International Chrysotile Association

THE TRUTH BEHIND NUMBERS – AN ICA ANALYSIS OF A UNEP DOCUMENT - IMPACTS OF ASBESTOS FIBERS ON HUMAN HEALTH

Highlights

- **A document published by the UNEP in February 2024[1], with the collaboration of the WHO and the ILO, estimated that in 2016 asbestos caused 209,481 deaths, which stands for more than 70 per cent of all deaths from work related cancers.**
- **The document's estimate was based on mixed exposures which occurred in the last century when amphiboles were often used – not today when only chrysotile is used.**
- **This estimate does not provide any indication of the risk associated with the current exclusive use of chrysotile.**
- **While the UNEP document takes potency differences between chrysotile and amphibole asbestos into account, especially for mesothelioma, the final risk assessment uses combined estimates across all asbestos types due, as was claimed, to the mixed nature of reported exposures over several decades. Separate risk estimates for chrysotile vs amphiboles fibers were not presented in the final exposure-risk relationship table.**
- **There is strong evidence today that chrysotile does not cause mesothelioma.**
    - **Data presented in a recent epidemiology study of the largest and oldest chrysotile mine has shown that chrysotile does not cause lung cancer.**
    - **As presented (in the supplementary data) in this epidemiology study on workers from this chrysotile mine in Russia, no statistically significant association with lung cancer in men based on chrysotile fibers/cm3-years was observed even with earlier high exposure levels.**
- **The UNEP calls for studies on alternative to chrysotile which we fully support. These studies should be conducted on an equivalent fiber exposure basis.**

Context

Early in 2024, a paper titled "Options for addressing asbestos contaminants in products and the environment" was distributed as an information document to the participants to the 6th session of the United Nations Environment Programme (UNEP)'s UN Environment Assembly[1], which was held in Nairobi, Kernya, between February 26th and March 1st 2024. In it, the authors stated that

[1] UNEP in cooperation with the World Health Organization (WHO) and with input from the International Labor Organization (ILO)) prepared the paper following a request from the participants to the 5th Session of the UN Environment Assembly of the UNEP.

in 2016, asbestos caused an estimated 209,481 deaths, which stand for more than 70 percent of all deaths from work-related cancers.

The ICA's analysis of the information concerning asbestos in the UNEP document does not provide any indication on the risk of using chrysotile only today. For chrysotile, the UNEP's estimate has no validity based on current scientific publications and data. In fact, the current use of chrysotile alone has little, if any, contribution to workplace mortality today.

On the other hand, the UNEP document does address the important issue of assessing alternatives to chrysotile fibers, as ICA has long been advocating. Those issues are summarized below.

Key issues

In the opening page of the UNEP document, the authors state that "Globally, in 2016, occupational exposure to asbestos caused an estimated 209,481 deaths, which stands for more than 70 percent of all deaths from work-related cancers." A review of the references cited by its authors[2] provides no clear explanation of how this impressive number was determined other than that it represents a cumulative sum of mesothelioma, trachea, bronchus, lung, ovary, and larynx cancers.

This number raises questions. The WHO itself uses a slightly different number: the WHO Global Health Estimates, stemming from the 2016 Global Burden of Disease (GBD) study[3], state that there were 218,827 asbestos attributed cancer deaths. It must be noted that in the same way, the ICA's review revealed that the number of deaths from mesothelioma worldwide in 2016, which has been historically associated with amphibole asbestos exposure, was reported by WHO to be 23,104, while in the GBD study estimates this number was 27,612.

[2] Mandrioli et al. 2018 WHO/ILO work-related burden of disease and injury: Protocol for systematic reviews of occupational exposure to dusts and/or fibres and of the effect of occupational exposure to dusts and/or fibres on pneumoconiosis. Environ Int. 2018 Oct;119:174-185. doi: 10.1016
- European Commission 2022. Commission staff working document impact assessment. Proposal for a Directive of the European Parliament and of the Council amending Directive 2009/148/EC on the protection of workers from the risks related to exposure to asbestos art work. https://eur-lex.europa.eu/legalcontent/
EN/TXT/PDF/?uri=CONSIL:ST_12863_2022_ADD_2&qid=1673446822849&from=EN. Accessed October 2023
- Schlünssen, et al. 2023. The prevalences and levels of occupational exposure to dusts and/or fibres (silica, asbestos and coal): A systematic review and meta-analysis from the WHO/ILO Joint Estimates of the Work-related Burden of Disease and Injury. Environment International 107980.

