Risk Assessment of Lung Cancer and Asbestosis in Workers Exposed to Asbestos Fibers in Brake Shoe Factory in Iran

Mansour R. AZARI^{1*}, Ali NASERMOADDELI¹, Mohammad MOVAHADI¹, Yadollah MEHRABI¹, Hossein HATAMI¹, Hamid SOORI¹, Elaheh MOSHFEGH¹ and Behnam RAMAZNI¹

¹Safety Promotion and Injury Prevention Research Center, Shahid Beheshti University of Medical Sciences, Chamran Highway, Evin District, Tehran, Iran

Received August 19, 2008 and accepted June 30, 2009

Abstract: Chrysotile asbestos fiber, imported from Russia, is used mainly for manufacturing purposes in Iran and related risks in the form of asbestosis and cancer were studied. Occupational exposure of all male workers (61 persons) to asbestos in a brake shoe factory was monitored. Cumulative exposures were determined through multiplication of typical exposure and work history. Risk assessment of exposed workers was estimated by risk criteria recommended by the American Environmental Protection Agency. Measurement of lung function parameters such as forced expiratory volume in one second (FEV₁), forced volume capacity (FVC) and FEV₁/FVC of exposed workers were obtained. Unadjusted correlation and adjusted correlation analysis for support of the association between cumulative exposure (fiber/ml-yr) and lung function parameters were used. Exposure of majority of exposed group was far greater than the occupational exposure limits (0.1 fiber/ml) in the range of 0.06-8.06 fiber/ml. Cumulative exposures in the range of 0.02 to 110.77 fiber/ml-yr were obtained. According to the risk criteria stated by ATSDR, risk assessment of workers in term of fibrotic changes was predicted for at least 24.6 percent of the exposed subjects. Again, according to the lung cancer risk criteria stated by EPA, 59 percent of workers will have excess risk. Negative correlation between lung function parameters (FEV1, FEV1/FVC) and cumulative exposures adjusted for age and BMI were significant (*p*<0.05).

Key words: Asbestos fibers, Occupational monitoring, Risk criteria, Risk assessment, Asbestosis and lung cancer

Introduction

Asbestos is a general term applied to certain fibrous minerals long popular for their resistance, tensile strength and acoustic insulation¹⁾. The use of asbestos for vehicular brakes takes advantage of its heat resistance and material strength. Asbestos concentrations in these materials are considerable, ranging from 30–80%. The number of American workers exposed to asbestos dusts brake and clutch work is estimated at 900,000. Exposure of

*To whom correspondence should be addressed. E-mail: mrazari@hotmail.com workers to asbestos fibers especially by routine practices of "blowing out" brake surfaces and beveling was reported considerable²⁾. Asbestos related health risks are well documented in epidemiological and basic science literature^{3, 4)}. Occupational exposures of workers due to drastic decline in asbestos usage in developed nation western countries are much lower than in the past⁵⁾. Asbestos related occupational disease still constitutes a major occupational health problem, especially for developing countries, where effective control of exposure has not been achieved properly yet^{6–8)}. Asbestos exposure may cause asbestosis, lung cancer or mesothelioma with long latency period^{9–14)}. One of the newest approaches of toxicology is risk assessment, whereby scientific information on the hazardous properties of toxic agents and the extent of exposure results in a statement as to the probability that exposed populations will be effected. This process is a scientific attempt to identify and estimate the true risks¹⁵⁾. Significant exposure-response relationship has been reported for both lung cancer and asbestosis. The cumulative exposure relation for lung cancer was reported to be linear on a multiplicative scale. Excess lifetime risk at permissible exposure level of Chrysotile (0.1 fiber/ml), was predicted to be about 5/1,000 for lung cancer, and 2/1,000 for asbestosis¹⁶.

In a recent review of the epidemiological evidence of exposure-response relationships for development of asbestosis, the World Health Organization Task Group on Environmental Health Criteria for Chrysotile asbestos (WHO 1998) concluded that fibrotic changes are common following prolonged exposures of 5 to 20 fiber/ml corresponding to cumulative exposures of 50-200 fiber/ml-yr for a 10-yr exposure¹⁷⁾. Signs of lung fibrosis and increased mortality associated with asbestosis or nonmalignant respiratory disease have been observed in groups of workers with chronic cumulative exposures as low as 15-70 fiber/ml-yr for signs of lung fibrosis and 32-1,271 fiber/ml-yr for asbestosis-associated mortality¹⁸⁾. Other authors have also explored pulmonary function of asbestos-exposed workers and their findings in order to support association between asbestos exposure and pleural fibrosis and restrictive lung function^{19, 20)}.

