Kaohsiung Journal of Medical Sciences (2016) 32, 44–49

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.kjms-online.com

ORIGINAL ARTICLE

Relationship of cumulative dust exposure dose and cumulative abnormal rate of pulmonary function in coal mixture workers

Qing-Zeng Qian [a], Xiang-Ke Cao [b], Qing-Qiang Qian [c], Fu-Hai Shen [a,*], Qian Wang [a], Hai-Yan Liu [a], Jun-Wang Tong [a]

[a] *College of Public Health, North China University of Science and Technology, Tangshan, People's Republic of China*
[b] *College of Life Science, North China University of Science and Technology, Tangshan, People's Republic of China*
[c] *Affiliated Hospital of North China University of Science and Technology, Tangshan, People's Republic of China*

Received 26 August 2015; accepted 12 November 2015
Available online 6 January 2016

KEYWORDS
Coal mixture workers;
Cumulative abnormal rate;
Cumulative dust exposure;
Pulmonary function;
Pulmonary function indices

Abstract To investigate the dose-response relationship between cumulative dust exposure (CDE) and cumulative abnormal rate of pulmonary function in coal mixture workers. Three hundred and twenty eight coal mixture workers (exposed group) and 169 nondust-exposed workers (control group) were recruited. Basic information data were collected and pulmonary function tests were performed. Pulmonary function was compared between the two groups after comparing smoking behaviors. Pulmonary function indices [forced vital capacity in 1 second after full inspiration (FVC)%, forced expiratory volume (FEV)1%, and FEV1/FVC%] were compared among groups stratified by service length (exposure duration). The relationship between CDE dose and cumulative abnormal rate of pulmonary function in coal mixture workers was analyzed. Abnormal rate of pulmonary function in the exposed group (35.1%) was significantly higher than the control group (10.1%; $p < 0.001$); FVC%, FEV1%, and FEV1/FVC% in the exposed group decreased significantly compared with the control group (all $p < 0.05$). Differences in FVC%, FEV1%, and FEV1/FVC% among coal mixture workers stratified by exposure duration in the exposed group were statistically significant (all $p < 0.05$). The discernible increase in the cumulative abnormal rate was observed, from $\geq$ 1000 mg/m^3·years group to $\geq$ 1700 mg/m^3·years group. Correlation analysis revealed a positive correlation between the CDE dose and the cumulative abnormal rate of pulmonary function. Higher abnormal

Conflicts of interest: All authors declare no conflicts of interest.

* Corresponding author. College of Public Health, North China University of Science and Technology, Number 57 South Jianshe Road, Tangshan 063000, People's Republic of China.
E-mail address: shenfuhai_0727@163.com (F.-H. Shen).

http://dx.doi.org/10.1016/j.kjms.2015.11.003

pulmonary function rate was found among coal mixture workers, characterized by decreased pulmonary function indices. Our results suggested a positive relationship between CDE dose and cumulative abnormal pulmonary function rate, and a rapid increase in cumulative abnormal rate within a certain range of CDE dose. A lower limit value of 1000 mg/m^3·years has reference significance.

Introduction

Cumulatively exposed to respirable coal mine dust, coal miners are at risk of developing chronic occupational lung diseases and suffer from irreversible lung damage, including coal workers' pneumoconiosis (CWP), progressive massive fibrosis, and chronic obstructive pulmonary disease (COPD) [1,2]. Previous studies have well documented the association between long-term exposure to coal mine dust and mortality, even after adjustment for smoking [3,4]. Further, the association of emphysema and COPD with coal dust exposure has been reported, and increased cumulative dust exposure (CDE) may also increase the death rate of these diseases [1,5]. With enforcement of occupational exposure limits for coal mine dust, declined CWP prevalence from 11.2% to 2.0% is found among underground coal miners [6,7]. However, miners working entirely according to the contemporary standard still continue to develop lung diseases, implying that further restrictions and regulations in occupational dust exposure are needed [6,8].