[3]See table 1 of GBD 2016 Occupational Carcinogens Collaborators. Global and regional burden of cancer in 2016 arising from occupational exposure to selected carcinogens: a systematic analysis for the Global Burden of Disease Study 2016. Occup Environ Med. 2020 Mar;77(3):151-159. doi: 10.1136/oemed-2019-106012. PMID: 32054819; PMCID: PMC7035689.

It also seems that the UNEP document presupposes that current asbestos use, strictly limited to chrysotile fibers, is similar to the situation that prevailed in the middle of the XXth century when extensive amounts of amphibole asbestos (amosite and crocidolite) were also used.

The use of amphibole asbestos in the last century may still result in mesotheliomas today, but those diseases are not a result of the current use of chrysotile. To cite but one example, Santos et al. (2022)[4] systematically reviewed the literature on asbestos exposure and malignant pleural mesothelioma and reported that the mean age of patients was approximately 66 years, with a mean latency period between the first exposure and diagnosis of approximately 42 years. Thus, the mesothelioma deaths occurring in 2016 were a result of exposures that occurred in the 1970s or even earlier and are not the result from any possible current exposures to chrysotile.

Until the 1970s, little or no distinction was made between the use of amphibole asbestos and that of chrysotile. Amphibole asbestos, need it be repeated, were banned in most of the Western world in the 1980s, and similar actions were undertaken worldwide in the following decade.

The UNEP document states that of the 209,481 deaths, 177,614 were from lung cancers, which its authors attributed to asbestos exposure. This derivation appears to be based on a ratio of mesothelioma to lung cancers in cohorts heavily exposed decades ago to both amphibole and chrysotile asbestos[5]. A search of the GBD Study database for risk factors associated with asbestos exposure shows three citations as the basis for their determination (Lentes et al., 2011; Goodman et al., 1999; Camargo et al.,2011). The oldest exposures cited in these publications range from 1904 to 1939, a period when exposures were exceedingly high and when there was little, if any, differentiation between amphibole and chrysotile asbestos.

As only chrysotile is used today, extrapolating asbestos-related deaths from mixed exposures at high exposure concentrations is meaningless. Gilham et al., 2015[6] reported that all mesothelioma in the UK could be accounted for from amosite exposure alone even though of the five million tons of UK asbestos imports since 1954, 4.45 million tons of chrysotile were imported (89 %), compared to 0.45 tons of amosite (9 %) and 0.1 tons of crocidolite (2 %). Their results confirm that chrysotile exposure was not a factor in explaining the UK mesothelioma incidence.

Another publication (McCormack et al., 2012)[7] estimated the asbestos-related lung cancer burden from mesothelioma mortality: it included 68 risk estimates drawn from 55 studies, in which excess

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[4] Cátia Santos, Maria dos Anjos Dixe, Ema Sacadura-Leite, Philippe Astoul, António Sousa-Uva; Asbestos Exposure and Malignant Pleural Mesothelioma: A Systematic Review of Literature. *Port J Public Health* 28 December 2022; 40 (3): 188–202.

[5] UNEP used an asbestos impact ratio (AIR) approach where the AIR was defined as the excess deaths due to mesothelioma observed in a population divided by the excess deaths in a hypothetical population heavily exposed to asbestos (without differentiating chrysotile form amphibole asbestos).

