A Lower Asbestos Exposure Limit!

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The Filter provides editorial space for opinion pieces from our members. Such pieces offer an author's perspective on a specific topic or issue, with the aim of persuading readers to consider their points of view.

As part of the Europe's Beating Cancer Plan, European Union (EU) ministers of employment agreed their position on a proposal to tighten EU legislation protecting workers from the risks of asbestos. Their position is that the current occupational exposure limit (OEL) should be lowered to 0.01 f/mL as an 8-hour time-weighted average (TWA) and that asbestos fibre-counting should be carried out based on a more modern method (electron microscopy - EM). This "balanced approach" was said to be underpinned by a public health objective aiming at the necessary safe removal of asbestos. Consequently, a 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 at work has been published. Before becoming law, the Council and the European Parliament need to agree on a joint position on the proposed revision. The Parliament is still in the process of defining its stance.

We (the authors) are of the opinion that such a reduction in the OEL is not justified for the following reasons:

1. The estimation of risk of adverse health effects due to asbestos exposure (e.g. mesothelioma & lung cancer) has been determined based on applying linear no threshold (LNT) exposure-response models. However, LNT risk extrapolation is being questioned in the peer-reviewed literature as to its validity. Calabrese *et al* (2022) go further to say that LNT was made policy based on fraudulent research, manipulation of scientific literature, and scientific misconduct by the US National Academy of Sciences.

2. There is mounting evidence that indicates there are protective mechanisms that can prevent carcinogenesis at low doses of genotoxic chemicals. Inflammation generally co-initiates cancer and transiently amplifies activated stem cells. Several non-genotoxic mechanisms have demonstrated threshold-shaped dose-response for cancer outcomes.
3. During the long latency period (typically 30 plus years) before the clinical diagnosis of cancer of the lung or of the larynx or diffuse malignant mesothelioma, genetic, chromosomal and epigenic alterations occur. Recent biochemical studies have confirmed that oxidative damage to cytosine is a plausible biological mechanism leading to epigenetic alterations and development of cancer in association with persistent inflammation (IARC, 2012).
4. Various authors have suggested the existence of a chronic inflammation-driven threshold concentration of asbestos fibres that causes asbestos-related cancers. What that threshold concentration is, remains uncertain, but certainly more than one fibre is required.
5. Even when using LNT exposure-response models, there are difficulties in attempting to apply the dose response data that does exist.
6. We believe that we are not seeing an increase in mesothelioma in Australia under current exposure scenarios. Due to the long latency period, most exposed workers in industries where asbestos exposure risk was high did not develop mesothelioma until decades later. The latest published data for Australia indicates a decrease in rates of mesothelioma between 2013 and 2020 (AIHW, 2021). The asbestos mining and manufacturing industries in Australia have disappeared and exposures have been significantly less since the early 1990s. In addition, workers are now well protected with PPE and mandated work practices in asbestos removal, such that the working life exposure profile results in an extremely low cumulative exposure and subsequently extremely low risk of asbestos related disease.
7. In terms of quantitative risk assessment all the epidemiology studies used to determine risk are based on phase-contrast optical microscopy (PCM) / membrane filter method (MFM) counts. PCM provides a relatively quick and cost-effective analysis of airborne asbestos samples, but it can't distinguish between asbestos and non-asbestos fibres, nor differentiate between the different types of asbestos, and the MFM detection limit is at 0.01 f/mL. While electron microscopy (EM) can assess asbestos fibre exposure more accurately and has a lower detection limit, it is significantly more expensive and takes more time to analyse than a PCM sample.

Most importantly, there is no simple relationship between results by PCM and EM counting methods. The ratio between PCM counts and EM counts varies considerably depending on fibre type and process/industry type and product type. The question that remains is, what EM-based OEL will be set as a PCM equivalent concentration?

8. In Australia, more than 90% of asbestos fibre monitoring is static sampling conducted for the purposes of background, control and clearance monitoring for asbestos removal works and not for exposure risk assessment (R Golec & L Apthorpe, pers comm). It seems somewhat academic whether the OEL is reduced to 0.01 f/mL as we should never be comparing these static monitoring results with an OEL in the first place as they were not taken in the breathing zone of workers. By default, we already use 0.01 f/mL as an action level which indicates the airborne fibre concentration is above the detection limit of the PCM method, and control actions are required to be implemented.

The AIOH (2016) position paper on asbestos notes that the "AIOH believes that current exposure standards used in Australia are adequate, and as with any carcinogen, exposures should be maintained as low as reasonably practicable (ALARP)." We are unaware of any published evidence that suggests levels of exposure below 0.01 f/mL will provide significant reductions in ill health compared to the current limit of 0.1 f/mL, particularly when airborne concentrations above the PCM detection limit of 0.01 f/ml require actions to control fibre concentrations. We believe that the 2016 AIOH position on asbestos is still relevant.

Whilst detection limits are improved with EM, it is impractical and expensive for most routine monitoring (i.e. background, control, and clearance monitoring) where quick results turnaround and reporting times are critical. The EM techniques can be used where lower detection limits are required and when identification of the fibre type in airborne samples is necessary.

References

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***Article featured in the Filter magazine May 2023**