Early effects of smoking and environmental pollution on lung function, respiratory symptoms and allergic disorders

Fathi M. El-Gamal (1)
Ahmed M. Alserihi (2)
Abdullah O. Alhasawi (2)
Abdulrahman S. Alshamrani.(2)
Eyad M. Alghamdi (2)
Hussain S. Althumali (2)
Mohammed A. Al-Ghamdi (2)

(1) Chairman of the Family Medicine department, Ibn Sina National College, Jeddah, KSA
(2) Medicine program, Ibn Sina National College, Jeddah, KSA

Corresponding author:
Prof. F. M. El-Gamal, Department of Family Medicine,
Ibn Sina National College. Al Mahjer Street. Jeddah, Kingdom of Saudi Arabia.
Tel: 6356555-6355882 / Fax: 6375344 – P.O. Box 31906 Jeddah 21418
Email: drfathimhelgamal1996@hotmail.com

Abstract

Background: Smoking and exposure to workplace environmental pollutants could be associated with adverse effects on respiratory health and occurrence of allergic disorders.

Objectives: To explore the early effects of exposure to cement dust in the workplace and smoking, on the occurrence of respiratory and allergic disorders in young adults.

Design: It is a cross-sectional study, where a convenient non-probability sample was selected.

Setting: The sstudy was conducted in a cement manufacturing factory at the North of Jeddah city and at a medical college.

Sample size: One hundred subjects were studied (50 workers exposed to cement dust, and 50 subjects, not exposed to any noxious substances).

Method: Each subject was asked to fill out a personal questionnaire (to collect socio-demographic, and health data), an occupational questionnaire, and the MRC questionnaire on respiratory symptoms and smoking habit, and ISAAC core questionnaire on asthma and allergy. Anthropometric measurements and forced spirometry (before and after administration of the bronchodilator), were conducted on each subject. Multi-nominal Logistic regression and multiple linear regression were used. Odds ratio (OR) and 95% confidence intervals (95% CI) were calculated. The level of significance for the study was 0.05.

Results: Smoking was significantly associated with chronic cough (OR=3.68; 95% CI: 0.99, 15.11 and p < 0.05), chronic phlegm production (OR=8.83; 95% CI: 2.33, 33.51, and p < 0.001), shortness of breath on exertion (OR=4.18; 95% CI: 1.49, 11.66, and p < 0.006), and eczema (OR=6.43; 95% CI: 1.33, 31.14, and p < 0.021). After allowing for age, height, weight and cement dust exposure, smoking subjects had significantly lower FEV1% compared to nonsmokers (Beta= -3.45%, p < 0.05). Cement dust exposure was not significantly associated with ill health.

Conclusions: Early effects of smoking are increased chronic respiratory symptoms and occurrence of eczema. FEV1% is the early affected lung function index in smokers, denoting airflow limitation. Cement exposure under the current environmental factory conditions seems to be safe.

Key words: Smoking, cement exposure, forced spirometry, allergic disorders

Introduction

Environmental pollution is a determinant of increased respiratory symptoms and impaired lung function [1-6]. It has been shown to be associated with occurrence and worsening of several respiratory disorders, such as bronchial asthma, and chronic obstructive pulmonary disease (COPD) [2-3]. Tobacco smoke is recognized as the most important risk factor for the development and the progression of COPD. Although tobacco smoke and combustion-related air pollution emit a range of pollutants in common, the role of ambient air pollution on the underlying chronic disease processes that ultimately lead to COPD are not well investigated. [7-9]. The cement industry provides building material for land-based and off-shore installations. Cement is typically produced by heating a homogenous blend of limestone and clay, which is then adjusted to a suitable content of calcium, silicon, aluminum and iron, in a kiln. During its heating to 1,450°C, clinker is formed, which contains calcium silicates, calcium aluminates and calcium ferrites. Clinker is subsequently ground with gypsum and other additives, resulting in a fine particulate powder called cement. In contact with water, clinker partly dissolves and forms an aqueous slurry of high alkalinity, giving clinker and cement strong irritant properties [1]. Cement production workers are exposed to airborne particles of raw materials, clinker, additives and to the final cement product, and their work has been linked to changes in lung function and airway symptoms [11]. Early studies on adverse respiratory effects of cement dust exposure include both non-positive studies and studies connecting cement production work with chronic airway inflammation and reduction of dynamic lung volumes [12, 13]. Other studies indicate a reduced forced vital capacity (FVC) or forced expiratory volume in 1 s (FEV1) [14–18], and a higher prevalence of chronic respiratory symptoms [16–19], and chronic obstructive pulmonary disease (COPD) [2], in cement production workers. Several other studies of lung function in cement production workers were non-positive [19, 21–23]. The literature is conflicting and conclusions about exposure–response relationships or safe levels of exposure cannot be drawn [11]. Thus the aim of the present study was to explore the impact of smoking, and exposure to cement dust on the occurrence of respiratory symptoms and allergic disorders; and to study its early effects on the forced flow-volume curve indices.

