Respiratory Health Following Long Term Occupational Exposure to Fiberglass Dust

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Abstract

Background: Despite the wide application of fiberglass, its respiratory effects have not been extensively studied. The purpose of this study was to further examine this issue among a group of workers occupationally exposed to this compound.

Methods: This is a historical-cohort study in which the respiratory health of 49 workers exposed to fiberglass as well as 42 unexposed employees as the referent group from a local fiberglass industry was evaluated. A standardized respiratory questionnaire was used. The subjects underwent chest x-ray and were examined by a physician for any possible respiratory abnormalities. Furthermore, pulmonary function tests were performed just before the work shift. Moreover, to assess the extent to which subjects had been exposed to fiberglass dust, respirable dust concentrations were measured in different dusty work sites, using standard methods.

Results: The average age of the exposed subjects and the duration of exposure were 39.6±7.34 and 11.2±5.59 years, respectively. Atmospheric concentrations of respirable fraction of fiberglass dust in dusty work sites, namely Line and Tissue units, were found to be 44.5 and 6.27 mg/m³, respectively. The prevalence of respiratory symptoms and abnormalities in the chest x-ray of the exposed and unexposed subjects was not significantly different. Lung function parameters, i.e., vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in the first second (FEV₁), and percentage ratio of FEV₁/FVC in the exposed workers were comparable with those of the control subjects. Significant associations between the length of exposure to fiberglass and age with FEV₁/FVC ratio were noted.

Conclusion: The results support that exposure to fiberglass dust is unlikely to be associated with respiratory symptoms, abnormal radiographic changes or functional impairments of the lungs.

Keywords: Fiberglass; Respiratory symptoms; Pulmonary function test; Occupational exposure; Chest x-ray

Introduction

Fiberglass is extensively used in the production of acoustic and thermal insulators and the manufacture of textiles to reinforce plastic materials. Fiberglass is an amorphous silicate manufactured from glass.¹ Fiberglass and other synthetic mineral fibers have replaced asbestos, a known human carcinogen, and their potential health effects have attracted more attention over the last three decades.²

The health effects of fiberglass, in general, and its respiratory effects, in particular, are diverse and controversial.³ Length, diameter and length to width ratio of synthetic fibers are critical factors in the expression of their toxicities.⁴,⁵ Intrapleural injection of synthetic fibers dispersed in saline in animal models resulted in formation of pleural tumors (mesothelioma).⁶ An increased risk of cancer and mortality associated with exposure to man made mineral fibers was also reported.⁶-¹⁰ However, these findings have not been reproduced by others.¹¹-¹³ Similarly, in some studies, exposure to fiberglass has been reported to be associated with lung fibrosis,⁵,¹⁴ chronic bronchitis,⁵,¹⁵ parenchymal disease of the lungs¹⁶ and pneumonia.¹⁷ Interestingly, even in some of these studies, the excess risk of mortality has been attributed to the
non-malignant respiratory diseases.\(^\text{17}\) On the contrary, in some studies no significant associations have been observed between exposure to fiberglass and the prevalence of these disorders or any other form of obstructive or restrictive pulmonary diseases.\(^\text{15}\) National Institute for Occupational Health and Safety (NIOSH) states that fibers having diameters less than 3.5 \(\mu m\) had not been extensively used until 1960. Therefore, neither sufficient exposure has taken place, nor enough time has lapsed for the chronic effects of such fibers, if any, to be manifested. Therefore, NIOSH recommends further studies to be undertaken to determine the effects of long term exposure to fibers with different dimensions.\(^\text{18}\) The purpose of this study was, therefore, to assess, more thoroughly, the pulmonary response to this compound, if any, after long term occupational inhalation exposure.

**Materials and Methods**

This historical cohort study was carried out at a local factory producing fiberglass in Shiraz, Southern Iran. The respiratory health of all 49 fiberglass exposed workers and 42 unexposed employees (referent group) from the same industry with almost identical socioeconomic and demographic status (sex, ethnic background, education, smoking habits, income as well as family size) was evaluated.

Both groups were volunteer subjects. No selected subjects refused to participate in the study. None of the exposed subjects had a past medical or family history of respiratory illnesses or any other chest operations or injuries as the exclusion criteria of the study. Similarly, none of the referent subjects had been exposed to fiberglass or other chemicals known to cause respiratory symptoms, ventilatory disorders or abnormal radiographic changes in the lungs during their employment in the plant or prior to it. The prevalence of respiratory problems and changes in the parameters of lung function were studied among the above-mentioned exposed and unexposed groups.

