Estimation and Health Risk Assessment of Respirable Silica in the Ambient Particulate Matter of Dhaka City

M N I Nahin[1], S Nahian[1], M S Islam[1], S Roy, F Jeba and A Salam[1]*

[1]*Department of Chemistry, Faculty of Science, University of Dhaka, Dhaka- 1000, Bangladesh*

(Received: 9 June 2022 ; Accepted: 25 August 2022)

Abstract

A simple, rapid, and efficient spectrophotometric method was developed for the quantification of respirable crystalline silica (SiO_2) in atmospheric particulate matter (PM). The PM samples were collected for 24 hours from two different locations at the University of Dhaka (Mukarram Hussain Khundker Building-MHK and Teacher Student Centre-TSC) using a low-volume air sampler from September to December 2021. The extraction of PM loaded filters followed by filtration resulted in a clear solution. Using UV-visible spectroscopy, the concentration of silica in the filtrate was evaluated by treating the filtrate with molybdate-3 reagent and an amino acid suspension. The average PM-bound silica concentrations at MHK and TSC were 7.72 and 33.86 μgm^{-3} respectively. Compared to MHK, the silica content in the ambient atmosphere of TSC was four times higher, owing to extensive metro-rail construction work at TSC. The contribution of silica to PM was 3.47% (Site 1) and 5.15% (Site 2). The Hazard Quotient (HQ) was used to characterize the non-cancer risk posed by respirable silica. TSC recorded a high value of 12.87 for the hazard quotient of respirable silica compared to 2.57 for MHK indicating that the exposed inhabitants may have adverse non-cancer health impacts. Carcinogenic risk assessment of silica showed that 1 in 341 individuals at MHK and 1 in 78 individuals at TSC could get cancer in their lifetime. This is the first study that demonstrated the high level of PM-bound respirable silica severely degraded air quality as well as potential human health in Dhaka.

I. Introduction

Air pollution has become a global concern nowadays due to prolonged exposure to various air pollutants that can lead to severe health risks. The Lancet Commission on Pollution and Health has recently declared that around 7.0 million people across the world die every year owing to air pollution[1]. The exposure of humans to Particulate matter (PM) is of special significance when considering the exposure of humans to airborne pollutants. The PM has a significant amount of silicon, as it is the second most abundant element on the Earth[2]. Respirable crystalline silica (RCS) refers to the respirable fraction of airborne crystalline silica that can enter the gas- exchange region of the lungs via inhalation[3]. The anthropogenic RCS from construction, ceramics, stone, and glass appliances are the major producers of PM[4]. Crystalline silica exhibits polymorphism in the form of quartz, tridymite, and crystoballite[5]. Prolonged exposure to such polymorphic forms in occupational settings may lead to lung cancer[6]. Construction workers are the worst sufferers as they inhale excessive amounts of crystalline silica present in closed spaces[7]. Crystalline silica is declared as a group-1 carcinogen by the International Agency for Research on Cancer[8].

Chronic exposure to RCS may cause pulmonary silicosis, bronchitis, tuberculosis, chronic obstructive pulmonary disease (COPD), kidney disease, etc.[9-12]. Since 1968, silicosis is considered the most common respiratory disease owing to silica dust exposure[13-15]. Non-occupational silica exposures are also observed in places where the soil has high silica content and dust storms occur frequently. In such regions, the exposed recipient may develop non-pneumoconiosis[16.17]. To conduct epidemiological research on the occupational and non-occupational exposure of a community to respirable crystalline silica, it is necessary to assess the silica quantitatively in the atmosphere of Bangladesh. Various methods have been proposed for determining the airborne silica concentration, including visible spectrophotometry by NIOSH (method no. 7601)[18], IR spectrophotometry by NIOSH (method no. 7602)[19], ICP-MS technique[20], X-ray diffraction (XRD) by OSHA-United States Occupational Safety and Health Administration[21]. In this work, a simple and easily accessible method for detecting the silica concentration in the atmospheric particulate matter by UV-visible spectroscopy.

However, this result will reflect the percentage of silica within the overall mass of suspended particulate matter.

In addition, a health risk assessment was conducted to monitor the impact of respirable crystalline silica inhalation on public health.