Methods

A cross sectional study was undertaken during January to April, 2020, in a cement factory at North of Jeddah and at a medical college at South of Jeddah, KSA.

The total number of studied subjects was 100; 50% male workers from a cement factory in North of Jeddah, and 50% male subjects not exposed to any noxious materials that can affect the chest or the skin. The total number of cases and controls was more than the necessary minimum number needed for this study (74 subjects, as assessed by G*power software [24], for α = 0.05, β = 0.95, effect size is 0.3, and 2-tail-t-test).

Data was collected on each subject, after we obtained written consent to participate in the study. Data were collected through: 1-Interview questionnaire which provided information on personal and socio-demographic characteristics of the subject; 2- Occupational questionnaire which provided information on nature of exposure, duration of employment in years, duration of exposure per day, and use of personal protective equipment; 3- MRC questionnaire on respiratory symptoms and smoking habit which is a standardized questionnaire that provides information on chronic respiratory symptoms and smoking habit [25-26]; 4-ISAAC core questionnaire on asthma and allergy which was used to diagnose bronchial asthma, allergic rhinitis and atopic eczema [27]; 5- Anthropometric measurements: weight and height of the subject, were measured using standard techniques and equipment [28]; 6- Lung function testing according to the standardization of procedure and maneuver cited by the ATS [28] Forced spirometry was measured where indices from flow volume curve and time volume curve were obtained, namely:

FVC = forced vital capacity; FEV1 = forced expiratory volume in one second; FEV1% = (FEV1 / FVC) * 100; PEFR = Peak expiratory flow rate; FEF25 – 75%= Flow rate between 25% and 75% of the FVC; FEF75% = Forced expiratory flow at 75% of FVC expired; FEF50% = Forced expiratory flow at 50% of FVC expired; FEF25% = Forced expiratory flow at 25% of FVC expired. Lung function was assessed before and 10 minutes after administration of Salbutamol (Ventolin) inhalation. Data analysis and statistical tests: Data was analyzed using the Statistical Package for Social Sciences (IBM SPSS, version 22, Armonk, NY: IBM Corp.). Multi-nominal logistic regression method was used where respiratory symptoms and allergic disorders were used as the dependent dichotomous variables; other variables were used as independent variables, where Odds ratios, 95% confidence interval (95% CI), and p values were calculated. Linear Multiple Regression Analysis was used to study continuous variables that could significantly predict lung function indices. The significance of the differences was calculated at 95% CI; $P < 0.05$ was considered as statistically significant.

Ethical considerations
Ethical clearance was obtained from the institutional review board (IRB) of the College of Ibn Sina (IEC Ref No: H-09-12092019). Permission was obtained from the director and foremen of the factory. Informed consent was obtained from the subjects, after providing information about the purpose of the study. In order to keep confidentiality of any information provided by study participants, the data collection procedure was anonymous.