[6] Gilham C, Rake C, Burdett G, et al. Occup Environ Med Published Online First: December 29, 2015. doi:10.1136/oemed-2015-103074 See: https://oem.bmj.com/content/73/5/290

[7] McCormack V, Peto J, Byrnes G, Straif K, Boffetta P. Estimating the asbestos-related lung cancer burden from mesothelioma mortality. Br J Cancer. 2012 Jan 31;106(3):575-84. doi: 10.1038/bjc.2011.563. Epub 2012 Jan 10. Erratum in: Br J Cancer. 2014 Dec 9;111(12):2381. PMID: 22233924; PMCID: PMC3273352.

cancer deaths were calculated for each cohort based on observed minus expected deaths, based on national/regional age- and sex-specific rates, to obtain Standardized Mortality Ratios (SMR)[8].

Again, the studies included high exposures that occurred many years ago. The authors estimated fiber-specific ratios which characterize the overall asbestos-related lung cancer to mesothelioma relationship across different exposure circumstances and over a long period of time. In these studies, there was a marked correlation between lung cancer SMR and mesothelioma cohorts exposed to the amosite asbestos (amphibole). For amphibole asbestos, estimates suggest there was between a 6 % and 10 % increase in lung cancer deaths for every mesothelioma death in 1,000 deaths. Chrysotile cohorts had a wider range of estimates, resulting from little correlation between excess lung cancers and mesotheliomas. When present, the authors state that it appears that many of the mesotheliomas were actually due to amphibole exposure. The authors state that "for chrysotile, widely consumed today, asbestos-related lung cancers cannot be robustly estimated from few mesothelioma deaths and the latter cannot be used to infer no excess risk of lung or other cancers". Their analysis does not exclude a lung cancer effect from these older cohorts but mentions that smoking can be a major contributor.

The UNEP document or the references cited therein provide no differential information on the potency of chrysotile alone at exposure levels that occur today.

But such information does exist: Schonfeld et al., 2017[9], reported on the airborne dust concentrations in one of the largest chrysotile asbestos operation since the 1890's and still operating today at Uralasbest in Russia from over 90,000 dust measurements collected across six factories and a mine covering five decades. In 1950, the total dust concentration ranged from 50 to 1,000 mg/m^{3,} but as early as 2000, control measures reduced the concentration to a range of 0.5 to 8 mg/m^3 depending on activity. In a follow-up publication on cancer mortality at the same mine, Schüz et al. (2024)[10] presented in the supplementary data to the study that no statistically significant difference was found for lung cancer in men based on chrysotile fibers/cm^3-years even with the earlier high exposure levels. The study's abstract[11] and pages 4-6 of the supplementary data[12] (Table 4) from the Schüz et al., (2024) publications are included in Annex 1 and 2 of the present document.

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[8] The Standardized Mortality Ratio (SMR) is a statistical measure to compare the mortality rate of a study group to that of a standard population.

[9] Schonfeld SJ, Kovalevskiy EV, Feletto E, Bukhtiyarov IV, Kashanskiy SV, Moissonier M, Straif K, McCormack VA, Schüz J, Kromhout H. Temporal Trends in Airborne Dust Concentrations at a Large Chrysotile Mine and its Asbestos-enrichment Factories in the Russian Federation During 1951-2001. Ann Work Expo Health. 2017 Aug 1;61(7):797-808. doi: 10.1093/annweh/wxx051. PMID: 28810689; PMCID: PMC6005011.

[10] Schüz J, Kovalevskiy E, Olsson A, Moissonnier M, Ostroumova E, Ferro G, Feletto E, Schonfeld SJ, Byrnes G, Tskhomariia I, Straif K, Morozova T, Kromhout H, Bukhtiyarov I. Cancer mortality in chrysotile miners and millers, Russian Federation: main results (Asbest Chrysotile Cohort-Study). J Natl Cancer Inst. 2024 Jun 7;116(6):866-875. doi: 10.1093/jnci/djad262. PMID: 38247448;

[11] See : https://academic.oup.com/jnci/article/116/6/866/7577290 Accessed March 2025

[12] Supplementary data https://academic.oup.com/jnci/article/116/6/866/7577290#supplementary-data Accessed October 3rd 2024.

Currently, as only chrysotile is used in controlled environments, no cancer mortality would be expected.