In 2013, a total of 23,152 occupational disease cases in China were reported, among which coal mining activities accounted for $>$ 50% of them [9]. Occupational dust and gas/fume exposure is associated with the increased prevalence of COPD in the Chinese population, independent of smoking, sharing a similar attributable fraction in Western populations [10]. Pulmonary ventilation function serves to evaluate the damaged pulmonary function and compensatory function in early stage of CWP patients who exhibit impaired pulmonary ventilation and diffusion function [11]. Forced expiratory volume in 1 second after full inspiration (FEV1) and FEV1/forced vital capacity (FVC) are widely used in pulmonary function examination [12,13]. Generally, an accelerated decline in FEV1 over time may result in COPD; while low FEV1 in early adulthood has been suggested to play important roles in causing COPD [14,15]. Pulmonary function tests (FVC, FEV1, and FEV1/FVC) have important implications for the labor ability appraisal of CWP patients. However, regulations for limiting coal mine dust exposure, and efforts to reduce the prevalence and severity of lung diseases still need to be further evaluated [1,6]. Therefore, the current study was performed to examine the relationship between CDE and cumulative abnormal rate of pulmonary function, and to propose a lower limit value for CDE in coal miners in China.

Methods

Study participants

A total of 328 coal mixture workers (exposed group) were recruited from a coal mine in Tangshan, and 169 nondust-exposed workers (control group) were selected from an instrument, electrical, and mechanical factory in the same area. Participants in the exposed group were enrolled if they met the following inclusion criteria: (1) dust-exposed time $>$ 1 year; (2) dust-exposed length of service in the coal mine accounted for more than half of one's dust exposure career; (3) complete physical examination results within 2 years; and (4) clear and complete records of occupational history. All enrolled individuals were men. The mean age of the 328 coal mixture workers was 46.8 $\pm$ 9.5 years (range, 21—60 years) and the mean body weight was 58.7 $\pm$ 11.3 kg (range, 42—86 kg). The mean age of the nondust-exposed workers was 46.5 $\pm$ 7.8 years (range, 21—59 years) and the mean body weight was 58.1 $\pm$ 8.5 kg (range, 43—85 kg). There were no significant differences in age or body weight between the exposed group and the control group (all $p > 0.05$). The study was approved by the Ethics Committee of our university. All participants provided written, informed consents. Study protocols followed the ethical principles for medical research involving human participants of the Helsinki Declaration [16].

Data collection

Information on baseline characteristics were collected using questionnaires including the following contents: (1) dust exposure history and demographic characteristics (date of birth, sex, initial working time, occupational history, current occupation, retirement age, etc.); and (2) pulmonary disease history (time of initial diagnosis, grades, complications, onset time of complications, etc.). Occupational data of all the participants were mainly extracted from the Capital and Labor Staff Database, and supplemented by Occupational Health Examination Records. Any contradictories or difficulties in obtaining complete information were solved by face-to-face interviews with employed workers or telephone interviews with retired workers. The pulmonary function data were obtained by a periodic check in the Occupational Disease Prevention and Treatment Institute of the coal miner, and supplemented

by the Capital and Labor Staff Database. Dust exposure data were collected based on the files in the Dust Detection Department of the coal mine ventilation area and supplemented by the Occupational Health Inspection and Management Department.

Pulmonary function tests were performed with a AS.507 spirometer for each individual (Minato AS-507; Minato Medical Science Co. Ltd., Osaka, Japan). FVC%, percentage of FEVl%, and FEVl/FVC ratio (FEVl/FVC%) were employed as pulmonary function indices. FVC% and FEVl% refer to percentages of absolute/predicted FEV1 and FVC values, respectively; and were estimated according to the individuals' age, height, and weight.

Evaluation criteria

Smoking: smoking index = the number of cigarettes smoked per day × smoking years, then smoking was defined as smoking index $\geq$ 20, and nonsmoking was $<$ 20 [17].

Abnormal pulmonary function: based on "Chinese professional medicine", FVC $<$ 80% and FEV1/FVC $>$ 70% was defined as restrictive pulmonary dysfunction; FVC $>$ 80% and FEV1/FVC $<$ 70% was defined as obstructive pulmonary dysfunction; and FVC $<$ 80% and FEV1/FVC $<$ 70% was defined as mixed type pulmonary dysfunction. Participants underwent a physical examination and having one of these conditions was considered as experiencing abnormal lung function.

Dust-exposed service length: a dust-exposed length of service was counted in years from the 1st year of dust exposure, in which participants had no occupational changes. Dust-exposed lengths of service were classified into four groups: $<$ 10 years, 10—19 years, 20—29 years, and $\geq$ 30 years group [18].

CDE dose [mg/(m^3·years)] was calculated as accumulated value of $\sum_{Ci \times Ti}$ (Ci, time-weighted average concentration in each year; Ti, dust exposure year).

