Work-related Respiratory Symptoms and Ventilatory Disorders among Employees of a Cement Industry in Shiraz, Iran

Masoud Neghab and Alireza Choobineh

Abstract: Work-related Respiratory Symptoms and Ventilatory Disorders among Employees of a Cement Industry in Shiraz, Iran: Masoud Neghab, et al. Occupational Health Department, School of Health, Shiraz University of Medical Sciences, Iran—Although the main hazard in cement processing is dust and respiratory tract disorders are the most important group of occupational diseases in this industry, evidence for associations between exposure to cement dust and either respiratory symptoms or functional impairment has not been conclusive. This study was, therefore, undertaken to more thoroughly examine the effects of occupational exposure to cement dust on the respiratory system. The study population consisted of a group of 88, randomly selected, male workers with current exposure to cement dust and 80 healthy male office workers without present or past history of exposure to dust that served as the referent group. Subjects were interviewed and were given respiratory symptom questionnaires to answer. They also underwent chest X-ray and lung function tests. Additionally, personal dust monitoring for airborne inhalable and respirable dust was carried out at nine different worksites. Moreover, X-ray diffraction (XRD) and X-ray fluorescence (XRF) techniques were performed to determine the silica phases and the SiO₂ contents of the dust samples. Levels of exposures to inhalable and respirable cement dust were estimated to be 53.4 ± 42.6 and 26 ± 14.2 mg/m³, respectively (Mean ± SD). Statistical analysis of the data revealed that symptoms like regular cough, phlegm, wheezing and shortness of breath were significantly (p<0.05) more prevalent among exposed workers. Similarly, chest radiographs of exposed workers showed various degrees of abnormalities including emphysematous changes, old calcified granulomas, emphysematous changes associated with inflammatory processes, evidence of chronic inflammatory processes, focal calcification of the lungs and infiltrative changes. However, no significant changes were noted in the radiographs of the referent group. Furthermore, exposed workers compared to their referent counterparts showed significant reductions in the parameters of lung function. In conclusions, our data provide corroborative evidence further substantiating the contention that exposure to cement dust is associated with respiratory symptoms and functional impairments. (J Occup Health 2007; 49: 273–278)

Key words: Portland cement dust, Pulmonary function, Functional impairment, Respiratory symptoms

Portland cement (commonly known as cement) is a fine, grayish green powder, with an aerodynamic diameter ranging from 0.05 to 5.0 μm, which is produced by heating ground cement rock or other limestone-bearing materials into a fused clinker that is then ground into a fine powder. It is composed of lime, alumina, silica, and iron oxide as tetra calcium alumino ferrate (4 CaO. Al₂O₃. Fe₂O₃), tricalcium aluminate (3CaO, Al₂O₃), tricalcium silicate (3CaO. SiO₂), and dicalcium silicate (2CaO. SiO₂). Small amounts of magnesia (MgO), Na, K, and S are also present. Sand is added to make concrete. Because most Portland cement is used in the construction of buildings, consumption parallels building trends. A high amount of cement is yearly produced in Iran. At the Shiraz cement factory, daily production reaches 25,000 to 30,000 tonnes. This indicates that large numbers of workers are occupationally exposed to cement dust.

The main occupational hazard during cement processing is dust. However, high ambient temperatures, radiant heat and high noise levels are also common. Pathological conditions encountered in cement industry workers include diseases of the respiratory system, digestive disorders, skin diseases, rheumatic and nervous conditions, and hearing and visual disorders.

Although the main hazard in cement processing is dust, and respiratory tract disorders are the most important
group of occupational diseases in this industry, evidence for associations between exposure to cement dust and either respiratory symptoms or functional impairment has not been conclusive\textsuperscript{4–7–9)). Additionally, the potential adverse health effects of Portland cement have not been extensively studied\textsuperscript{10}). Therefore the main purpose of this study was to more thoroughly examine the effects of occupational exposure to Portland cement dust on the respiratory system of workers at a local cement producing factory in Shiraz, Iran.

**Materials and Methods**

**Study subjects**

This cross-sectional study was carried out at a local cement-producing factory in Shiraz, Iran. A total of 190 workers with past history and current exposure to cement dust were found to be eligible for the study. They were working in three major dusty areas, as detailed in the section on measurement of atmospheric dust. After consultation with statisticians and epidemiologists, 88 of these subjects were selected based on their current job classification by stratified random sampling.

