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Syracuse Research Corporation
Merrill Lane
Syracuse, New York 13210

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ABSTRACT: This information profile on*amorphous silicas (763189, 60676860, 1343982) is part of a group of 46 such profiles that provide information about chemicals or industrial processes considered to be potential occupational hazards. Each profile contains summary data on known and suspected health effects, the extent of worker exposure and the industrial importance of either a single chemical, class of chemicals, or a particular industrial process. The report was developed for use by occupational safety and health professionals in industry, and labor and other areas, to provide them with a synopsis of information in their workplaces.

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INTRODUCTION

An information profile is a working paper used by the National Institute for Occupational Safety and Health (NIOSH) to assist in establishing Institute priorities. It is an initial step in determining the need to develop comprehensive documents or to initiate research. Each profile summarizes data on known and suspected health effects, the extent of worker exposure, physical and chemical properties, and the industrial importance of individual chemicals and classes of chemicals. The profile may also be used by industry, labor, and the occupational health community as a synopsis of information on each subject and to identify possible health hazards associated with their workplaces.

Although detailed literature searches are conducted using computerized and manual searching techniques to identify pertinent and recent information, not all the literature obtained is incorporated in the report due to the summary nature of the profiles. Further, literature published after 1978 may not be included in these profiles because it was generally unavailable at the time the search was completed.

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AMORPHOUS SILICAS

SUMMARY

Amorphous silicas are distinguished from crystalline silica in that the arrangement of silicon dioxide molecules is nonperiodic and random. It has been suggested that truly amorphous silica should contain less than one percent quartz; thus, diatomaceous earth may not strictly qualify for consideration under the classification of amorphous silicas. In this information profile, emphasis is placed upon the so-called "synthetic amorphous silicas": silica gel, colloidal silica, precipitated silica, and fumed silica. Studies on the biological activity of all types of amorphous silicas, however, including diatomaceous earth, are considered for the purpose of establishing a basis for health hazard assessment.

Several forms of amorphous silica are commercially produced, and production quantities may approach 800 million pounds per year. Amorphous silica products are widely used in industry: silica gel in dessication, dehumidification (adsorption), thickeners, catalysts, and surface coatings; colloidal silica in coatings and as binders; precipitated silica in fillers, coatings, chemical carriers, and for viscosity control; and fumed silica as reinforcing and thickening or thixotropic agents in the plastics and rubber industries.

The biological activity of amorphous silicas has been examined occasionally over the past several decades. Acute administration by injection and intratracheal instillation is known to be lethal to animals following relatively small doses (10-400 mg/kg body weight); however, the main area of concern over the toxicity of amorphous silica is its potential for induction of pulmonary fibrosis. This action is well-known following exposure to crystalline silica, but the fibrogenic activity of amorphous silica has traditionally been regarded as minimal. Whereas the fibrogenic activity of diatomaceous earth may be attributed to the presence of small amounts of crystalline silica, truly amorphous silica is also implicated as a fibrogenic agent in several animal studies. Moreover, a recent examination of workers exposed to amorphous silica dust revealed evidence of pulmonary fibrosis that could be attributed to this occupational exposure.

Present workplace exposure limits for amorphous silica reflect the fact that it has been regarded only as a nuisance dust. A reevaluation of the role of amorphous silica in fibrogenic disease may be necessary; however, at present, the available data base is inadequate to perform a reliable health risk assessment.

1. Synonyms

2. Chemical Abstracts Service (CAS) Number

3. Registry of Toxic Effects of Chemical Substances (RTECS) Number

4. Molecular Formula

5. Chemical Structure

6. Physical and Chemical Properties

The above information for amorphous silica is presented in Table 1.

7. Producer and User Data

Amorphous silica is a substantially dehydrated, polymerized silica which may be considered to be a condensation polymer of silicic-acid. Amphorous silicas are usually further categorized as silica gel, colloidal silica, precipitated silica, or fumed silica. These subclassifications are related to the method of preparation of each (Maher, 1969).

It is also possible to consider vitreous silica as an amorphous form (Coyle, 1969); however, the classes of amorphous silicas as defined by Maher (1969) are used in this report.

