Crystalline silica in High Temperature Insulation Wool (HTIW) products after use in high temperature applications

In our everyday lives we are all exposed to dusts containing crystalline silica (CS). While this rarely causes any harm, occupational exposures to CS particles that are fine enough to enter the lung (respirable CS), typically during mining, quarrying, stone masonry and sand blasting activities, can cause disease in exposed workers – possibly including lung cancer.

The high temperature insulation industry has long been concerned with any possible effects of exposure to dusts from their products that may occur during maintenance or wrecking of used furnaces. Such dusts can contain respirable CS. Numerous studies have been undertaken, independently or commissioned by the industry, to study this possibility and the results of these are described below. Overall, the available data indicate that exposure to CS in after-use HTIW products, as may occur during furnace maintenance and wrecking, is unlikely to cause disease.

Background

Refractory materials, including bricks, castables and man made mineral fibre products, are usually composed of silicates – silicon in various combinations with oxygen and other elements. Most high temperature insulation wools consist of amorphous glass fibres, composed of either alkaline earth silicates (AES[1]) or alumino silicates (refractory ceramic fibres; ASW/RCF[2]). A third group are the polycrystalline wools (PCW). Although types of silica may be used in their manufacture, none of these materials contain free crystalline silica in the product as sold and installed. However, it is now known that AES and ASW/RCF may transform into a mixture of crystalline phases, including CS, following prolonged heating. PCW, though, does not contain CS even after use.

Amorphous HTIWs (ASW/RCF and AES) are produced from a molten glass stream which is aerosolised by a jet of high pressure air or by letting the stream impinge onto spinning wheels. The droplets are drawn into fibres; the mass of both fibres and remaining droplets cool very rapidly so that no crystalline phases form.

When amorphous HTIW products are installed and used in high temperature applications, such as industrial furnaces, at least one face (the hot face) may be exposed to conditions causing the fibres to partially devitrify. Depending on the chemical composition of the fibres and the time and temperature to which the materials are exposed, different stable crystalline phases may form.

Devitrification involves separation within the glass of phases with similar compositions to those of stable silicate minerals such as mullite, enstatite and wollastonite or diopside. These phases crystallise within a matrix of silica-rich glass from which, in turn, silica also crystallises, predominantly in the form of cristobalite[3].

Regulation and classification of dusts containing respirable crystalline silica dust

In 1997 IARC[4] reviewed the available literature on crystalline silica exposure and concluded that there was sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources; IARC therefore classified crystalline silica in these situations as a Group 1 carcinogen.

In making their overall evaluation, the IARC Working Group noted that carcinogenicity in humans was not detected in all industrial circumstances. Carcinogenicity may be dependent on inherent characteristics of the crystalline silica or on external factors affecting its biological activity or distribution of its polymorphs.

In Europe, as of December 2010, suppliers of materials have to classify, label and package hazardous substances according to the Classification, Labelling and Packaging Regulation (EC) 1272/2008. As a consequence, mixtures and substances containing CS (fine fraction) above 1% are classified as hazardous and must be appropriately labelled. In addition, various national regulations exist in almost all EU member States. Such regulations entail various controls including regulatory limit values fixing the maximum allowed exposures to respirable airborne crystalline silica.

DETAILS OF THE FIBRES TESTED IN THE FRAUNHOFER INSTITUTE CYTOTOXICITY STUDIES

- A number of commercially available AES fibres were tested including calcium magnesium silicates (AES 1, 2, 3) and magnesium silicate (AES 4).
- Fibres were heated to the indicated temperatures, representing either normal maximum continuous use conditions or the classification temperature.
- Non heated samples were also tested.