* Author for Correspondence. e-mail: asalam@gmail.com; asalam@du.ac.bd, Tel: +8801817061160

II. Methodology

Sampling procedure

The first sampling location, Site 1, was an educational institute with coordinates of 23.7282^o latitude and 90.3982^o longitude. The second sample point, Site 2 was next to the metro-rail construction site and had coordinates of 23.7320^o latitude and 90.3991^o longitude. Ambient PM samples were collected for 24 hours on pre-weighed polytetrafluoroethylene (PTFE) filters (Ultipor N_{66} Nylon 6,6 Membrane filter, 47 mm diameter) using a TSP low volume air sampler. The sampler was placed 2 meters above the ground and the volume of air was recorded from the difference of the initial and final gas meter reading[22]. A total of 18 samples were collected, 10 from Site 1, 6 from Site 2, and 2 blank samples. Finally, the filters were preserved in petri-dishes under room temperature. The weight of the PM loaded filters was measured using a microbalance (Model: AS220.R2, Radwag) with a sensitivity of 0.0001 g. The difference between the initial and final weight of the filters corresponded to 24 hr mass of PM, which being divided by the volume of air, results in 24 hr average concentration of PM. Finally, the filters were kept in the refrigerator below 0°C until sample extraction followed by chemical analysis.

Extraction of PM loaded PTFE filters

Half of each filter paper was taken in a 250 mL polystyrene beaker and 10 mL of 5% NaOH was added to it. The mixture was subjected to shake by a mechanical shaker at 25°C for 90 min. Then the beaker was kept in a sonicator at 25°C for 10 min. After that, 12 mL of 5% HNO_3 was added into the beaker followed by sonication at 25°C for 10 min[2]. The mixture was allowed to stand overnight and was filtered on the next day using Whatman 40 filter paper. 5 mL filtrate of each sample was preserved in a centrifuge tube for silica determination by UV-visible spectrophotometer. During the entire extraction process, the pH of the solution was maintained less than 4.0 using pH meter.

Health risk assessment

The United States Environmental Protection Agency (USEPA) defined health risk assessment as the model used to estimate the possible health effects arising from exposure to carcinogenic and non-carcinogenic species[23]. Both non-cancer and cancer risk imposed by silica exposure were calculated using the average crystalline silica concentration.

Non-cancer risk characterization

Non-cancer risk of silica refers to the exposure concentration of silica below which severe health hazards are less likely to occur[24]. The exposure concentration (EC) in $\mu g m^{-3}$ was calculated using the following equation:

$$EC = \frac{CA \times ET \times EF \times ED}{AT} \qquad (1)$$

where, CA represents the silica concentration in air in $\mu g m^{-3}$, ET denotes the exposure time in hours per day, EF stands for the exposure frequency in days per year, ED indicates the exposure duration in years and AT is the averaging time, which was calculated as exposure duration in years × 365 days per year × 24 hours per day[25]. ET and ED were taken as 24 hours per day and 70 years, respectively[26]. A maximum EF of 365 days a year was used to represent a worst scenario where exposure was estimated for the whole year[24].

Non-cancer risk of respirable silica is characterized by Hazard Quotient (HQ). HQ is a unit less parameter, defined as the ratio of the predicted exposure concentration (EC) to the reference concentration (RfC):

$$HQ = \frac{EC}{RfC} \qquad (2)$$

The reference concentration (RfC) of silica was taken as 3 $\mu g m^{-3}$ [27,28]. If HQ of silica is less than 1, it implies no possible risk to develop a non-cancer health effect, whereas an HQ higher than 1 indicates a possible risk[29].

Cancer risk characterization

Cancer risk characterization signifies the possibility of an individual developing cancer from lifelong exposure to a carcinogenic substance[30]. The cancer risk imposed by respirable silica can be estimated using the following equation[25]:

$$Cancer\ risk = IUR \times EC \qquad (3)$$

Where, IUR is the inhalation unit risk of silica, expressed in $\mu g^{-1} m^3$, and EC is the exposure concentration of silica in $\mu g m^{-3}$. The reference value of IUR was taken as 3.8×10^{-4} $\mu g^{-1} m^3$ [31,32].

Quality control

Quality control was maintained throughout the sampling and analytical procedures to ensure reliability of the data. In every step of this study, use of borosilicate apparatus was avoided so that no contamination into the samples could occur.