Cumulative abnormal pulmonary function rate = 1 − cumulative normal rate; cumulative normal rate = (1 − abnormal rate in the present stage) × (1 − abnormal rate in the upper stage); abnormal rate = morbidity/adjusted population; adjusted population = (population of the beginning of the period—population of the end of the period)/2. The population of the beginning of the period referred to individuals consistent with the CDE dose, and the population of the end of the period refereed to individuals consistent with the range of certain CDE dose.

Statistical analysis

SPSS version 17.0 software (SPSS Inc., Chicago, Il, USA) was used for statistical analysis. Continuous variables were presented as means ± standard deviation; categorical variables were presented as frequencies and percentages. Comparisons between continuous variables were conducted with *t* test and *F* test. The Chi-square test was used for categorical variables. Correlations were analyzed with Pearson correlation analysis. A p value $<$ 0.05 was considered statistically significant.

Results

Comparisons in smoking rate, pulmonary function, and pulmonary function indices

The similar smoking rate in the exposed group and the control group was observed (53.7% vs. 50.9%, $p > 0.05$), but the abnormal rate of pulmonary function was observed in the exposed group when compared with the control group (35.1% vs. 10.1%, $p < 0.05$). In addition, decreased FVC%, FEV1%, and FEV1/FVC% were found in the exposed group compared with the control group (all $p < 0.05$; Table 1).

Comparisons in pulmonary function indices stratified by length of service

Compared with the $<$ 10 years group, decreases in FVC%, FEV1%, and FEV1/FVC% were found in the 10—19 years group, the 20—29 years group, and the $\geq$ 30 years group, respectively (all $p < 0.05$). Additionally, FVC%, FEV1%, and FEV1/FVC% decreased obviously in the 20—29 years group and the $\geq$ 30 years group compared with the 10—19 years group (all $p < 0.05$). Moreover, significant differences in FVC%, FEV1%, and FEV1/FVC% were observed between the

Table 1 Comparisons in smoking rate, pulmonary function, and pulmonary function indices between the exposed group and the control group.

Group	Exposed group (n = 328)	Control group (n = 169)	χ^2/t	p
Smoking distribution			0.344	0.558
Nonsmoking	152 (46.3)	83 (49.1)		
Smoking	176 (53.7)	86 (50.9)		
Pulmonary function			35.743	<0.001
Normal	213 (64.9)	152 (89.9)		
Abnormal	115 (35.1)	17 (10.1)		
Pulmonary function indexes				
FVC%	78.2 ± 6.9	90.4 ± 7.2	4.560	0.045
FEV1%	72.5 ± 5.4	89.4 ± 6.0	5.331	0.037
FEV1/FVC%	86.1 ± 6.3	98.3 ± 1.2	4.417	0.046

Data are presented as n (%) or mean ± standard deviation.
FEV 1 = forced expiratory volume in 1 second after full inspiration; FVC = forced vital capacity.

Table 2 Comparisons in pulmonary function indices stratified by length of service in the exposed group.

Length of service (y)	FVC%	FEV1%	FEV1/FVC%
< 10 group (n = 60)	88.6 ± 5.8	85.5 ± 6.3	97.2 ± 1.0
10–19 group (n = 112)	81.2 ± 4.7[a]	77.0 ± 5.1[a]	89.9 ± 4.2[a]
20–29 group (n = 107)	73.5 ± 5.9[a,b]	68.8 ± 7.6[a,b]	82.3 ± 6.7[a,b]
≥ 30 group (n = 49)	67.6 ± 7.1[a,b,c]	55.2 ± 8.4[a,b,c]	73.4 ± 7.5[a,b,c]

FEV 1 = forced expiratory volume in 1 second after full inspiration; FVC = forced vital capacity.
[a] Compared with < 10 group, $p < 0.05$.
[b] Compared with < 10–19 group, $p < 0.05$.
[c] Compared with < 20–29 group, $p < 0.05$.

20–29 years group and the ≥ 30 group (all $p < 0.05$; Table 2).

Comparison of pulmonary function index stratified by smoking

We stratified individuals in the exposed group by smoking, and the subgroup analysis showed that FVC%, FEV1%, and FEV1/FVC% of smokers were significantly lower than those of nonsmokers (all $p < 0.05$; Table 3).