The risk of lung cancer associated with exposed occupational group indicated linear relationship with cumulative exposure. Integrated Risk Information System (IRIS) reported that occupational exposure for 20 yr at levels of 0.1 fiber/ml and 10 fibers/ml (measured by Phase Contrast Method) corresponds to 2 to 200 excess risks respectively per 1,000 persons²¹). Agency for Toxic Substances and Disease Registry has also presented linear doseresponse relationship model for computation of lung cancer risk²²).

The objective of this study was to assess risk of young exposed workers to asbestos through determination of cumulative dose.

Materials and Methods

This study was carried out in brake shoe and clutch disk manufacturing plant in Iran. Brake shoe and clutch disk are manufactured by mixing components consisting of chrysotile asbestos with chemicals such as toluene, sulfur, ferrous oxide, carbon black, graphite, lead, resins and fillers. The mixtures are injected into molds. Asbestos products are further processed by finishing machines such as grinding, drilling, and cutting. Asbestos fibers and some of the compounds used for brake shoe and clutch disk are released into the indoor air mainly during weighing, mixing, pressing and finishing processes. In this study occupational exposure of all workers to asbestos fibers involved in a brake shoe manufacturing processes (61 male workers) with mean age of 34, ranging from 21 to 58 yr old were monitored for exposure to airborne asbestos and lung function tests. Consent of each worker for personal monitoring of exposure to asbestos, lung function tests and demographic information were obtained prior to actual examinations in routine work shifts.

Personal monitoring of worker's exposure to asbestos fibers for at least 4 h was conducted according to the NIOSH method²³⁾. In this study, personal sampling of all exposed workers (61 persons), weighing (3), mixing (11), pressing (25) and finishing processes (22), were done by drawing a known volume of air through a 25mm diameter cassette containing a mixed-cellulose ester filter. The cassette was equipped with an electrically conductive 50-mm extension cowl. Analysis of asbestos fibers in samples was done with a phase contrast microscope. Cumulative exposure as an index of the workers' exposure (fiber/ml-yr) was calculated by multiplication of personal exposure and duration.

Most studies of the risk of asbestos-related lung cancer in occupationally exposed workers indicate that the dose-response relationship is best described by a relative risk model, given by the equation below. Since this is a relative risk model, the absolute risk of lung cancer due to asbestos exposure depends not only on cumulative asbestos dose, but also on the underlying risk of lung cancer due to other causes²².

Relative Risk = $1.0 + K_1$ (cumulative dose)

Absolute Risk = Relative Risk • Underlying Risk

Based on American national average lung cancer risk data for male and female smokers and nonsmokers, EPA calculated the cumulative exposures of 0.035 fiber/ml-yr and 0.35 fiber/ml-yr represent excess lung cancer risks of 10^{-4} for smokers and non-smokers respectively²⁴). In this study, EPA method of calculation of excess lung cancer risk for smokers and non-smokers were used to compute proportionally excess lung cancer risks for categorized quartile ranges of cumulative exposure in our study group.

Lung function tests (FVC, FEV_1 and FEV_1/FVC) were conducted on exposed population by a spirometer (Clinical ST-300 made by Fukoda Sungou Company) according to the method of American Thoracic Society²⁵).

Statistical analysis

Unadjusted and adjusted correlation analysis was used to support the association between cumulative exposure (fiber/ml-yr) and lung function parameters (FEV₁, FVC and FEV₁/FVC) adjusted for age and body mass index.

Results

Basic characteristics of workers including age, BMI, height, smoking status and their typical exposure were organized in Table 1. Work history of 42 percent of workers was less than a year and at the same time just 10 percent of workers had work history more than 20 yr. Exposure of study group was in the range of 0.06–8.06 fiber/ml, with a mean of 1.65 and standard deviation of 1.74 fiber/ml. Cumulative exposure were in the range of 0.02 to 110.77 fiber/ml-yr and geometric mean of cumulative exposure of different working groups in regard to their work histories were in the range of 0.14 to 53.68 fiber/ml-yr (Table 2).