Results

The mean age of the exposed group was 34.24 years (7.88), while mean age of non-exposed group was 37.08 years (13.16). This difference was not statistically significant (t = -1.31, and   p < 0.19). Mean duration of exposure to cement dust among cement exposed groups was 7.38 years (5.02). Among the whole subjects, 24% were smokers and mean duration of smoking was 13.12 years (10.25), while 76% were non-smokers. Chronic cough was encountered among 10% of the whole subjects, while chronic phlegm production was encountered among 14%, and shortness of breath on exertion was found in 23% of the subjects. Bronchial asthma was encountered among 7% of the subjects, and hay fever in 19%; while eczema was found in 8% of the subjects.

Table 1 reveals the multi-nominal logistic regression for respiratory symptoms and allergic disorders, in relation to age, exposure to cement dust and smoking habit. Chronic cough was 4 times more likely to be encountered among smoking subjects compared to non-smokers (OR=3.68; 95% CI: 0.99, 15.11' and p < 0.05). Neither advancing age nor exposure to cement dust were significantly associated with occurrence of chronic cough.

Chronic phlegm production, was about 9 times more likely to occur in smoking subjects compared to those who do not smoke (OR=8.83; 95% CI: 2.33, 33.51, and p < 0.001). Cement exposed workers were less likely to suffer from chronic phlegm production compared to the non-exposed subjects (OR=0.20; 95%CI: 0.05, 0.83; p value < 0.027). Shortness of breath on exertion, was about 4 times more likely to occur among smoking subjects compared to those who do not smoke (OR=4.18; 95% CI: 1.49, 11.66, and p < 0.006).

Eczema, was about 6 times more likely to occur among smoking subjects compared to those who do not smoke (OR=6.43; 95% CI: 1.33, 31.14, and p < 0.021). Neither bronchial asthma nor hay fever, were significantly associated with smoking habit or with exposure to cement dust   (p >0.05).

Table 2 shows the comparison of mean values of forced spirometric tests (pre and post bronchodilator administration) between cement exposed workers and non-exposed subjects. The mean values of forced flow volume curve indices and time-volume curve indices were similar for both cement exposed workers and non-exposed subjects except for Pre-FEF75%, where mean value was higher in the cement exposed workers (1.78 L/S (0.81)) compared to non-exposed subjects (1.35L/S (0.53) where t = -2.19 and p < 0.03.

Table 3 reveals the comparison of mean values of forced spirometric tests (pre and post bronchodilator administration) between smokers and non-smoking subjects. The mean values of Forced flow volume curve indices and time-volume curve indices were similar in both smokers and nonsmokers (p>0.05).

Table 4 shows correlation/regression relationship between lung function tests and age, height, weight, smoking habit, and exposure to cement dust. It was found that after allowing for confounding factors such as age, height weight and smoking habit the mean values of the cement exposed workers and non-exposed subjects were similar, and no significant difference was found (p > 0.05). After allowing for age, height, weight and cement dust exposure, smoking subjects had significantly lower FEV1% compared to nonsmokers (Beta= -3.45%, p < 0.05). Other lung function indices were not significantly different for smokers compared to non-smokers (p> 0.05).

Duration of exposure to cement dust was, also, not associated with significant changes in the lung function indices (Table 5).

Table 1: Multi-nominal Logistic regression between health conditions and age, smoking habit and exposure to cement dust

Independent variables	B	Sig.	Exp(B)	95% Confidence Interval for Exp(B)	
				Lower Bound	Upper Bound
Chronic cough					
Intercept	0.899	.468			
Age in years	0.015	.642	1.015	.952	1.083
Exposure to cement	-0.235	.735	.790	.202	3.086
Smoking habit	1.352	.052	3.863	.988	15.108
Chronic phlegm production					
Intercept	1.239	.256			
Age in years	.004	.883	1.004	.952	1.059
Exposure to cement	-1.596	.027	.203	.049	.834
Smoking habit	2.178	.001	8.827	2.325	33.509
Shortness of breath on exertion					
Intercept	.069	.942			
Age in years	-.004	.858	.996	.949	1.044
Exposure to cement	.684	.189	1.981	.714	5.498
Smoking habit	1.430	.006	4.179	1.498	11.663
Doctor diagnosed Bronchial asthma					
Intercept	.715	.695			
Age in years	.038	.460	1.039	.939	1.150
Exposure to cement	1.865	.092	6.454	.736	56.627
Smoking habit	.003	.998	1.003	.173	5.804
Doctor diagnosed Hay fever					
Intercept	-.068	.946			
Age in years	.035	.184	1.035	.984	1.089
Exposure to cement	-.589	.273	.555	.193	1.591
Smoking habit	.885	.120	2.423	.795	7.389
Doctor diagnosed eczema					
Intercept	3.199	.022			
Age in years	-.056	.110	.946	.883	1.013
Exposure to cement	.577	.500	1.781	.332	9.540
Smoking habit	1.861	.021	6.429	1.327	31.136

