

SILICA

AND SANDBLASTING

According to the ILO, some 2.3 million workers die as the result of the work they do, every year. Both IndustriALL Global Union and Building and Wood Workers' International consider this number, shocking as it is, to be a gross underestimate.

For every worker killed in a sudden, violent accident, four to five die of occupational diseases. Silica looms large among occupational causes of lung diseases and cancer - perhaps second only to asbestos.

What is silica?

Silica, generally, is the crystalline form of silicon dioxide (SiO₂). Silica is one of the most common minerals on earth, and it is the crystalline form of silica that poses the greatest problem. The most common crystalline mineral form is quartz, and less commonly found crystalline varieties include tridymite, cristobalite and other forms. It is a major component of many rocks, including quartz, granite, flint, slate and sandstone. Common sand is mostly quartz particles.

Amorphous silica is SiO₂ in a non-crystalline form, generally considered to be much less hazardous than crystalline silica. Silica gel is nearly-pure synthetic amorphous silica, and diatomaceous earth is a naturally-occurring mineral that is mostly - but not entirely - amorphous silica. However, some amorphous silica also contains crystalline silica (diatomaceous earth always does) and the safety of amorphous silica itself remains a matter of debate.



Silica dust

Inhalation of respirable silica can cause silicosis or lung cancer. Exposure to silica dust can result during any excavation, disturbance, working, cutting, breaking, crushing, drilling, grinding or transportation of rock, sand or materials that contain silica. Abrasive blasting (sandblasting) is a particularly dangerous activity that guarantees a high exposure to silica dust unless done in an isolated space such as a glove-box or with well-fitted respiratory protection of the correct type, preferably with a positive pressure mask.

Particles smaller than about five micrometers are easily respirable, deep into the lung, where the crystalline particles tend to lodge and stay once inhaled.

Silicosis

Silicosis is a fibrotic lung disease caused by inhalation of crystalline silica. Silicosis was one of the first recognized occupational diseases. Bernardino Ramazzini (1633-1714) - sometimes called the father of occupational medicine - described this respiratory disease in 1705. He found a sand-like substance in the lungs of deceased stonecutters. Other names for silicosis include "Potter's Rot" and "Grinder's Disease", which gives a clue as to its occupational causes.

The prevalence of silicosis increased markedly with the introduction of mechanized mining. More dust being produced in a shorter time results in much greater exposure. The Hawk's Nest Tunnel catastrophe near Gauley Bridge, West Virginia, USA, in the 1930s resulted in the deaths of at least 764 tunnel workers due to silicosis and led to the first efforts to regulate silica exposure in that country.

Today, despite regulations and control measures that have been implemented in many areas, thousands of new cases of silicosis are diagnosed every year.

Silicosis is unfortunately often misdiagnosed even today, with early symptoms of shortness of breath being attributed to anything from asthma to pneumonia. Because most people do not need their full lung capacity, even when working, a significant percentage of the lungs can be destroyed before most people notice a problem. A proper medical diagnosis requires lung x-rays for evidence of inflammation and scarring; nodular lesions in the upper lobes of the lungs.

Silicosis is an occupational disease risk for any worker exposed to crystalline silica dust, including: miners (surface and underground), rock drillers, sandblasters (industrial and garment), quarry workers, ceramic-tile-glass- and brick-makers, foundry workers, grinders, stonecutters, cement workers, and workers involved in construction, repair, rehabilitation or demolition of concrete structures. Because silica is so common, many other occupations may be implicated.

Silicosis in general is under-diagnosed and its true extent is usually underestimated. This is in part because silicosis can be identified as chronic obstructive pulmonary disease, pneumoconiosis, or other generic lung disease. Misdiagnosis is common, with the cause of death sometimes mis-attributed to pneumonia or tuberculosis even on death certificates. Doctors frequently pay little attention to the occupational history of their patients, or mistakenly believe an industry to be low risk.

Silicosis can be classified according to its differing rates of development and progression as chronic, accelerated or acute. Silicosis victims have increased vulnerability to infections (including tuberculosis), autoimmune diseases (e.g. systemic sclerosis), and diseases of other systems (e.g. kidneys), further complicating correct diagnosis. The end stage of silicosis disease may include progressive massive fibrosis of the lungs, fluid filling of the alveoli (small air sacs in the lungs), emphysema, or chronic bronchitis. It is said to be a particularly unpleasant way to die, with the feeling of slowly suffocating over a period of months, or years, as you fight to breathe.

