and asbestos insurance entities is to refuse compensation and fight criminal prosecution for asbestos-related diseases (ARD). An example is mesothelioma in Italy, where it is falsely stated that such disorders are exclusively due to initial, but not to subsequent asbestos exposure [6-8].

The recent publication by Feder et al. on asbestos fibre burden in human lungs [9] fits very well in the studies casting doubt about the well-documented adverse health effects of chrysotile. Their non-substantiated claim that chrysotile fibres are persistent in human lung is contradicted by the collective experience of the Institute for Occupational and Social Medicine at the Justus Liebig-University of Giessen. Here, we have analyzed lung specimens of more than 350 patients with and 150 without occupational asbestos exposure by analytical scanning and transmission electron microscopy (ARTEM) in combination with detailed clinical and occupational histories. In contrast to our experience, Feder et al. reported SEM or TEM fibre analysis based on only 6 patients at autopsy. Their publication shows several severe shortcomings. It is not clear from which analytical method fibre data presented in the supplementary material employed. The same is true for asbestosis grading, since the authors mention that they followed different heterogeneous definitions.

In the additional Table 1 of their supplementary material Feder et al. show concentrations of asbestos bodies and free fibres in lung specimens of 12 subjects. Here, the method of fibre analysis is not reported. There is an unexplained huge variation of up to a factor of 5 between samples of the same patient. As opposed to the known ratio of asbestos bodies versus free fibres of c. 1:1000 [10] the authors present nearly always much higher numbers of asbestos bodies compared to free fibres (for example 500:0, and 1900:0, respectively, in samples 1 and 2 of patient 1 at surgery). Another puzzle is the

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mostly ca. 3 fold increase in asbestos bodies and fibres from time of surgery till time of autopsy as shown in their additional Table 2 and partially also in additional Table 1. The authors unsubstantially speculate that this may be due to higher concentrations in probes taken at autopsy compared to surgery; from the literature there is no evidence supporting such a speculation. Uniformly, experienced research groups described a halflife of amphiboles of a few years with an extreme of 20 years [11-15] whereas half-life of chrysotile was found to be in the range of several months [15-17] (see also below). These inconsistencies with facts well-documented in the literature are not even discussed by the authors; their data are obviously due to mismeasurements or significant methodological problems.

Feder et al.'s conclusion about the biopersistence of chrysotile lacks credibility, given the small number of patients examined of which 2 show rather low chrysotile proportions (10-35%) indicating its low biopersistence. This conclusion is not even congruent with their own website where they have stated "usually, chrysotile is cleared from the lungs very rapidly". It also contradicts the authors most recent publication [18] where their figures show a strong decline of asbestos bodies in the human lung after a latency period of 25 years and only a few remaining asbestos bodies after latency periods of more than 4 decades.

Previous literature

There is no dispute among independent scientists about the cancerogenic and fibrogenic effects of chrysotile. Chrysotile like amphibole asbestos causes mesothelioma, cancer of the lung, larynx, and ovary, asbestosis, pleural fibrosis [19–32].

Mineralogist Heidermanns et al. analyzed asbestos fibres in lung tissue from 17 asbestosis cases using infrared spectrography and x-ray structure analysis (detection limit of chrysotile fibres was 2%). They concluded, "In no case could we identify chrysotile fibres" [33]. Kern et al. [34] investigating 2 cases of malignant mesothelioma, in which occupational history indicated only chrysotile exposure, found that lung fibre burden analysis revealed the presence of amosite but not chrysotile.

Velasco-Garcia et al. [35] recently analysed lung tissues of 20 Spanish ex-shipyard workers by means of scanning electron microscopy (SEM) and energy-dispersive x-ray spectroscopy (EDX); no chrysotile fibres were found although chrysotile was the predominant asbestos type used.

By means of ARTEM we demonstrated in our detailed investigations of lung tissues from 10 subjects after operation because of suspected asbestos-induced lung cancer that, on average, only 2% of asbestos bodies comprise chrysotile fibres. If chrysotile was biopersistent, about 95% would be expected [36]; (**> Fig. 1**).

In another study of 47 patients with malignant mesothelioma ARTEM analysis of lung specimen exhibited ca. 10 times higher amphibole fibre concentrations compared to chrysotile fibres; this was true for the dominating short fibres with a length of less than $5 \mu m$ as well as for those longer (**> Table 1**).



▶ Fig. 1 ARTEM analyses of asbestos bodies in lung tissue. In contrast to c. 94% chrysotile consumption in the workplaces in the Federal Republic of Germany, the AB central fibres (cores) exhibited in only 2% chrysotile, but in 98% amphiboles. Data are from 134 investigated asbestos bodies from lung specimen of 10 asbestos-exposed workers with 0 to 2 fibres per specimen (total number of fibres ranged from 15 to 50 per worker), and a chrysotile/amphibole ratio from 0 to 8%, mean = 2.2%). For details see [36] and pp.66ff of [68].