Production and Trends

The estimated annual production volumes of the amorphous silicas are listed below (SRC estimate):

Amorphous Silica	Annual Production (millions of lbs)
Silica gel	560
Colloidal silica	<100
Precipitated silica	<100
Fumed silica	20-50

A specific growth figure is not available for the amorphous silica market; however, there are no indications at this time that any significant increases or decreases are to be expected in the near future.

Uses

Two of the more commercially important uses of silica gel are applications in desiccation and dehumidification (adsorption) and as catalysts. Silica gel is particularly useful in drying air and other gases in both static and dynamic systems and for selective adsorptions, especially for hydrocarbons. Alumina-silica gel complexes are used as catalysts for petroleum cracking (Maher, 1969; Lawler, 1977).

Table 1. Amorphous Silicas

	Silica, amorphous fumed	Silica, amorphous fused (<0.1 μ)	Silica, precipitated, silica gel
Synonyms	Aerosil Amorphous silica dust Silica aerogel Silica anhydride	Cab-o-sil; fused silica Colloidal silica; SG 67 Colloidal silicon dioxide; silicon dioxide Diatomaceous earth; silicon dioxide Dri-die; Silikill Ent 25,550	Silica acid Metasilicic acid
CAS Number	7631-86-9	60676-86-0	1343-98-2
RTECS Number	VV7310000	VV7320000	VV8850000
Molecular Formula	SiO_2	SiO_2	H_2SiO_3
Chemical Structure	SiO_2	SiO_2	H_2SiO_3
Physical and Chemical Properties			
Molecular Weight	60.09	60.09	78.11
Physical State	White powder	White powder	White powder precipitate lustrous granules (gel)
Boiling Point,°C	2230°C	2230°C	
Melting Point,°C	1710°C	1710°C	Room temperature
Vapor Pressure	10 mm at 1732°C	10 mm at 1732°C	
Evaporation Rate			
Solubility			Insoluble in H_2O or acids Soluble in hydrofluoric acid
Specific gravity	2.2 to 2.6	2.2 to 2.6	
Stability			

Fine-particle silica gel is used as a flattening agent for incorporation into surface coatings, synthetic fabrics, and plastics to make the surface appear more attractive. Silica-gels are used as thickeners for forming pharmaceutical ointments, inks, cements, and adhesives and as anti-caking agents for sulfur, zinc oxide, and food spice powders. In addition, silica is used to formulate anti-soil agents for carpets and rugs (Maher, 1969; Lawler, 1977).

Colloidal silica is used commercially as a coating for blueprint and photosensitive papers to improve definition, clarity, and brightness of the exposed print. Another important application of colloidal silica involves its incorporation into floor wax to impart increased skid resistance. Other uses include applications in the textile industry, modification of organic elastomers and resins adhesives, and as a binder for sand molds and glass fibers (Maher, 1969).

Precipitated silicas are widely used in the rubber industry as fillers and reinforcing pigments, and in the paper industry both as a filler and in coating formulations. Other applications include use as a carrier and diluent for high-concentration insecticides and agro-chemicals, anticaking substances, absorbing liquids and sticky solids, as well as use for viscosity control in the formation of plastics, inks, textiles, coatings, and pharmaceuticals (Maher, 1969).

Fumed silicas find wide application in the plastics and rubber industries as reinforcing and thickening or thixotropic agents. One of the most important uses of these silicas as a thixotropic agent in the plastics industry is in the field of polyester-glass reinforced plastics. Fumed silicas are also used as a means of increasing the thixotropic consistency of epoxy resins, plastisols, and organosols (Maher, 1969).

Producers and Distributors

The following companies manufacture and distribute the various amorphous silicas:

Silica Gel (Chem. Week, 1978; Maher, 1969)

Eagle Chem. Co.
Monsanto Chem.
Sacony-Mobil Oil Co.
W.R. Grace
Penn. Glass Sand Corp.
PQ Corporation

Colloidal Silica (Chem. Week, 1978; Maher, 1969)

Dupont
Monsanto
Nalco Chem.
Degussa, Inc.
Nyacol, Inc.