The leak of air sampler was tested and calibrated before sampling. Weight of the filter paper, pump reading of the machine, sampling date and time were noted carefully before and after sampling. Blank samples were collected during the experiment and analyzed to maintain accuracy of the data. The filter papers were preserved in clean, dry and airtight plastic petri-dishes. After sampling, the petri-dishes were kept in the refrigerator to avoid contamination before chemical analysis. Just before extraction, the loaded filters were taken out of the

refrigerator and kept in a dessicator for 30 min so that no moisture is present. Apparatus used in chemical analysis were cleaned with acetone and rinsed properly with

deionized water. Analytical grade reagents were used during chemical analysis. Reagent blanks were also analyzed by UV-visible spectrometers for correction of instrument readings. The spectrometer was properly calibrated before operation.

Method development

Preparation of standard solution

For the UV-visible spectrophotometric technique of determining silica content in ambient particulate matter samples, a stock solution is required to construct an analytical calibration curve. Sodium metasilicate non-ahydrate ($Na_2SiO_3.9H_2O$) was dissolved in water at room temperature to prepare a stock solution of 100 ppm. The stock solution was diluted to produce a series of standard solutions with concentrations ranging from 0.05 to 2.50 ppm. In order to avoid contamination from glassware, borosilicate glass equipment was avoided. Instead, a polystyrene apparatus was used to prepare standard solutions and analyse PM samples[33].

Construction of analytical calibration curve

5.0 mL aliquot from each of the standard solutions was taken in a polystyrene test tube and 125 µL of molybdate-3 reagent was added. All the mixtures were subjected to sonication and later they were allowed to settle down for 4 min. After that, 125 µL of citric acid reagent was added to each mixture and allow it for 1 min to complete the reaction. Exactly 250 µL of amino acid F suspension was further added to each tube. The tubes were immediately put into the mechanical shaker for vortex mixing. After mixing, each solution was allowed to settle down for a while. Later, absorbance of each solution was measured at 815.6 nm by a double beam UV-visible spectrophotometer (Model: UV-1800, SHIMADZU, Japan). The same procedure was performed on PM samples. Similarly, the blanks were analyzed using the same methodology. Necessary corrections were made by excluding the blank results from the measured data.

III. Results and Discussion

Determination of silica concentration in PM samples

Phosphates and silicates are well-known inorganic constituents of particulate matter as well as primary water supplies. As they co-exist in PM samples, phosphate interference can occur in UV-visible spectrophotometric method[33]. So, the phosphate should be masked before subjecting the samples to UV-visible analysis. Both phosphates and silicates react with molybdate-3 reagent (at

pH<7) forming molybdophosphoric acid and molybdosilicic acid complexes respectively. The first complex gives citric acid hydrolysis reaction, but the molybdosilicic acid complex remains inert to hydrolysis. Hence, excess citric acid was used to remove the phosphate complex. After that, addition of amino acid F suspension turns the yellow molybdosilicic acid complex into an intense blue colored complex, which absorbs light at 815.6 nm. According to Beer- Lambert law, this concentration is proportional to the silica concentration. Therefore, concentration of silica in the PM samples were determined using the calibration curve constructed using the standard solutions.
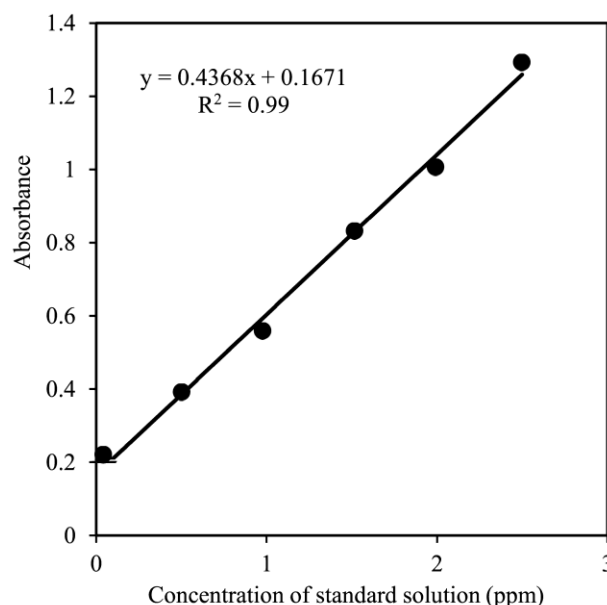

