December 5, 2001

Nomination of

Abrasive Blasting Agents

For Chronic Inhalation Studies in Rats

National Institute for Occupational Safety and Health

Abrasive Blasting Agents

EXECUTIVE SUMMARY

Abrasive blasting involves forcefully projecting a stream of abrasive particles onto a surface, usually with compressed air or steam. Because silica sand is commonly used in this process, workers who perform abrasive blasting are often known as sandblasters. Tasks performed by sandblasters include removing irregularities from foundry castings; cleaning and removing paint from ship hulls, stone buildings, metal bridges, and other metal surfaces; and finishing tombstones, etching or frosting glass, and performing certain artistic endeavors.

When workers inhale the crystalline silica used in abrasive blasting, the lung tissue reacts by developing fibrotic nodules and scarring around the trapped silica particles (Silicosis and Silicate Disease Committee, 1988). This fibrotic condition of the lung is called silicosis. If the nodules grow too large, breathing becomes difficult and death may result. Silicosis victims are also at high risk of developing active tuberculosis (Myers et al., 1973; Sherson and Lander, 1990; Bailey et al., 1974).

In 1974, NIOSH recommended that silica sand (or other substances containing more than 1% free silica) be prohibited as abrasive blasting material and that less hazardous materials be used in blasting operations (NIOSH, 1974b). Alternatives to silica sand are recommended by NIOSH. The recommendations, however, are based on the presence or absence of known hazards such as respirable quartz, arsenic, beryllium, cadmium, lead, chromium, manganese, nickel, titanium silver, and vanadium (NIOSH 2001). Recommendations are not based on potential risks of exposure to specific blasting agents. This is due to a lack of comprehensive studies that have evaluated the health effects of chronic inhalation of most of the alternative blasting abrasives. Because potential alternatives have not been adequately evaluated in a comparative manner, it is difficult to make explicit recommendations of less hazardous alternatives to silica sand.

To begin the process of obtaining health effects data on silica sand substitutes, NIOSH has recently assessed acute pulmonary toxicity in rats following intratracheal instillation of nine commercially available substitutes (Hubbs et al., 2001; Porter et al., 2002). The nine substitutes tested were coal slag, specular hematite, steel grit, copper slag, nickel slag, crushed glass, staurolite, garnet, and olivine. Of these nine, NIOSH is nominating five (5) alternative blasting materials for testing in order to establish the potential for these agents to induce lung fibrosis as result of whole body inhalation exposure. Testing data is needed because of the high production volumes of the these agents, the large number of workers exposed, and the inadequacy of present toxicity data to determine safe exposure levels. Data from testing will provide a foundation on which NIOSH can base recommendations for use of alternatives to silica sand, and should provide dose-response toxicity data upon which to recommend occupational exposure limits.

Coal slag is selected for testing because it is a high use product that may have toxicity

comparable to silica sand and may not be a less hazardous alternative. Specular hematite and steel grit are two abrasive blasting materials that appear to have low pulmonary toxicity potentials and confirmation of this will provide a basis for recommending them as alternatives to silica sand. Garnet exposure induces pulmonary inflammation comparable to silica sand but induces a lower fibrotic response following acute exposure. Therefore, garnet may also be less hazardous than silica sand during chronic exposure, but the consequence of chronic inflammation is of concern. Crushed glass presently has limited market share and little is known regarding its pulmonary toxicity. A demonstration that it is a less hazardous substitute than silica sand could markedly increase its use since it is manufactured from a vast supply of recycled glass. Blasting sand will also need to be tested as a positive control material.

The other blasting agents are not being nominated for testing at this time because they would not likely be recommended by NIOSH as less hazardous alternatives than silica sand. For example, nickel and copper slags produce significant airborne quantities of arsenic, nickel and chromium (NTIS 1999a, 1999b), which are considered occupational carcinogens. Olivine has also been demonstrated to cause tumors in vivo (Wilson et al., 1988) and produced significant air borne quantities of nickel and chromium (NTIS 1999a and 1999b). In laboratory and field studies, staurolite generated respirable quartz concentrations above the NIOSH REL.

REQUESTED STUDIES

NIOSH is requesting that whole body inhalation studies be conducted in rat with coal slag, specular hematite, steel grit, garnet, crushed glass and blasting sand containing approximately 50 % crystalline silica. The source material for study should be the same as that used in the Hubbs et. al., 2001, and Porter et al., 2002 studies. The particle size distributions and characteristics need to be representative of the materials to which workers are exposed.

As the end point of concern is pulmonary fibrosis, limiting studies to male rats may be possible. There is no evidence that would indicate a sex difference in fibrotic response to inhaled particles. The mouse is not considered a good model to assess fibrotic response to particle exposure as some mice strains are not responsive to silica and may be unresponsive to other blasting agents.

