Silicosis and industrial bronchitis by exposure to silica powders and cement

María Martha Méndez-Vargas,[a] Fabiola Berenice Báez-Revueltas,[b]
Pablo López-Rojas,[c] José HoracioTovalín-Ahumada,[a]
José Othón Zamudio-Lara,[d] Irma Araceli Marín-Cotoñieto,[e] Francisco Villeda[f]

Objective: to identify the association between the exposure of workers to inorganic dust in a quarry and a cement factory and pulmonary diseases.
Methods: a transversal study on data from 32 quarry workers (QWs) and 57 cement production line workers (CFWs) to inorganic-dust exposure was performed. Pulmonary function tests and chest X ray were done in both groups.
Results: there were differences between QWs and the CFWs; QWs were younger, shorter and thinner. The amount of sick individuals might be considered significantly different, showing a smaller proportion on the cement production line. The quarry workers have been on the company for a shorter period of time and, in the end, they present more serious pulmonary problems with an average of 3 years working.
Conclusions: the amount of free-silica that is managed on the quarry affects the workers in some way, even when literature states that the evolution of industrial bronchitis and silicosis have a period development of approximately 10 years.

Key words
silicosis
pneumoconiosis
occupational exposure
occupational diseases

Silicosis, pneumoconiosis, industrial bronchitis, tuberculosis and some types of cancer are among the diseases that have been related to the exposure to inorganic dust. Silicosis is a disease that is known since Herodotus' times, where mines' workers were exposed to silica dust and silicates during the extraction activities, and in some quarries areas where silica was employed as a raw material. Currently studies show that mines and quarries workers presented a high dead rate due to this disease.[1] In 1870 Visconti gave to Silicosis its name. At the present, a physiopathology pattern had been identified[2] and today is well known that it is the most frequent pneumoconiosis and cause a chronic fibrotic disease produced by the exposure to free silica.[3]

The silica to be harmful has to be in the form of silicon dioxide or free-silica, which is generally found as quartz. The particles size has to be from 2 to 6 microns, so they can get into the human alveolus. When the particles are either bigger or smaller, they are less harmful for the exposed individual.[4]

When a person is exposed to silica particles trigger a whole mechanism in the body: When breathing, the particles get into the terminal part of the respiratory tree and then into the respiratory bronchioles.[5] Then, the macrophages start the phagocytes process on the silica particles trough the pinocytic phenomenon in an effort to destroy them via the proteases and elastase within the lysosome in the macrophage´s cytoplasm. Generally the silica particles cannot get destroyed by this process, instead, the macrophages die due to the lysosome enzymes effect, and a production of new macrophages start over and over because they get destroyed every time, causing with this an inflammation and increase in lymphocyte *T* which produce lymphokines that inhibit the migration of the macrophages and increase the destructiveness of the lysosome enzymes. This whole process causes the neutrophils to produce a chemoattracans substance to fibroblasts and creates collagen fibers that surround the silica particles. The silicosis nodules are the characteristically histopathology elements of the disease.[6]

There is an increase of the cellular response produced by the monocytes due to the interleukin 1 produced by the macrophages. The alveolar fibrosis starts giving a big amount of interstitial macrophages that get into the alveoli. This sets a constant response at the alveolar tissue that keeps going even when the subject is not exposed to the silica powders anymore.

Silicosis, like all pneumoconiosis, is asymptomatic in early stage. The disease gives signals of its presence in two different situations: when is already very advanced or when the subject suffers from other pathologies, such as, bronchitis, tuberculosis, chronic obstructive pulmonary disease, emphysema and spontaneous pneumothorax, among others.