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8. Biological Effects of Exposure

a) Acute Effects

The acute toxicity data are summarized in Table 2.

Table 2. Acute Toxicity Data

Species	Route	Dose	Result	Reference
Silica, amorphous fused				
rat	oral	3160 mg/kg	LD_{50}	NIOSH, 1977
rat	ipr	400 mg/kg	LD_{Lo}	Policard et al., 1954
rat	itr	120 mg/kg	LD_{Lo}	Policard et al., 1954
mouse	ivn	9 mg/kg	LD_{Lo}	NIOSH, 1977
mouse	ipr	40 mg/kg	LD_{Lo}	NIOSH, 1977
cat	ivn	15 mg/kg	LD_{Lo}	NIOSH, 1977
rabbit	ivn	35 mg/kg	LD_{Lo}	NIOSH, 1977
guinea pig	ipr	120 mg/kg	LD_{Lo}	NIOSH, 1977
Silica, amorphous fumed				
rat	ipr	50 mg/kg	LD_{Lo}	NIOSH, 1977
rat	ivn	15 mg/kg	LD_{50}	NIOSH, 1977
rat	itr	10 mg/kg	LD_{Lo}	NIOSH, 1977

Intraperitoneal and intratracheal injections of amorphous silica (silica condensate of heated silica, particles <1 μ) at doses of 30, 50, and 100 mg/kg were administered to rats to determine the toxic and local fibrosing effects (Policard et al., 1954). Intratracheal injections of 30-50 mg/kg caused immediate death to 80-90 percent of the animals involved. Death resulted from pulmonary edema with alveolar hemorrhage and dilatation of pulmonary blood vessels. Intraperitoneal injections were less toxic; 20-30 percent of the rats given 100 mg/kg died, while all those given 50 mg/kg survived. Rats given intraperitoneal injections were examined further; weight loss, diarrhea, and prostration occurred within four days of administration. Autopsies revealed a generalized "stress reaction" in the victims' bodies consisting of changes in the histiocytes of the peritoneal cavity and omentum, enlarged lymph nodes, atrophy of the thymus, hypertrophy of the spleen, and a 50-97 percent weight increase of the adrenal glands. The local fibrosing action was studied in the animals receiving intraperitoneal injections. Peritoneal edema gradually lessened and disappeared 60 days after treatment. Small, spherical nodules 1.5-20 mm in diameter appeared on the omentum. The lesions resembled those formed by quartz, but occurred earlier and were more accentuated. The fibrosis gradually increased after 30-60 days. The authors felt that the lack of a fibrosing effect reported in previous studies may have been due to the small size of the silica particles used; these particles may have been so toxic that fibrosis did not have time to develop before death occurred.

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Precipitated Silica (Maher, 1969)

Mallinckrodt Chem.
PPG, Ind.
PQ Corporation
J.M. Huber Co.

Fumed Silica (SRI, 1978)

Cabot Corp.
Degussa Corp.
Reynolds Metals

Additional distributors of amorphous silicas include (Chem. Week, 1978):

Berkshire Chem.
Miller-Stephenson Chem.
George Uhe Co.
Woelm Pharma. GMBH & Co.
McKesson Chem.
Thompson-Hayward Chem.
Van Waters and Rogers Co.

Manufacturing Processes

There are two commercial methods for the manufacture of silica gel -- the bulk method and the slurry method (Maher, 1969). The bulk method consists of preparing a silica hydrosol by mixing sodium silicate with a strong mineral acid, allowing the hydrosol to rigidly set, and then mechanically breaking the hydrosol. The particles are then washed and dried. The slurry process consists of mixing sodium silicate with an acid at a pH and silica concentration such that a gelantinous precipitate is formed. The silica hydrosol can then be washed and dried, usually via spray drying.