NIOSH has previously demonstrated that an 18-week exposure to crystalline silica at 15 mg/m^3, 5 days/week, 6 hours/day (Porter et al., 2001) produces histological evidence of fibrosis. This is likely to occur later with less toxic materials. Study duration should be 26 or even 39 weeks. An option of continuing studies beyond 39 weeks should be considered for materials that do not elicit a fibrotic response.

For risk assessment purposes, selected doses should include concentrations that are relevant to human exposures. Rat lung burdens achieved should be comparable to those estimated in humans with a working lifetime exposure even if this results in overloading doses. To adequately assess effects, lung burden and lung associated lymph node burdens are needed as well as well as pulmonary responses.

CHEMICAL IDENTIFICATION

Silica Sand

There are several major producers of blast sand that each provide about 2 to 3% of the total sales of silica sand for abrasive blasting operations. Fairmount Minerals accounted for about 12.6% of the 1992 sales. Their Best Sand subsidiary provided about 8.3% of all blast sand consumption, and their Wedron Silica subsidiary provided about 4.2% of all blast sand consumption.

A California Silica Products blast sand bulk sample that NIOSH archived for future studies contains 52% quartz. California Silica Products is one of several producers that supplied 40,000 tons of blast sand that comprised about 2 to 3% of the silica sand blasting abrasive market.

Total 1992 consumption of silica sand was 1.75 million tons (Paumanok, 1992).

Composition of blast sand reported in

California Silica Products MSDS:		**Range of All Silica Sand Blasting Abrasives:**
Silicon Dioxide	100%	87% to 99.9%
Crystalline Silica	52%	37% to 99.9%
Aluminum oxide	-------	0.1% to 5%
Ferric Oxide	-------	0.06% to 0.15%
Calcium Oxide	-------	0.01% to 0.1%
Potassium Oxide	-------	0.03% to 3.5%
Magnesium Oxide	-------	0.002% to 0.007%
Titanium Oxide	-------	0.02% to 0.15%
Sodium Oxide	-------	0.01% to 0.44%

The percentage of each compound for California Silica Products silica sand falls within the range of percentages for all silica sand blasting abrasives.

Coal Slag

Coal slag is the most commonly consumed alternative blasting abrasive to silica sand (42% of alternative abrasives consumption) (Paumanok, 1992). It is the least expensive alternative abrasive in terms of purchase price per ton, which many end-users still emphasize.

Coal slags are waste products formed from the burning of coal in electric power plants. Slag is formed during the burning of a powdered coal that produces an ash with a relatively low melting point. During the burning process the molten ash falls to the bottom of the furnace into water where it frits. The resulting product is a hard black solid with less than 1% silica and is an excellent abrasive blasting material (Stettler et al., 1983). "Black Beauty¤" coal slag is a product of Reed Minerals (Highland, IN). Reed reports that in a typical analysis the most abundant compounds are silicon dioxide, aluminum oxide, ferric oxide, calcium oxide, potassium oxide, magnesium oxide, and titanium dioxide (Reed Minerals, 1992). The typical free-silica

crystalline content is stated to be less than 0.1% (Reed Minerals, 1992).

Total 1992 consumption was 442,000 tons (Paumanok, 1992).

Major producer: Reed Minerals: 268,000 tons (61% of total production)

Composition of material reported in:

Reed Minerals MSDS:		**Range of All Coal Slag Blasting Abrasives:**
Silicon Dioxide	47.2%	46% to 51%
Aluminum oxide	21.4%	17.2% to 25.5%
Ferric Oxide	19.2%	18% to 21%
Calcium Oxide	6.8%	4.3% to 7.2%
Potassium Oxide	1.6%	<1.7%
Magnesium Oxide	1.5%	1.0% to 1.5%
Titanium Oxide	1.0%	<1.3%
Crystalline Silica	<0.1%	<1.0%

The percentage of each compound for Reed Minerals Black Beauty coal slag falls within the range of percentages for all coal slag blasting abrasives.

Specular Hematite (Iron Oxide)

Specular hematite or iron oxide is relatively new to the abrasive blasting market.

Major producers of specular hematite are Barnes Environmental, Inc and Crystalgrit, Inc.

Recent annual consumption is estimated to be about 25,000 to 30,000 tons (Hansink, 2000).

