Exposure to PM₁₀ as a risk factor for the development of nasal obstruction and chronic obstructive pulmonary disease

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Objectives: To investigate whether air pollution is a potential risk factor for airways obstruction.

Methods: A prospective cohort study (11.3 ± 2.9 years) that took place in two areas (Eordea where concentration of PM₁₀ was high and Grevena, Greece). We used the MRC questionnaire, spirometry, and anterior rhinomanometry at both visits.

Results: Initially we examined 3046 subjects. After excluding chronic obstructive pulmonary disease (COPD) patients, we re-examined 872 subjects and 168 of them had developed COPD (Grevena: 24.3%, Eordea: 18.5%). Multivariable logistic regression analysis showed that the area of residence and thus exposure to air pollution was not a risk factor for the development of COPD (OR: 0.51, 95% CI: 0.18–1.46, $P=0.21$). On the other hand, residence in Eordea was strongly related to the development of severe nasal obstruction (OR: 11.47, 95% CI: 6.15–21.40, $P<0.001$). Similar results were found after excluding patients with COPD stage I as well as in the subgroup of never smokers.

Conclusion: Air pollution was associated with severe nasal obstruction but not with COPD development.

Keywords: COPD, Air pollution, PM₁₀, Spirometry, Rhinomanometry

Introduction

Chronic obstructive pulmonary disease (COPD) is considered a major public health problem as it is one of the most common causes of morbidity and mortality worldwide. Chronic obstructive pulmonary disease was the sixth cause of death worldwide in 1990 and it is estimated that this will rise to the third range in place by 2020, mainly as a result of increased life expectancy.¹ The direct and indirect economic costs of COPD for the insurance system and society are extremely high.^{2,3} Common problems in everyday clinical practice are: (a) underdiagnosis,^{4,5} (b) misdiagnosis,⁶ and (c) the varying prevalence of the disease according to different spirometric criteria, age group, and geographic area.^{7,8}

The recognition of etiologic factors for the development of the disease is considered an important step

toward the implementation of preventive measures and treatments. Cigarette smoking is the most important risk factor for COPD development. Many longitudinal studies have proved that there is an increased risk of COPD among smokers compared to non-smokers while the rate of decline in respiratory function was much slower after smoking cessation.^{9,10} Even though up to one fourth of patients with mild to severe COPD are estimated to be non-smokers, the exact risk factors in this subgroup are still not clear.^{11,12} There are some cohort studies that concluded that improvement of particulate air pollution had lessened decline in respiratory function and symptoms.^{13,14}

On the other hand, lifetime prevalence of rhinitis is estimated between 10 and 20% worldwide with a variety of causative factors and negative effects on the quality of life.^{15,16} However, there are not many long-term, cohort studies that have investigated the importance of air pollution for the development of nasal obstruction. The purpose of the present study is to investigate whether air

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pollution is a potential risk factor for the development of COPD and nasal obstruction by following up two subgroups of general population who have been exposed to different levels of air pollution.

Methods

The current study was a prospective cohort study (baseline visit: 1994–1995 and follow-up: 2006–2007) that took place in two different geographic areas in Greece.

Areas

Area A was the basin of Eordea (Kozani, Greece). The basin of Eordea has an elongated diameter of approximately 30 km, along a north-western to south-eastern axis, with a transverse diameter of 8–12 km, and occupies a surface area of 450 km². It is surrounded by mountains whose altitude is approximately 1000 m. In the basin there are five coal-fired electrical power factories (each with 4–6 steam-powered stations) which belong to the National Electrical Power Corporation. They use exclusively lignite (40 million tons annually) as there are large surface deposits in the same area. These factories represent ≈70% of the national electric power production (3.6 GW annually). Mining and burning of the lignite in association with poor air recycling because of geographic location lead to particulate air pollution that was usually out of permissible levels. Concentrations of airborne particles with an aerodynamic diameter ≤10 μm (PM₁₀, could be inhaled) were measured and recorded over time (2–3 times per week) through a network of 14 monitoring stations in the basin of Eordea. Measurements of PM₁₀ concentration were based on absorption of beta-radiation. We used mean monthly values based on longitudinal measurements of all 14 monitoring stations. From 1993 to 2008 there was a gradual placement of electrostatic precipitators that has significantly decreased air pollution levels. The mean annual income per capita in the county of Kozani was 13 003 Euros (98.7% of the mean national income) for 2004 according to the National Statistical Administration.¹⁷

Area B was a mountainous region in the county of Grevena, Greece with no factories within a radius of 50 km and no other significant sources of air pollution. A modeling system consisting of 5th generation NCAR/Penn State Misoscale Model (MM5) and comprehensive air quality model with extensions (CAMx) has been setup to simulate atmospheric pollution in Grevena as there were no monitoring stations for air pollution in this specific area. Meteorological conditions and data from air pollution monitors settled in the nearby counties were incorporated in the above model. We applied the simulation model four times annually (separately for every season) for the years 1994 and 2007.

