Comparative Health Risk Assessment of Asbestos in Tehran, Iran

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Abstract

Health risk assessment has been used to investigate the cancer and non-cancer risk of Asbestos in the air of Tehran, Iran. This study focused on the risk of lung cancer and mesothelioma on the residents of the region. It presents an overview of Asbestos concentration in 31 samples with the average concentration of 0.011/ml in different districts in Tehran. Results provided by EPA (IRIS) analysis showed the total lifetime cancer risk of 46.3 x 10⁻⁵. Based on the risk calculations presented in EPA (1986a), the average cancer risk value of lung cancer and mesothelioma was calculated as a discrete value for smokers and non-smokers. Assuming lifetime continuous exposure due to inhalation, the expected incidence is 46 and 152 mesothelioma deaths, and 42 and 13 lung cancer deaths per 100,000 persons for smokers and nonsmokers, respectively. In addition, In accordance with the Air Quality Guidelines of the World Health Organization database, the extra risk of lung cancer between 2.42x10⁻⁵ and 1.13x10⁻⁵, for smokers and 2.86x10⁻⁶ and 1.13x10⁻⁷ for nonsmokers was calculated.

Keywords: Health risk, Asbestos, EPA, Inhalation

1 Introduction

Asbestos is a general term given to a group of six different fibrous forms of highly durable silicate minerals, (amosite, chrysotile, crocidolite, and the fibrous varieties of tremolite, actinolite, and anthophyllite) that occur naturally in the environment. Chrysotile belongs to the serpentine family of minerals, while all of the others belong to the amphibole family [1].

Because asbestos is composed of silicates that has particularly interesting physicochemical properties such as flexibility and resistance to traction, heat, and chemical reactions, it is used commercially into numerous products of industrial activities such as cement, asphalt, and brake pads [2-5].

The primary diseases associated with asbestos exposure are asbestosis (caused by the inhalation and retention of asbestos fibers), mesothelioma (an otherwise rare form of cancer associated with the lining around the lungs), and lung cancer [6-10]. Inhaled asbestos, however, is reported carcinogenic in populations non-occupationally exposed to asbestos, giving rise to lung tumors and mesotheliomas [11-17]. Different factors are involved in the impacts of Asbestos on human health such as concentration of Asbestos, residence time, size, shape and chemical form of Asbestos fibers, source of emissions, individual risk agents like smoking and suffering from lung disease before affected by Asbestos.

Asbestos fibers have no detectable odor or taste. They do not dissolve in water or evaporate and are resistant to heat, fire, chemical and biological degradation [1]. Levels of asbestos (fibers.m⁻³) can be detected in almost any air sample (A cubic meter is the amount of air that we breathe in 1 hour).

By asbestos fibers inhalation into lungs, some of the fibers will be deposited in the air passages and on the cells that make up your lungs. Most fibers are removed from your lungs by being carried away or coughed up in a layer of mucus to the throat, where they are swallowed into the stomach. Amphibole asbestos fibers are retained in the lung longer than Chrysotile asbestos fibers [1]. One study found that nearly 70 percent of WTC rescue and recovery workers suffered new or worsened respiratory symptoms while performing work at the WTC site. The study describes the results of the WTC Worker and Volunteer Medical Screening Program, which was established to identify and characterize possible WTC-related health effects in responders. The study found that about 28 percent of those tested had abnormal lung function tests, and 61 percent of those without previous health problems developed respiratory symptoms [19].

Risk of lung cancer is expected to be up to ten times higher in smokers than in non-smokers exposed to the same inhaled asbestos concentrations [15, 6]. Therefore, in order to quantify degree of Asbestos risk, it is essential to assess health risk associated with Asbestos.

The case study's location is Tehran which is the most populated city in Iran with a population of approximately 8,245,000 people. Asbestos is vastly used in Tehran in a wide range of car brake caliper, manufactured products, mostly in building materials, friction products, and heat-resistant fabrics. In 1390, Tehran with land measurement of 730 square kilometers encompassed about 8 million and 245

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At the end of autumn 2013, the number of cars with license plate of Tehran exceeded 2 million and 422 thousand people. Tehran’s air pollution is critical for 318 days in 2010. Smokers in the population of Tehran, who are more inclined to the lung cancer risk, included 25.4 percent of the whole population [21]. Carcinogenic nature of asbestos (classified as Group I human carcinogens based on the International Agency for Research on Cancer), inferior building stuff, climate, geographic pattern and traffic promote this study to evaluate the recent asbestos exposure status in Tehran’s air.