Silicosis y bronquitis industrial por exposición a polvos de sílice y cemento

Resumen

Objetivo: asociar la exposición a polvos inorgánicos en trabajadores de una cantera y una cementera y la generación de alteraciones pulmonares.
Métodos: estudio transversal con 32 trabajadores expuestos a polvos inorgánicos en una cantera y 57 en una cementera. Se les realizaron pruebas de función respiratoria y telerradiografías de tórax. Se midieron polvos totales.
Resultados: existieron diferencias demográficas significativas entre las dos líneas de producción: los empleados de la cantera fueron más jóvenes, menos altos y ligeramente más delgados. La proporción de enfermos fue significativamente diferente entre las dos áreas: en la cementera fue menor que en la cantera. Aun cuando los trabajadores de la cantera tuvieron una antigüedad menor, presentaron problemas pulmonares más graves.
Conclusiones: la cantidad de sílice libre que se maneja en la cantera afecta gravemente la salud de los trabajadores, ya que con un promedio de tres años de antigüedad laboral presentaban silicosis. Por ello, deben establecerse mejores programas de higiene industrial para el control de los polvos

Palabras clave
silicosis
neumoconiosis
exposición profesional
enfermedades profesionales

There are three different clinical pictures caused by inhaling silica powders:

- *Chronic silicosis:* that is the most common type. Requires 10 or more years of exposure to powders containing up to 30 % of free silica. It develops slowly and if the exposure is not so intense, its effects are not so harmful.[5]
- *Acute silicosis:* It is usually seen on subjects that work with sand blasts. It is shown after 2 to 3 years of exposure to silica powders with a 90 to 100 % of free-silica. This clinical picture of silicosis is also known as silicoproteinosis. It is a very malign pneumoconiosis that rapidly produces respiratory insufficiency, abnormalities in the lungs and emphysema. It is characterized by multiple small immature nodules.[7]
- *Accelerated silicosis:* This type of silicosis is worse clinical presentation. It is produced when the inhaled powder contains 40 to 84 % of free-silica. This silicosis as the acute one is dependent on the magnitude of the exposure to silica. An occupational environment that has this kind of exposure occurs during abrasion operations in mines, in these operations, it is believed that silica is more toxic to the alveolar macrophages, given the increase of REDOX that is present on the surface of the powder, which produces highly reactive radicals OH- such as carbon and, in some cases, nitrogen.[6-8]

The accurate diagnosis of silicosis has a protocol:[9-12]

- Information about the exposure level of the individual to silica powders. This is obtained via the work history of the employee and it is confirmed by a sensory visit to the work place and quantified by air sampling in the area.
- Alterations on the thorax X-ray agree with the ones listed on the International Classification of Radiographs of pneumoconiosis for the ILO 2000.[13]
- Spirometry, the pulmonary function test is used to identify functional alterations defined by the American Thoracic Society (ATS).[14-16] We can establish three different patterns: obstructive, restrictive and mixed. When the individual presents a parenchymal problem a restrictive pattern is observed, like the one in silicosis. When the patient shows airways problems, such as bronchitis or asthma the pattern that is seen is the obstructive one. When the pathology has a long last evolution of the pathology, usually presents a mixed pattern.

The prevalence and gravity of the silicosis depends both, on the intensity to the exposure to free-silica powders and on the individual's susceptibility.[17]

Bronchitis is another disease known to be related to the exposure to cement powders,[18] as well as to smoke, sulfur dioxide,[19,20] exposure to chemicals agents[21] and pollution. However it is a common disease, there is not enough research done on acute industrial bronchitis caused by the exposure to inorganic dust.

Table I Results of the environmental study on the concentration of inorganic powders

Location	Area	Concentration (mg/m^3)
Quarry	Drill 1	7.98
	Drill 2	89.67
	Quarry tunnel 1	3.94
	Control house	0
	Shreder	15.19
	Cellar 1	17.08
	Cellar 2	65.9
	Gammametric	18.17
Production cellar	Packaging 1	3.4
	Packaging 2	30.56
	Packaging 3	25.45
Maintenance	Sand blast	46.89

Table II T-test for quarry workers *vs.* cement production line workwers

Variable	Averages		*p*-value for $H_0:\mu_1=\mu_2$
	Quarry workers	Cement workers	
Age	31.34	38.72	0.001
Weight	74.06	79.55	0.099
Height	162.18	167.86	0.001
Body mass index	28.24	27.8	0.714
Old	2.94	8.16	0.001
Sick	0.59	0.4	0.086
Diagnosis	2.47	1.81	0.020