The mean annual income per capita in the county of Grevena was 9510 Euros for 2004 (72.2% of the mean national income) according to the National Statistical Administration.

Subjects

Initially a population sample (≥30-year-old subjects) was randomly selected from the local telephone books. Subjects were invited via a mailed letter by a network of local general practitioners and pulmonologists and all subjects who were willing to take part in a screening program for pulmonary diseases comprised the initial cohort of the study (3046 subjects: 2687 in area A and 359 in area B, mean age ± SD: 56.5 ± 13.8 years). All participants were permanent inhabitants of the above geographic areas. Exclusion criteria were: (a) medical diagnosis of any obstructive pulmonary disease (e.g. asthma, COPD, and bronchiectasis) documented in the health booklet of the Social Fund, (b) history of other pulmonary diseases (e.g. tuberculosis, interstitial lung disease, and lung cancer) or thoracic surgery (c) symptoms indicating uncontrolled cardiac disease (e.g. coronary heart disease, congestive heart failure, and arterial hypertension) and (d) inability to perform technically acceptable spirometry or anterior rhinomanometry. After excluding those who were diagnosed with COPD during the first visit (269 subjects, 8.8%, mean age ± SD: 65.8 ± 11.1 years), we re-examined (2006–2007) the subjects who agreed to participate (872 out of 2777 subjects: 757 in area A and 115 in area B). Patients who died (83 subjects in Eordea and 13 in Grevena), refused to undergo the second round of medical examinations (987 in Eordea and 129 in Grevena), or had changed residence (626 in Eordea and 67 in Grevena) were not included in the follow-up visit. The Medical Ethics Committee of the “G. Papanicolaou” Hospital, Thessaloniki, Greece approved the study protocol.

Outcome measurements

We used exactly the same diagnostic tools for both visits. Medical history was evaluated using the Medical Research Council Questionnaire (1986). We collected data on: (a) current employment and occupational history, (b) respiratory symptoms (e.g. cough, phlegm, dyspnea, and wheezing), (c) diagnosis of cardiopulmonary diseases, and (d) smoking habits. All subjects performed spirometry (Vitalograph Ltd, Buckingham, England) according to American Thoracic Society/European Respiratory Society guidelines.¹⁸ Predicted values of the European Respiratory Society were used.¹⁹ All measurements were performed before and 15–30 minutes after inhalation of 400 μg salbutamol via a metered dose inhaler. Patients with forced expiratory volume in 1 second (FEV₁)/forced vital capacity (FVC) <0.7 after bronchodilation were defined as COPD patients and categorized according

Figure 1 Flow diagram of the study. During the follow-up period 18.5% of the subjects in Eordea and 24.3% in Grevena developed COPD.

to GOLD (global initiative for obstructive lung disease) stages (FEV₁% predicted after bronchodilation: I (mild) $\geq 80\%$, II (moderate) 80–50%, III (severe) 50–30% and IV (very severe) $<30\%$). We measured nasal flows using anterior rhinomanometry, Rhinotest mP 500.²⁰ In order to record the flow, the subject breathed quietly in the seated position via an air-tied face mask with one nostril open. An intranasal sponge with a catheter was introduced into the other nostril, transferring the pressure variations to the device. Flow was measured at 150 Pa and reported in ml/second. Flow sum for both nostrils was considered normal if >850 ml/second while severe nasal obstruction was defined as flow sum ≤ 500 ml/second.

We used the following definitions: *current smoker*: a person who smoked at least one cigarette during the last month; *ex-smoker*: a person who gave up smoking at least 1 month before the evaluation; *chronic bronchitis*: the presence of productive cough for 3 months yearly for the last 2 years; *rhinitis*: the presence of nasal congestion, discharge, and sneezing without respiratory infection during one or more seasons; *employee in the*

National Company of Electricity production: a person who worked there for the last 5 years.