Figure 1: Probable biomarkers which can be used to determine oxidative, inflammatory and genotoxic effects of fibers/particles, like asbestos, Adapted from Bhattacharya et al., 2005 [18]
2 Materials and Methods

Determination of the dose-response is the first step to start this study. For monitoring asbestos in air, a known volume of air is drawn through a 25-mm diameter cassette containing a mixed-cellulose ester filter (OSHA method ID-160). The cassette must be equipped with an electrically conductive 50-mm extension cowl. The sampling time and rate are chosen to give a fiber density of between 100 to 1,300 fibers.mm\(^{-2}\) on the filter. A portion of the sample filter is cleared and prepared for asbestos fiber counting by Phase Contrast Microscopy (PCM) at 400X [22]. Sampling rate was 1.5 L.min\(^{-1}\), air volume was 2000 Liter, the instruments were calibrated, and the detection limit is 0.001 fibers.cc\(^{-1}\). There are thirteen monitoring stations located in the city. The monitoring data for asbestos is consisting of values of concentrations observed at each station in different dates from months of 2010. The monitoring station data were obtained from the Air quality control company, City of Tehran. Exposure assessments on Tehran’s population were conducted based on the route of inhalation. The input parameters for evaluating the exposure assessment and risk calculations are summarized in Table 1.

The equations used to calculate the chronic daily intakes are shown below:

\[
CDI_{\text{inhal}} = \frac{C_{\text{air}} \times EF \times ED \times ET}{AT} \quad (\text{Eq. 1})
\]

Where, CDI is chronic daily intake (f.ml\(^{-1}\)), \(C_{\text{air}}\) is mean concentration of asbestos in Tehran’s air (f.ml\(^{-1}\)), EF is number of days exposed to Asbestos in a year with assumption of 350 days, year\(^{-1}\), ED is average age equal to 70 years, ET is number of hours exposed to Asbestos during a day with assumption of 5 hours, day\(^{-1}\) inhalation in the open air, and AT is possible time for Tehran residents being threatened by cancer which is equal to life-time. In order to assess total cancer risk caused by inhalation of Asbestos equations used is shown below:

\[
\text{Total Risk} = CDI \times UR \quad (\text{Eq. 2})
\]

where, UR summarizes the cancer unit risk for inhalation of asbestos (f.ml\(^{-1}\)). These values were taken from California EPA or IRIS (1986). The lifetime cancer risk for inhabitants of Tehran was calculated using the asbestos concentrations and the input parameters above mentioned.

In addition, this study provides the calculation of life time cancer risk due to exposure assessment of asbestos based on other scientific researches. A linear dose-response relationship was assumed for lung cancer and mesothelioma respectively, and the corresponding slopes were defined as potency factors: \(K_l\) for lung cancer and \(K_M\) for mesothelioma [2]. Most studies specially evaluated 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 [1]:

\[
\text{Relative RISK} = 1.00 + K_l \times \text{(cumulative dose)} \quad (\text{Eq. 3})
\]

Using this equation, EPA (1986a) calculated the value of KL (the fractional increase in relative risk of lung cancer per f-year.ml\(^{-1}\)) for 14 sets of lung cancer mortality data from the past studies. The resulting geometric mean value was 0.010 (f-yr/ml\(^{-1}\)) [1]. Based on national average lung cancer risk data for male and female and smokers and nonsmokers, EPA (1986a) calculated that lifetime exposure to 0.0001 f.ml\(^{-1}\) corresponded to the excess lung cancer risks [1]. For the purposes of calculating risk levels of lung cancer, the results were presented for both men and women, and smokers and nonsmokers. Also, based on several studies on Mesothelioma, EPA (1986a) fit exposure-incidence data from four studies to the following equation [23-26]:

\[
\text{Incidence} = KM \times f \times [(T - 10)^3 - (T - 10 - d)^3] \quad (\text{Eq. 4})
\]

In which KM equals an empirical constant, f is intensity of exposure (f.ml\(^{-1}\)), \(T\) is latency (years since first exposure) and d is the duration of exposure (years). Based on an analysis of the relative cancer risk of mesothelioma, a value of 1x10\(^8\) was identified as the most reasonable estimate for KM (method EPA 1986a) [1]. Finally, there is another practical assumption for a quantitative risk assessment of lung cancer due to inhalation of asbestos presented in the paper (method WHO 1987). However, the lung cancer incidence can still be expressed as a linear function as [27]:

\[
I_L = \frac{f}{\text{age,smoking, ...}} \times \text{fibre exposure} = f_L (\text{age,smoking, ...}) \times [1 + K_L \times f \times d] \quad (\text{Eq. 5})
\]

where \(f_L\) the underlying lung-cancer is risk with no asbestos exposure, and \(K_L\) is a constant representing the increase in relative risk due to exposure to asbestos.