Colloidal silicas are prepared either by peptizing silica hydrogel or by gradual growth of silica micelles (Maher, 1969). Hydrogel peptization is achieved by hydrothermal treatment at elevated pH, temperature, and pressure; small silica micelles solubilize and gradually repolymerize to larger particles to form the final 20 nm colloidal silica. Alternatively, dilute active silica (or silicic acid) species of about 300 mol wt are formed by ion-exchange removal of sodium ions from sodium silicate. This active silica is gradually fed to a tank of silica micelles in water at 80-100°C. The fresh active silica reacts with the heel particles, causing gradual growth until the desired 20-50 nm size is achieved. The sol is then concentrated by evaporation to the desired level and small amounts of sodium hydroxide are added to achieve maximum stability.

Precipitated silicas are prepared by mixing sodium silicate and mineral acid in a solution containing a calcium salt, which limits silica polymerization and causes precipitation of very fine silica particles; the silica particles are washed free of electrolytes and dried (Maher, 1969).

The preferred commercial method of preparing fumed silica involves reacting silicon tetrachloride with hydrogen and oxygen in a flame to form a very fine silica plus hydrochloric acid (Maher, 1969).

Vitums and coworkers (1977) summarized the results obtained in a NIOSH study in which rats received intratracheal instillations of amorphous silica dust derived from vaporized crystalline silica (quartz). Their findings confirmed the earlier work of Policard et al. (1954) by demonstrating the production of granulomatous inflammation and fibrosis of the lungs. The dust samples reportedly induced pulmonary lesions that were similar to those induced by a quartz reference sample.

b) Subchronic Effects

The toxicity of Degussa dust, an amorphous silica powder, was investigated by Schepers et al. (1957) in a series of inhalation and injection studies. Rats were exposed to an average concentration of 1.5 mg per cubic foot for 8 hours each weekday for one year or for six months with six months of monitoring their recovery. Of the 65 rats used, 8 had died after 3-5 months and 17 after 6 months; all deaths were considered pulmonary in origin. Seven of the 20 rats removed after 6 months of exposure died; 3 died from pulmonary causes and 4 succumbed to rete cell sarcomatosis of the lymphoid tissue. Tissue analysis revealed a direct correlation between length of exposure and mortality, accumulation of SiO_2 in the lungs, and severity of the tissue reaction. The main lethal factor was considered to be pulmonary insufficiency due to emphysema in combination with the obstruction of the pulmonary vasculature by macrophage nodules. Although 74 percent of the animals died during exposure, almost complete recovery occurred in 65 percent of the animals removed from the dust after six months. The silica was removed from the lung tissue and cellular nodules; perivascular infiltrations and emphysema were almost completely resolved.

c) Chronic Effects

i. Carcinogenicity

No data were encountered.

ii. Mutagenicity

No data were encountered.

iii. Teratogenicity

No data were encountered.

iv. Other Effects

Amorphous silica in the form of diatomaceous earth may contain varying amounts of crystalline silica, depending upon source and subsequent treatment. Diatomaceous earth has been linked to pulmonary fibrogenic effects in experimental animals and humans (Hamilton and Hardy, 1974; Hunter, 1975; ACGIH, 1974). Since crystalline silica impurities may account for the biological effects attributed to diatomaceous earth, it may not be appropriate to assume that truly amorphous silica will produce the same effects.

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Tebbens and coworkers (1957) exposed guinea pigs to atmospheres containing an average daily dust level of diatomaceous earth of 70 gm dispersed in a room of 4-1/2 by 7 by 13 feet for a period of 1-50 weeks. The dust dispersed into the room was 190 gm per day for the first 9 weeks but, this dust level was lowered after 13 of 32 animals died from pneumonia, pericarditis, or emphysema. The amount of silica (percent in dry tissue) increased from 0.1 to 3.0 percent, showing significant accumulation in the lungs over the 50 weeks. Those animals that survived the 37-50 weeks of exposure showed no external signs of pulmonary damage. Extensive gross and microscopic changes of the lungs were observed upon autopsy; these included thickening of the alveolar septa by infiltration of macrophages, accumulation of large numbers of multinuclear cells with dust particles, and epithelization of the alveoli. These changes were evident after as little as five weeks but were clouded by infectious processes. After 16 weeks of exposure, the peribronchial lymph nodes were invaded by dust-laden cells, and, by the end of 37 to 50 weeks, almost complete replacement of the lymph nodes had occurred. Lymph nodes at the lung roots increased in size up to 15 mm. Similar changes, but no gross enlargement, were seen in the spleen. The liver, kidneys, intestines, esophagus, and trachea were normal.

d) Human Effects

Historically, amorphous silica has been regarded to have only slight, if any, pulmonary fibrogenic activity; in comparison to crystalline silica, its threat to human health was considered minimal. Present threshold limit values for exposure to amorphous silica reflect the fact that it has been treated in much the same manner as nuisance dust.