Table 2 shows comparison of forced spirometric tests (pre and post bronchodilators) between cement exposed workers and non-exposed subjects

LFT	Exposure	Mean	Standard Deviation	t-test	p-value
Pre-FVC	not exposed	3.5528	.76210	-.52	.582
	exposed	3.6484	.95744		
Pre- FEV1	not exposed	3.0010	.52858	-1.322	.189
	exposed	3.1620	.67948		
Pre- FEV1%	not exposed	79.8100	8.22989	-.380	.705
	exposed	80.4042	7.39206		
Pre- PEFR	not exposed	5.9010	1.91745	.118	.907
	exposed	5.8594	1.60628		
Pre- FEF25-75%	not exposed	2.9898	.99034	-.696	.488
	exposed	3.1220	.90809		
Pre- FEF75%	not exposed	1.3518	.52925	-2.194	.031
	exposed	1.7870	1.29860		
Pre- FEF50%	not exposed	3.7826	.80514	.256	.798
	exposed	3.7386	.90992		
Pre- FEF25%	not exposed	4.9972	2.10419	-.315	.754
	exposed	5.1190	1.74936		
Post-FVC	not exposed	3.6110	.69550	-.401	.689
	exposed	3.6794	.98593		
Post-FEV1	not exposed	3.1122	.45492	-.858	.393
	exposed	3.2220	.78246		
Post-FEV1%	not exposed	81.7400	7.98419	-.633	.528
	exposed	82.7000	7.16639		
Post-PEFR	not exposed	6.9138	1.85069	..428	.670
	exposed	6.7564	1.82766		
Post-FEF25-75%	not exposed	3.7226	1.06328	.654	.515
	exposed	3.5968	.84849		
Post-FEF75%	not exposed	1.5818	.62640	-1.411	.161
	exposed	1.8474	1.17418		
Post-FEF50%	not exposed	4.2742	.86359	1.751	.083
	exposed	3.9632	.91166		
Post-FEF25%	not exposed	5.5756	2.08943	.588	558
	exposed	5.3448	1.82341		

Table 3: Comparison of forced spirometric tests (pre and post bronchodilators) between smoking and non-smoking subjects

	Smoking habit	Mean	Std. Deviation	t-test	p-value
Pre-FVC	Nonsmoker	3.5914	.84207	-.188	.851
	Smoker	3.6296	.94172	-,.77	.860
Pre- FEV1	nonsmoker	3.0905	.61486	.262	.794
	Smoker	3.0529	.61084	.263	.794
Pre- FEV1%	nonsmoker	80.7975	7.82851	1.590	.115
	Smoker	77.9208	7.39500	1.638	.109
Pre- PEFR	nonsmoker	5.8726	1.87642	-.076	.939
	Smoker	5.9042	1.35989	-.090	.929
Pre- FEF25-75%	nonsmoker	3.1014	.96669	.854	.395
	Smoker	2.9117	.88851	.893	.377
Pre- FEF75%	nonsmoker	1.5857	1.00016	.285	.776
	smoker	1.5179	1.06289	.276	.784
Pre- FEF50%	nonsmoker	3.8217	.86740	1.276	.205
	smoker	3.5671	.80138	1.330	.191
Pre- FEF25%	nonsmoker	5.1180	1.96836	.552	.582
	smoker	4.8683	1.81250	.576	.568
Post-FVC	nonsmoker	3.6495	.81803	.089	.929
	smoker	3.6317	.96136	.082	.935
Post-FEV1	nonsmoker	3.1891	.65590	.610	.543
	smoker	3.0975	.59061	.644	.523
Post-FEV1%	nonsmoker	82.6447	7.27132	.999	.320
	smoker	80.8750	8.44580	.924	.362
Post-PEFR	nonsmoker	6.8451	1.82048	.097	.923
	smoker	6.8033	1.90570	.095	.925
Post-FEF25-75%	nonsmoker	3.6647	.96099	.093	.926
	smoker	3.6438	.97355	.092	.927
Post-FEF75%	nonsmoker	1.7176	.90865	.057	.955
	smoker	1.7050	1.07584	.052	.959
Post-FEF50%	nonsmoker	4.1822	.88087	1.264	.209
	smoker	3.9175	.93750	1.223	.229
Post-FEF25%	nonsmoker	5.4889	1.98415	.261	.795
	smoker	5.3692	1.89558	.267	.791