Table 1: Input parameters for exposure assessment

<table>
<thead>
<tr>
<th>Input parameter</th>
<th>Unit</th>
<th>Value</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unit Risk</td>
<td>f.ml(^{-1})</td>
<td>0.23</td>
<td>IRIS</td>
</tr>
<tr>
<td>Concentration of asbestos</td>
<td>f.ml(^{-1})</td>
<td>0.010068977</td>
<td>Air quality control co. (2011)</td>
</tr>
<tr>
<td>Exposure frequency (EF)</td>
<td>Days per year</td>
<td>350</td>
<td>Lee et al. (2004)</td>
</tr>
<tr>
<td>Exposure duration (ED)</td>
<td>years</td>
<td>70</td>
<td>Lee et al. (2004)</td>
</tr>
<tr>
<td>Exposure time (ET)</td>
<td>Hours per day</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Average lifetime (AT)</td>
<td>days</td>
<td>70*365</td>
<td>Lee et al. (2004)</td>
</tr>
</tbody>
</table>

The US Environmental Protection Agency (1985) and World Health Organization (1987) found values for \(K_L\) ranging from 0.0004 to 0.016 (expressed per fiber-year per ml air = f-y.ml\(^{-1}\)) [28, 29]. Smoking has no bearing upon the risk of mesothelioma, but increases the risk of lung cancer from asbestos exposure approximately tenfold. The World Health Organization (1987) used a range of \(K_L\) to calculate a range for the lifetime lung-cancer risk due to asbestos.
exposure, assuming that the risk of lung cancer in the absence of asbestos exposure (I°) is roughly 10% for smokers and ten times lower for non-smokers [27]. So, the risk evaluation includes all the above-mentioned calculations are presented in results.

3 Results and Discussion

1.1 Spatial distribution of asbestos air contamination

The spatial distribution map of asbestos air contamination (Figure 2) showed the maximum concentrations in east districts with 0.024 f.mL⁻¹ while the lowest concentrations in north districts were 0.006 f.mL⁻¹ and the mean concentration were seen 0.01 f.mL⁻¹. In addition, the lowest contaminant levels for asbestos are predominantly along with a north-south axis.

This study shows that the maximum and minimum concentrations of asbestos were seen in point 12 and 2 with 0.027 and 0.004 f.mL⁻¹ respectively, while the mean concentrations were 0.010 f.mL⁻¹. Although the variance in sample point 2 is high, the median asbestos concentrations for most points are near the mean values (Table 2). The occupational exposure level of 0.1 f.mL⁻¹ is equivalent to the PEL proposed by OSHA.

3.2 Lifetime cancer risk of asbestos

Using the average concentration of each THM species, the lifetime cancer risks through inhalation, was calculated based on the assumptions on Table 1, Equation 1 and Equation 2. Table 3 presented the result for CDI and total cancer risk due to asbestos exposure (inhalation). As it is shown, the expected incidence is 47 deaths per 100,000 persons.

The results of these calculations from lung cancer indicate that the concentration of 0.01007 f.mL⁻¹ corresponds to a lifetime excess risk level of 25×10⁻⁵ and 2×10⁻⁵ for men, smoker and nonsmoker, and 15×10⁻⁵ and 2×10⁻⁵ for women, smoker and nonsmoker, respectively (Figure 3). The data show that asbestos exposure and cigarette smoking do not interact with mesothelioma. The research estimates lifetime cancer risks from mesothelioma based on levels of asbestos detected 18×10⁻⁵ and 23×10⁻⁵ for men, smoker and nonsmoker, and 26×10⁻⁵ and 28×10⁻⁵ for women, smoker and nonsmoker, respectively (Figure 4). Assuming the population of 6,150,000 nonsmokers in Tehran, the expected incidence is 13 lung cancer deaths and 152 mesothelioma deaths per 100,000 persons. The results estimated that continuous lifetime exposure to air containing 0.01007 f.mL⁻¹ of asbestos for a population of 2,100,000 smokers in Tehran would result in about 42 cases of lung cancer and 46 cases of mesothelioma per 100,000 persons.