The subjects were interviewed and a respiratory symptom questionnaire, as suggested by the American Thoracic Society\(^\text{19}\) (ATS 1978), with a few modifications, was applied for all. This standardized questionnaire included questions on respiratory symptoms (presence or absence of regular dry and/or productive cough, wheezing, shortness of breath, etc.), smoking habits and medical and family history of each subject. Additionally, it contained detailed occupational history and specific questions concerning all the jobs undertaken before employment at the plant under study, particularly those associated with the risk of respiratory morbidity. These were then used to obtain symptom prevalence data among exposed and unexposed groups.

To assess the extent to which the subjects had been exposed to fiberglass dust, standard protocols\(^\text{16}\) personal dust monitoring for respirable fraction was used in different dusty work sites. These included two major areas of Tissue and Line units. For estimation of the atmospheric dust concentration in these units, a personal dust sampler (Casella, London, UK), calibrated by a digital automatic calibrator connected to a filter holder equipped with a 25 mm cellulose membrane filter (pore size 0.8\(\mu m\)) through which air was pumped by a battery-powered motor at a constant flow rate of 3.5 lit/min was used. Based on a few preliminary tests, to avoid overloading of the filters, the optimum sampling time was evaluated to be a few minutes. Dust concentration expressed in mg/m\(^3\) was calculated from the changes in the dried filter for respirable fraction as measured by a digital scale at a sensitivity of 0.1 mg, before and after sampling, divided by the volume of the air sampled.

Pulmonary Function Tests (PFTs) including mean percentage predicted Vital Capacity (VC), Forced Vital Capacity (FVC), Forced Expiratory Volume in the first second (FEV\(_1\)), Peak Expiratory Flow (PEF), Forced Expiratory Flow between 25\% and 75\% of the FVC (FEF\(_{25-75}\)), were done with a portable calibrated vitalograph spirometer (Vitalograph- COMPACT, Buckingham- England) on site, following the guidelines given by the ATS (1979)\(^\text{20}\) and the percentage predicted lung values were observed capacities as measured by spirometer device divided by predicted or expected capacities (based on gender, age, weight, height, ethnic background, etc, as calculated and adjusted by spirometer device) multiplied by 100.

The subjects who had been annually examined, as part of their periodic medical examinations, by the resident physician of the industry, were referred to a medical center and underwent Posterior-Anterior (PA) chest x-ray, using a Siemens instrument. Standard PA chest X-rays being read and reported by a radiologist. The size of the film was 35 X 35 cm, the distance of the subject with X-ray tube was about six feet, and the electrical voltage was 100 kv.

The data were statistically analyzed, using student's
t and Chi-Square tests or Fisher exact test, where applicable (with a preset probability of \( p < 0.05 \)). Experimental results are presented as arithmetic mean±SD. Statistical tests were conducted, using SPSS software (version 11.5, Chicago, IL, USA) on a personal computer. Additionally, the simultaneous effects of confounding variables such as smoking, age, duration of exposure on the prevalence of respiratory symptoms and changes in the parameters of pulmonary function were evaluated, using simple and multiple logistic regression analysis. As all of the individuals with current exposure to fiberglass were included in the study, statistical power was not applicable to our study. However, one should note that in some studies\(^{21}\) the incidence rate of asthma among fiberglass exposed workers has been reported to be about 14%. Given this figure and an \( \alpha=0.05 \) and a statistical power of 80%, the sample size is estimated to be 48.

### Results

Age, duration of exposure to fiberglass dust and the number of smokers are shown in Table 1. As shown, the mean±SD of age and duration of exposure to dust for the exposed group were 39.6±7.34 and 11.2±5.59 years, respectively. The corresponding values for the referent group were 42.76±7.57 and ±0. Atmospheric concentrations of respirable fractions of fiberglass dust in Line and Tissue units were found to be 44.5 and 6.27 mg/m\(^3\), respectively. These data indicate that unexposed subjects are, to some extent, older than their exposed counterparts, although other socioeconomic and demographic factors (ethnic background, level of education, income and family size) of both groups were identical and their data are not shown for the sake of clarity.

The results of lung function tests are presented in Table 2. As shown, exposure to fiberglass dust was not associated with significant decrements in VC, FVC, FEV\(_1\) values or FEV/FVC ratio.