Composition of material reported in:

Barnes Environmental BarShot MSDS:		**Range of All Specular Hematite Blasting Abrasives:**
Iron Oxide	98% to 99%	96.8% to 99%
Crystalline Silica	0.2% to 0.7%	<0.1% to 0.7%
Silicon Dioxide	0.5% to 1.2%	0.5% to 3.2%
Sodium Oxide	<0.03%	<0.03%
Calcium Oxide	<0.05%	<0.05% to 0.26%
Magnesium Oxide	<0.05%	<0.05%
Aluminum Oxide	0.2% to 0.3%	0.2% to 0.63%
Titanium Oxide	0.1% to 0.3%	0.08% to 0.3%
Potassium Oxide	<0.03%	<0.03%

The percentage of each compound for Barnes Environmental, Inc. BarShot.50 specular hematite falls within the range of percentages for all specular hematite blasting abrasives.

Steel Grit

Steel Grit is the second most commonly used alternative blasting abrasive in terms of tons consumed. Due to steel grit s excellent recycling properties, it is the abrasive that is used to prepare the most square feet of surfaces for blasting operations.

Major producers are Ervin Industries, Inc. with annual production of 95,000 tons (47% market share) and National Metal Abrasives with annual production of 36,000 tons (19% market share).

Total 1992 consumption was 195,700 tons (Paumanok, 1992).

Composition of material reported in:

Ervin Industries, Inc. MSDS		**Range of All Steel Grit Blasting Abrasives:**
Iron	>96%	>95%
Silicon Dioxide	<1.2%	0.3% to 1.5%
Carbon	<1.2%	0.7% to 1.3%
Manganese	<1.3%	0.5% to 1.3%
Chromium	<0.25%	<0.25%
Nickel	<0.2%	<0.2%
Crystalline Silica	None	None

The percentage of each compound for Ervin Industries, Inc. Amasteel steel grit falls within the range of percentages for all steel grit blasting abrasives.

Garnet

Garnet. is a naturally occurring mixture of almandite garnet (Fe3Al2(SiO4)3) with some magnesium and manganese substitution for iron. Other components include mica and less than 0.1% crystalline silica (Gorrill, 1996). Typical chemical analysis noted that the major components were silicon dioxide, ferric oxide, aluminum oxide, magnesium oxide, calcium oxide, and manganese oxide.

Total 1992 consumption was 24,000 tons (Paumanok, 1992).

Major producers are Emerald Creek Garnet Co. (Fernwood, ID) with10,000 tons or 42% of the garnet market and Barton Mines with 8,000 tons or 33% of the garnet market.

Composition of material reported in

Emerald Creek MSDS		**Range of All Garnet Blasting Abrasives:**
Silicon Dioxide	36.8%	36% to 40%
Ferric oxide	32.7%	30% to 33%

Aluminum Oxide	25.5%	20% to 26%
Magnesium Oxide	3.1%	1.0% to 6.0%
Calcium Oxide	1.2%	1.0% to 2.0%
Manganese Oxide	1.0%	1.0%
Titanium Oxide	____	<2.0%

The percentage of each compound for Emerald Creek garnet falls within the range of percentages for all garnet blasting abrasives.

Crushed Glass

Crushed glass is relatively new to the abrasive blasting market. For the U.S. shipbuilding and repair industry, a survey of dry abrasive blasting media projected 2,725 tons as the annual usage rate for crushed glass. No other consumption data were identified for crushed glass.

Annual consumption is unknown.

Major producers of crushed glass blasting abrasive are TriVitro Corporation, Universal Groun Cullet, Inc. and Strategic Materials, Inc. TriVitro markets Vitrogrit VG 3050.

Composition of material reported in **TriVitro Corp MSDS**		**Range of All Crushed Glass Blasting Abrasives:**
Silicon Dioxide	73%	72% to 81%
Sodium Oxide	14%	13% to 14%
Calcium Oxide	10%	8.6% to 10.0%
Magnesium Oxide	<1%	1.0% to 4.0%
Aluminum Oxide	<1%	0.2% to 1.0%
Sulfur Trioxide	<1%	<1%
Potassium Oxide	___	<0.4%
Iron Oxide	___	<0.4%

The percentage of each compound for TriVitro Corp Vitrogrit VG 3050 crushed glass falls within the range of percentages for all crushed glass blasting abrasives.

EXPOSURE INFORMATION

Number of Exposed Workers

NIOSH estimates that approximately 150,000 workers are employed as abrasive blasters (NIOSH provisional). The National Occupational Exposure Survey indicates that the construction industry employs the largest number of sandblasters, with the highest proportion in the special trades industries (NIOSH 1988b; 1998c; 1990b). Silica sand represents approximately 63% of all blasting abrasives consumed. Over-exposures to crystalline silica and

silicosis are still prevalent among abrasive blasting workers (NIOSH Alert 1992 and unpublished SENSOR data). For many abrasive blasting operations, several nearby workers are without any respiratory protection (pot tenders, blaster helpers, shipyard workers, painters, welders and laborors), so that the total number of workers exposed to abrasive blasting materials is likely a multiple of the estimated abrasive blasters.