Table 4: Correlation/regression relationship between lung function tests and age, height, weight, smoking habit, and exposure to cement dust

Independent variables	Lung function indices							
	FVC	FEV1	FEV1%	PEFR	FEF25-75	FEF75	FEF50	FEF25
Constant	3.287	3.508	57.804	4.890	4.254	3.266	5.110	5.162
Age (years	-.003	-.005	-.150**	.001	-.007	-.007	-.007	-.006
Height (Cm)	.000	-.003	.180	.003	-.007	-007	-.007	-.001
Weight	.004	.004	-.034	.005	.002	-.006	003	.004
Smoking habit	.024	-.057	-3.455*	.035	-202	-.134	-.236	-.271
Exposure to cement dust	.106	.164	.837	-.009	.132	.383	-.037	.155

Table 5: Correlation/regression relationship between lung function tests and age, height, weight, smoking habit, duration of employment in cement industry

Independent variables	Lung function indices							
	FVC	FEV1	FEV1%	PEFR	FEF25-75	FEF75	FEF50	FEF25
Constant	3.519	3.823	59,463	4.895	4.497	4.016	5.049	5.462
Duration of employment	-.007	.012	.033	-.013	.016	.021	-.008	.010
Age (years	-.003	-.007	-.158*	.003	-.010	-.011	-.006	-.008
Height (Cm)	.000	-.005	.174	.003	-.007	-.009	-.007	-.002
Weight	.003	.003	-.035	.004	.002	-.007	.003	.004
Smoking habit	.054	-.049	-3.370	056	-.206	-.103	-.229	-.262

Discussion

Chronic obstructive pulmonary diseases, characterized by long-term poorly irreversible airway limitation and persistent respiratory symptoms, are a common and preventable disease [29]. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, there are three criteria to diagnose the disease: 1- a post-bronchodilator FEV1% of less than 70%, 2- symptoms of respiratory system as shortness of breath on exertion, chronic cough, production of sputum or wheezing, and 3- significant exposure to noxious environmental stimuli such as smoking or chemical environmental hazards [30]. Therefore in the present study, as the main objective was to assess the impact of smoking and environmental pollution, particularly exposure to cement dust, on the respiratory system, lung function was assessed before and after administration of bronchodilator. MRC questionnaire was used and occupational questionnaire was asked of each subject. As this study was interested in evaluating the early effects of smoking and exposure to noxious materials on the lung and occurrence of allergic disorders, the studied subjects were relatively of young age where the mean age of the exposed group was 34.24 years (7.88), while the mean age of the non-exposed group was 37.08 years (13.16). The mean duration of exposure to cement dust and smoking were relatively low (7.38 years, and 13.12 years respectively).