There is clear evidence provided in the scientific literature that today, the use of chrysotile alone without mixed amphibole exposures and with considerably lower exposure concentrations (than which occurred when many of the epidemiology studies cited were performed) does not cause mesothelioma and certainly would not be associated “with 70 % of work-related cancers”.

About alternatives

The need for rigorous database referencing and differentiation between fiber types is especially important in light of increasingly vocal calls for using alternatives to the chrysotile fiber. The UNEP document is remarkably honest in its remarks on the lack of scientific data on health hazards related to so-called "safer alternatives"[13]:

> "*As in any case of chemical substitution, supplementary research (including life-cycle assessments (LCA)) and monitoring of the asbestos alternatives is warranted to avoid any unintended health and environmental consequences and regrettable substitutions. To make well-informed decisions on asbestos replacement, it is essential to conduct a LCA of potential alternatives. (…) However, only some of the substitute materials have been assessed for health hazards, and health hazard data has not been sufficient in many cases. The examination of alternatives in a study conducted by Park (2018) concluded that initiatives should be undertaken to reduce workers' exposure to replacement materials devoid of asbestos.*
>
> *According to patent data from the United States and Europe, fibrous materials may be considered as an alternative to asbestos. There are many kinds of fibrous materials, which can be classified into synthetic and natural fibres. However, recent studies brought to lights evidence on health hazards, including links to cancers, of fibrous materials used as asbestos substitutes."*

The ICA welcomes this recognition by UNEP of the need for more research on alternatives to chrysotile and hopes that its call will be heard within the WHO, the ILO as well as by all parties to the Rotterdam Convention. The ICA encourages authorities to evaluate the potential toxicity of all fibers, including alternatives, based on equal fiber number exposure.

[13] See https://documents.un.org/doc/undoc/gen/k24/003/25/pdf/k2400325.pdf pp. 14-15. Accessed October 3rd 2024.

Annex 1

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Article

Cancer mortality in chrysotile miners and millers, Russian Federation: main results (Asbest Chrysotile Cohort-Study)

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Abstract

Background: We investigated mortality in workers of the world's largest chrysotile mine and enrichment factories located in the town of Asbest, Russian Federation.

Methods: This historical cohort study included all workers employed for at least 1 year between 1975 and 2010 and follow-up until the end of 2015. Cumulative exposure to dust was estimated based on workers' complete occupational history linked to dust measurements systematically collected from the 1950s. Exposure to chrysotile fibers was estimated using dust-to-fiber conversion factors. Relative risks (RRs) and 95% confidence intervals (CIs) were estimated as mortality rate ratios in Poisson regression models.

Results: A total of 30 445 (32% women) workers accumulated 721 312 person-years at risk and 11 110 (36%) died. Of the workers, 54% had more than 30 years since their first exposure. We found an exposure-response between cumulative dust and lung cancer mortality in men. No clear association with dust exposure but a modest increase in the highest category of fiber exposure was seen for lung cancer in women. Mesothelioma mortality was increased (RR = 7.64, 95% CI = 1.18 to 49.5, to at least 80 fibers per cm^3 years and RR = 4.56, 95% CI = 0.94 to 22.1, to at least 150 mg/m^3 years [dust]), based on 13 deaths. For colorectal and stomach cancer, there were inconsistent associations. No associations were seen for laryngeal or ovarian cancer.

Conclusion: In this large-scale epidemiological study in the world's largest active asbestos mine, we confirmed an increased risk of mesothelioma with high fiber exposure and an increasing mortality for lung cancer in men with increasing dust exposure. Less clear-cut increased lung cancer mortality was seen in the women. Continued mortality follow-up is warranted.

Annex 2

Supplementary Data

Supplementary material to: Schüz, et al., Cancer mortality in chrysotile miners and millers, Russian Federation: main results (Asbest Chrysotile Cohort-Study). J Natl Cancer Inst. 2024 Jun 7;116(6):866-875. doi: 10.1093/jnci/djad262. PMID: 38247448;

https://academic.oup.com/jnci/article/116/6/866/7577290#supplementary-data Accessed October 3rd, 2024.