Current Exposure Limits

The current OSHA permissible exposure limit (PEL) for respirable crystalline silica (quartz) is 100 $\mu g/m^3$ as an 8-hour time-weighted average (TWA) (29 CFR** 1910.1000). The NIOSH recommended exposure limit (REL) for respirable crystalline silica is 50 $\mu g/m^3$ as a TWA for up to 10 hours/day during a 40-hour workweek (NIOSH 1974b). This REL is intended to prevent silicosis. However, evidence indicates that crystalline silica is a potential occupational carcinogen (NIOSH 1988a; IARC 1997; DHHS 1991) and NIOSH is reviewing the data on carcinogenicity.

NIOSH, OSHA and ACGIH do not have specific REL, PELS or TLVs for any abrasive blasting alternatives to silica sand. Specular hematite is a specific form of iron oxide and the NIOSH REL and ACGIH TLV for iron oxide dust and fume (as Fe) is 5 mg/m^3 and the OSHA PEL is 10 mg/m^3. Steel grit is composed of greater than 95 % iron, therefore, exposures to steel grit would be limited by exposure limits for iron oxide dust and fume. Most of the abrasives have several metals (arsenic, beryllium, cadmium, etc. see Table 1) and exposure to these abrasives could be limited by exposure limits for these constituents.

Respiratory Protection Practices

Acute silicosis is less common today than it was in the 1930s because engineering controls are available to reduce exposure to respirable crystalline silica and because the use of alternative abrasives is increasing. However, data indicate that many abrasive blasters continue to work without adequate respiratory protection (NIOSH, 1974a).

Ventilation controls for reducing crystalline silica exposures are not used in most industries (NIOSH, 1990b). Samimi et al. (1974) found that even in short-term sandblasting operations (less than 2 1/2 hours of blasting during an 8-hour workday), the average concentration of dust was 764 micrograms per cubic meter ($\mu g/m^3$), with an average crystalline silica content of 25.5%. This average crystalline silica concentration was twice the 1974 standard of the Occupational Safety and Health Administration (OSHA).

In a 1974 study of respiratory protection practices during abrasive blasting (NIOSH 1974a), the protection factors for supplied-air respirators with helmets ranged from 1.9 to 3,750. This wide range was attributed to the varied conditions of the equipment rather than to the superiority of any brand. Maintenance was universally poor or nonexistent, and the persons responsible for selecting respiratory protection for abrasive blasting were inadequately informed about the proper use and maintenance of such equipment. The higher protection factors were associated with high rates of helmet air flow, but these high flow rates increased noise levels as a result of

air turbulence. The study also indicated that the air inlets were too noisy and that the blasters' helmets tended to fall from the wearers' shoulders when they stooped.

Restriction of Silica in Abrasive Blasting

Because of the high risk for silicosis in sandblasters and the difficulty in controlling exposures, the use of crystalline silica for blast cleaning operations was restricted in Great Britain in 1950 (Factories Act 1949) and in other European countries in 1966 (ILO, 1972). In 1974, NIOSH recommended that silica sand (or other substances containing more than 1% crystalline silica) be prohibited as abrasive blasting material and that less hazardous materials be used in blasting operations (NIOSH 1974b) due to the silicosis hazard and difficulty with controlling crystalline silica exposure associated with sandblasting. Alternatives to silica sand are recommended by NIOSH. However, few comprehensive studies have been conducted to evaluate the health effects of most of the alternative blasting abrasives.

Use of Alternative Abrasives

The risk of silicosis is high in workers exposed to abrasive blasting with silica, and the hazard is difficult to control. NIOSH has therefore recommended since 1974 that silica sand (or other substances containing more than 1% crystalline silica) be prohibited as abrasive blasting material (NIOSH 1974b, NIOSH 1990a). A variety of materials (iron oxide, slags, steel grit, olivine, staurolite, garnet, and crushed glass) are available as alternative blasting media (NIOSH 1974c; Mackay et al. 1980; Stettler et al. 1988). However, no comprehensive inhalation studies have been conducted to evaluate the health effects, especially the pulmonary toxicity, of these substitute materials. Engineering controls and personal protective equipment should be used with any of the alternative abrasives to reduce hazards generated by blasting abrasives and substrates.

In addition to the health hazards of abrasive blasting materials, the finely fractured particles of material being removed (lead paint, for example) may also create health risks for workers (NIOSH 1991a).

HEALTH EFFECTS

A 1980 Department of Labor report to Congress on occupational disease estimated that 1 million workers were exposed to crystalline silica. Regardless of the use of respiratory protection sporadic clusters of silicosis continue to occur in the sand blasting industry (Beck et al., 1982; Kavet et al., 1978). Over exposure to crystalline silica and silicosis are still prevalent among abrasive blasting workers (NIOSH, Alert, 1992, and unpublished SENSOR data). Although blasting with silica is restricted in several other industrialized nations, its use in the United States is widely practiced despite the recommendation of banning by NIOSH in 1974.