If *p*-value ≤ 0.05 (significance level), then we reject the null hypothesis

In order to have a good preventive program in work places with silica contaminated environments is convenient to keep a preventive program, where the workers that are exposed are checked periodically.[15,16]

Silica is the most abundant element in nature.[9] The outermost layer of the earth's surface is made of 98.6 % silica and silicates. The aim exposure to silica dust occurs in industries that perform extraction (mines and quarries) and transformation industries (companies that produce ceramics, refractory products, glass, paints, enamels, paper and cement).[22]

The International Labor Organization in 1971 in Bucharest, Romania, defined the pneumoconiosis as "accumulation of dust in the lungs tissue and the physiological reactions in the presence of this dust", where powder is defined as "inanimate solid aerosol particles".[18] Pneumoconiosis are considered real working diseases given that they are originated in the presence of contaminants that are usually found on specific work places and they are rarely found outside of them.

When cement powders are inhaled in small amounts they cause, as a first side-effect, irritation of the respiratory tree and after some time of exposure, the particles that are deposited in the terminal portion at the level of the respiratory bronchioles can produce pneumoconiosis. The exposure time required to develop chronic silicosis is 10 years. The other two types of silicosis require a different time of exposure that can be reached by the use of sand blasts. Although bronchitis is a complication of pneumoconiosis and especially of silicosis. It is known that the development of industrial bronchitis is shorter than the development of pneumoconiosis.

The raw materials needed for the production of cement are: limestone, sand, clay, shale, magna and oxides of shale. These have to be mixed with silica, aluminum and iron as sand, clay, bauxite, shale, iron and blast furnace slag. Plaster is added at the end of the process.[23] Portland cement is a material that is compound by calcium oxides (60-70 %), silica (5 %) in the form of free-silica, aluminum trioxide (4-7 %), ferric oxide (2-6 %) and magnesium oxide (less than 5 %). The exposure to silica and silicates on a quarry occur specially on the warehouse, the crushing and the packing areas.

Some cautionary measurements that can be taken reduce the exposure level in work places is the use of fans, extractors and respirators. The only problem with this measures is that most of the activities on a quarry are done outdoors, therefore is really hard to follow these cautionary measurements. Whole employees should wear personal protection, such as breathing filter equipment.

On the 60th Assembly of the World Health Organization and the International Labor Organization (WHO/ILO), in 2007, was approved a Comprehensive Workers Health Plan, which aimed to reduce all types of pneumoconiosis and to eliminate silicosis by the year 2000 completely[24]. Unfortunately this goal was not reached, especially in developed countries. The reason was that many old work processes are still in use.

Among the 10 main work related diseases that have been found during the last 10 years in Mexico[25,26] are (in order or importance): Hearing impairment, respiratory conditions due to fumes of chemical vapors denominated industrial bronchitis or chemical bronchitis, all the pneumoconiosis produced by silica and silicates, antracosilicosis and pneumoconiosis produced because of the exposure to other inorganic powders (not silica nor cement). The auditory pathology is found with one single exam, while the lung one needs at least five different ones, for that reason is important to establish a good identification method of the disease as well as a more effective prevention mechanism.

Methods

An observational, descriptive, retrospective and transversal study was realized. The data was provided by a private company that has both, a quarry, where they are in charge of the extraction of silica rocks in an open environment, and a line of cement and concrete production. The sample of 32 quarry workers and 57 cement production line workers were considered with the following criteria: workers that were exposed to inorganic powders and who had completed full check-ups.* The exposure level of workers to inorganic dust was considered as the interaction that the workers

*Administrative workers that are usually indoors and well isolated from the inorganic powders did not qualify for the study. Given that the study was done to gather the data was observational and the use of the obtained information is completely confidential, there are not ethic violation

have from their work environment to inorganic dust, whether they use it as raw material or as a sub product from the production lines. Several measurements were taken in the different areas of the company accounting for the total amount of dust and for the breathable dust reporting values in mg/m.[3] In an environmental study on the company (table I), in a monitoring was taken into account only the total amount of powders and it was not possible to determine the amount of silica that they contained. By a periodic inspection of the work area, the inspectors observed: the age of the subject (age), weight (in kilograms), height (in centimeters), body mass index (BMI), workers´ seniority (old), the location where the subject works (loc, where loc = 0 if the subject works in the quarry and loc = 1 if works in the cement production line), whether the subject has a disease or not (where sick = 1 if the subject is sick, sick = 0 if not), the diagnosis of the subject (diagnosis, where sick = 1 if normal, sick = 2 if industrial bronchitis, sick = 3 if silicosis, sick = 4 if mixed).