SAMPLE	HEATING DURATION (DAYS)	HEATING TEMPERATURE (° C)	CRYSTALLINE SILICA CONTENT (WEIGHT %)
AES 1	28	950	0.3
	7	1100	18
AES 2	28	1050	10
	7	1200	23
AES 3	28	1150	34
	7	1300	32
AES 4	1	1260	18

AES 1/2/3/4 describe samples of calcium-magnesium- and magnesium-silicate wools, representing a wide range of commercially available AES products

Fig. 1

1 AES stands for Alkaline Earth Silicate, a type of wool exonerated from the carcinogen classification under Directive 97/69/EC and Regulation (EC) 1272/2008.

2 ASW/RCF stands for Alumino Silicate Wool, also called Refractory Ceramic Fibre (RCF).

3 Brown TP, Harrison PTC (2014) Crystalline silica in heated man-made mineral fibres: a review. Reg. Toxicol. Pharmacol. 68, 152-159.

4 IARC Monograph on the Evalaution of Carcinogenic Risk to Humans. Volume 68: Silica, some silicates, coal dust and para-aramid fibrils.

Summary of scientific information available for CS in after-service ASW/RCF and AES

When, in the 1980's, RCF's were tested in a series of animal inhalation experiments (the so-called RCC studies), the samples tested included a specimen of heated (devitrified or crystallised) ASW/RCF estimated as containing 27 % cristobalite, to simulate after-use fibres. This sample caused less lung effects than any other sample tested and no excess of tumours.

Further studies at the IOM in Edinburgh also found this sample to be inert when injected into the peritoneum of rats[5]. These early results with ASW/RCF already gave an indication that after-use (devitrified) fibres do not constitute a health hazard.

Some forms of CS can accumulate in the lungs causing inflammation, tissue damage and silicosis. This is especially true of freshly-cleaved CS which is toxic to the cells (macrophages) that normally clear dusts from the lungs. This prevents the silica from being removed. The damaged cells release substances that attract more macrophages and inflammatory cells, and a cascade of deleterious effects ensues.

The ease with which macrophages may be kept alive (cultured) in the laboratory - whilst retaining their ability to ingest particles - has enabled their interaction with silica to be studied intensively in so-called in vitro experiments. Forms of silica that are toxic to macrophages in vitro also cause disease in animals.

As there are ethical and other reasons for trying to avoid animal experiments, heated AES fibres have been tested in vitro using cultured macrophages. As with heated ASW/RCF's, AES fibres were not toxic to these cells[6] even after complete devitrification.

More recently at the Fraunhofer Institute for Toxicology and Experimental Medicine, four samples of AES (three calcium-magnesium-silicate wools and one magnesium silicate wool), with classification temperatures between 1100°C and 1300°C, were heated for different durations to their classification temperature and/or their normal maximum continuous use temperature (approx 150° C below their classification temperature)[7]. These heated samples contained between 0.3 and 32 % CS (see Fig. 1).

Unheated and heated fibres were then tested in cultures of macrophages. Two measures of toxic activity were used. Firstly the ability of the fibres to cause the cells to leak was determined by measuring the amount of an enzyme (lactate dehydrogenase; LDH) normally found inside the cells that had leaked into the medium outside (results are shown in Fig. 3). Secondly the amount of DNA damage was measured using the Comet assay in which the level of DNA strand breakage is quantified in individual cells.

The control quartz sample (DQ12) was clearly positive in both these assays. However none of the heated or unheated fibres showed significant activity. The cytotoxicity results for all four types of AES wools are illustrated in Fig 3.