Fig. 1. Absorbance of standard silicate solutions   nm for the calibration curve to determine silica concentration using UV-visible spectrophotometer.
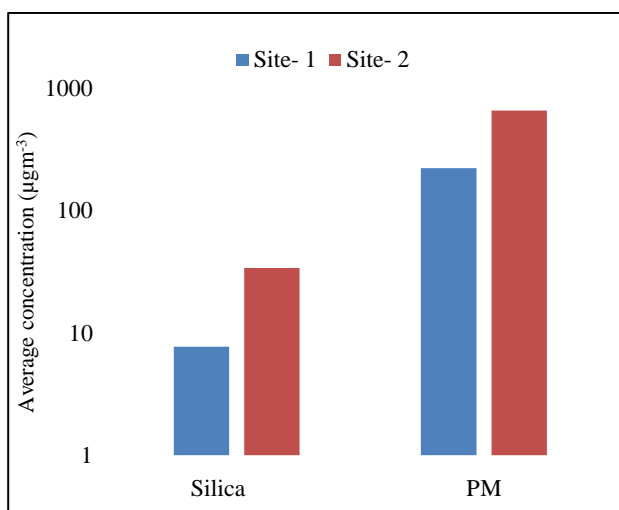
The linear plot depicted in Fig. 1 has an R^2 value of 0.99. The following equation can be used to calculate the silica content in PM samples:

y = 0.4368x + 0.1671, where y = absorbance of the PM samples, and x = concentration of silica in samples. In the calibration curve an intercept of absorbance was shown which eliminated from the absorbance of the samples to get the actual concentration of silica.

The average concentration of silica at Site 1 was 7.716 μgm^{-3}, while the average amount of PM was 222.0 μgm^{-3} (Table 1). However, 3.47% of the total PM mass was composed of silica. The highest crystalline silica concentration at Site 1 was 19.5 μgm^{-3}, while the lowest value was 0.22 μgm^{-3}.

Table 1. **Demonstration of obtained silica concentration in PM samples and the individual PM concentration at Sites 1 and 2, respectively.**

Sample No.	Concentration of silica in PM sample (μgm^{-3})		Concentration of PM sample (μgm^{-3})	
	Site 1 (MHK)	Site 2 (TSC)	Site 1 (MHK)	Site 2 (TSC)
01	3.36	81.81	205.7	1405.4
02	0.22	29.85	66.4	663.1
03	1.28	28.22	159.9	597.9
04	1.37	16.42	160.3	380.1
05	1.75	21.96	185.6	413.4
06	16.91	24.95	303.3	485.2
07	19.50	-	324.2	-
08	10.06	-	265.6	-
09	6.79	-	234.9	-
10	15.91	-	314.2	-
Average	7.72	33.87	222.0	657.5

Fig. 2. Variation of average concentration of crystalline silica (SiO_2) and PM at both Site 1 (MHK) and Site 2 (TSC) in Dhaka city.

On the other hand, the average concentration of silica at Site 2 was found to be 33.87 μgm^{-3}, while the average concentration of PM was found to be 657.5 μgm^{-3}. Therefore, the PM at Site 2 contained 5.15% silica. A number of parameters, such as particle size, wind speed, wind direction, variation in silica sources, turbulence, etc., affect the quantity of silica in dust. The average silica concentration in PM samples from Site 2 was 4.39 times higher than those of Site 1. This is due to PM samples being obtained in an indoor environment inside at Site 1, whereas sampling at Site 2 was done outdoors near a metro rail construction site. At Site 2, occupational silica from drilling, chipping, cutting, grinding, and hammering of concrete, bricks, rods, etc. was about 4 times more than non-occupational silica at Site 1.

Comparison of silica concentration in Dhaka with other global cities

Fig. 3. compares the concentration of silica in Bangladesh with values from the other nations. This study determined that the average silica content in the PM fraction in Dhaka was 20.79 μgm^{-3}. However, the guideline values of USEPA and COEHHA (California Office of Environmental Health Hazard Assessment) for silica are 5.0 and 3.0 μgm^{-3}, respectively[34]. However, silica content in Dhaka was also lower than the NIOSH and ACGIH guideline value 25.0 μgm^{-3} [35]. The amount of silica in the ambient air of Dhaka is much higher than in cities in developed countries (Fig. 3).