Table 3 illustrates the frequency of abnormal findings in chest radiographs of exposed and referent subjects. As displayed, most of the radiographs of both groups were normal (chests were clear). Additionally, no significant differences were noted between the prevalence of abnormal radiographic findings (emphysematous changes, focal calcification, chronic inflammatory process and fibrotic changes) in both groups. The odd ratios (OR) for developing abnormal

### Table 1: Physical characteristics and exposure data (mean±SD) and smoking habits of study subject

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n=49)</th>
<th>Unexposed (n=42)</th>
<th>( p )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age(yr)</td>
<td>39.6±7.34</td>
<td>42.76±7.57</td>
<td>0.043*</td>
</tr>
<tr>
<td>Duration of exposure to dust(yr)</td>
<td>11.2±5.59</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Number of smokers</td>
<td>19 (38.76 %)</td>
<td>10 (22.22%)</td>
<td>0.273**</td>
</tr>
</tbody>
</table>

\* Mean values were significantly different (student’s t test), **Number of smokers were not significantly different (Fisher’s Exact test, \( p>0.05 \)), NA: not applicable

### Table 2: Percentage predicted lung function among exposed and referent subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n=49)</th>
<th>Unexposed (n=42)</th>
<th>( p )-value(^{11})</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC</td>
<td>79.58±16.00</td>
<td>82.55±14.37</td>
<td>0.359</td>
</tr>
<tr>
<td>FVC</td>
<td>95.71±19.95</td>
<td>92.9±18.81</td>
<td>0.493</td>
</tr>
<tr>
<td>FEV(_1)</td>
<td>103.27±18.51</td>
<td>102.37±19.99</td>
<td>0.827</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
<td>107.53±9.13</td>
<td>110.12±8.18</td>
<td>0.168</td>
</tr>
</tbody>
</table>

\(^{11}\) There was no statistically significant difference between mean values (student’s t. test, \( p>0.05 \)).

### Table 3: Frequency (percent) and odds ratio (%95 CI) of abnormal findings in chest radiographs of exposed and unexposed subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n=49)</th>
<th>Unexposed (n=42)</th>
<th>Odds ratio (CI)</th>
<th>( p )-value(^{11})</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Normal</td>
<td>37 (75.5)</td>
<td>33 (78.6)</td>
<td>1</td>
<td>NA</td>
</tr>
<tr>
<td>Emphysematous changes</td>
<td>10 (20.4)</td>
<td>5 (11.9)</td>
<td>1.78 (0.55-5.76)</td>
<td>0.247</td>
</tr>
<tr>
<td>Focal calcification of lungs</td>
<td>1 (2)</td>
<td>0 (0)</td>
<td>0.97 (0.92-1.03)</td>
<td>0.535</td>
</tr>
<tr>
<td>Chronic inflammatory process</td>
<td>1 (2)</td>
<td>3 (7.1)</td>
<td>0.30 (0.03-2.99)</td>
<td>0.287</td>
</tr>
<tr>
<td>Fibrotic changes</td>
<td>0 (0)</td>
<td>1 (2.4)</td>
<td>1.03 (0.97-1.09)</td>
<td>0.497</td>
</tr>
</tbody>
</table>

\(^{11}\) there was no statistically significantly difference between mean values (chi-square and fisher’s exact test, \( p>0.05 \)), NA: not applicable
changes in chest radiographs after exposure to fiberglass dust are also presented in Table 3. As shown, none of these ORs reached statistical significance.

The association between age and exposure to fiberglass dust and changes in the parameters of pulmonary function was studied, using simple linear regression analysis. As shown in Table 4, while there was a general tendency for these values to become smaller as estimated age and cumulative exposure increased, none, but FEV1/FVC ratio reached statistical significance.

Similar observations were made, using stepwise multiple linear regression analysis, including independent variables of age, smoking and length of exposure to fiberglass dust in the model. A significant linear relationship between FEV1/FVC ratio and age was observed, using log transformed data for the length of exposure. The regression coefficients in this model were as follows: constant coefficient (88.486, \(p<0.0001\)), age variable coefficient (0.856, \(p=0.001\)), and variable coefficient of (log transformed) duration of exposure (-15.277, \(p=0.017\)).

The odd ratios (ORs) of both groups for developing abnormal PFT (FEV1<80, FVC<80, FEV1/FVC < 75, restrictive or obstructive pattern)\(^{22}\) were not significantly different (\(p>0.05\)).