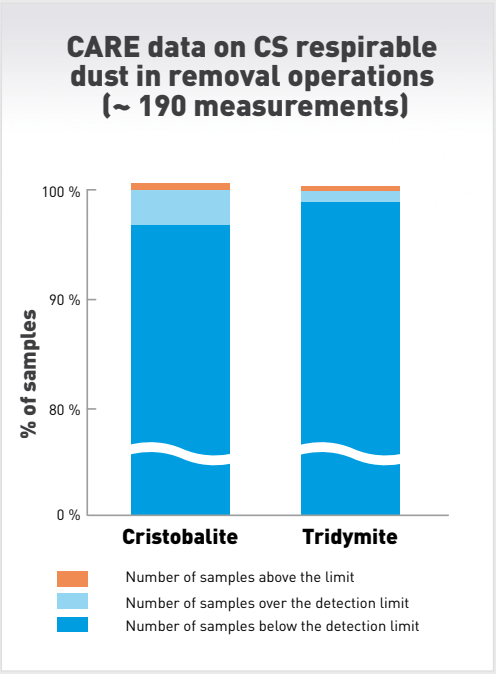
The authors of this study[7] concluded that heating AES wools mediates changes in CS content and in fibre length/shape, with heated fibres containing varying amounts of CS (principally in the form of cristobalite) and generally being more particulate in nature than the original unheated forms. While the changes in fibre morphology did impact biological activity, with the heated material showing less activity, CS content had little or no bearing on the measured cellular responses to heated AES wools.

These results were confirmed in yet another series of in vitro experiments conducted at Heriot-Watt University in Edinburgh, using mouse macrophage and human alveolar epithelial cell lines. These cells were exposed to virgin HTIW of different compositions (incl. RCF/ASW and several types of AES), and corresponding heat-treated samples. DQ12 was used as positive control. Cell death, cytokine release, and reactive oxygen species (ROS) formation were assessed in both cell types. The researchers concluded that *"HTIW did not induce cell death or intracellular ROS, and their ability to induce pro-inflammatory mediator release was low. In contrast, DQ12 induced cytotoxicity, a strong pro-inflammatory response and ROS generation."*[8]

Why is CS in after-service HTIW not a health concern?

ECFIA has developed, as part of its Product Stewardship Programme, the so-called CARE Programme (Control and Reduced Exposure) as described elsewhere[9]. CARE occupational hygienists have had

WORKPLACE MEASUREMENTS



TYPE	CRISTOBALITE	TRIDYMITE
Total number of samples	187	163
Sample above the detection limit	6 (3%)	2 (1%)
Applied Limit value [mg/m³]	0.05	0.05
Samples above the workplace limit value	1 (0.5%)	0 (0%)

Fig. 2

5 Miller BG, Searl A, Davis JMG, Donaldson K, Cullen RT, Bolton RE, Buchanan D, Soutar CA (1999) Influence of fiber length, dissolution and biopersistence on the production of mesothelioma in the rat peritoneal cavity. Ann Occup Hyg; 43:155-66.

6 Brown, R.C. (1999). Regulation of crystalline silica: where next? Indoor and Built Environment 8: 113-120.

7 Ziemann C, Harrison PTC, Bellmann B, Brown RC, Zoitos BK, Class P (2014) Lack of marked cyto- and genotoxicity of cristobalite in devitrified (heated) alkaline earth silicate wools in short-term assays with cultured primary rat alveolar macrophages. Inhalation Toxicol. 26, 113-127.

8 Matthew S. P. Boyles, David Brown, Jilly Knox, Michael Horobin, Mark R. Miller, Helinor J. Johnston & Vicki Stone (2018): Assessing the bioactivity of crystalline silica in heated high-temperature insulation wools, Inhalation Toxicology. Available online: https://doi.org/10.1080/08958378.2018.1513610

9 Maxim LD, Allshouse JN, Deadman J, Kleck C, Kostka M, Webster D, Class P and Sébastien P. (1998) CARE – A European programme for monitoring and reducing refractory ceramic fibre dust at the workplace: initial results. Gefahrstoffe Reinhaltung der Luft. 58(3):97-103.

the opportunity to collect about 190 samples during after-service operations where HTIW insulation materials were being repaired or removed. In only six samples was cristobalite detected, and only one sample showed a level of respirable airborne cristobalite above 0.05 mg/m^3, the occupational limit recommended by the EU Scientific Committee for Occupational Exposure Limits (SCOEL). This confirms that, in most cases, respirable CS is not detectable and even more rarely is it above occupational limit values (see Fig. 2).