	<5µm		>5µm	
	geom. mean	geom. SD	geom. mean	geom. SD
Chrysotile	0.26	4.3	0.03	2.7
Amphiboles	2.28	11.3	0.34	7.1

Table 1 ARTEM analysis of lung specimen of 47 patients suffering from malignant mesothelioma. Geometric means and geometric

standard deviations are given in 106 fibres/g dry lung tissue [37].

Our experience is consistent with the above findings as well as with those of leading lung pathologists and fibre experts [38-50]. Even in highly exposed workers in chrysotile asbestos mines, the percentage of chrysotile fibres identified by means of energy dispersive X-ray spectroscopy (EDX) and selected area electron diffraction (SAED) was mostly in the range of 3 – 30%.

Similar findings were reported in animal studies where pulmonary accumulation of crocidolite was 3 to 4 times greater than that of chrysotile [51].

The Helsinki expert consensus report [52] concluded: "Chrysotile fibres do not accumulate within lung tissue to the same extent as amphiboles because of faster clearance rates; therefore, occupational histories (fibre-years of exposure) are probably a better indicator of lung cancer risk from chrysotile than fibre burden analysis is."



Fig.2 Concentration of chrysotile fibres in human lung tissue by year of examination and interim time since asbestos exposure [58]. Lung specimen of 257 asbestos-exposed patients were analyzed by ARTEM. 28 of them suffered from asbestosis, 105 from lung cancer, 44 from malignant mesothelioma, 35 did not have a lung disorder; in 46 cases the cause of lung disorder remained unknown. Mean chrysotile fibre concentrations were 50,000/g lung tissue (mean) at low and 190,000/g lung tissue at high asbestos exposure levels. Low exposure: < 20 fibre-years, high exposure: >20 fibre-years. Note that highest concentrations were found in the 1980s with an interim period of less than one year, whereas concentrations decreased later, after an interim time of circa 30 years and generally after the year 2000 fibres could mostly not be detected any more. Modified from [58], courtesy of Springer Medizin Verlag GmbH, in: Schneider J, Arhelger R, Brueckel B. Lungenstaubanalysen in der Begutachtung asbestverursachter Erkrankungen. Zbl Arbeitsmed 2015; 65: 305-309.

Selected and misinterpreted literature

Feder et al. do not present and discuss the literature appropriately. Their literature review is very limited and, indeed, biased. All publications by independent researchers overwhelmingly exhibiting low biopersistence of chrysotile are ignored, including those noted above. Feder et al. cite a discredited article by Bernstein et al. funded by the asbestos lobby [53]. David Bernstein, after working for the tobacco industry and Union Carbide, a company facing asbestos litigation, now works as a consultant to the asbestos industry. When testifying in court on behalf of asbestos interests, Bernstein acknowledged that no scientific body in the world supports his conclusions regarding the supposed harmlessness of chrysotile asbestos [54]. Feder et al. completely misinterpret LaDou et al. [55] and Qwinn et al. [56] who never had "an ongoing debate about the hazardous nature of chrysotile".

The fact that Feder et al. only studied subjects with the arbitrary level of at least 500 fibres per gram wet lung begs the question of what might have been observed had they included subjects with lower concentrations of asbestos fibres? Also, what would be the relationship with fibre-years if evaluated by a detailed occupational history (the recommended standard of practice of the Medical Advisory Board of the German Ministry of Labour and Social Affairs)? Of note, although one of the authors is a pneumologist, detailed occupational histories are completely missing in their publications.

Feder et al. claim that the reduction of chrysotile fibres in human lungs is found in many studies and occurs within the broad period of 3–29 years as experienced by their 6 cases. They argue that this "is best explained by the natural defense mechanism of the lung, for example mucociliary clearance in the bronchi and expectoration, and possible acid hydrolysis by lysosomes" [9]. In fact, the peripheral airways and alveoli do not have ciliated cells and the assumption that fibre reduction occurred solely before the given interim time periods from 3 years to almost 3 decades is not credible. At best, they are being highly speculative in reference to the cited work of Churg and DePaoli [57].

Schneider et al. recently updated ARTEM fibre analysis in the lung tissue of 257 cases of asbestos-exposed patients with suspected asbestos-related diseases. 28 of them suffered from asbestosis, 105 from lung cancer, 44 from malignant mesothelioma, 35 did not have a lung disorder; in 46 cases the cause of lung disorder remained unknown. Mean chrysotile fibre concentrations were 50,000/g lung tissue (mean) at low and 190,000/g lung tissue at high asbestos exposure level. Highest chrysotile concentrations were found in the 1980s with an interim period of less than 1 year whereas the concentrations decreased later. After an interim time of circa 30 years and generally after the year 2000, fibres could mostly not be detected anymore. A highly significant decline of measured chrysotile fibre counts was not only observed over the last 3 decades, it was also associated with the interim period; interim periods of more than 30 years were associated in more than 50% without detectable chrysotile fibre levels, in 90%, the level was below 0.2×10⁶ fibres/gram tissue [58, 59] (▶ Fig. 2).

What about undeclared authors' Conflicts of Interest (COI)?