A recent study, conducted by Kotaki et al in 2019, revealed that in addition to the impact of smoking, the elderly who were chronically exposed to air pollution had exacerbated respiratory symptoms and impaired respiratory function [31]. Similar findings were reported by other studies [32, 33]. In the present study most of the studied subjects were relatively young, and only 2 subjects, who were nonsmokers and not exposed to cement dust, fulfilled the criteria of COPD. However, in line with previous studies (31-33), smoking was significantly associated with chronic cough (OR=3.68; 95% CI: 0.99, 15.11), chronic phlegm production (OR=8.83; 95% CI: 2.33, 33.51), and shortness of breath on exertion (OR=4.18; 95% CI: 1.49, 11.66). These findings from the present study, support the incrimination of smoking in the genesis of chronic inflammatory diseases of the lung airways and COPD. Several studies have assessed the association between smoking exposure and allergic diseases. In each of the allergic conditions, results were conflicting and alternated between the harmful effects of smoking, [34, 35, 36] and protection [37–39], while some studies could not find evidence of any effect [40–42]. In the present study smoking was only significantly associated with occurrence of eczema (OR=6.43; 95% CI: 1.33, 31.14, and p < 0.021). In the present study exposure to cement dust in the studied factory was not significantly associated with increased occurrence of chronic respiratory symptoms or allergic disorders. This is contradictory to the results of some studies [43-46]. This could be due to the relatively young age of the exposed workers or due to the control measures applied in this factory compared to work place exposures in other studied factories. Forced spirometric indices, also, in the present study were, similar in cement exposed workers and non-exposed subjects. Some lung function indices were better in the cement exposed workers e.g. FEF75% which reflects airflow in the small airways, compared to the employee in the medical college, and this seems to be due to the exercise effect of working as a blue collar worker in a factory compared to the sedentary life style adopted by the control subjects.

As for smokers, the early lung function index affected was FEV1% (Beta = -3.45%, p < 0.03) which was decreased in the smokers compared to non-smokers, which denotes the beginning of airflow limitation at this relatively young age, and before development of COPD.

Conclusions

Early effects of smoking are increased chronic respiratory manifestations and reduced FEV1%, which indicated obstructive impairment. Cement exposure under the current environmental factory conditions seems to be safe. Smoking cessation programs should be implemented among workers in industries, and to the population in the community to combat the major risk of COPD.

Strengths and limitations of this study
In this study flow volume curve indices which are sensitive to early changes in the small airways were used, in addition to time volume curve indices which, mainly, measure late effect on large airways. Lung function was measured before and after administration of bronchodilator to categorize subjects with COPD whose post bronchodilator FEV1% were less than 70%, to meet GOLD criteria.

ISAAC questionnaire used in this study has been validated worldwide. Multifactorial statistical tests were, also, employed to allow for the confounders during assessing the different associations. However, among the limitations of this study were that the questionnaire data depended on the recall of the subjects. It was also based on workers from only one factory; thus we can't exclude self-selection bias.

Competing interests
All authors declare that they have no competing interests.

References

[1] Saskia C. van der Zee, Paul H. Fischer b, Gerard Hoek c. Air pollution and subclinical interstitial lung disease: the Multi-Ethnic Study of Atherosclerosis (MESA) air–lung study. Environmental Research 2016; 148: 475-469.

[2] Guarnieri M, Balmes JR. Outdoor air pollution and asthma. Lancet 2014; 383: 1581–1592.

[3] Adar SD, Kaufman JD, Diez-Roux AV, et al. Air pollution and percent emphysema identified by computed tomography in the multi-ethnic study of atherosclerosis. Environ Health Perspect 2015; 123: 144–151.

[4] EL-Gamal, F. M; Kordy, M. N; Ibrahim, M. A Study of respiratory symptoms and lung function in polyvinyl chloride fabrication workers. Saudi medical journal. 1995, 16,(1): 36-41..

[5] Rice MB, Rifas-Shiman SL, Oken E, et al. Exposure to traffic and early life respiratory infection: a cohort study .Pediatr Pulmonol 2015; 50: 252–259.

[6] Chinn DJ, Cotes JE, El-Gamal FM, Wollaston JF. Respiratory health of young shipyard welders and other tradesmen studied cross sectionally and longitudinally. Occup Environ Med. 1995;52:33–42.

[7] Ku □nzli N, Perez L, Rapp R. Air Quality and Health. Lausanne, European Respiratory Society, 2010.