Statistical analysis

Quantitative variables were expressed using mean values with standard deviations (SD) and median values with ranges (minimum, maximum). Initially, the quantitative variable normality of distribution was assessed by Kolmogorov–Smirnov test. The non-parametric Mann–Whitney *U* test was applied to compare quantitative variables between regions. The analysis of covariance (ANCOVA) was used to adjust the effect of age and smoking on the rate of decline in variables between sites. The qualitative variables are expressed in frequencies and percentages. The relationship between qualitative variables was tested by the χ^2 . The logistic regression analysis was used to find etiologic factors for COPD and nasal obstruction in the second visit. Initially, we applied univariate analysis and then the multivariable model was built with all the significant univariate parameters ($P < 0.05$) adjusting for area of residence and smoking. Odds ratios as well as the 95% confidence intervals were

Table 1 Data of mean PM₁₀ concentration (µg/m³) measured through a network of 14 monitoring stations in the basin of Eordea and a simulation model for the area of Grevena

	Eordea (area A)			Grevena (area B)
Year of measurements	1991	2001	2009	1994 and 2007
Number of measurements/year	147	321	224	4
Mean annual concentration	75	49	54	16
Maximum	180	209	206	19
Minimum	26	11	11	11
Percentage of days per year with concentration >50 µg/m ³	78%	39%	42%	0%

presented. Sensitivity analysis was performed by excluding smokers or ex-smokers from the analysis. In all statistical tests the level of significance was set as 0.05 and two-sided. The statistical analyses were performed with the statistical package SPSS 17.0.

Results

The flow diagram of the study is shown in Fig. 1. Mean follow up of study subjects was 11.3 ± 2.9 years. The subgroup of subjects who were lost to follow-up visit (e.g. died, declined to participate, or changed residence) had comparable mean age and lung function parameters to those who were included in the study analysis (data not shown).

Regarding the EC standard levels of air quality for PM₁₀ the mean annual concentration should be less than 40 µg/m³, while the daily average concentration should not exceed 50 µg/m³ more than 35 days per year.²¹ As shown in Table 1, the average annual concentration and the percentage of days per year with concentrations of PM₁₀ more than 50 µg/m³ were both above the permissible levels for area A. Decrease in PM₁₀ concentrations could be attributed to placement of electrostatic precipitators during the same time period. On the other hand, calculated PM₁₀ concentrations for the area of Grevena were below 20 µg/m³ at all recorded times.

Tables 2 presents data about anthropometric measurements, MRC questionnaire, spirometry, and rhinomanometry for individuals who have been examined at both sites ($N=872$). One hundred and sixty-eight subjects, 28 in the region of Grevena (24.3%) and 140 in Eordea (18.5%) developed COPD during the follow-up period (cumulative incidence: 19.3%, Fig. 1). Seventy-six patients (45.2%) had developed mild COPD, 79 moderate (47%), 12 severe (7.2%), and one patient suffered from very severe COPD (0.6%). We found that patients who developed COPD were older; most of them were males; with worse smoking status. There was a greater prevalence of chronic bronchitis among COPD compared to non-COPD subjects. There were 69 non-smokers who developed COPD, 52 of them (75.4%) had stage I disease while 24/99 (24.2%) of smokers with COPD had stage I disease.

The mean ($\pm$ SD) annual rate of decline of FEV₁ was -61.5 ± 41.4 for those who developed the disease compared to -39.2 ± 37 ml for the remaining subjects ($P<0.001$). Also the mean ($\pm$ SD) rate of decline of FEV₁ was -50.3 ± 35 ml/year for patients with mild disease versus -71.5 ± 44.2 ml/year for those who developed moderate or severe disease ($P=0.002$). We also found that patients in Eordea with severe nasal obstruction (flow sum ≤ 500 ml/second) at the

Table 2 Anthropometric, medical history, spirometric, and rhinomanometric data of the study population (Eordea) and control group (Grevena) at baseline and 11.4 ± 2.7 years later