Similarly, 80 healthy male office unexposed employees as the referent group were randomly selected from persons who were employed in the same industry with identical sex and ethnic background, and almost the same education and income as well as family size. Both groups were volunteer subjects. No selected subject refused to participate in the study. Additionally, the study was conducted in accordance with the Helsinki Declaration of 1964 as revised in 1989. All of the participants signed an informed consent form before commencement of the study. The study was reviewed and approved by ethics committee of Shiraz University of Medical Science.

The prevalence of respiratory problems and changes in the parameters of lung function were studied among the above mentioned exposed and unexposed groups. None of the exposed subjects had past medical or family history of respiratory illnesses or any other chest operations or injuries. Similarly, none of the referent subjects had been exposed to cement or other chemicals known to cause respiratory symptoms, ventilatory disorders or abnormal radiographic changes in the lungs during their employment with the plant or prior to it. However, based on the preliminary interviews conducted by the authors, prior to subjects’ participation in the study, and referring to the pre-employment medical examinations history, three of the randomly selected unexposed individuals had past history (prior to their employment in the site) of TB (fully treated before employment), asthma and allergic rhinitis (also currently suffering from it). In order to minimize the effects of confounding variables, these three were not included in the study.

**Measurements of study variables**

**Respiratory illness:** Subjects were interviewed and the respiratory symptom questionnaire, as suggested by the American Thoracic Society (ATS, 1978)\textsuperscript{11}), with a few modifications, was given to all of them. This standardized questionnaire includes questions on respiratory (presence or absence of regular dry and/or productive cough, wheezing, shortness of breath, etc), nasal and eye symptoms, smoking habits, medical and family history of each subject, detailed occupational history and specific questions concerning all jobs held 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 the exposed and unexposed groups.

**Measurement of atmospheric dust:** To assess the extent to which workers had been exposed to cement dust, personal dust monitoring for airborne inhalable (particle size \( \geq 5 \mu m \)) and respirable fractions (particle size \(< 5 \mu m \)) was carried out at different dusty worksites. Dusty worksites included three major areas: (1) blending and grinding raw materials, (2) grinding of a mixture of clinker and gypsum, and (3) screening and storing of cement in silos. For estimation of the atmospheric dusts in these units, a personal dust sampler (Casella, London-LTD), calibrated by a digital automatic calibrator connected to a filter holder equipped with a 25 mm membrane filter (pore size 0.45 \(\mu m\)) through which air was pumped by a battery-powered motor at a constant flow rate of 2 l/min was used. Based on a few preliminary tests, the optimum sampling time, to avoid overloading of the filters, was evaluated to be 120 minutes.

Dust concentration expressed in mg/m\(^3\) was calculated from the changes in the dried filter (for respirable fraction) or cyclone collector weight (for inhalable fraction), as measured by a digital scale at a sensitivity of 0.1 mg, before and after sampling, divided by the volume of air sampled. All measurements were conducted during the fall and winter seasons (from November to March) of 2004.

**Pulmonary function tests:** 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\(_{25-75}\))\textsuperscript{13}), followed guidelines given by the American Thoracic Society (1979)\textsuperscript{13}) and were measured on-site with a portable calibrated vitalograph spirometer (Vitalograph-COMPACT, Buckingham, England). Pulmonary function tests were performed with the assistance of a trained, skilled technician under supervision. The spirometer was calibrated twice a day with a 1-liter syringe in accordance.
to the standard protocol for the instrument used. The mean percentage predicted value was based on subject age, weight, standing height, sex and ethnic group as calculated and adjusted by the spirometer device. Subjects were requested not to take a shower or smoke for at least two hours prior to the test. Additionally, they were trained to become familiar with the maneuvers. The standing height and weight of each subject were measured in normal working clothes. Before the tests, subjects rested in a sitting position for about 5 min. They were then asked to stand in front of the spirometer, as comfortably as possible, and a nose clip was put on. At least, three acceptable maneuvers were performed. If a subject showed great variability between the various forced vital capacity (FVC) volumes, up to eight maneuvers were obtained. The largest volumes (as percentage predicted lung function) were selected for analysis. 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.