The COI declarations of the authors do not disclose their important financial COIs.¹

¹ In fact, one of the senior authors is directly working in the IPA institute which is owned by the accident insurance system for the raw/crude material industry and chemical industry (BGRCI). Further, it is not mentioned that the Institute for Pathology at the Ruhr-Universität with its German Mesotheliomregister, where the other authors work, has been owned and sponsored since its beginning in the 1970s by the accident insurance agency for the mining industry/BGRCI till 2013 and is nowadays the foundation under the name: "Georgius Agricola Stiftung Ruhr" with a board dominated by accident insurance interests. They still financially support this foundation and are responsible for acceptance and compensation of all types of occupational-related diseases. There is also evidence that at least one of the authors, the head of this Pathology Institute, has a highly significant direct financial conflict of interests since she has been paid for the Institute's analysis of asbestos fibres and its histological examination of lung tissues. As mentioned, accident insurance institutions routinely mandate exclusively this author to do such analysis of lung issues in compensation cases (c. 1000 cases per year) and to perform decision-making expert opinions (Expert opinions: c. 2000 annually) [60-65].

Table 2 Figures from 2013 – 2016 on reported, acknowledged, newly compensated asbestos workers in Germany. Also figures on deaths of acknowledged subjects are given. Out of the cases overwhelmingly reported by physicians percentage of acknowledged cases from those reported is in the range of 71% for mesothelioma, of 55% for asbestosis/pleural fibrosis or plaques, but only of 20% for lung cancer (including a few cases of larynx cancer), respectively. Asbestos-induced occupational diseases (OD) in Germany in 2016, 2015, 2014, 2013 from

https://www.baua.de/DE/Angebote/Publikationen/Berichte/Suga-2015.html; https://www.baua.de/DE/Angebote/Publikationen/Berichte/Suga-2016.html; http://www.dguv.de/de/zahlen-fakten/bk-geschehen/index.jsp.

Occupational disease no.	Reported OD	Acknowledged OD	Newly compensated OD	Deaths	Year
4103 Asbestosis, Pleural fibrosis/plaques	3654 3712 3602 3636	2189 2002 1997 1926	580 541 603 582	168 165 153 159	2016 2015 2014 2013
4104 Lung or larynx cancer	4478 4482 4343 4079	915 773 834 794	817 715 766 711	622 593 595 559	2016 2015 2014 2013
4105 Mesothelioma	1336 1417 1380 1425	1040 958 1048 978	952 881 976 904	872 812 817 734	2016 2015 2014 2013
4114 Lung cancer due to asbestos + PAH	126 138 132 142	22 33 23 24	19 28 20 24	15 11 18 17	2016 2015 2014 2013
Total (2016)	9594	4166	2366	1677	

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Conclusion

In conclusion, the article by Feder et al. is irreparably flawed. It puts forth dangerous disinformation that the asbestos industry has used for decades to manufacture doubt about the scientific evidence relating to the various asbestos fibre types. The authors continue to foment uncertainty by falsely claiming that there is a bona fide scientific debate still going on among reputable scientists as to whether or not chrysotile asbestos is harmful, without mentioning that the articles that deny harm caused by chrysotile asbestos are funded by the chrysotile asbestos industry or insurance-affiliated scientists. The patently wrong statements in their article and the expert opinions of the authors that chrysotile shows biopersistence in human lungs has been applied to more than 10,000 cases in Germany to unjustly dismiss workers' compensation claims [60-65]. This is especially relevant for asbestos-caused lung cancer where, of the annual 4,000 claims for compensation, only about 800 (c. 20%) have been accepted in recent years. But also many asbestosis and some mesothelioma cases have not been accepted as occupational diseases due to misleading interpretation of low fibre counts in lung tissue (> Table 2). Official bodies should cease using unrealistic chrysotile biopersistence criteria in depriving workers suffering from asbestos-related diseases of their deserved compensation. In accordance with the Collegium Ramazzini [66], Begin and Christman [67] and Irving Selikoff (personal communication: "Patients should be compensated if there is documented history of occupational exposure to asbestos"), we argue not to exclude causation by asbestos in lung and pleural disorders, mesothelioma in other organs as well as

in larynx or ovary cancer, before a detailed occupational history has been taken for relevant asbestos exposure.

In summary, the authors provide findings which do not allow any conclusion. Interestingly, from time to time they state the opposite to their "new" message on their website ("usually, chrysotile is cleaved from the lung very rapidly"). Further, the authors' confusing presentation of selected literature cites asbestos industry-funded work which claims that chrysotile is quickly expelled from the lungs and therefore is harmless. At the same time, they reject the argument that chrysotile asbestos fibres are quickly expelled from the lungs and claim that chrysotile asbestos fibres in the lungs provide necessary evidence of harm causation. Our concern relates to their unsubstantiated statements about "new insights into the chrysotile debate", and especially that "there is no significant reduction of asbestos fibre concentrations in lung tissues over time after exposure cessation". These unsubstantiated claims are neither consistent with their own findings nor with the literature which shows that chrysotile in contrast to amphiboles is rapidly cleared from the lungs.

Conflict of interest

During recent 3 years, H-JW and XB have testified in ligitation, XB also on behalf of statutory insurance institutions.

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