Risk assessment of different classes of workers in regard to lung cancer incident rate was computed according to respective geometric mean of cumulative exposures. Risk of lung cancer incidence for smokers according to the geometric mean of cumulative exposures 0.14, 1.42, 10.98 and 53.68 fiber/ml-yr for various cumulative exposure ranges, were projected 0.4, 4.06, 31.37 and 153.37 excess lung cancer per thousand exposed, respectively. Corresponding risk of lung cancer for groups of nonsmoker workers were estimated tenfold lower than smoking workers (Table 2). Generally, 59 percent of all workers from smoker and nonsmokers have excess risk of lung cancer more than one per thousand (Table 2).

Based on ATSDR association of cumulative exposure with lung fibrosis, 24.6 percent of our exposed workers had cumulative exposure in range of 15–70 fiber/ml-yr,

Table 1. Demographic characteristics of the workers studied

81		
Work history (yr)	Mean ± SD	8.84 ± 8.39
Height (cm)	Mean ± SD	169.3 ± 6.05
BMI (kg/m ²)	Mean \pm SD	24.86 ± 3.86
Smoker	N (%)	25 (41)
Exposure range	(fiber/ml)	0.06-8.06
Worker's with exposure higher than PEL	%	95

could result in signs of lung fibrosis¹⁸⁾.

Correlation coefficients of FEV₁ and FVC adjusted for age and BMI of exposed total population with their cumulative exposure to asbestos (fiber/ml-yr) were significant (R=-0.37 and -0.26, respectively). Correlation coefficients of FEV₁ and FVC adjusted for age and BMI of exposed nonsmoker population with cumulative dose of exposure (fiber/ml-yr) were also significant (R=-0.40 and -0.48, respectively).

Discussion

Ninety five percent of workers in manufacturing brake shoe in this study had higher exposure than US Occupational of Safety and Health Administration (OSHA) permissible exposure level (PEL) at 0.1 fiber/ml. Since, acceptable risks according to OSHA, for carcinogens correspond to lifetime risks of less than 1 in thousand exposed populations²⁶⁾ and corresponding risks of lung cancer and asbestosis at OSHA's permissible exposure level are 5/1,000 and 2/1,000, respectively¹⁶), the permissible exposure level of 0.1 fiber/ml is debatable. By using the lung cancer risk model by EPA²⁴, excess risk of workers with cumulative dose of 0.14-53.68 fiber/mlyr were in range of 0.40-153.30 and 0.04-15.33 per thousand for smokers and nonsmokers, respectively. In comparison, according to IRIS²¹⁾ estimated excess risk of lung cancer for cumulative dose of 2-200 fiber/ml-yr was reported in range of 2-200 per thousand, which is an overestimate for nonsmoking population compared to results obtained in this study.

As stated by Gustavsson *et al.*²⁷⁾, cumulative exposure in low range (0–2.5 fiber/ml-yr) underestimates the true risk of cancer incidence. However, the majority of workers in this study, i.e. 52.5 percent had higher cumulative exposure than Gustavsson *et al*²⁷⁾. Lung cancer risks computed in this study according to the criteria stated by the EPA were substantial, especially for smokers. Generally, 59 percent of all exposed group from smoker and nonsmokers had excess risk of lung cancer more than one per thousand.

ATSDR has explored association of lung fibrosis with

Table 2. Risk assessment of lung cancer of workers based on quartile geometric mean of cumulative exposure (fiber/ml-yr)

	Mean of cumulative	Geometric mean of cumulative exposure	No. of workers (Risk per thousand)	
	exposure		Smoker	Non-smoker
0.02–0.54	0.21	0.14	8 (0.40)	8 (0.04)
0.55-4.74	1.59	1.42	6 (4.06)	9 (0.40)
4.75-30.82	12.32	10.98	4 (31.4)	11 (3.1)
30.83-110.77	58.27	53.68	7 (153.4)	8 (15.3)

cumulative exposure as low as 15–70 fiber/ml-yr¹⁸), and in this study 24.6 percent of workers had cumulative exposure in that range, which could have sign of lung fibrosis. Meanwhile, according to the WHO, 13.1 percent of our workers had cumulative exposures of 50–200 fiber/ml-yr, for which fibrotic changes were commonly reported¹⁷).