It was reported that the PM-bound silica concentration in California (USA) ranged from 1-8 μgm^{-3} [36], whereas the weekly average silica concentration in PM in Rome (Italy) ranged from 0.3-2.9 μgm^{-3} [37]. However, PM-bound silica content in Dhaka was similar to that in Raipur, India (30.0 μgm^{-3}) and Johannesburg, South Africa (19.0 μgm^{-3})[38]. On the other hand, Tehran, Iran recorded high amounts of respirable silica (130.0 μgm^{-3}) in ambient air[21]. Excessive ambient silica (120-300 μgm^{-3}) was also found in Henan, Hunan and Jiangxi provinces of China[39].

Health risk assessment of PM-bound crystalline silica

Table 2 summarizes the non-cancer risk and cancer risk owing to exposure to PM-bound respirable crystalline silica at Site 1 (MHK). The non-cancer risk was characterized by hazard quotient (HQ). For Site 1, the average HQ of silica was 2.572. As HQ> 1, there was a possible risk of facing non-cancer health effects while being exposed to respirable silica. On the other hand, the average cancer risk at Site 1 was found to be 2.932×10^{-3}. For regulatory purposes, the acceptable value of cancer risk is in the range of 1×10^{-6} to 1×10^{-4} [40]. Hence, the average value of cancer risk was higher than the acceptable limit and 1 in 341 individuals at Site 1 had a possibility to develop cancer in his lifetime.
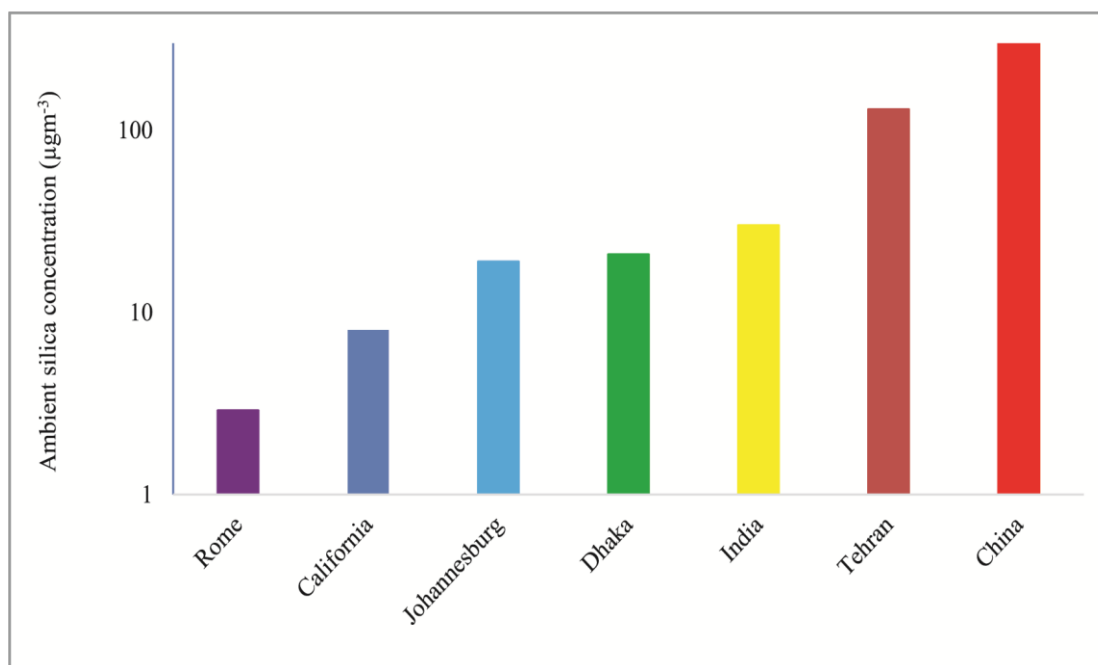
Fig. 3. Comparison silica concentration in the ambient atmosphere among global cities

Table 2. Non-cancer and cancer risk from exposure to PM-bound respirable crystalline silica at Site 1 (MHK) and Site 2 (TSC) in Dhaka, Bangladesh.