Silica

The unique biological reactivity of crystalline silica has been attributed, in part, to surface properties of silica. Hydrogen bonding between silica particles and biological membranes has been implicated in cell membrane injury and lung disease.

In vivo pulmonary reactions to crystalline silica resulting in the development of pulmonary fibrosis and carcinogenesis are well documented in experimental animal models. Although the development of pulmonary fibrosis in rats, mice and monkeys is well documented, the classical silicotic nodular lesion seen in humans is not identified in animals exposed to crystalline silica. Similarly carcinogenesis by crystalline silica was observed in rats and not found in mice and hamsters. Therefore, the biological response of certain species or strains of animals not responding to a pathogen is probably not negative evidence of the pathogenicity or carcinogenicity of an agent.

Human Pathologic Reactions

Silicosis

When workers inhale the crystalline silica used in abrasive blasting, the lung tissue reacts by developing either acute or chronic silicosis depending on the exposure dose. Acute silicosis is characterized by alveolar lipoproteinosis and a decrease in gas exchange. Chronic silicosis is characterized by fibrotic nodules and scarring around the trapped silica particles [Silicosis and Silicate Disease Committee 1988]. If the nodules grow too large, breathing becomes difficult and death may result. Silicosis victims are also at high risk of developing active tuberculosis (Myers et al., 1973; Sherson and Lander 1990; Bailey et al., 1974). The silica sand used in abrasive blasting typically fractures into fine particles and becomes airborne. Inhalation of freshly fractured silica appears to produce a more severe lung reaction than aged silica that is not freshly fractured (Vallyathan et al., 1988; Vallyathan et al., 1995; Castranova et al., 1996). This factor may contribute to the development of acute and accelerated forms of silicosis among sandblasters.

Simple and Complicated Silicosis

Chronic exposure to low concentrations of crystalline silica promote the formation of fibrotic nodules in the lung parenchyma with a typical morphologic appearance of discrete, rounded, whorled, hyalinized lesions (Axelson and Sjoberg, 1979). These fibrotic nodules are usually less than a centimeter in diameter and are sharply separated from the surrounding lung tissue. This simple silicosis rarely will result in any clinically apparent disease. Simple silicosis with continued exposure and time will lead to increased nodular density and decremental changes in pulmonary function. Progressive massive fibrosis is a common feature of such complicated silicosis resulting in compromised lung function.

Acute and Accelerated Silicosis

Acute silicosis is associated with alveolar lipoproteinosis caused by highly reactive fractured silica/ and/or intense silica dust exposure with a high mortality rate. Acute silicosis is associated with sand blasting, silica flour mill operations, rock drilling, tunneling and other occupations where crystalline silica is fractured (Saffiotti et al., 1968, Nettesheim et al., 1975). Clinically it

is characterized by severe shortness of breath and hypoxemia. Secondary infection is a common terminal complication.

Accelerated silicosis is also associated with lipoprotenosis and is often associated with sand blasting and other similar operations described earlier (Kavet et al., 1978; Steinhoff et al., 1991). The condition refers to the rate of progression to classic nodular silicosis at an accelerated rate of five to ten years. The disease progresses to nodular state rapidly with no abnormalities other than increasing breathlessness. The patient often dies within 7 to12 years with respiratory failure.

Bronchogenic Carcinoma

The International Agency for Research on Cancer (IARC) comprehensively reviewed recently the human and animal experimental studies and concluded that there is sufficient evidence for the carcinogenicity of inhaled crystalline silica in the forms of quartz or crystobalite from occupational sources (IARC, 1987a, 1997). The agency classified crystalline silica under a 1A, i.e., a carcinogen to humans (IARC, 1997). However, the human epidemiologic studies are debatable, mainly because of the lack of control studies well matched for smoking and other exposure variables to known carcinogens. A supporting quantitative relationship between crystalline silica exposure and bronchogenic cancer was also demonstrated in workers without radon exposure such as Vermont granite workers, German slate workers, North Carolina Dusty Trade Workers and ceramic and pottery workers in Sweden and Italy. Although information is not available for never-smokers with silicosis, from these studies it can be concluded that silicosis predisposes one to an increased risk for bronchogenic carcinoma. The National Toxicology Program s 9th Report on Carcinogens lists respirable crystalline silica as Known Human Carcinogen (NTP, 2000).