Asbestos mining and milling have well characterized relations with a number of hazards for the respiratory system^{28, 29)}. In the current study, lung function performances were used to support the results of risk assessment of asbestosis through lung restrictive functional damage. Generally, correlations of lung function parameters such as FEV1 and FVC adjusted for BMI of exposed groups were significant with cumulative exposure of workers. Recently, Bagatin et al.³⁰⁾ using a similar approach, also found a significant effect of lower cumulative exposure on lung function levels in Brazilian workers and role of control measures in reduction of nonmalignant respiratory morbidity in asbestos exposed occupational groups was demonstrated. To remove the interference of smoking with lung function³¹⁾, correlation of cumulative exposure with lung function parameters such as FEV1 and FVC adjusted for BMI from exposed nonsmoker group, were also studied in this study and found to be significant.

Regardless of variations in exposure between workers within job tasks, incomplete information on earlier exposure levels, and possible errors in work histories of subjects, results of this study demonstrated high risk of asbestos exposed group for development of asbestosis and lung cancer. Unfortunately, the Iranian brake shoe factory has not paid much attention for controlling worker's exposure and from the age and work experience of workers, it is evident that there is high turn over in working group and large number of workers (42.6 percent) have less than 1 yr of work history. Despite the high turn over of workers in brake shoe factory, their cumulative exposures are considerable compared with other exposed group abroad¹⁻⁴). It must be emphasized that aging of workers even with discontinuation of their employment at brake shoe factory, could be accompanied with higher risk of asbestos relevant diseases. Considering successes of control measures in term of substitution of asbestos as raw material, application of engineering control systems, improvement of work practices in reduction of occupational exposure to asbestos fibers and encouraging workers to quit smoking in developed countries^{30, 31)}, risk management of high risk exposed group could be rationalized.

References

1) LaDou J, Landrigan P, Bailar JC, Foa V, Frank A,

Collegium R (2001) A call for an international ban on asbestos. CMAJ **164**, 489–90.

- Paustenbach DJ, Finley BL, Lu ET, Brorby GP, Sheehan PJ (2004) Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to Present): a "State-of-the-Art" review. J Toxicol Environ Health 7, 25–80.
- Butnor KJ, Sporn TA, Roggli VL (2003) Exposure to brake dust and malignant mesothelioma: a study of 10 cases with mineral fiber analyses. Ann Occup Hyg 47, 325–30.
- 4) Oliveira MCB, Frazao EB, Coutinho JMV, Valarelli JV (1997) Technological characteristics of the serpentines in the Cana Brava Mine, GO, Brazil. Proceedings of the International Symposium on Mine Planning and Equipment, 77–80, AA Balkema, Rotterdam.
- Victor L, Roggli TA, Oury TD (2006) Occupational and environmental exposure to asbestos. In: Pathology of Asbestos-Associated Diseases, 2nd Ed., 17–33, Springer, New York.
- Becklake MR (1991) The epidemiology of asbestosis. In: Mineral fibers and health, Liddell D and Miller K (Eds.), 103–19, CRC Press, Boca Raton.
- 7) Emamhadi MA, Nouraei M, Tahbaz MO, Bakhshayes KM, Zahirifar S, Bahadori M, Masjedi MR (2004) Evaluation of Asbestos related pulmonary changes, a study on Hajat-Nehbandan Chrysotile mine & factory workers. Oroumieh Medical Journal 16, 128–35.
- Emamhadi MA, Halvaee A, Nouraei M, Jabari HR, Masjedi MR (2004) Evaluation of smoking effects on pulmonary function in Asbestos workers. Shahid Sadoughi Med J 4, 17–24.
- Becklake MR, Fournier-Massey G, Rossiter CE, McDonald JC (1972) Lung function in chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health 24, 401–9.
- 10) McDonald JC, Becklake MR, Gibbs GW, McDonald AD, Rossiter CE (1974) The health of chrysotile asbestos mine and mill workers of Quebec. Arch Environ Health 28, 61–8.
- Liddell D, Eyssen G, Thomas D, McDonald C (1975) Radiological changes over 20 years in relation to Chrysotile exposure in Quebec. In: Inhaled particles. Walton WH (Ed.), 799–813, Pergamon Press, Oxford.
- 12) Selikoff IJ (1977) Clinical survey of Chrysotile asbestos miners and millers in Baie Verte, Newfoundland-1976. Report to the National Institute of Environmental Health Sciences, Research Triangle Park.
- 13) Enarson DA, Embree V, MacLean L, Grzybowski S (1988) Respiratory health in chrysotile asbestos miners in British Columbia; a longitudinal study. Br J Ind Med 45, 459–63.
- Weiss W (1999) Asbestosis: a marker for the increased risk of lung cancer among workers exposed to asbestos. Chest 115, 536–49.
- 15) Robert CJ (2001) Risk Assessment. In: Principles of toxicology, environmental and industrial applications,