The cumulative abnormal rate of pulmonary function increased with the increase of CDE dose in the exposed group. The cumulative abnormal rate increased from 0.33% in the ≥ 100 mg/m^3·years group to 98.41% in the ≥ 1700 mg/m^3·years group. There was a rapid increase in the cumulative abnormal rate from ≥ 1000 mg/m^3·years group to ≥ 1700 mg/m^3·years group (Table 4, Figure 1). A positive correlation between the CDE dose and the cumulative abnormal rate of pulmonary function was observed ($r = 0.759$, $p < 0.001$).

Discussion

Coal mine and silica dust may lead to impaired pulmonary function and also lung diseases which remain a relevant occupational hazard for miners [19,20]. The influence factors of CWP incidence have been extensively studied by researchers, and likely contributing factors for increasing CWP prevalence include mine size and low seam mining [6,21]. Findings in the present study revealed that abnormal rate of pulmonary function in the exposed group (35.1%) was much higher than the control group (10.1%), suggesting that coal mixture workers are more likely to have abnormal pulmonary function because of respiratory system function changes caused by long-term coal dust exposure [22]. Partially consistent with our findings, Kuempel et al [23] reported that cumulative exposure to respirable coal mine dust and cigarette smoking may increase emphysema severity in US coal miners. Respirable coal dust concentration in working area, CDE, and free silica content are significant risk factors for pulmonary function abnormality [1]. In addition, more than a quarter of 3771 eligible miners had evidence of CWP, abnormal pulmonary function, or both [24].

The pulmonary function indices (FEV1%, FEV1/FVC%, and FVC%) in the exposed group were significantly lower than those in the control group, showing that the pulmonary function indices of the coal mixture workers had decreased significantly, and serious pulmonary function damage was observed. An official report of the American Thoracic Society documented the relationship between occupational exposures and an increased risk in chronic cough, lower FEV1, and a lower FEV1/FVC ratio [25]. In addition, dust exposure was found to be associated with a routine decline in FEV1 in workers of Norwegian silicon carbide plants each year [26]. Compared with healthy miners, pulmonary function (FVC, FEV1, FEF50, FEF75, and FEF25–75% of predicted values) declined among miners with CWP, suggesting that changes in pulmonary function parameters are associated with the development of CWP among coal mine workers [27]. Pulmonary function indices (FVC%, FEV1%, and FEV1/FVC%) in the exposed group significantly declined from the < 10 years group to 10–19 years group, 20–29 years group. and ≥ 30 years group successively, demonstrating that the pulmonary ventilation function of dust-exposed workers would decrease with the increasing service length. The dust-exposed length may be associated with impaired pulmonary function since long-term exposure to coal dust can cause the occurrence of lung nodules and interstitial fibrosis, thereby affecting the function of lung ventilation and air exchange [4,11]. Correlation analysis

Table 3 Comparison of pulmonary function index stratified by smoking.

	Case	FVC%	FEV1%	FEV1/FVC%
Smoker	176	74.16 ± 4.49	69.34 ± 3.52	82.41 ± 4.11
Nonsmoker	152	82.88 ± 6.21	76.16 ± 4.86	90.38 ± 5.68
t		14.37	16.35	17.36
p		< 0.001	< 0.001	< 0.001

FEV 1 = forced expiratory volume in 1 second after full inspiration; FVC = forced vital capacity.

Table 4 Relationship between cumulative dust exposure dose and cumulative abnormal rate of pulmonary function.

CDE dose ($mg/m^3 \cdot y$)	Beginning of the period (*n*)	End of the period (*n*)	Pulmonary function abnormal population (*n*)	Adjusted population (*n*)	Abnormal rate	Cumulative abnormal rate
≥ 0	328	0	0	328	0.0000	0.0000
≥ ≥ 100	311	17	1	302.5	0.0033	0.0033
≥ ≥ 200	298	13	1	291.5	0.0034	0.0067
≥ ≥ 300	284	14	2	277	0.0072	0.0106
≥ ≥ 400	266	18	3	257	0.0117	0.0188
≥ ≥ 500	247	19	5	237.5	0.0211	0.0326
≥ ≥ 600	226	21	6	215.5	0.0278	0.0483
≥ ≥ 700	203	23	7	191.5	0.0366	0.0634
≥ ≥ 800	181	22	7	170	0.0412	0.0763
≥ ≥ 900	165	16	7	157	0.0446	0.0840
≥ ≥ 1000	147	18	8	138	0.0580	0.1000
≥ ≥ 1100	116	31	12	100.5	0.1194	0.1705
≥ ≥ 1200	87	29	12	72.5	0.1655	0.2651
≥ ≥ 1300	61	26	12	48	0.2500	0.3741
≥ ≥ 1400	37	24	14	25	0.5600	0.6700
≥ ≥ 1500	22	15	8	14.5	0.5517	0.8027
≥ ≥ 1600	12	10	6	7	0.8571	0.9359
≥ 1700	7	5	4	4.5	0.8889	0.9841