Variables	Baseline visit		Follow-up visit	
	Eordea ($N=757$)	Grevena ($N=115$)	Eordea ($N=757$)	Grevena ($N=115$)
Age (years)	55.8 (11.7)*	60.3 (9.3)	67.1 (11.2)	71.7 (9.0)
Females, n (%)	349 (46.1)	54 (47)	349 (46.1)	54 (47)
Height (cm)	162.9 (8.7)	162.0 (8.5)	162.5 (9.1)	160.8 (8.7)
Smokers, n (%)	318 (42.0)	37 (32.2)	317 (41.9)	39 (33.9)
Packs/year, Median (IQR)	33 (18, 50)	44 (29, 53)	45 (26, 72)	55 (30, 68)
Chronic bronchitis n (%)	287 (37.9) [†]	18 (15.7)	392 (51.8) [†]	29 (25.2)
Rhinitis, n (%)	257 (33.9) [‡]	18 (15.7)	320 (42.3) [‡]	17 (14.8)
FVC (ml)	3,505 (1009)	3,307 (995)	2,842 (939)	2,728 (833)
FVC% pred.	101.5 (18.4)	100.4 (16.0)	90.1 (20.5)	92.5 (17.8)
FEV ₁ (ml)	2,808 (843)	2,650 (717)	2,308 (794)	2,135 (681)
FEV ₁ % pred.	106.0 (19.9)	106.2 (19.4)	101.1 (25.5)	101.7 (22.6)
FEF ₂₅₋₇₅ % pred.	103.5 (37.7)	96.9 (38.1)	110.3 (51.6)	101.4 (54.6)
FEV ₁ /FVC%	80.8 (6.7)	80.4 (7.6)	82.2 (9.2)	78.3 (9.4)
Sum of nasal flows (ml/second), Median (IQR)	637 (416, 874) [§]	882 (692, 1082)	458 (294, 600) [§]	737 (599, 884)
Severe nasal obstruction, n (%)	250 (33.0)	13 (11.3)	436 (58.5)	12 (10.5)

Smokers: ex- and current, FEF₂₅₋₇₅: forced expiratory flow 25–75% FVC, IQR: interquartile range, severe nasal obstruction: flow sum ≤ 500 ml/second. All parameters are expressed as mean (SD) if not otherwise noticed.

* $P<0.001$, [†] $P<0.001$ (Chi-square test), [‡] $P<0.001$ (Chi-square test), [§] $P<0.001$ (Mann-Whitney *U* test), ^{||} $P<0.001$ (Chi-square test).

first visit showed faster annual rate of decline of FEV₁ compared to the rest of the subjects in the group of Eordea (-6.5 vs -3.3 ml/year, $P=0.018$).

Trying to identify etiologic factors for developing COPD, we estimated odds ratios for the following factors: geographic area of residence, age, sex, smoking, quantitative smoking history (packs/year), employment in the National Electric Company production (we hypothesize that these workers were more intensively exposed to particulate air pollution), presence of chronic bronchitis, symptoms of rhinitis, FEV₁, FEF_{25–75}, the ratio FEV₁/FVC%, and the presence of severe nasal obstruction (Table 3). The area of residence and thus exposure to air pollution did not appear to be an etiologic factor for the development of the disease. Additionally the percentage decrease of FEV₁ ($-4.3 \pm 0.6\%$ for Eordea vs $-4 \pm 1.7\%$ for Grevena, $P=0.885$) and FEF_{25–75} ($-9.2 \pm 2\%$ for Eordea vs $-13.2 \pm 5.1\%$ for Grevena, $P=0.474$) did not differ between the two areas after adjustment for age and smoking status. On the other hand, we found that measured nasal flows among inhabitants of Grevena were higher compared to Eordea (flow sum 1st visit: 897 ± 343.3 vs 672.6 ± 342.4 ml/second, $P<0.001$, flow sum 2nd visit: 758.6 ± 259.7 vs 470.9 ± 230.8 ml/second, $P<0.001$). Table 4 presents logistic regression analysis for possible etiologic factors for severe nasal obstruction development. It was found that residence in area A was strongly related to severe nasal obstruction (OR: 11.47, 95% CI: 6.15–21.40).

After excluding patients with COPD stage I (Table 5) we found similar results as for the whole COPD group; FEV₁/FVC% and FEF_{25–75} pred. were the only statistically significant etiologic factors. Additionally in the subgroup of never smokers, the multivariable logistic regression analysis showed that living in Eordea was not a risk factor for development

of COPD (OR: 0.584, 95% CI: 0.219–1.557, $P=0.282$) but was associated with severe nasal obstruction (OR: 15.113, 95% CI: 6.406–35.651, $P<0.001$) as was estimated for the whole study sample.

Discussion

The main conclusions of the present study were: (a) residence in an area of increased particulate air pollution was associated with severe nasal obstruction but not with increased risk of developing COPD and (b) the presence of severe nasal obstruction was associated with more rapid decline in FEV₁ among inhabitants of a polluted area. To our current knowledge this is the first prospective study that provides evidence on the relationship between particulate air pollution and increased risk of developing severe nasal obstruction as objectively assessed by rhinomanometry.