Sample No.	Site 1(MHK)			Site 2(TSC)		
	Silica concentration (μgm^{-3})	Non-cancer risk (HQ)	Cancer risk ($\times 10^{-3}$)	Silica concentration (μgm^{-3})	Non-cancer risk (HQ)	Cancer risk ($\times 10^{-3}$)
01	3.36	1.120	1.280	81.81	27.269	31.086
02	0.22	0.074	0.084	29.85	9.949	11.342
03	1.28	0.427	0.486	28.22	9.405	10.722
04	1.37	0.457	0.521	16.42	5.473	6.239
05	1.75	0.582	0.663	21.96	7.321	8.345
06	16.91	5.638	6.427	24.95	8.318	9.482
07	19.50	6.500	7.411	-	-	-
08	10.06	3.354	3.824	-	-	-
09	6.79	2.264	2.582	-	-	-
10	15.91	5.304	6.046	-	-	-
Average	7.72	2.572	2.932	33.87	11.289	12.869

Table 2 represents the non-cancer risk and cancer risk imposed by PM-bound respirable crystalline silica at Site 2 (TSC). The average HQ of silica at this location was 11.289. As HQ> 1, the residents were subjected to adverse non-cancer effects while being exposed to respirable silica. On the contrary, the average cancer risk at Site 2 was 12.869×10^{-3}, which exceeds the USEPA guideline value (10^{-4}- 10^{-6}). This implies that 1 in 78 individuals at Site 2 may fall a victim to cancer in his lifetime.

IV. Conclusion

This study aims to validate an analytical method for determining the quantity of respirable crystalline silica in airborne particulate matter for a developing nation like Bangladesh. The proposed UV-visible spectrophotometric method was found to be reliable with reasonable detection limit and the novelty of this method lies in its low cost, effortless handling and easy maintenance. This proposed spectrophotometric method was applied for the determination of silica concentration in PM samples at two different locations in Dhaka. The average concentration of silica measured in the ambient air at both locations below the USEPA and OEHHA-recommended guideline values. The amount of silica from construction work area (Site 2) was almost 4 times higher than the amount from non-construction area (Site 1). Consequently, people at Site 2 are four times more likely to experience non-cancer health consequences than those in Site 1. In addition, one person at every 78 people living at Site 2 has a lifetime risk of developing cancer. The findings of this study suggest that being exposed to PM-bound respirable silica might have a significant negative impact on the health of the general population in Dhaka. In order to reduce air pollution and ensure the safety of construction workers, the government should increase public awareness of air pollution and establish stringent restrictions.

References

1. Zhang, Q., X. Jiang, D. Tong, S. J. Davis, H. Zhao, G. Geng, T. Feng, B. Zheng, Z. Lu, D. G. Streets, R. Ni, and D. Guan, 2017. Transboundary health impacts of transported global air pollution and international trade. *Nature*, **543(7647)**, 705-709.

2. Lu, D., J. Tan, X. Yang, X. Sun, Q. Liu, and G. Jiang, 2019. Unraveling the role of silicon in atmospheric aerosol secondary formation: a new conservative tracer for aerosol chemistry. *Atmospheric Chemistry and Physics*, **19(5)**, 2861-2870.

3. Stacey, P., A. Thorpe, P. Roberts, and O. Butler, 2018. Determination of respirable-sized crystalline silica in different ambient environments in the United Kingdom with a mobile high flow rate sampler utilising porous foams to achieve the required particle size selection. *Atmospheric Environment*, **182**, 51-57.

4. Healy, C. B., M. A. Coggins, M. Van Tongeren, L. MacCalman, and P. McGowan, 2014. Determinants of respirable crystalline silica exposure among stoneworkers involved in stone restoration work. *Annals of occupational hygiene*, **58(1)**, 6-18.

5. National Institute for Occupational Safety and Health (NIOSH), 1994. Manual of analytical methods. Method 7602, Silica Crystalline by IR.

6. Vida, S., J. Pintos, M. É. Parent, J. Lavoué, and J. Siemiatycki, 2010. Occupational exposure to silica and lung cancer: pooled analysis of two case-control studies in Montreal, Canada. *Cancer Epidemiology and Prevention Biomarkers*, **19(6)**, 1602-1611.

7. Pocock, D. 2012. On-tool controls to reduce exposure to respirable dusts in the construction industry. Health and Safety Executive

8. International Agency for Research on Cancer (IARC), 2009. Monographs on the Evaluation of Carcinogenic Risk to Humans. Metals, Particles and Fibres. Vol. 100C, Lyon, France.