We will consider two different scenarios. First we considered sick as a response variable, in order to observe if there is a pattern on what can trigger the disease. Then we will consider the diagnosis so we can observe if there is something that differentiates one kind of diagnosis to another.

The lung alterations identifying silicosis were described as "the accumulation of dust in the lungs and the reaction of the body in the presence of it" together with chest X-rays that showed the presence of rounded opacities and small irregularities. Industrial bronchitis was defined as "respiratory tract disease due to an acute or chronic exposure to irritants in the environment and adversely effects on the anatomy and function of the tracheobronchial tree". An industrial bronchitis diagnosis was made when the lung function tests showed an obstructive pattern on any stage and when alterations where found on X-ray as an increase of the outer third frame and irregular opacities. Chest X-ray interpretation was according to the code of the ILO International Classification of Radiographs in Pneumoconiosis from 2000 as well as when the spirometric studies showed a restrictive or mixed pattern.

A sensory reconnaissance visit was made to the company (both to the quarry and to the cement production line), where a vast amount of dust was observed all over the place. Afterwards, air samples were obtained using air pumps and filters and a gravimetric method in order to identify the levels of organic dusts in the environment and to be able to compare these levels with the levels allowed by the Mexican Official Standard NOM-10-STPS-1999.[27] The data that was collected was analyzed with statistical techniques according to the characteristics of the data.

Results

A sample of 89 subjects was gathered (32 from the quarry and 57 from the cement production line). All were men, with an age average of 38.07 years. The time of exposure to inorganic dusts was measured according to the amount of years the individual has been in the company (6.28 years in average). There were significant differences between the subjects that worked for the quarry and the ones that work for the cement production line (table II). The first group was significantly younger, and has been exposed to a "contaminated" environment for a shorter period. But there were not a significant difference on the amount of sick people on each group. We compare the characteristics of healthy subjects and non-healthy ones, we observed that there were no significant differences between both groups. The difference was observed in the correlations of all the variables involved in the study (table III), we saw that even though the fact of being sick or not it was not correlated with any other characteristic. There was a significant correlation between the place where the subject was located (either quarry or cement production line) and the diagnosis.

Table III Pearson correlation coefficients

Variable	Age	Weight	Height	BMI	Old	Loc	Sick	Diagnosis
Age	1.000	0.114	**0.221**	–0.03	**0.432**	0.423	–0.053	–0.029
Weight		1.000	**0.324**	**0.793**	0.075	0.187	0.041	0.022
Height			1.000	–0.214	0.163	**0.371**	–0.09	–0.182
Body mass index				1.000	–0.096	0.042	0.098	0.130
Old					1.000	**0.498**	–0.048	–0.071
Loc						1.000	–0.183	**–0.271**
Sick							1.000	**0.944**
Diag								1.000

The bold cells are the significant coefficients under a significance level of $p \leq 0.05$

The exposure levels by location (table I) we had the highest exposure in the quarry (89.67 mg/m^3 in one of the drills) followed by the cement production line (65.90 mg/m^3 in one of the cellars). The difference could be the amount of silica that each environment had, unfortunately that could not be measured.

Discussion

The study confirmed the fact that there are still many silicosis and industrial bronchitis cases in Mexico as well as in other countries, such as, India, Australia and Italy. We have to on improving the working conditions and the preventive actions. We have to remember that silicosis is a progressive condition that even when we stop the exposure of the subject, if the destruction process produced by the alveolar macrophages induced by the dust is started and fibrosis is created after that, there is no way back until the subject dies. It is a big problem given that the disease is unnoticed and there is not a specific treatment. Given the high volumes of dust in some of the areas we were not surprised with no difference on the amount of sick people by location, neither would we are surprised that the quarry workers have worst problems than the cement production line ones. There are individuals that are susceptible and others that are resistant to silicosis but there has not been found a pattern that predicts the susceptibility level of each subject but it is thought that genetics is involved, but this can just be confirmed by more studies in molecular biology and genetics on subjects that are exposed, and this will also help us to respond even more questions about these pathologies and, therefore, create better preventive programs.