Chest radiography: Subjects were invited to a medical center to be examined by a pneumologist and underwent Posterior-Anterior (PA) chest X-ray, using a Siemens instrument. Standard PA chest X-rays were read by a radiologist and a pneumologist. The size of the film was 35 × 35 cm, the distance of the subject from the X-ray tube was about six feet, and the electrical voltage was 100 kv.

Determination of silica phases and SiO₂ content: Wet chemistry, X-ray diffraction (XRD) and X-ray fluorescence (XRF) techniques were performed to determine the silica phases and the SiO₂ contents of the dust samples.

Data analysis and statistical procedures

The data were statistically analyzed using Student’s t-test (or Welch alternate t-test, when the standard deviations of two comparable variables with the F test were significantly different) and Chi-square or Fisher exact test, where applicable (with a preset probability of p<0.05). When the detection of an effect caused by an independent variable in any particular field was not predictable, statistical analysis was conducted using a two-sided p-Value. Experimental results are presented as arithmetic means ± standard deviations. Statistical tests were conducted using GraphPad InStat tm, V204a, 1990–1993 on a personal computer.

Results

Table 1 depicts subjects’ physical characteristics, level and duration of exposure to cement dust, SiO₂ content of the dust samples and length of smoking. Although exposed individuals were, on average, slightly older than unexposed subjects, neither this difference nor the differences noted between other parameters were statistically significant.

Table 2 represents the distribution of smokers and non-smokers in both groups. As shown, there was no significant difference between the number of smokers in both groups.

Table 3 illustrates the frequency of abnormal clinical findings among exposed and unexposed subjects. Exposed workers, regardless of their smoking habits, had higher prevalence of regular cough, phlegm, wheezing and shortness of breath. These symptoms were more frequent among the smokers for both groups (data not shown).
Table 2. Distribution of study subjects by smoking habits

<table>
<thead>
<tr>
<th></th>
<th>Non smokers</th>
<th>Smokers</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposed</td>
<td>48</td>
<td>40</td>
<td>88</td>
</tr>
<tr>
<td>Unexposed</td>
<td>36</td>
<td>44</td>
<td>80</td>
</tr>
<tr>
<td>Total</td>
<td>84</td>
<td>84</td>
<td>168</td>
</tr>
</tbody>
</table>

χ²*  

p-Value 0.2795

*Chi-square statistics (with Yates correction).

Table 3. Frequency (%) of abnormal clinical findings in exposed and unexposed subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n=88)</th>
<th>Unexposed (n=80)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wheezing</td>
<td>28.4% *</td>
<td>5%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>17% *</td>
<td>5%</td>
<td>0.006</td>
</tr>
<tr>
<td>Phlegm</td>
<td>26.1% *</td>
<td>15%</td>
<td>0.03</td>
</tr>
<tr>
<td>Cough</td>
<td>31.8% *</td>
<td>20%</td>
<td>0.04</td>
</tr>
</tbody>
</table>

*Significantly different from its corresponding value in the unexposed group (chi-square, p<0.05).

Table 4. Abnormal findings in chest radiographs

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Exposed (n=88)</th>
<th>Unexposed (n=80)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emphysematous changes</td>
<td>15.9%</td>
<td>0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Old calcified granulomas</td>
<td>5.7%</td>
<td>0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Emphysematous changes associated</td>
<td>4.5%</td>
<td>0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>with inflammatory process</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic inflammatory process</td>
<td>4.5%</td>
<td>0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Focal calcification of the lungs</td>
<td>4.5%</td>
<td>0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Infiltrative changes</td>
<td>2.3%</td>
<td>0</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table 5. Percentage predicted lung function among exposed and unexposed subjects

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Exposed (n=88)</th>
<th>Unexposed (n=80)</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VC</td>
<td>88.9 ± 30.6*†</td>
<td>109.7 ± 33.4</td>
<td>0.002</td>
</tr>
<tr>
<td>FVC</td>
<td>87.5 ± 25.4*</td>
<td>108.6 ± 26.9</td>
<td>0.0006</td>
</tr>
<tr>
<td>FEV₁</td>
<td>90.8 ± 26.8*</td>
<td>113.5 ± 30.5</td>
<td>0.0006</td>
</tr>
<tr>
<td>FEF₂₅–₇₅%</td>
<td>98.7 ± 33.4 *</td>
<td>134.7 ± 50.6</td>
<td>0.0003</td>
</tr>
<tr>
<td>PEF</td>
<td>81.1 ± 35.9*</td>
<td>101.7 ± 37.3</td>
<td>0.01</td>
</tr>
<tr>
<td>FEV₁/VC</td>
<td>108.2 ± 22.4</td>
<td>110.0 ± 30.0</td>
<td>0.66</td>
</tr>
<tr>
<td>FEV₁/FVC</td>
<td>104.0 ± 9.16</td>
<td>105.3 ± 11.1</td>
<td>0.42</td>
</tr>
</tbody>
</table>