CDE = cumulative dust exposure.

further revealed a positive correlation between the CDE dose and the cumulative abnormal rate of pulmonary function, and there was a rapid increase in the cumulative abnormal rate from ~1000 $mg/m^3 \cdot$years group to ~1700 $mg/m^3 \cdot$years group, suggesting that a lower limit value of 1000 $mg/m^3 \cdot$years has reference significance.

In conclusion, higher abnormal pulmonary function rate was found in coal mixture workers, characterized by decreased FVC%, FEV1%, FEV1/FVC%, and dust-exposed length may be a critical risk factor for pulmonary function. The positive correlation between CDE dose and cumulative abnormal rate suggested a lower limit value of 1000 $mg/m^3 \cdot$years for CDE in coal miners in a Chinese population. We failed to put the chest X-ray changes into statistical analysis due to the incomplete data regarding the chest X-ray; therefore, further studies are required to confirm our findings in a larger population with different work types in coal miners and more comprehensive information.

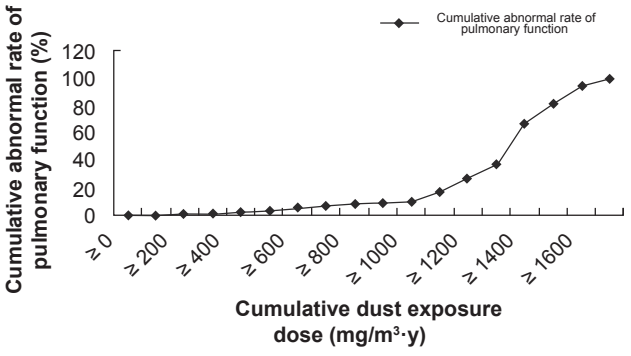

Figure 1. Does-response relationship between cumulative dust exposure and cumulative abnormal rate of pulmonary function in the exposed group. Along with the increase of cumulative dust exposure dose in the exposed group, the cumulative abnormal rate of pulmonary function also increased. There was a rapid increase in the cumulative abnormal rate from ≥ 1000 $mg/m^3 \cdot$years group to ≥ 1700 $mg/m^3 \cdot$years group.

Acknowledgments

We would like to thank our researchers for their hard work and colleagues for their valuable advice.

References

[1] Petsonk EL, Rose C, Cohen R. Coal mine dust lung disease. New lessons from old exposure. Am J Respir Crit Care Med 2013;187:1178—85.
[2] Seaman DM, Meyer CA, Kanne JP. Occupational and environmental lung disease. Clin Chest Med 2015;36:249—68.
[3] Miller BG, MacCalman L. Cause-specific mortality in British coal workers and exposure to respirable dust and quartz. Occup Environ Med 2010;67:270—6.
[4] Graber JM, Stayner LT, Cohen RA, Conroy LM, Attfield MD. Respiratory disease mortality among US coal miners; results after 37 years of follow-up. Occup Environ Med 2014;71:30—9.
[5] Santo Tomas LH. Emphysema and chronic obstructive pulmonary disease in coal miners. Curr Opin Pulm Med 2011;17:123—5.
[6] Suarthana E, Laney AS, Storey E, Hale JM, Attfield MD. Coal workers' pneumoconiosis in the United States: regional differences 40 years after implementation of the 1969 Federal Coal Mine Health and Safety Act. Occup Environ Med 2011;68:908—13.
[7] Centers for Disease C, Prevention. Pneumoconiosis and advanced occupational lung disease among surface coal miners—16 states, 2010—2011. MMWR Morb Mortal Wkly Rep 2012;61:431—4.
[8] Vallyathan V, Landsittel DP, Petsonk EL, Kahn J, Parker JE, Osiowy KT, et al. The influence of dust standards on the