Supplementary Table 4. Mortality rate ratios (RR) and 95% confidence intervals (CI) for categories of cumulative dust exposure and cumulative fibre exposure, by deaths from different causes and cancer sites, by applying lag times of 10 years and of 20 years, by sex, adjusted for age and time since last employment

NOTE: Mortality rate ratios (RR) are considered statistically significant only when the 95% confidence interval (CI) does not include 1.0. Specifically, for a mortality rate ratio to be statistically significant, the lower limit of the 95% CI must be greater than 1.0. For Lung Cancer Fibers/cm^3-years all values are not statistically significant.

Supplementary Table 4. Mortality rate ratios (RR) and 95% confidence intervals (CI) for categories of cumulative dust exposure and cumulative fibre exposure, by deaths from different causes and cancer sites[a], by applying lag times of 10 years and of 20 years, by sex, adjusted for age and time since last employment

Dust category (mg/m³-years)	Men				Women			
	10-year lag		20-year lag		10-year lag		20-year lag	
	N deaths	RR (CI)	*N* deaths	RR (CI)	*N* deaths	RR (CI)	*N* deaths	RR (CI)
	All deaths				All deaths			
0[b]	536	1.10 (0.99–1.23)	1595	0.94 (0.87–1.01)	76	0.79 (0.59–1.05)	303	0.96 (0.82–1.14)
>0–20	2208	1.00	2097	1.00	506	1.00	512	1.00
≥20–65	2261	0.94 (0.89–1.00)	2096	0.92 (0.86–0.98)	823	1.03 (0.92–1.15)	768	0.99 (0.89–1.11)
≥65–150	2079	0.90 (0.84–0.96)	1513	0.93 (0.86–1.00)	821	0.94 (0.84–1.05)	708	0.93 (0.83–1.05)
≥150	1186	0.98 (0.90–1.06)	969	1.00 (0.92–1.09)	614	1.01 (0.89–1.14)	549	1.01 (0.89–1.14)
p for trend		0.11		0.83		0.77		0.96
	All cancers (main ICD group C)				All cancers (main ICD group C)			
0	44	1.20 (0.87–1.67)	161	1.04 (0.86–1.27)	15	0.90 (0.50–1.61)	67	1.03 (0.73–1.45)
>0–20	285	1.00	323	1.00	97	1.00	99	1.00
≥20–65	435	1.06 (0.91–1.24)	468	1.06 (0.91–1.22)	169	1.09 (0.85–1.41)	145	1.04 (0.81–1.35)
≥65–150	494	1.10 (0.94–1.29)	362	1.13 (0.96–1.34)	137	0.88 (0.67–1.15)	121	1.03 (0.78–1.36)
≥150	268	1.14 (0.95–1.37)	212	1.12 (0.93–1.36)	117	1.13 (0.85–1.50)	103	1.24 (0.93–1.66)
p for trend		0.17		0.20		0.79		0.26
	Lung cancer				Lung cancer			
0	13	1.14 (0.63–2.05)	54	1.03 (0.74–1.44)	1	1.49 (0.17–13.08)	6	4.02 (1.09–14.90)
>0–20	95	1.00	116	1.00	7	1.00	4	1.00
≥20–65	161	1.19 (0.92–1.55)	177	1.12 (0.88–1.43)	10	0.74 (0.28–1.95)	9	1.25 (0.38–4.06)
≥65–150	190	1.34 (1.02–1.76)	135	1.27 (0.97–1.67)	11	0.65 (0.25–1.70)	10	1.33 (0.41–4.31)
≥150	105	1.44 (1.06–1.95)	82	1.31 (0.97–1.79)	12	1.07 (0.42–2.75)	12	2.34 (0.75–7.38)
p for trend		0.01		0.06		0.78		0.07
	Laryngeal cancer[c]				Ovarian cancer			
0	2	2.46 (0.50–12.18)	3	0.47 (0.14–1.67)	1	0.61 (0.07–5.12)	5	0.81 (0.26–2.59)
>0–20	8	1.00	15	1.00	10	1.00	10	1.00
≥20–65	18	1.41 (0.61–3.29)	11	0.46 (0.21–1.03)	10	0.65 (0.26–1.58)	7	0.58 (0.22–1.56)
≥65–150	9	0.63 (0.23–1.71)	13	0.83 (0.37–1.88)	9	0.70 (0.27–1.83)	8	1.08 (0.39–2.95)
≥150	9	1.21 (0.44–3.35)	4	0.44 (0.14–1.39)	5	0.64 (0.21–2.01)	5	1.01 (0.32–3.20)
p for trend		0.72		0.29		0.45		0.77