Coal Slag

The acute in vivo pulmonary reactions to coal slag have also been assessed in experimental animals (Hubbs et al., 2001). One month after intratracheal instillation of 10 mg/rat coal slag or silica sand there was significant PMN infiltration, potentiation of particle-stimulated oxidant generation from alveolar macrophages (chemiluminescence), and lung cell damage (elevated LDH in lavage fluid). These acute pulmonary reactions tended to be greater for coal slag than blasting sand. In contrast, Stettler et al. (1995) found that coal slag was considerably less fibrogenic than crystalline silica following a single intratracheal instillation.

The chronic response of rats to exposure to coal slag has been reported by Mackay et al. (1980). Ten months after intratracheal instillation of 20 mg/rat coal slag significant pulmonary fibrosis was observed. Compared to a fibrotic score of 10 for crystalline silica, coal slag rats had a fibrotic score of 4.

The trace metal content of various coal slag samples has been reported (Stettler et al., 1982). Compared to copper and nickel slags, coal slag tended to have the lowest levels of contamination with metals suspected to be carcinogens, such as beryllium, chromium, nickel and arsenic (Table 1 shows the elemental concentrations of the blasting abrasive substitutes used by Hubbs et al.,

2001 and Porter et al., 2002). However, coal slag had significantly higher levels of beryllium than any blasting abrasive when used under a NIOSH contract conducted by KTA-Tator Inc. (NTIS 1999a, 1999b).

The available data suggest that coal slag exhibits cytotoxicity and acute inflammatory potential similar to abrasive sand samples. Evidence indicates the coal slag can cause significant pulmonary fibrosis although to a lesser extent than crystalline silica. Additional studies are needed to define the fibrotic potential of coal slag relative to silica sand.

Iron Oxide (Specular Hematite)

Iron oxide appears to exhibit low toxicity in vivo compared to crystalline silica. For example, intratracheal instillation of 10 mg/hamster crystalline silica resulted in recruitment of PMN and elevation of RBC, LDH and albumin in lavage fluid which was significant 1 day post-exposure (Beck et al., 1982). However, Beck and colleagues found that iron oxide caused much less inflammation and damage than silica, and that these pulmonary reactions were quickly resolved. This agrees with results of a NIOSH study (Hubbs et al., 2001) with rats 30 days after instillation of 10 mg/rat iron oxide where bronchoalveolar lavage markers of inflammation and damage were not different from control levels and were significantly lower than values after silica sand instillation. Mild, transient inflammation has also been reported after short term (3 hr) inhalation of high levels (274 mg/m^3) of iron oxide (Kavet et al., 1978). Iron oxide, in contrast to silica, failed to induce significant secretion of inflammatory cytokines (TNF-α and IL-1) by rat lungs after intratracheal exposure (Antonini et al., 1996). The lack of sustained inflammation and lung damage after exposure of animal models to iron oxide is consistent with absence of pulmonary fibrosis reported in rat after intratracheal instillation of rouge (Harding, 1945; Hubbs et al., 2001).

Animal investigations of the carcinogenicity of iron oxide have been clearly negative. Intratracheal instillation (380 mg iron oxide by multiple injections) resulted in no lung tumors in a 2 1/2 year study with rats (Steinhoff et al., 1991). Similar results were reported by Saffiotti et al. (1968) after 15 weekly instillations of 3 mg iron oxide each. Inhalation of iron oxide (40 mg/m^3 for 2 years) also gave negative results in hamsters (Nettesheim et al., 1975). Therefore the International Agency for Research on Cancer views evidence for the lack of carcinogenicity of iron oxide in animals as convincing (IARC, 1987b).

The relatively low fibrogenicity of iron oxide reported for animal models is generally consistent with epidemiology studies of exposed workers. For example, Teculescu and Albu (1973) studied 14 workers exposed for an average of 10 years to pure iron oxide dust and found no pulmonary function changes that would be consistent with pulmonary fibrosis. Lay et al., (1999) reported that the presence of inflamation following the intrapulmonary installation of iron oxide in human subjects which resolved rapidly after the exposure ended. Iron oxide particulates are also administered intravenously in humans as radiologic contrast agents (Semelka and Helmberger, 2001).

Early epidemiology studies reported an elevated incidence of lung cancer in workers exposed to

iron oxide. However, most of these studies are plagued with confounding exposures (Stokinger, 1984). For example, Boyd et al. (1970) reported a 70% higher than normal lung cancer mortality in underground iron-ore miners. However, surface iron mine workers did not exhibit higher lung cancer mortality and underground miners were exposed to high radon levels (100 p Ci/L). In addition, a case control study of factory workers exposed to high amounts of iron oxide dust found no excess cancer in the respiratory system or other sites (Axelson and Sjoberg, 1979). Therefore, the human data for iron oxide-induced cancer is viewed as negative (Stokinger, 1984).