[8] Zanobetti A, Bind MA, Schwartz J. Particulate air pollution and survival in a COPD cohort. Environ Health 2008; 7: 48.

[9] Schikowski T, Mills IC, Anderson HR, et al. Ambient air pollution: a cause of COPD? Eur Respir J 2014; 43: 250–263.

[10] Ahmadi H, Durrant CAT, Sarraf KM, et al . Chemical burns: a review. Curr Anaesth Crit Care 2008; 19: 282–286.

[11] Health and Safety Executive. Portland cement dust – hazard assessment document EH75/7. Watch committee. Merseyside, Health and Safety Executive, 2004.

[12] Bazas T. Effects of occupational exposure to dust on the respiratory system of cement workers. J Soc Occup Med 1980; 30: 31–36.

[13] Fell AK, Sikkeland LI, Svendsen MV, et al. Airway inflammation in cement production workers. Occup Environ Med 2010; 67: 395–400.

[14] Meo SA. Health hazards of cement dust. Saudi Med J 2004; 25: 1153–1159.

[15] Mwaiselage J, Bratveit M, Moen B, et al. Cement dust exposure and ventilatory function impairment: an exposure-response study. J Occup Environ Med 2004; 46: 658–667.

[16] Yang CY, Huang CC, Chiu HF, et al. Effects of occupational dust exposure on the respiratory health of Portland cement workers. J Toxicol Environ Health 1996; 49: 581–588.

[17] Noor H, Yap CL, Zolkepli O, et al. Effect of exposure to dust on lung function of cement factory workers. Med J Malaysia 2000; 55: 51–57.

[18] Al-Neaimi YI, Gomes J, Lloyd OL. Respiratory illnesses and ventilatory function among workers at a cement factory in a rapidly developing country. Occup Med 2001; 51: 367– 373.

[19] Abrons HL, Petersen MR, Sanderson WT, et al. Symptoms, ventilatory function, and environmental exposures in Portland cement workers. Br J Ind Med 1988; 45: 368–375.

[20] Vestbo J, Rasmussen FV. Long-term exposure to cement dust and later hospitalization due to respiratory disease. Int Arch Occup Environ Health 1990; 62: 217–220.

[21] Catenacci G, Tringali S, Brunetti G, et al. Decrement of respiratory function indices in a case series of workers exposed to cement dust: a longitudinal study. G Ital Med Lav 1988; 10: 123–129.

[22] Fell AK, Thomassen TR, Kristensen P, et al. Respiratory symptoms and ventilatory function in workers exposed to Portland cement dust. J Occup Environ Med 2003; 45: 1008–1014.

[23] Abu Dhaise BA, Rabi AZ, al Zwairy MA, et al. Pulmonary manifestations in cement workers in Jordan. Int J Occup Med Environ Health 1997; 10: 417–428.

[24] Faul F, Erdfelder E, Lang A-G, Buchner A. G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behavior Research Methods 2007; 39, 175-191.

[25] Medical Research Council on the Aetiology of Chronic Bronchitis. Standardised questionnaire on respiratory symptoms. Br Med J 1960; 2: 1665.

[26] Cotes JE, Chinn DJ. MRC questionnaire (MRCQ) on respiratory symptoms
Occupational Medicine 2007; 57 (5): 388, https://doi.org/10.1093/occmed/kqm051

[27] Mitchell R, Beasley B, Björkstén J, Crane L, García□ Marcos U, Keil G. The association between BMI, vigorous physical activity and television viewing and the risk of symptoms of asthma, rhino-conjunctivitis and eczema in children and adolescents: ISAAC Phase Three. Clinical & experimental Allergy 2013; 43 (1): 73 -84.

[28] Cotes JE, Chinn DJ, Miller MR. Lung Function. 6th ed. Chichester, United Kingdom:
Wiley-Blackwell 2006. ISBN13: 0632064939.

[29] Vogelmeier CF, Criner GJ, Martinez FJ, et al. Global strategy for the diagnosis, management, and prevention

of chronic obstructive lung disease 2017 report. GOLD executive summary. Am J Respir Crit Care Med. 2017;195(5):557–582.