	Stomach cancer				Stomach cancer			
0	6	1.12 (0.45–2.80)	22	1.02 (0.57–1.81)	5	2.01 (0.66–6.13)	11	0.90 (0.39–2.08)
>0–20	30	1.00	34	1.00	15	1.00	18	1.00
≥20–65	54	1.33 (0.84–2.12)	57	1.33 (0.85–2.06)	24	1.00 (0.52–1.92)	18	0.74 (0.38–1.44)
≥65–150	64	1.55 (0.96–2.51)	49	1.69 (1.04–2.74)	13	0.55 (0.26–1.20)	11	0.56 (0.26–1.23)
≥150	33	1.54 (0.90–2.66)	25	1.45 (0.83–2.54)	14	0.91 (0.42–1.94)	13	0.94 (0.44–2.00)
p for trend		0.06		0.08		0.38		0.47
	Colorectal cancer				Colorectal cancer			
0	3	1.59 (0.46–5.54)	12	1.54 (0.74–3.21)	1	0.44 (0.05–3.81)	6	0.72 (0.24–2.13)
>0–20	18	1.00	20	1.00	14	1.00	13	1.00
≥20–65	41	1.36 (0.78–2.40)	41	1.23 (0.71–2.13)	22	0.94 (0.48–1.84)	19	0.96 (0.47–1.95)
≥65–150	43	1.09 (0.61–1.95)	35	1.23 (0.68–2.21)	22	0.83 (0.42–1.65)	24	1.27 (0.63–2.56)
≥150	29	1.35 (0.72–2.51)	26	1.53 (0.82–2.85)	18	0.99 (0.48–2.04)	15	1.12 (0.52–2.41)
p for trend		0.65		0.18		0.90		0.66

Fibre category (f/cm^3-years)	Men				Women			
	10-year lag		20-year lag		10-year lag		20-year lag	
	N deaths	RR (CI)	*N* deaths	RR (CI)	*N* deaths	RR (CI)	*N* deaths	RR (CI)
	All deaths				All deaths			
0[b]	536	1.10 (0.98–1.22)	1595	0.93 (0.86–1.00)	76	0.79 (0.59–1.06)	303	0.95 (0.81–1.12)
>0–12	2256	1.00	2248	1.00	524	1.00	545	1.00
≥12–40	2621	0.94 (0.89–1.00)	2524	0.90 (0.85–0.96)	916	1.03 (0.92–1.15)	870	0.98 (0.88–1.09)
≥40–80	2064	0.86 (0.80–0.92)	1341	0.88 (0.81–0.95)	758	0.92 (0.82–1.03)	622	0.90 (0.80–1.01)
≥80	793	0.97 (0.89–1.06)	562	1.02 (0.92–1.12)	566	0.95 (0.84–1.07)	500	0.98 (0.87–1.11)
p for trend		0.01		0.16		0.17		0.63
	All cancers (main ICD group C)				All cancers (main ICD group C)			
0	44	1.14 (0.82–1.57)	161	0.97 (0.80–1.17)	15	0.91 (0.51–1.64)	67	1.01 (0.72–1.43)
>0–12	309	1.00	374	1.00	95	1.00	103	1.00
≥12–40	495	0.98 (0.85–1.14)	557	0.95 (0.83–1.09)	179	1.10 (0.86–1.42)	163	1.04 (0.81–1.33)
≥40–80	489	1.00 (0.86–1.17)	295	0.92 (0.78–1.09)	134	0.95 (0.72–1.24)	111	1.02 (0.78–1.35)
≥80	189	1.13 (0.93–1.36)	139	1.18 (0.96–1.46)	112	1.12 (0.84–1.48)	91	1.17 (0.87–1.57)
p for trend		0.34		0.57		0.75		0.51
	Lung cancer				Lung cancer			
0	13	1.03 (0.57–1.85)	54	0.94 (0.68–1.31)	1	1.86 (0.21–16.64)	6	5.76 (1.38–24.01)
>0–12	106	1.00	135	1.00	6	1.00	3	1.00
≥12–40	183	1.07 (0.83–1.36)	214	1.01 (0.81–1.27)	12	1.02 (0.38–2.71)	12	2.14 (0.60–7.61)
≥40–80	187	1.17 (0.90–1.52)	109	1.03 (0.78–1.37)	9	0.74 (0.26–2.10)	9	2.00 (0.54–7.47)
≥80	75	1.36 (0.99–1.86)	52	1.32 (0.94–1.86)	13	1.49 (0.56–3.97)	11	3.36 (0.92–12.22)
p for trend		0.04		0.24		0.44		0.05