Table 3: Chronic daily index and lifetime cancer risk via inhalation of asbestos in Tehran

<table>
<thead>
<tr>
<th>CDI (f/ml)</th>
<th>0.002011497</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer Risk</td>
<td>4.63E-04</td>
</tr>
</tbody>
</table>
Table 2: Statistical information for all water districts

<table>
<thead>
<tr>
<th>Points</th>
<th>Minimum asbestos (f.mL⁻¹)</th>
<th>Maximum asbestos (f.mL⁻¹)</th>
<th>Mean asbestos (f.mL⁻¹)</th>
<th>Median asbestos (f.mL⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. 1</td>
<td>0.0032</td>
<td>0.0098</td>
<td>0.007075</td>
<td>0.00765</td>
</tr>
<tr>
<td>No. 2</td>
<td>8.3E-06</td>
<td>0.009</td>
<td>0.004504</td>
<td>0.004504</td>
</tr>
<tr>
<td>No. 3</td>
<td>0.008</td>
<td>0.008</td>
<td>0.008</td>
<td>0.008</td>
</tr>
<tr>
<td>No. 4</td>
<td>0.0071</td>
<td>0.0071</td>
<td>0.0071</td>
<td>0.0071</td>
</tr>
<tr>
<td>No. 5</td>
<td>0.005</td>
<td>0.005</td>
<td>0.005</td>
<td>0.005</td>
</tr>
<tr>
<td>No. 6</td>
<td>0.008</td>
<td>0.008</td>
<td>0.008</td>
<td>0.008</td>
</tr>
<tr>
<td>No. 7</td>
<td>0.007</td>
<td>0.017</td>
<td>0.012</td>
<td>0.012</td>
</tr>
<tr>
<td>No. 8</td>
<td>0.0041</td>
<td>0.0041</td>
<td>0.0041</td>
<td>0.0041</td>
</tr>
<tr>
<td>No. 9</td>
<td>0.005</td>
<td>0.012</td>
<td>0.008333</td>
<td>0.008</td>
</tr>
<tr>
<td>No. 10</td>
<td>0.001</td>
<td>0.0058</td>
<td>0.00376</td>
<td>0.0048</td>
</tr>
<tr>
<td>No. 11</td>
<td>0.001</td>
<td>0.021</td>
<td>0.008825</td>
<td>0.00665</td>
</tr>
<tr>
<td>No. 12</td>
<td>0.00053</td>
<td>0.097</td>
<td>0.026808</td>
<td>0.00485</td>
</tr>
<tr>
<td>No. 13</td>
<td>0.018</td>
<td>0.018</td>
<td>0.018</td>
<td>0.018</td>
</tr>
<tr>
<td>Tot.</td>
<td>0.00000083</td>
<td>0.097</td>
<td>0.010069</td>
<td>0.007</td>
</tr>
</tbody>
</table>

As it has mentioned, the World Health Organization (1987) used another equation to calculate a range for the lifetime lung-cancer risk due to asbestos exposure. With the lowest and highest values of 

\[ K_i \] (0.0004 and 0.016 f-yr.mL⁻¹, respectively) calculated by Liddell from cohort studies, a lifetime lung-cancer risk after exposure to 0.01007 f.mL⁻¹ of asbestos can be estimated. The extra risk of lung cancer is between 2.42×10⁻⁶ and 1.13×10⁻³, for smokers and 2.86×10⁻⁶ and 1.13×10⁻³ for nonsmokers (Figure 5). So, the average extra risk of lung cancer for a population with 25.4% smokers would be 1.9×10⁻⁴. The approximate content of the risk of lung cancer due to asbestos exposure estimated in this way is 19 lung cancer deaths per 100,000 persons.

4 Conclusion

This study provided a lifetime general population risk assessment of asbestos inhalation in which the estimated value of the risk was assessed seriously high. The southeast and western part of Tehran inhaled mostly polluted air which leads to higher cancer cases caused by asbestos fibers. The determination of cancers for 8,245,000 total inhabitants is close to 50 per 100,000. Also, the expected incidence is estimated 13 lung cancer deaths and 152 mesothelioma deaths per 100,000 persons among nonsmokers and 42 cases of lung cancer and 46 cases of mesothelioma per 100,000 persons among smokers (method EPA 1986a). According to WHO 1987 guidelines the extra risk of lung cancer accounts for a range between 2.42×10⁻⁶ and 1.13×10⁻³, for smokers and 2.86×10⁻⁶ and 1.13×10⁻³ for nonsmokers which will probably be acceptable in comparison with other methods. However; it is important to keep in mind that the validity of the given risk calculations is difficult to judge but it surely helps risk management considerations to make a good choice for the general population of Tehran.

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