9. Vupputuri, S., C. G. Parks, L. A. Nylander-French, A. Owen-Smith, S. L. Hogan, and D. P. Sandler, 2012. Occupational silica exposure and chronic kidney disease. *Renal failure*, **34(1)**, 40-46.

10. Milovanović, A., D. Nowak, A. Milovanović, K.G. Hering, J.N. Kline, E. Kovalevskiy, I.Y. Kundiev, B. Peruničić, M. Popević, B. Šuštran, and M. Nenadović, 2011. Silicotuberculosis and silicosis as occupational diseases: Report of two cases. *Srpski arhiv za celokupno lekarstvo*, **139(7-8)**, 536-539.

11. Hnizdo, E., and V. Vallyathan, 2003. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: a review of epidemiological and pathological evidence. *Occupational and environmental medicine*, **60(4)**, 237-243.

12. Craighead, J. E. 1988. Diseases associated with exposure to silica and non-fibrous silicate minerals. *Archives of Pathology & Laboratory Medicine*, **112**, 673-720.

13. Bang, K. M., J. M. Mazurek, J. M. Wood, G. E. White, S. A. Hendricks, and A. Weston, 2015. Silicosis mortality trends and new exposures to respirable crystalline silica-United States, 2001-2010. MMWR. *Morbidity and mortality weekly report*, **64(5)**, 117.

14. Kachuri, L., P. J. Villeneuve, M. É. Parent, K. C. Johnson, Canadian Cancer Registries Epidemiology Group, and S. A. Harris, 2014. Occupational exposure to crystalline silica and the risk of lung cancer in Canadian men. *International Journal of Cancer*, **135(1)**, 138-148.

15. National Institute for Occupational Safety and Health (NIOSH), 2003. Silica crystalline No. 7602. NIOSH Manual of Analytical Methods.

16. National Institute for Occupational Safety and Health (NIOSH), 2003. Silica crystalline No. 7601. NIOSH Manual of Analytical Methods.

17. Aureli, F., M. Ciprotti, M. D'Amato, do E. Nascimento da Silva, S. Nisi, D. Passeri, A. Sorbo, A. Raggi, M. Rossi, & F. Cubadda, 2020. Determination of total silicon and SiO_2 particles using an ICP-MS based analytical platform for toxicokinetic studies of synthetic amorphous silica. *Nanomaterials*, **10(5)**, 888.

18. Tavakol, E., M. Azari, R. Zendehdel, S. Salehpour, S. Khodakrim, S. Nikoo, and B. Saranjam, 2017. Risk evaluation of construction workers' exposure to silica dust and the possible lung function impairments. *Tanaffos*, **16(4)**, 295.

19. Blondeau, P., V. Iordache, O. Poupard, D. Genin, and F. Allard, 2005. Relationship between outdoor and indoor air quality in eight French schools. *Indoor air*, **15(1)**, 2-12.

20. United States Environmental Protection Agency (USEPA), 2001. Toxics Release Inventory: Public Data Release Report.

21. Andraos, C., W. Utembe, and M. Gulumian, 2018. Exceedance of environmental exposure limits to crystalline silica in communities surrounding gold mine tailings storage facilities in South Africa. *Science of the total environment*, **619**, 504-516.United States Environmental Protection Agency (USEPA), 2009. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part F, Supplemental Guidance for Inhalation Risk Assessment).

22. United States Environmental Protection Agency (USEPA), 2011. Exposure Factors Handbook. 2011 Edition (Final). U.S. Environmental Protection Agency, Washington, DC, USA (EPA/600/R-09/052F).

23. Office of Environmental Health Hazard Assessment (OEHHA), 2005. Adoption of Chronic Reference Exposure Levels for Silica (Crystalline, Respirable).

24. Collins, J. F., A. G. Salmon, J. P. Brown, M. A. Marty, and G. V. Alexeeff, 2005. Development of a chronic inhalation reference level for respirable crystalline silica. *Regulatory Toxicology and Pharmacology*, **43(3)**, 292-300.

25. Kolluru, R. 1996. Risk Assessment and Management Handbook, for Environmental. Health and Safety Professionals, **16**.