In summary, in vivo animal data for fibrosis and lung cancer for iron oxide are consistently negative. When confounding exposures are considered, human data are consistent with this conclusion.

Steel grit

The pulmonary effects of respirable steel grit have not been extensively studied. Although it is predominately comprised of iron, it cannot be considered equivalent to iron oxide since its biological persistence may be very different from iron oxide. Steel grit, in contrast to blasting sand, did not cause significant inflammation, lung damage, or fibrosis 30 days following tracheal installation (Porter et al., 2001).

In environmentally-controlled laboratory and field studies, steel grit generates significantly lower concentrations of respirable dust than blast sand. However, it generated significantly higher concentrations of possible carcinogens such as nickel and arsenic than most of the blasting abrasives. No studies evaluating the carcinogenicity of inhaled steel grit are available.

Garnet

Garnet produced pulmonary inflammation and damage in vivo at a potency similar to that for silica sand but induces reduced fibrotic type response following acute exposure (Hubbs et al., 2001). In addition, freshly blasted garnet produced more hydroxyl radicals than silica sand in vitro as assessed by ESR spectroscopy (NIOSH unpublished data). No studies evaluating the carcinogenicity of inhaled garnet are available.

Crushed glass

Insufficient data are available for assessment of the pulmonary fibrogenic or carcinogenic potential of inhaled crushed glass. Intratracheal instillation exposure to crushed glass causes inflammation, cell damage to a degree similar to blast sand (Porter et al., 2002). Crushed glass also caused significant pulmonary fibrosis in this study. Preliminary data suggest the production of hydroxyl radical in vitro (NIOSH, unpublished data). No studies evaluating the carcinogenicity of inhaled crushed glass are available.

General Information

Under a NIOSH contract, KTA-Tator Inc. collected consumption data and technical and economic data for ten types of blasting abrasives (including coal slag, specular hematite, and steel grit) in an environmentally-controlled laboratory and partially controlled field setting (NTIS 1999a, 1999b) available at www.cdc.gov/niosh/abrpt946.html). All alternative abrasives were

technically and economically competitive with silica sand.

KTA-Tator also reported bulk and airborne concentrations of thirty different potentially toxic components. For both the laboratory and field studies, steel grit generated significantly less respirable dust than blast silica. Coal slag and specular hematite generated respirable dust concentrations that were not significantly different than blast sand. Coal slag generated airborne beryllium concentrations that were significantly greater than all of the other blasting abrasives. Steel grit generated airborne arsenic, chromium, manganese, and nickel concentrations that were significantly greater than most of the other blasting abrasives. NIOSH HHEs and an OSHA inspection found similar bulk and airborne arsenic, chromium, manganese, and nickel concentrations in blasting operations which used steel grit. For twelve potentially toxic components that were emphasized in analysis of the laboratory and field studies by KTA-Tator, specular hematite and crushed glass generated concentrations that were below the limit of detection, or lower than all of the other blasting abrasives.

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Table 1. Elemental Analysis of Abrasive Blasting Substitute Agents

Element	Sand 1	Sand 2	Coal Slag	Specular Hematite	Steel Grit	Garnet	Crushed Glass	Olivine	Copper Slag	Nickel Slag	Stauro -lite
Aluminum	2200	1200	2600	270	300	5700	120	330	41000	1100	220
Arsenic					30				23		
Barium	11		13	7		1			0.91		
Beryllium		0.03						0.01		0.01	
Cadmium							0.02			.	
Calcium	4900	2800	650	210			110	44	170000	1200	38
Chromium	4	2.7			1200	630		51	810	550	3
Cobalt	2	0.73		7	39	5		74	32	14	
Copper	4	4		4	870	1	1	1	1500	6	
Iron	5300	2900	4200	230000	560000	15000	170	42000	140000	2600	300
Lead		0.47			7		0.7	0.3	1.5	0.7	5
Lithium	2	1.8					0.1	1.7	27	0.1	
Magnesium	3000	1900	100	310	21		35	220000	24000	35	5
Mangenese	100	88	6	190	6400	700	3	470	3400	3	13
Molybdenum					300						
Nickel					700			1700	22		
Phosphorus	100	85			130	93	8		1200	8	40

Platinum				280	92			20			
% Quartz	49	55				1					
Selenium								530	1500		
Silver									1		
Sodium	99	22	80	58		25	230		880	230	90
Tellurium					28						
Thallium									80		
Titanium	230	82	88	66	5.3	94	6	8	1200	6	520
Vanadium	9	4	5	20	76	2		0.5	96		4
Yttrium	3	3.2			0.43	31			11		4
Zinc	8	5.5		19	12	3	70	17	91	70	2
Zirconium	5	3		13		2	4		30	4	8

Source of data is Hubbs et al., 2001 and Porter et al., 2001.
Concentrations are ug/g bulk material except quartz which is expressed as percentage.
Blank cell indicates element below the limit of detection.