prevalence and severity of coal worker's pneumoconiosis at autopsy in the United States of America. Arch Pathol Lab Med 2011;135:1550—6.
[9] Wu B, Ji X, Han R, Han L, Wang T, Yang J, et al. GITR promoter polymorphism contributes to risk of coal workers' pneumoconiosis: a case-control study from China. Immunol Lett 2014; 162:210—6.
[10] Lam KB, Yin P, Jiang CQ, Zhang WS, Adab P, Miller MR, et al. Past dust and GAS/FUME exposure and COPD in Chinese: the Guangzhou Biobank Cohort Study. Respir Med 2012;106: 1421—8.
[11] Bian LQ, Zhang Y, Jiang R, Mao L. Impairment of pulmonary function and changes in the right cardiac structure of pneumoconiotic coal workers in China. Int J Occup Med Environ Health 2015;28:62—70.
[12] Leaker BR, Barnes PJ, Jones CR, Tutuncu A, Singh D. Efficacy and safety of nebulized glycopyrrolate for administration using a high efficiency nebulizer in patients with chronic obstructive pulmonary disease. Br J Clin Pharmacol 2015;79: 492—500.
[13] Nishimura M, Makita H, Nagai K, Konno S, Nasuhara Y, Hasegawa M, et al. Annual change in pulmonary function and clinical phenotype in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2012;185:44—52.
[14] Ramirez-Venegas A, Sansores RH, Quintana-Carrillo RH, Velazquez-Uncal M, Hernandez-Zenteno RJ, Sanchez-Romero C, et al. FEV1 decline in patients with chronic obstructive pulmonary disease associated with biomass exposure. Am J Respir Crit Care Med 2014;190:996—1002.
[15] Lange P, Celli B, Agusti A, Boje Jensen G, Divo M, Faner R, et al. Lung-Function Trajectories Leading to Chronic Obstructive Pulmonary Disease. N Engl J Med 2015;373: 111—22.
[16] Glas J, Seiderer J, Bues S, Stallhofer J, Fries C, Olszak T, et al. IRGM variants and susceptibility to inflammatory bowel disease in the German population. PLoS ONE 2013;8:e54338.
[17] Huang YJ, Yuan XL, Miao ZY, Guo WH. The analysis on comparing pulmonary function of smoking COPD patient with that of non-smoking COPD patient. Jilin Med J 2011;32: 2542—3.
[18] Wu QL, Ding XP, Xu XH, Wang HD, Cheng XR. Investigation on pulmonary functional indices in workers exposed to dust in a coal mine industry group. China Occup Med 2010;37:261—2.
[19] Cohen RA, Patel A, Green FH. Lung disease caused by exposure to coal mine and silica dust. Semin Respir Crit Care Med 2008;29:651—61.
[20] Laney AS, Weissman DN. Respiratory diseases caused by coal mine dust. J Occup Environ Med 2014;56(Suppl. 10):S18—22.
[21] Laney AS, Attfield MD. Coal workers' pneumoconiosis and progressive massive fibrosis are increasingly more prevalent among workers in small underground coal mines in the United States. Occup Environ Med 2010;67:428—31.
[22] Wang ML, Beeckman-Wagner LA, Wolfe AL, Syamlal G, Petsonk EL. Lung-function impairment among US underground coal miners, 2005 to 2009: geographic patterns and association with coal workers' pneumoconiosis. J Occup Environ Med 2013;55:846—50.
[23] Kuempel ED, Wheeler MW, Smith RJ, Vallyathan V, Green FH. Contributions of dust exposure and cigarette smoking to emphysema severity in coal miners in the United States. Am J Respir Crit Care Med 2009;180:257—64.
[24] Blackley DJ, Halldin CN, Wang ML, Laney AS. Small mine size is associated with lung function abnormality and pneumoconiosis among underground coal miners in Kentucky, Virginia and West Virginia. Occup Environ Med 2014;71:690—4.
[25] Eisner MD, Anthonisen N, Coultas D, Kuenzli N, Perez-Padilla R, Postma D, et al. An official American Thoracic Society public policy statement: novel risk factors and the global burden of chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2010;182:693—718.
[26] Johnsen HL, Bugge MD, Foreland S, Kjuus H, Kongerud J, Soyseth V. Dust exposure is associated with increased lung function loss among workers in the Norwegian silicon carbide industry. Occup Environ Med 2013;70:803—9.
[27] Zou J, du Prel Carroll X, Liang X, Wang D, Li C, Yuan B, et al. Alterations of serum biomarkers associated with lung ventilation function impairment in coal workers: a cross-sectional study. Environ Health 2011;10:83.