**Discussion**

Given the data provided, there should be very few socioeconomic and demographic differences between exposed and unexposed subjects because they were from the same industry with almost identical age, annual income, length of employment, education, sex, ethnic background, smoking habits, etc. Additionally, none of the subjects had a past medical or family history of respiratory illnesses or any other chest operations or injuries. Furthermore, confounding variables were controlled and accounted for in this study. Therefore, the absence of any significant differences in the prevalence of respiratory symptoms and radiographic changes as well as similar values for the parameters of lung function in exposed and referent subjects may well indicate that exposure to fiberglass is unlikely to induce respiratory symptoms, ventilatory disorders or radiographic abnormalities in the lungs. Similar findings have been reported by others.\(^{21,23,24}\)

It is known that at least 15% of the lung small airways are to be involved and a decrement of about 50% in FEV1 is required before a subject is likely to complain from a reduced ventilatory capacity and the appearance of respiratory abnormalities.\(^{19,25,26}\) This could explain the negative findings of this study and indicates that under the exposure scenario explained in this study, fiberglass is unlikely to adversely affect the workers’ respiratory health.

Conversely, the findings of some other studies indicate that respiratory symptoms have been associated with fiberglass exposure.\(^{6,15}\) This discrepancy could be explained, at least in part, by a difference in the subjects’ age, smoking habits, length of exposure, past occupations, concentration and particle size of the dust to which they had been exposed. Likewise, some authors have shown that chronic bronchitis as well as other respiratory disorders associated with fiberglass increase the risk of contracting coronary disease and coronary deaths.\(^{27,28}\)

In our study, most of the chest x-rays were clear. This finding which corresponds well with the results of PFT and with the absence of any significant in-

<table>
<thead>
<tr>
<th>Parameter</th>
<th>constant coefficient (p-value)</th>
<th>Regression coefficient (p-value)</th>
<th>constant coefficient (p-value)</th>
<th>Regression coefficient (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC</td>
<td>90.464 (&lt;0.001)</td>
<td>-0.275 (0.388)</td>
<td>79.991 (&lt;0.001)</td>
<td>0.07 (0.76)</td>
</tr>
<tr>
<td>FVC</td>
<td>122.182 (&lt;0.001)</td>
<td>-0.668 (0.089)</td>
<td>97.066 (&lt;0.001)</td>
<td>-0.199 (0.492)</td>
</tr>
<tr>
<td>FEV1</td>
<td>119.744 (&lt;0.001)</td>
<td>-0.414 (0.277)</td>
<td>104.613 (&lt;0.001)</td>
<td>-0.133 (0.646)</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>87.707* (&lt;0.001)</td>
<td>0.498* (0.006)</td>
<td>106.637 (&lt;0.001)</td>
<td>0.155 (0.240)</td>
</tr>
</tbody>
</table>

\(^{(*)}\)Simple linear regression analysis, *Statistically significant different (\(p<0.05\))
crease in the prevalence of respiratory symptoms among exposed employees is also in full agreement with the observations of Hill et al., Janet et al., Wright et al. and Nasr et al. In contrast, using the criteria of International Labor Organization (ILO), Kilburn et al. have shown pathologic abnormalities in the chest radiographs of fiberglass exposed subjects, although it appears that the role of potential confounders such as cigarette smoking has not been adequately controlled.

The absence of any significant differences in the VC, FVC, FEV/FVC ratios of exposed and referent subjects observed in our study is similar to and in line with the findings of other studies. When these investigators controlled the effects of confounders, no significant decrements in the parameters of pulmonary functions of fiberglass workers were noted.

Although significant associations between the length of exposure to fiberglass dust, age and cigarette smoking with VC, FVC and FEV₁ were not observed, a significant and reverse association between age and FEV₁/FVC was noted, i.e. with increasing age, FEV₁/FVC ratio decreased (0.482 unit per year). Although, to the best of our knowledge, this has not been previously reported by others, the role of age on decreasing FEV₁ value in fiberglass workers has been demonstrated earlier.

However, one should not ignore the physiologic function of growth and age, per se, on reducing the FEV₁ value. For instance, while Miller et al. report this coefficient for fiberglass workers for each year of age to be -0.0233 unit, the corresponding values reported by Knudson et al. and Cotes et al. only for each year of growth and aging in the subjects without exposure to fiberglass are - 0.0292 and - 0.033, respectively. These figures are higher than that reported by Miller for age and exposure.

In conclusion, our data are in line with and provide further evidence to support the notion that fiberglass, even at concentrations higher than its current TLV of 5 mg/m³, is unlikely to induce respiratory symptoms, ventilatory disorders or abnormal changes in chest radiographs. However, given the small sample size of our study as well as some other unavoidable limitations such as length and intensity of exposure, additional studies with larger sample sizes and more sufficient follow up as well as employing populations exposed to higher atmospheric concentrations of fiberglass are clearly needed to conclusively demonstrate the presence or lack of a causal relationship between fiberglass exposure and respiratory morbidity.

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Conflict of interest: None declared.

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Neghab et al.


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