Silica related fibrosis and cancer in humans have most clearly been observed following exposures to freshly cleaved respirable silica dust. In after-use HTIW, crystalline silica crystals are embedded in a matrix composed of other crystals and glasses and do not seem to be biologically available or capable of damaging the lung[10]. It has been suggested that there is a possibility that such "passivated" silica could be attacked in the body and any coating eventually removed, thus re-activating the potential for the deposited dust to do harm. However, silica particles that do not damage macrophages would be cleared by the normal processes and therefore would not accumulate in the lungs.

Most effects of inhaled or injected fibres are not due to the silica content but to their fibrous shape and size. The absence of effects from devitrified fibres in animal experiments is probably due to the brittle nature of the heated fibres. They readily fragment into shorter pieces which are then easily and rapidly cleared from the lung. A recent extensive review of the literature on crystalline silica in heated man-made vitreous fibres[3] has confirmed that the formation of CS in heated HTIW does not render them more toxic but that concomitant alterations to their fibrous shape actually serve to reduce their biological activity.

Overall, therefore, experimental studies on the biological activity of after-use HTIW have not demonstrated any hazardous activity that could be related to any form of silica they may contain. This, coupled with the inability to detect airborne CS during most after-use activities, means that there is unlikely to be any risk of CS related disease from employment in furnace maintenance or wrecking. However, worker protection measures that comply with regulations must be applied. Where no regulation or code of practice is available, ECFIA recommends that you consult its handling advice available at www.ecfia.eu.

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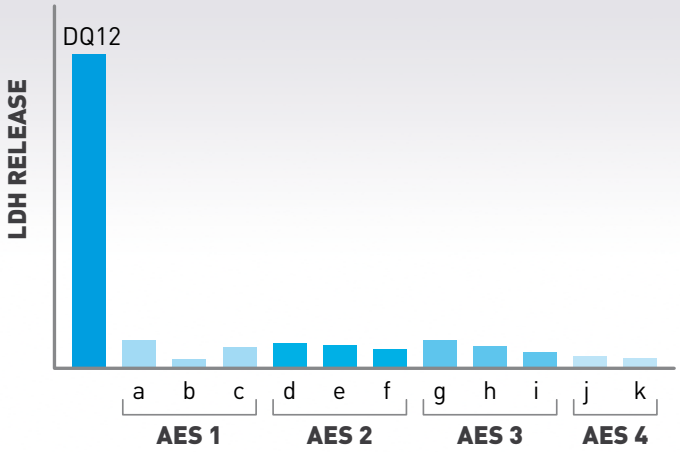
10 Techniques used to evaluate levels of crystalline silica in the workplace do not allow differentiation between free CS and forms where the CS is embedded within a particle of different (mixed) composition. Most regulations apply therefore to all forms of CS whether available biologically or not, and whether aged or freshly cleaved.

CYTOTOXICITY OF NORMAL (UNHEATED) AND HEATED AES WOOLS IN CULTURED RAT ALVEOLAR MACROPHAGES



All the wool samples, whether heated or unheated, showed low biological activity. Heating generally reduced rather than increased their toxicity.

LDH release in culture supernatants after 2 h of incubation with 200 µg/cm^2 of the normal or heated AES 1, AES 2, AES 3 and AES 4 wool samples or the quartz DQ12 positive control, over and above that found for Al_2O_3, a particulate negative control (not shown).

SAMPLE		HEATING TEMPERATURE (° C)	HEATING DURATION (WEEKS)
AES 1	a	-	-
	b	950	4
	c	1100	1
AES 2	d	-	-
	e	1050	4
	f	1200	1
AES 3	g	-	-
	h	1150	4
	i	1300	1
AES 4	j	-	-
	k	1260	24 hours

Fig. 3