	Laryngeal cancer[c]				Ovarian cancer			
0	2	1.65 (0.35–7.73)	3	0.45 (0.13–1.55)	1	0.59 (0.07–4.96)	5	0.69 (0.23–2.12)
>0–12	12	1.00	17	1.00	10	1.00	12	1.00
≥12–40	16	0.74 (0.34–1.57)	12	0.39 (0.18–0.84)	10	0.60 (0.25–1.46)	7	0.43 (0.17–1.10)
≥40–80	9	0.41 (0.16–1.01)	10	0.71 (0.30–1.68)	9	0.72 (0.28–1.85)	10	1.09 (0.44–2.66)
≥80	7	0.89 (0.34–2.37)	4	0.73 (0.23–2.28)	5	0.60 (0.19–1.84)	1	0.16 (0.02–1.32)
p for trend		0.33		0.47		0.42		0.25
	Stomach cancer				Stomach cancer			
0	6	1.05 (0.43–2.59)	22	0.96 (0.55–1.69)	5	1.79 (0.59–5.38)	11	0.84 (0.37–1.92)
>0–12	33	1.00	39	1.00	17	1.00	20	1.00
≥12–40	60	1.19 (0.76–1.84)	73	1.30 (0.87–1.95)	22	0.75 (0.40–1.43)	18	0.61 (0.32–1.16)
≥40–80	70	1.55 (0.98–2.46)	40	1.40 (0.85–2.29)	15	0.60 (0.30–1.23)	14	0.72 (0.35–1.46)
≥80	18	1.18 (0.64–2.17)	13	1.25 (0.64–2.42)	12	0.68 (0.32–1.46)	8	0.57 (0.24–1.34)
p for trend		0.14		0.27		0.22		0.13
	Colorectal cancer				Colorectal cancer			
0	3	1.75 (0.50–6.13)	12	1.39 (0.68–2.82)	1	0.43 (0.05–3.70)	6	0.76 (0.25–2.25)
>0–12	17	1.00	24	1.00	15	1.00	13	1.00
≥12–40	46	1.46 (0.83–2.57)	51	1.11 (0.68–1.83)	24	0.90 (0.47–1.72)	23	1.08 (0.55–2.15)
≥40–80	44	1.21 (0.67–2.19)	25	0.82 (0.45–1.49)	20	0.80 (0.40–1.58)	21	1.33 (0.65–2.69)
≥80	24	1.91 (1.00–3.66)	22	1.98 (1.07–3.66)	17	0.94 (0.46–1.91)	14	1.22 (0.56–2.64)
p for trend		0.16		0.13		0.78		0.60

[a] ICD codes for cancer sites are: lung, C33–C34; larynx, C32; ovary, C56; stomach, C16; colon and rectum, C18–C21.

[b] Because the 10-year or 20-year lag time was applied, some workers had no occupational exposure to dust or fibres; as the counting of risk time started with first exposure, they are kept as a separate group and displayed only for the purpose of completeness (see Materials and Methods)

[c] Only 1 case of laryngeal cancer in women; therefore, analysis for women was not carried out.