A recent study, however, has presented evidence that may justify a reevaluation of the role of amorphous silica in producing pulmonary fibrosis in man. Vitums and coworkers (1977) examined 40 men employed in the production of silicon metal. Exposures to amorphous silica dust (<0.05-0.75 μ), which was a product of crystalline silica vapor, had occurred intermittently over an 11-18 year period. Roentgenograms of the chest revealed abnormalities in 11 of the 40 workers. Additional clinical studies performed on four of the men showed no evidence of restrictive impairment of lung function (total lung capacity and vital capacity); however, a minimal-to-moderate reduction in the diffusing capacity for carbon monoxide was noted in two workers, and a moderate-to-severe decrease in $FEV_{1.0}$ was noted in the same two patients. Lung biopsies from two of the workers showed fibrosis, emphysema, and numerous intraalveolar macrophages containing material identified by X-ray diffraction studies as amorphous silica dust (6.7% silica on a dry weight basis). Since all of these workers were also cigarette smokers, it was considered probable that smoking was the cause of the observed emphysema. An evaluation of biopsy material made at the Armed Forces Institute of Pathology, however, concluded that "the fibrohistiocytic nodules present in this case have some similarity to descriptions of experimental pneumoconiosis due to amorphous silica."

Diatomaceous earth has been used for centuries and medical studies of pneumoconiosis in workers have appeared in the literature since 1932 (Hamilton and Hardy, 1974). The summarized results revealed only chest X-ray changes with little or no incapacitation. The ailment is characterized, as are other chronic chest diseases, by dsypnea, cough, and weight loss. X-rays show linear, nodular, or coalescent lesions. Delays in injurious effects up to eleven years after exposure have been reported. The pathologic changes of diatomaceous earth pneumoconiosis are produced by the combination of diffuse alveolar thickening produced by amorphous silica and the effects of the fibrogenic action of the crystalline form.

The American Conference of Governmental Industrial Hygienists (ACGIH, 1974) relates several cases of the adverse effects of amorphous silica on humans. Several authors found no serious lung pathology in workers exposed to diatomaceous earth. Others found doubtful linear-nodular changes in workers exposed for five or more years. Mild silicosis was noted in diatomite workers and in one worker after a 12-year exposure to tripoli (diatomaceous earth). One death was reported in a worker employed in the quarrying, drying, and pulverizing of diatomaceous earth.

Of 108 men handling a diatomaceous deposit in California, 68.5 percent developed silicosis. Fifteen of the cases were moderately advanced and six were advanced (Hunter, 1975).

Medical observations of workers engaged in the production of aerosil (fumed silica) revealed diffuse pneumofibrosis in 4.3 percent of the men (Evtushenko et al., 1973). The concentration of aerosil was 266 mg/cu m in the production unit. A high frequency of atrophic inflammatory alterations in the upper respiratory tract and anterial dry rhinitis of the nasal and throat mucosa were observed.

9. Threshold Limit Values, OSHA Standards, NIOSH Recommended Standards

The current TLV adopted by the ACGIH for amorphous silica is 20 ppm/cu ft. A reduction in this value to 5 mg/cu m total dust (all sampled sizes) and 2 mg/cu m for respirable dust (<5 μ) has been proposed in the Notice of Intended Changes for 1977. In addition, a separate TLV -- 1.5 mg/cu m respirable dust -- has been proposed for natural diatomaceous earth. These are considered trial limits, to be adopted after two years in lieu of new evidence (ACGIH, 1974; 1977).

The OSHA Standard (TWA) is 80 mg/cu m/% SiO_2 (NIOSH, 1977).

10. Other Standards

No other standards were encountered.

11. Occupational Exposures

No data were available.

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