Williams PL, James RC and Roberts SM (Eds.), 437–77, Wiley & Sons, Hoboken.

- 16) Stayner L, Smith R, Bailer J, Gilbert S, Steenland K, Dement J, Brown D, Lemen R (1997) Exposureresponse analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. Occup Environ Med 54, 646–52.
- 17) WHO (1998) Chrysotile asbestos: environmental health criteria. World Health Organization, Geneva.
- 18) U.S. Department Of Health And Human Services (a). Toxicological Profile for Asbestos. Agency for Toxic Substances and Disease Registry. [Online] September 2001. http://www.atsdr.cdc.gov/toxprofiles/tp61.html. Accessed July 30, 2008.
- 19) Brodkin CA, Barnhart S, Anderson G, Checkoway H (1993) Correlation between respiratory symptoms and pulmonary function in asbestos-exposed workers. Am Rev Respir Dis 148, 32–7.
- Schwartz DA, Galvin JR, Yagla SJ, Speakman SB (1993) Restrictive lung function and asbestos-induced pleural fibrosis. A quantitative approach. J Clin Invest **91**, 2685–92.
- IRIS. Asbestos. Integrated Risk Information System US EPA. [Online] 2 22, 2001. http://www.epa.gov/iris/ subst/0371.htm. Accessed July 28, 2008.
- 22) U.S. Department Of Health And Human Services (b) (2001) Toxicological profile for asbestos. Agency for toxic substances and disease registry. http://www.atsdr. cdc.gov/toxprofiles/tp61.html. Accessed July 30, 2008.
- 23) US Department of Health and Human Services (1984) NIOSH manual of analytical methods, DHHS Publication No. 84–100. US Government Printing Office, Washington, DC.

- 24) Nicholson WJ (1986) Airborne asbestos health assessment update. U.S. Environmental Protection Agency, Washington, DC.
- American Thoracic Society (1995) Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 152, 1107–36.
- 26) Rodricks JV, Brett SM, Wrenn GC (1987) Significant risk decisions in federal regulatory agencies. Regul Toxicol Pharmacol 7, 307–20.
- 27) Gustavsson P, Nyberg F, Pershagen G, Scheele P, Jakobsson R, Plato N (2002) Low-dose exposure to asbestos and lung cancer: dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. Am J Epidemiol 155, 1016–22.
- 28) Chen CR, Chang HY, Suo J, Wang JD (1992) Occupational exposure and respiratory morbidity among asbestos workers in Taiwan. J Formos Med Assoc 91, 1138–42.
- 29) Algranti E, Mendonca EMC, DeCapitani E, Freitas JBP, Silva HC, Bussacos MA (2001) Non-malignant asbestos-related diseases in Brazilian asbestos-cement workers. Am J Ind Med 40, 240–54.
- 30) Bagatin E, Neder JA, Nery LE, Terra-Filho M, Kavakama J, Castelo A, Capelozzi V, Sette A, Kitamura S, Favero M, Moreira-Filho DC, Tavares R, Peres C, Becklake MR (2005) Non-malignant consequences of decreasing asbestos exposure in the Brazil chrysotile mines and mills. Occup Environ Med 62, 381–9.
- 31) Waage HP, Vatten LJ, Opedal E, Hilt B (1996) Lung function and respiratory symptoms related to changes in smoking habits in asbestos-exposed subjects. J Occup Environ Med 38, 178–83.