Exposure to silica also increases the risk of lung cancer.

Disease mechanisms

The most easily respirable particle size is between 0.5 to 3 micrometers (μm or microns). Respirable particles are too small to be visible - but if you can see dust in the air, there is likely some dust that is small enough to penetrate deep into the lungs.

Once in the lungs, the natural defenders of the lung, macrophage cells, attempt to engulf particles, and cilia remove the dead macrophages along with the particles. However, the macrophages are usually only partly successful in this, and remaining silica particles cause localized cell death in the lungs. The body responds by forming fibrous tissue (as when a wound scabs over) at the point of injury. At the same time natural defenses try to engulf, kill and digest silica particles as if they were invading bacteria. Unfortunately, silica particles can't be killed, so poisonous natural chemicals and enzymes accumulate and more healthy lung cells die. The cycle of injury, cell death, and scarring repeats. Eventually, regions of the lung become stiff and fibrous, normal expansion and contraction of the lung is restricted, and damaged portions are unable to exchange oxygen.

The cycle of injury and repair, and the presence of powerful (even though natural) cell-killing enzymes and chemicals increases the risk of DNA damage and cancer. Meanwhile, the body's reaction to the constant inflammatory response can eventually implicate other components of the immune system, making the victim much more vulnerable to infections and causing the frequently-observed auto-immune diseases and damage to other systems of the body.

Some doctors have observed that fresh silica particles (newly fractured) seems to be more toxic than aged silica (such as beach sand). This may have something to do with the surface chemistry of the particles.

Exposure intensity, length and peak exposure determine the nature and progression of silicosis. Chronic silicosis takes many years to kill. Greater exposure can kill within a few years via accelerated silicosis. Acute silicosis due to massive exposure can kill in a few months. Not every individual will have the same response to the same exposure.

Silicosis is considered to be the most common occupational lung disease worldwide. Reported cases are probably less than a third of the real rate due to misdiagnosis and under-reporting. It is believed to be particularly common in developing countries.

There is NO CURE for silicosis except perhaps heart-lung transplantation. By the time you notice symptoms, you may have lost up to 85 per cent of your lung capacity. Now you are fighting to slow the destruction of the remaining 15 per cent. In general, treatment involves easing symptoms and dealing with complications such as infection.

Dying for fashion

Sandblasting of denim jeans for a worn look remains a popular finishing treatment for denim jeans. It is fast and cheap (if the effects on workers are ignored). How many workers are at risk from this practice is difficult to identify and quantify since the fashion industry is heavily outsourced, and many brands claim to have switched to other methods of creating this popular look. Workers are often uninformed of the hazards of this practice, and even if informed, sometimes are willing to "accept" the risk of silicosis or lung cancer.

Exposure limits

Although greater levels of exposure result in greater risk, there is no verifiable safe level of exposure.

Most jurisdictions have enacted "legal" exposure limits. Find out what legal limits apply at your workplace. Legal exposure limits must not be exceeded.

Principles of control

Basic industrial hygiene demands that protective measures be fully adequate and not create new hazards. Control of the hazard at the source is to be preferred: this means complete elimination, substitution, or complete isolation using engineering controls. If control at the source cannot be fully achieved, control along the path of exposure is the next best possibility, as close to the source as possible using measures like effective local ventilation. If control at or near the source do not eliminate the hazard then control at the worker is the least-preferred, but often necessary, option. Control at the worker means the use of personal protective equipment such as respirators. This is always less ideal because it raises problems of availability, fit, cleanliness, maintenance and compliance that are non-issues if the hazard is eliminated at the source.

Workers' representatives and health and safety committee members must be fully aware of and prepared to defend every worker's basic health and safety rights. These are:

- ▶ the Right to Refuse (or shut down) unsafe work without fear of repercussions
- ▶ the Right to Participate in occupational health and safety decision-making. This means full partnership in the development and implementation of all health and safety policies, procedures, and programmes including risk assessments, inspections, audits, and accident/incident investigations. The only people with the moral authority to assess a risk are those who face the risk.
- ▶ the Right to Know, fully, about the hazards of the work and to receive the necessary information, education, and training to do the job safely.

SILICA IS DEADLY.

Don't take your rights to the grave!