There is enough evidence about the acute effects of air pollution on the respiratory system. In a systemic review analysis²² it was concluded that concentration of airborne coarse particles, usually defined and measured as particles >2.5 and <10 μm , were associated with respiratory, COPD, and cardiovascular hospital admissions. Similarly, a national study in 36 US cities during the period 1986–1999 showed that during the warm season hospital admissions for COPD were associated with PM₁₀ exposure on the day before (1.47% increase for every 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀) while admissions for pneumonia were associated with PM₁₀ exposure on the same day of exposure (0.84% increase for every 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀).²³

According to an international follow-up study²⁴ that took place 8.9 years after baseline evaluation among 6924 persons, new onset of chronic phlegm was 4.0% (4.5% in males and 3.5% in females). PM_{2.5} content at center level did not show any association with prevalence or new onset of chronic phlegm.²⁴ Additionally, a prospective study among children

Table 3 Logistic regression analysis for possible etiologic factors for chronic obstructive pulmonary disease (COPD) development

Variables	Univariate			Multivariable		
	OR	95% CI	P-value	OR	95% CI	P-value
Area of residence (Eordea vs Grevena)	0.705	0.443, 1.121	0.14	0.51	0.18, 1.46	0.210
Age (at follow-up)*	1.692	1.411, 2.042	<0.001	1.14	0.82, 1.60	0.441
Gender (male)	2.543	1.764, 3.666	<0.001	2.12	0.30, 15.10	0.455
Smoking	2.435	1.728, 3.431	<0.001	1.95	0.98, 3.86	0.057
Packs/year†	1.195	1.094, 1.305	<0.001	1.10	0.97, 1.24	0.135
Employment in the National Company of Electricity production	1.291	0.83, 2.007	0.257			
Chronic bronchitis	1.877	1.334, 2.647	<0.001	1.14	0.58, 2.22	0.713
Rhinitis	0.856	0.967, 0.672	0.843			
FEV ₁ % pred. ‡	0.618	0.556, 0.679	<0.001			
FEF _{25–75} % pred. ‡	0.562	0.511, 0.618	<0.001			
FEV ₁ /FVC%	0.657	0.615, 0.701	<0.001	0.71	0.66, 0.77	<0.001
Severe nasal obstruction	1.417	0.994, 2.019	0.054			

Smoking: ex- and current, FEF_{25–75}: forced expiratory flow 25–75% FVC, severe nasal obstruction: flow sum ≤ 500 ml/second, for other variables: see definitions. *For every additional 10 years, †for every additional 10 packs/year, ‡for every additional 10% pred.

Table 4 Logistic regression analysis for possible etiologic factors for nasal obstruction development

Variables	Univariate			Multivariable		
	OR	95% CI	P-value	OR	95% CI	P-value
Area of residence (Eordea vs Grevena)	11.99	6.48, 22.20	<0.001	11.47	6.15, 21.40	<0.001
Age (at follow-up)*	1.07	0.95, 1.21	0.282			
Gender (male)	1.01	0.77, 1.31	0.990			
Smoking	1.19	0.91, 1.57	0.207	1.07	0.80, 1.44	0.650
Packs/year†	1.07	0.90, 1.16	0.108			
Employment in the National Company of Electricity production	1.15	0.80, 1.67	0.446			
Chronic bronchitis	1.60	1.20, 2.12	0.001	1.17	0.86, 1.60	0.309
Rhinitis	1.79	1.34, 2.41	<0.001	1.48	1.08, 2.03	0.015
FEV ₁ % pred. ‡	0.93	0.88, 1.00	0.055			
FEF ₂₅₋₇₅ % pred. ‡	0.96	0.93, 0.99	0.035	0.95	0.91, 0.98	0.006
FEV ₁ /FVC%	0.98	0.96, 1.00	0.077			

Smoking: ex- and current, FEF₂₅₋₇₅: forced expiratory flow 25–75% FVC, severe nasal obstruction: flow sum ≤ 500 ml/second, for other variables: see definitions. *for every additional 10 years, †for every additional 10 packs/year, ‡for every additional 10% pred.

with asthma in southern California found no association between coarse particles and bronchitis symptoms.²⁵