The best way to fight a disease is with prevention, in the case of silicosis, this is done by epidemiological surveillance on exposed individuals and by implementing industrial hygiene measurements and safety like a wet processes, air injection and extraction and the use of personal protective equipment.[28]

Unfortunately we don't have the correct correlation between the amounts of functional alterations vs. X-ray alterations on early stages, but when pneumoconiosis is advanced the correlations is obvious.

Industrial bronchitis evolves depending on the exposure of the subject, which can be acute, subacute or chronic. Most of the patients can have an inflammation that is not only located on the respiratory system but also on all the mucous membranes of upper respiratory, initially the nose and following the pharynx, larynx, trachea, bronchi, bronchioles, reaching up to the alveoli and interstitial . Irritation can be simple or chemical; is considered simple when the agent exerts an irritating effect on its own, and chemical when the agent does not have a respiratory irritant effect until it is transformed and its metabolites are those who attack the respiratory mucosa.[29,30]

According to the literature, in order to develop silicosis, the subject needs to be exposed to the hazardous environment for about 10 years and we observed in our study the average exposure that the sick personas had was 6.5 years; this might be because of the amount of inorganic dusts the workers were exposed to dust and the silica percentage must be quite in order for it to considerably accelerate the development of the disease.

Conclusions

We can say that as long as a subject was exposed to powders with silica content there was a big chance that the individual will develop a pulmonary disease. Depending on the amount of silica powders the person was exposed it will define how severe will be the disease as we observed in the quarry workers, who presented more severe disease than the cement production line ones.

On the cement production line the workers showed mainly, industrial bronchitis which can be reversible as long as the person is no longer exposed to the dusty environment. Companies that work with dusty environments should have more protective measurements for its workers.

Conflict of interest disclosure: the International Committee of Medical Journal Editors form for disclosure of potential conflicts of interest has been completed and delivered by the authors. It hasn't been reported any conflict in regards to this article.

[a]Facultad de Estudios Superiores Zaragoza, Universidad Nacional Autónoma de México
[b]Departamento de Estadistica, Instituto Tecnológico Autónomo de México
[c]Clínica Tasqueña, Sistema de Transporte Colectivo
[d]Universidad de la Villa Rica, Veracruz, Veracruz, Mexico
[e]Hospital General de Zona 27, Instituto Mexicano del Seguro Social
[f]Coordinacion de Salud Publica, Instituto Mexicano del Seguro Social

[a,b,c,e,f]Mexico City, Mexico

Correspondence: María Martha Méndez-Vargas
Phone: (55) 5523 4778
E-mail: ramazzini.1700@gmail.com