* Significantly different from referent value (Student’s t test, p<0.05). †% predicted lung function=% observed / predicted.
from the same industry with almost identical annual income, education, age, sex, ethnic background, etc. Additionally, none of the subjects had past medical or family history of respiratory illnesses or any other chest operations or injuries. Since there were no significant differences in the major confounding variables of age, cigarette smoking, past history of medical illnesses, family history, socioeconomic and ethnic factors, the decrements in the parameters of pulmonary function, increased prevalence of respiratory symptoms and abnormal radiographic findings are likely to be the direct results of exposure to cement dust. Similarly, as dust exposed and unexposed subjects had smoked the same length of time, it is unlikely that cigarette smoking accounted for the differences in symptoms between the two groups.

Studies of chronic changes in pulmonary functions in cement workers have yielded conflicting results. For instance, in a few studies a higher prevalence of respiratory symptoms and varying degrees of airflow obstruction among men exposed to Portland cement dust have been shown\(^4\),\(^13\)–\(^15\). In contrast, some other studies have failed to find any significant differences in the parameters of pulmonary function and/or the prevalence of respiratory symptoms among cement workers when compared with other blue collar workers with similar smoking habits\(^1\),\(^16\),\(^17\).

Our findings indicate that there exists a general tendency for some parameters of pulmonary function to become smaller with chronic exposure to cement dust.

Respiratory tract disorders, the most important group of occupational diseases in the cement industry, are the result of inhalation of airborne dust. Chronic bronchitis, often associated with emphysema, has been reported to be the most frequent respiratory disease\(^3\). Additionally, silicosis followed by mixed dust pneumoconiosis have been claimed to be the greatest risks for cement workers\(^18\). Although normal Portland cement dust due to its low silica content does not generally cause silicosis, exposure to raw materials which present great variations in free silica content is likely to be associated with silicosis. Compared to referent subjects, workers at the plant were exposed to significant concentrations of (Table 1) cement dust (several times higher than the current standards). Consistent with other studies\(^1\),\(^4\),\(^7\),\(^9\),\(^13\)–\(^15\), an increased prevalence of respiratory symptoms (cough, phlegm, dyspnea and wheezing) among workers was demonstrated in this study (Table 3). Similarly, occupational exposure to cement dust has been reported to lead to reduction of ventilatory capacities\(^4\),\(^9\),\(^19\),\(^20\). In accordance with these findings, we show that exposure to cement dust resulted in a significant reduction in some parameters of lung function such as VC, FVC, FEV\(_1\), FEF\(_{25-75}\) and PEF (Table 5). However, no significant difference was noted in FEV\(_1\)/VC and FEV\(_1\)/FVC ratios. This observation may indicate that occupational inhalation exposure to cement dust is likely to induce minor degrees of restrictive ventilatory impairment. This finding is in full agreement with the views expressed by Kumar et al. indicating that in restrictive pulmonary diseases FVC is reduced but the expiratory flow rate, usually measured by FEV\(_1\), is normal or reduced proportionately. Hence, the ratio of FEV\(_1\)/FVC is near normal\(^1\). In this study, exposed workers had a significantly higher rate of pulmonary emphysema and other radiographic abnormalities than their unexposed counterparts. This finding is also in line with other studies which reported higher rates of chronic bronchitis and pulmonary emphysema in cement workers when compared to normal population\(^5\),\(^22\).

Conclusions

In conclusion, our data provide further evidence in favor of the notion that occupational exposure to cement dust is associated with respiratory symptoms, radiographic changes and functional impairments. These results which are in full agreement with our preliminary observations\(^5\), further justify the use of proper personal protective equipment, while at work, and the reduction or elimination of dust, using appropriate ventilation systems, to protect the workers from developing more severe chronic respiratory diseases in the future.

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References

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