On the other hand, there are some studies about the relationship between improvement on the air quality and lung function. A recently published cohort study in Germany with a follow-up of 20 years¹³ among elderly women concluded that parallel to the decline of ambient air pollution in the Ruhr area (PM₁₀ declined in average by 20 $\mu\text{g}/\text{m}^3$) there was a corresponding significant reduction of the age-related increase in prevalence of respiratory symptoms (prevalence of chronic cough with phlegm was estimated at 21.4% without air pollution reduction, observed prevalence 13.3%) and COPD (mild COPD was estimated at 39.5% without air pollution reduction, observed prevalence 18.2%). In a prospective study conducted in Switzerland among 9651 adults over an 11-year follow-up period, the net effect of a decline of 10 μg of PM₁₀ per cubic meter was to reduce the annual rate of decline in FEV₁ by 9% and of FEF₂₅₋₇₅ by 16%.¹⁴

DIMCA study in Denmark²⁶ as well as another prospective study from Northern Sweden²⁷ provide sufficient evidence about the prognostic role of spirometric parameters, specific respiratory symptoms, smoking status, and increasing age for the

development of COPD. Similarly in the present study we found that spirometric parameters and cigarette smoking were related with the development of COPD.

Of those who developed COPD in our study, 41.1% were non-smokers. In NHANES III survey among subjects 30–80 years old, 23% of patients with obstructive spirometric disorder were non-smokers of whom only 31.5% had a prior medical diagnosis of asthma and/or COPD.²⁸ Taking into account that COPD prevalence for subjects >60 years old is quite similar using the FEV₁/FVC < lower limit of normal or GOLD stages 2–4 criterion instead of the FEV₁/FVC < 0.7 criterion,⁸ we believe that the high portion (75.4%) of COPD stage I among non-smokers in the present study may represent false positive cases attributed to age-related biases.

In Lung Health Study 3 the subgroup of participants who continued to smoke 11 years after the initial assessment (25.9%) showed a rate of decline of FEV₁ 60 ml/year, while 38% of them showed FEV₁ < 60% of predicted value.¹⁰ In contrast, those who continued to abstain from smoking (16.7%) showed a rate of decline of FEV₁ 27 ml/year, while only 10% of them showed FEV₁ < 60% of predicted value. The above rate was similar in the subgroup of those who developed COPD (61.5 $\pm$ 41.4 ml/year) in our study. Recent guidelines of the GOLD, as in previous ones, have withdrawn stage 0 (at risk) from the severity scale as symptoms of chronic bronchitis are not a reliable predictor of development of COPD.²⁹ Similarly we concluded that the presence of chronic bronchitis was not associated with the development of the disease.

An interesting finding of the present study is the higher rates of severe nasal obstruction among subjects exposed to increased levels of PM₁₀ and the relationship between severe nasal obstruction and rate of decline in FEV₁. There are no prospective studies investigating the effects of particulate air pollution on nasal flows measured by rhinomanome-

Table 5 Multivariable logistic regression analysis for possible etiologic factors for chronic obstructive pulmonary disease (COPD) (stages II–IV) development

Variables	OR	95% CI for OR		P-value
		Lower	Upper	
Gender (male)	2.030	0.821	5.021	0.125
Age (at follow-up)	1.018	0.986	1.050	0.275
Smoking	1.346	0.592	3.064	0.479
FEV ₁ % pred.	0.988	0.961	1.017	0.115
FEF ₂₅₋₇₅ % pred. *	0.638	0.479	0.851	0.002
FEV ₁ /FVC% *	0.272	0.129	0.568	0.001
Chronic bronchitis	1.065	0.584	1.942	0.205

* For every additional 10% pred.

try. Hwang *et al.* found that the prevalence of allergic rhinitis was weakly related to PM₁₀ (aOR = 1.00, 95% CI: 0.99–1.02) among children.³⁰ It has to be mentioned that in the above study the diagnosis of rhinitis based on questionnaires and the exposure time to PM₁₀ was substantially less compared to our study. In a previous study of our department, we found that lignite miners in the same area demonstrated high prevalence of severe nasal obstruction (above 70%) and atrophic rhinitis (14%).³¹ We hypothesized that coarse particles may be trapped in the nasal mucosa leading to pathologic lesions and eventually to obstruction.

The main limitations of the present study are: (a) the control group was much smaller compared with the study sample, (b) the follow-up period may be not long enough (11.3 ± 2.9 years) to study a chronic progressive disease such as COPD, (c) the high proportion of participants who were lost to follow-up, and (d) we had no data about socioeconomic conditions of the participants that are usually associated with prevalence of COPD.

Conclusion

We concluded that particulate air pollution was associated with the development of severe nasal obstruction but not with COPD. Subjects with severe nasal obstruction demonstrated faster rate of decline of FEV1 while spirometric variables were associated with COPD development as it was anticipated.

Conflict of Interest

All authors of the present study have no conflict of interest to declare.

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