[30] Mirza S, Clay RD, Koslow MA, Scanlon PD. COPD guidelines: a review of the 2018 GOLD report. Mayo Clinic Proc. 2018;93(10):1488–1502.

[31] Kotaki K, Ikeda H, Fukuda T, Yuhei K, Yuki F, Kawasaki M, et al. Trends in the prevalence of COPD in elderly individuals in an air-polluted city in Japan: a cross-sectional study. International Journal of COPD 2019; 14:791–798

[32] Schikowski T, Sugiri D, Ranft U, et al. Long-term air pollution exposure and living close to busy roads are associated with COPD in women. Respir Res. 2005;6:152.

[33] Li J, Sun S, Tang R, et al. Major air pollutants and risk of COPD exacerbations: a systematic review and meta-analysis. Int J Chron Obstruct Pulmon Dis. 2016; 11: 3079–3091.

[34]. Lannerö E, Wickman M, van Hage M, Bergström A, Pershagen G, et al. Exposure to environmental tobacco smoke and sensitisation in children. Thorax 2008; 63: 172–176.

[35]. Cakir E, Ersu R, Uyan ZS, Oktem S, Varol N, et al. The prevalence and risk factors of asthma and allergic diseases among working adolescents. Asian Pac J Allergy Immunol 2010; 28: 122–129.

[36]. Lee CH, Chuang HY, Hong CH, Huang SK, Chang YC, et al. Lifetime exposure to cigarette smoking and the development of adult-onset atopic dermatitis. Br J Dermatol 2011; 164: 483–489.

[37] Bendtsen P, Grønbaek M, Kjaer SK, Munk C, Linneberg A, et al. Alcohol consumption and the risk of self-reported perennial and seasonal allergic rhinitis in young adult women in a population-based cohort study. Clin Exp Allergy 2010; 38: 1179–1185.

[38] Ludvigsson JF, Mostrom M, Ludvigsson J, Duchen K. Exclusive breastfeeding and risk of atopic dermatitis in some 8300 infants. Pediatr Allergy Immunol 2005; 16: 201–208.

[39] Metsälä J, Lundqvist A, Kaila M, Gissler M, Klaukka T, et al. Maternal and perinatal characteristics and the risk of cow's milk allergy in infants up to 2 years of age: a case-control study nested in the Finnish population. Am J Epidemiol 2010; 171: 1310–1316.

[40] McKeever TM, Lewis SA, Smith C, Collins J, Heatlie H, et al. Siblings, multiple births, and the incidence of allergic disease: a birth cohort study using the West Midlands general practice research database. Thorax 2001; 56: 758–762.

[41] Wang IJ, Guo YL, Lin TJ, Chen PC, Wu YN. GSTM1, GSTP1, prenatal smoke exposure, and atopic dermatitis. Ann Allergy Asthma Immunol 2010; 105: 124–129.

[42] Tariq SM, Matthews SM, Hakim EA, et al. The prevalence of and risk factors for atopy in early childhood: a whole population birth cohort study. J Allergy Clin Immunol 1998; 101: 587–593.

[43] Gizaw, Z., Yifred, B. & Tadesse, T. Chronic respiratory symptoms and associated factors among cement factory workers in Dejen town, Amhara regional state, Ethiopia, 2015. Multidiscip Respir Med 2016; 11: 13. https://doi. org/10.1186/s40248-016-0043-6

[44] Erhabor O, Kebbe BI, Zama II, Abdullahi N, Marafa Y, Okwesili AN, et al. Effect of occupational exposure of cement dust on some haematological parameters of workers in a cement company in Sokoto, Nigeria. Int J Med Sci Health Care. 2013; 1(7): 21–25.

[45] Rodríguez E, Ferrer J, Martí S, Zock JP, Plana E, Morell F. Impact of occupational exposure on severity of chronic obstructive pulmonary disease. Chest 2008; 134: 1237–43.

[46] Kakooei H, Gholami A, Ghasemkhani M, et al. Dust exposure and respiratory health effects in cement production. Acta Medica Iranica 2012; 50 (2): 122-6.