Table 2. Occupational Exposure Limits (OELs) for Blasting Abrasive Components

Components	NIOSH RELs	OSHA PELs	ACGIH TLVs
Aluminum Oxide (Al_2O_3)	None Established	15 mg/m^3 total 5 mg/m^3 resp.	10 mg/m^3, A4
Arsenic (As) metal & inorganic	**CARCINOGEN** 0.002 mg/m^3 [15-min]	0.01 mg/m^3	0.01 mg/m^3, A1
Barium (Ba) soluble cmpds.	0.5 mg/m^3	0.5 mg/m^3	0.5 mg/m^3, A4
Beryllium (Be) metal & cmpds.	**CARCINOGEN** 0.00005 mg/m^3 [ceiling]	0.002 mg/m^3 0.00005 mg/m^3 [ceiling]	0.002 mg/m^3, 0.01 mg/m^3 STEL A1
Cadmium (Cd)	**CARCINOGEN**	0.005 mg/m^3	0.01 mg/m^3, A2 respirable
Calcium Oxide (CaO)	2 mg/m^3	5 mg/m^3	2 mg/m^3
Carbon (C)	3.5 mg/m^3 **CARCINOGEN**	3.5 mg/m^3	3.5 mg/m^3, A4
Chromium (Cr) metal	0.5 mg/m^3	1 mg/m^3	0.5 mg/m^{3,} A4
Chromic Acid & Chromates	**CARCINOGEN** 0.001 mg/m^3	0.1 mg/m^3	0.05 mg/m^3 soluble, A1 0.01 mg/m^3 insoluble, A1
Cobalt (Co) metal, dust & fume	0.05 mg/m^3	0.1 mg/m^3	0.02 mg/m^3, A3
Copper (Cu) dusts & mists	1 mg/m^3	1 mg/m^3	1 mg/m^3
Iron Oxide (Fe_2O_3) dust & fume	5 mg/m^3	10 mg/m^3	5 mg/m^3, A4
Lead (Pb)	0.05 mg/m^3	0.05 mg/m^3	0.05 mg/m^3, A3
Magnesium Oxide (MgO)	None Established	15 mg/m^3	10 mg/m^3
Manganese (Mn) cmpds.	1 mg/m^3 3 mg/m^3-15 minute	5 mg/m^3 - ceiling	0.2 mg/m^3

Table 2 (continued)
Occupational Exposure Limits (OELs) for Blasting Abrasive Components

Components	NIOSH RELs	OSHA PELs	ACGIH TLVs
Molybdenum (Mo)	None Established	15 mg/m^3	5 mg/m^3 soluble, respirable, A3. 10 mg/m^3 inhalable and 3 mg/m^3 respirable insoluble & metal
Nickel (Ni) metal & other cmpds.	CARCINOGEN 0.015 mg/m^3	1 mg/m^3	inhalable: 0.2 mg/m^3 insol., A1 0.1 mg/m^3 soluble, A4 1.5 mg/m^3 metal, A5
Phosphorous (P)	0.1 mg/m^3	0.1 mg/m^3	0.02 ppm
Platinum (Pt)	1 mg/m^3	None Established	1 mg/m^3 as metal 0.002 mg/m^3 soluble salts
Selenium (Se) & cmpds.	0.2 mg/m^3	0.2 mg/m^3	0.2 mg/m^3
Crystalline Silica (SiO_2) as respirable quartz dust	CARCINOGEN 0.05 mg/m^3	respirable dust: 250 mppcf/(%SiO2 +5) or 10 mg/m3/(%SiO2 + 2) total dust: 30 mg/m3/(%SiO2 + 2)	0.05 mg/m^3, A2
Silver (Ag)	0.01 mg/m^3	0.01 mg/m^3	0.01 mg/m^3 soluble 0.1 mg/m^3 metal
Titanium Dioxide (TiO_2)	CARCINOGEN	15 mg/m^3	10 mg/m^3, A4
Vanadium (V_2O_5) dust & fume	0.05 mg/m^3 [15-minute]	0.5 mg/m^3 respirable dust 0.1 mg/m^3 fume	0.05 mg/m^3, respirable A4
Yttrium (Yt) & cmpds.	1 mg/m^3	1 mg/m^3	1 mg/m^3
Zinc oxide dust	5 mg/m^3 15 mg/m^3 ceiling	15 mg/m^3 total dust 5 mg/m^3 resp. dust	10 mg/m^3
Zirconium (Zr) & cmpds.	5 mg/m^3 10 mg/m^3 - STEL	5 mg/m^3	5 mg/m^3, A4 10 mg/m^3 - STEL