26. Lu, X., X. Zhang, L. Y. Li, and H. Chen, 2014. Assessment of metals pollution and health risk in dust from nursery schools in Xi'an, China. *Environmental research*, **128**, 27-34.

27. Rice, F. L., R. Park, L. Stayner, R. Smith, S. Gilbert, and H. Checkoway, 2001. Crystalline silica exposure and lung cancer mortality in diatomaceous earth industry workers: a quantitative risk assessment. *Occupational and* Mazurek, J. M., & M. D. Attfield, 2008. Silicosis mortality among young adults in the United States, 1968-2004. *American journal of industrial medicine*, **51(8)**, 568-578.

28. Norboo, T., P.T. Angchuk, M. Yahya, S.R. Kamat, F.D. Pooley, B. Corrin, I.H. Kerr, N. Bruce, and K.P. Ball, 1991.

29. Saiyed, H.N., Y.K. Sharma, H.G. Sadhu, T. Norboo, P.D. Patel, T.S. Patel, K. Venkaiah, and S.K. Kashyap, 1991. Non-occupational pneumoconiosis at high altitude villages in central Ladakh. *Occupational and Environmental Medicine*, **48(12)**, 825-829.

30. *environmental medicine*, **58(1)**, 38-45.

31. Goldsmith, D. F., R. P. Ruble, and C. O. Klein, 1995. Comparative cancer potency for silica from extrapolations of human and animal findings. *Scandinavian Journal of Work, Environment & Health*, 104-107.

32. Manno, B. R., I. K. Abukhalaf, and J. E. Manno, 1997. A simple spectrophotometric assay for the measurement of soluble silica in water. *Journal of analytical toxicology*, **21(6)**, 503-505.

33. United States Environmental Protection Agency (USEPA), 1996. Ambient Levels and Non-cancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment, Washingtong, DC, USA (EPA/600/R-95/115).

34. Prapamontol, T. 2020. Crystalline Silica Exposure and Air Quality Perception of Residents Living Around Home Stone Factories. *Sains Malaysiana*, **49(3)**, 573-581.

35. Davis, B. L., L. R. Johnson, R. K. Stevens, W. J. Courtney, and D. W. Safriet, 1984. The quartz content and elemental composition of aerosols from selected sites of the EPA inhalable particulate network. *Atmospheric Environment*, **18(4)**, 771-782.

36. De Berardis, B., E. Incocciati, S. Massera, G. Gargaro, and L. Paoletti, 2007. Airborne silica levels in an urban area. *Science of the total environment*, **382(2-3)**, 251-258.

37. Patel, K. S., S. Gupta, S. Ramteke, K. P. Rajhans, S. Nava, and F. Lucarelli, 2016. Silica particulate pollution in central India. *Journal of Environmental Protection*, **7(02)**, 170.

38. Chen, W., Y. Liu, H. Wang, E. Hnizdo, Y. Sun, L. Su, X. Zhang, S. Weng, F. Bochmann, F.J. Hearl, J. Chen, and T. Wu, 2012. Long-term exposure to silica dust and risk of total and cause-specific mortality in Chinese workers: a cohort study. *PLoS medicine*, **9(4)**, e1001206.

39. United States Environmental Protection Agency (USEPA), 1989. Risk Assessment Guidance for Superfund Volume 1: Human Health Evaluation Manual (Part A); Office of Emergency and Remedial Response: Washington, DC, USA.

40. Chapman, E. M. 1932. Acute silicosis. *Journal of the American Medical Association*, **98(17)**, 1439-1441.

41. Moore, M. 1999. Crystalline silica: occurrence and use. *Indoor and Built Environment*, **8(2)**, 82-88.

42. Rushton, L., S.J. Hutchings, L. Fortunato, C. Young, G.S. Evans, T. Brown, R. Bevan, R. Slack, P. Holmes, S. Bagga, and J.W. Cherrie, 2012. Occupational cancer burden in Great Britain. *British journal of cancer*, **107** (S92-S103).

43. Stacey, P., A. Thorpe, and P. Roberts, 2011. Levels ofrespirable dust and respirable crystalline silica at construction sites RR878 Levels of respirable dust and respirable crystalline silica at construction sites RR878. Health and Safety Executive.

Silicosis in a Himalayan village population: role of environmental dust. *Thorax*, **46(5)**, 341-343.