References

1. Proceedings of the Fourth International Pneumoconiosis Conference, Bucharest, 1971 27 Sep- 2 Oct, Apimondia Publishing House, Bucharest, Hungary.
2. Heppleston AG, Styles JA. Activity of a macrophage factor in collagen formation by silica. Nature. 1967; 214(5087):521-22.
3. Parkes WR. Occupational lung disorders. Third edition. England: Butterworth's & Co; 1994.
4. Weiss SJ, Lo Buglio AF. Phagocyte-generated oxygen metabolites and cellular injury. Lab Invest. 1982; 47(1):5-18.
5. NIOSH. Criteria for a recommended standard: occupational exposure to crystalline silica, Cincinnati, OH, U.S.: Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health. HEW publication Nº (NIOSH). p. 75-120.
6. Mossman BT, Churg A. Mechanism of pathogenesis of asbestosis and silicosis. Am J Respir Cit Care Med. 1998;157(5 Pt 1):1666-80.
7. Vallyathan V, Shi XL, Dalal NS, et al. Generation of free radicals from freshly fractured silica dust. Potential role in acute silica-induced lung injury. Am Rev Respir Dis. 1988;138(5):1213-219.
8. Fubini B. The surface chemistry of crushed quartz dust in relation to its pathogenicity. Org Chem Acta. 1987;138(3):193-97.
9. Ziskind M, Jones RN, Weill H. Silicosis. Am Rev Resp Dis. 1976;113(5):643-65.
10. Maldonado-Torres L, Méndez-Vargas MM. Enfermedades broncopulmonares de trabajo. México: Auroch; 1999.
11. Morgan WKC, Seaton A. Occupational lung diseases. Philadelphia: W.B. Saunders Co; 1975.
12. Ziskind M, Jones RN, Weill H. State of the art. Silicosis. Am Rev Resp Dis. 1976;113(5):643-65.
13. Guidelines for the use of ILO International Classification of Radiographs of Pneumoconiosis. Geneva: International Labour Office; 2000 (Occupational Safety and Health Series N° 22 (rev. 2000).
14. American Thoracic Society. Snowbird workshop on standardization of spirometry. Am Rev Respir Dis. 1979;119(5):831-38.
15. American Thoracic Society. Lung function testing selection of reference values and interpretative strategies. Am Rev Respir Dis. 1991;144(5):1202-18.
16. American Thoracic Society. Standardization of spirometry 1944 update. Am J Respir Crit Care Med. 1995;152(3):1107-136.
17. Peters J. Silicosis. En: Merchant JA, Boehlecke BA, Pickett-Harner M, editores. Occupational respiratory diseases. US: Department of Health and Human Services: National Institute for Occupational Safety and Health; 1986.
18. Dubois AB. Industrial bronchitis and the function of the lungs. Arch Environ Health. 1962;4:128-34.
19. Brinkman GL, Block DL, Cress C. Effects of bronchitis and occupation on pulmonary ventilation over eleven year period. J Occup Med. 1972;14(8):615-20.
20. Lowe CR, Campbell H, Khosla T. Bronchitis in two integrated steel workers III. Respiratory symptoms and ventilatory capacity related to atmospheric pollution. Br J Ind Med. 1970;27(2):121-29.
21. Lowe CR. Chronic bronchitis and occupation. Proc R Soc Med. 1968;61(1):98-102.
22. Merchant JA, Boehlecke BA, Pickett-Harner M. Occupational respiratory diseases. US: Department of Health and Human Services: National Institute for Occupational Safety and Health; 1986.
23. López RP, Nava LR Salinas TS, et al. Neumoconiosis en trabajadores expuestos a polvos inorgánicos. Rev Med Inst Mex Seguro Soc. 2008;46(2)163-70.
24. Eijkemans G. Seminario la patología pulmonar por sílice y silicatos. Su prevención. En: Plan Global de Salud de los Trabajadores. México: Instituto Nacional de Enfermedades Respiratorias; 2008.
25. Instituto Mexicano del Seguro Social. Memoria estadística del IMSS (2001-2006). México: IMSS, Dirección de Finanzas y Sistemas; 2006.
26. Yang CY, Huang CC, Chiu HF, Chiu JF, Lan SJ, Ko YC. Effects of occupational dust exposure en the respiratory health of Portland cement workers. J Toxicol Environ Health. 1996;49(6):581-88.
27. Norma Oficial Mexicana NOM-010-STPS-1999, condiciones de seguridad e higiene en los centros de trabajo donde se manejen, transporten, procesen o almacenen sustancias químicas capaces de generar contaminación en el medio ambiente laboral. Diario Oficial de la Federación. (Sep 13, 2000).
28. Norma Oficial Mexicana 017 STPS 2001, equipo de protección personal.- selección y uso y manejo en los centros de trabajo. Diario Oficial de la Federación. (Nov 5, 2001).
29. Maldonado-Torres L, Méndez-Vargas MM, González-Zepeda A. Enfermedad de las vías aéreas por contaminantes en el sitio de trabajo. México: IMSS, Coordinación General Comunicación Social; 1990.
30. Soto de la Fuente AE, Aguilar-Loya M, Méndez-Vargas MM, et a. Bronquitis industrial en trabajadores expuestos a hidroalcoholes. Rev Med Inst Mex Seguro Soc. 2007;45(6):565-72.