

# Association between low pulmonary function and metabolic risk factors in Korean adults: the Korean National Health and Nutrition Survey

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Received 24 February 2009; accepted 2 December 2009

## Abstract

Impaired lung function is a risk factor for cardiovascular events and mortality. In addition, lung function impairment is also associated with insulin resistance and type 2 diabetes mellitus. It is well known that a common mechanism, such as insulin resistance and obesity, underlies metabolic syndrome. Our aim was to evaluate the association between impaired lung function and metabolic risk factors using data from a nationwide survey of chronic obstructive pulmonary disease prevalence in Korea and the Korean National Health and Nutrition Survey in 2001. The study population included 4001 subjects (aged  $\geq 18$  years) who underwent spirometry at least twice. We analyzed the association of low pulmonary function with metabolic syndrome components using multiple linear regression and also analyzed the association of metabolic syndrome with restrictive lung disease and obstructive lung disease using multiple logistic regression adjusted for waist to height ratio, sex, age, smoking, and the other covariates. Waist girth, systolic blood pressure, and triglyceride were associated with forced vital capacity (FVC); and only triglyceride was so with forced expiratory volume in 1 second ( $FEV_1$ ), but not with  $FEV_1/FVC$  ratio. The odds ratio of metabolic syndrome for restrictive lung disease ( $FVC < 80\%$ ,  $FEV_1/FVC > 0.7$ ) was 1.40 (95% confidence interval, 1.01–1.98), and that for obstructive lung disease ( $FEV_1/FVC < 0.7$ ) was 0.93 (95% confidence interval, 0.67–1.28) after adjustment for covariates. These results indicate that low pulmonary function in the general population is associated with clustering of metabolic syndrome risk factors and, furthermore, that restrictive lung disease is also related to metabolic syndrome.

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## 1. Introduction

Impaired lung function, as measured by forced vital capacity (FVC) or forced expiratory velocity in 1 second ( $FEV_1$ ), is a risk factor for cardiovascular events and mortality [1–4]. Even modest reductions in expiratory flow volumes increase the risk of ischemic heart disease, stroke, and sudden cardiac death by 2- to 3-fold [1,3,5–7]. Cardiovascular conditions are the leading causes of mortality

among those with impaired lung function [1,3,6]. In addition, lung function impairment is also associated with insulin resistance [8,9] and type 2 diabetes mellitus [9,10].

Metabolic syndrome, characterized by clustering of abdominal obesity, hypertension, impaired glucose tolerance, and atherogenic dyslipidemia, is implicated in subsequent development of type 2 diabetes mellitus [11,12], cardiovascular disease, and mortality [12–14]. It is well known that a common mechanism, such as insulin resistance and obesity, underlies metabolic syndrome.

Results of several studies suggest that there is link between obesity and impaired lung function in healthy adults [15,16] and that fasting serum insulin levels and insulin

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resistance are inversely associated with FVC and FEV<sub>1</sub> [8]. In light of these findings, a positive link between impaired lung function and metabolic syndrome is to be expected.

Therefore, we conducted this study to evaluate the association of lung function, as measured by FVC or FEV<sub>1</sub>, and metabolic risk factors using data from a nationwide survey of chronic obstructive pulmonary disease (COPD) prevalence in Korea and the Korean National Health and Nutrition Survey (KNHNS) in 2001 [17].

## 2. Subjects and methods

### 2.1. Participants

Data were obtained from COPD prevalence in Korea and KNHNS, conducted in 2001; this was a cross-sectional and nationally representative survey. A stratified multistage clustered probability sampling design was used to select a representative sample of civilian, noninstitutionalized Korean adults aged at least 18 years. Two hundred sampling frames (13 200 households) from primary sampling units were randomly sampled, and 37 769 individuals from these sampling frames were included in the 2001 KNHNS. The COPD prevalence survey was completed by 9243 of 12 647 individuals who participated in the 2001 KNHNS. Among 9243 subject, 88.8% (8209 subjects, aged  $\geq 18$  years) responded to the questionnaire, of whom 4785 completed spirometry and 4001 underwent at least 2 spirometry measurements. The study was approved by the Institutional Review Board of Hallym University Sacred Heart Hospital.

### 2.2. Measurements

#### 2.2.1. Lung function tests

Trained interviewers visited the homes of subjects and administered a standardized questionnaire on physician-diagnosed diseases. A few days later, spirometry was performed by specially trained technicians according to the 1994 American Thoracic Society recommendations [18], using a dry rolling seal spirometer (Model 2130; Sensor-Medics, Yorba Linda, CA). We analyzed only data from subjects with 2 or more acceptable spirometry performances. Values used in the study were FVC, FEV<sub>1</sub>, and FEV<sub>1</sub> to FVC ratio (FEV<sub>1</sub>/FVC). *Restrictive lung disease* was defined as FVC less than 80% and FEV<sub>1</sub>/FVC greater than 0.7 of predicted value. Chest x-rays were obtained when spirometry was performed, using specially equipped mobile examination vehicles; and 2 radiologists evaluated the chest x-ray film. Procedures of performing spirometry and taking chest x-ray films are described in detail elsewhere [19].

#### 2.2.2. Laboratory measurements

A venous blood sample was collected from each subject after a 12-hour fast and was centrifuged and refrigerated at the examination site, and transferred in an icebox to a central laboratory in Seoul on the same day. Plasma glucose, total

cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglyceride (TG) levels were measured using a Hitachi 747 autoanalyzer (Tokyo, Japan).

We used the definition of metabolic syndrome proposed by the National Cholesterol Education Program Adult Treatment Panel III [20] as the presence of 3 or more of the following risk factors: (1) blood pressure (BP) of 130/85 mm Hg or higher, (2) TG of 1.7 mmol/L or higher (150 mg/dL), (3) low HDL-C of less than 1.0 mmol/L (40 mg/dL) for men and less than 1.3 mmol/L (50 mg/dL) for women, (4) fasting glucose of 6.1 mmol/L or higher (110 mg/dL), and (5) abdominal obesity as defined by Asia-Pacific cutoff limits (waist circumference  $>90$  cm for men and  $>80$  cm for women) [21]. If participants were using antihypertensive or diabetes medication, they were considered to show high BP or high fasting glucose.

Self-reported alcohol intake, smoking, and physical exercise habits were estimated from questionnaire responses. Individuals were classified as nonsmokers, exsmokers, or current smokers. Cigarette pack-years were calculated by multiplying the number of years of smoking by the average number of cigarettes smoked per day and dividing by 20. Alcohol consumption was estimated from reported intakes and use frequencies, divided into none, at least 1 cup per week, at least 5 cups once a week or more, and at least 5 cups 3 times a week or more [22]. Physical activity was divided according to estimated weekly energy consumption, as follows: none, less than 7.5 kcal/kg, 7.5 to 15 kcal/kg, and greater than 15 kcal/kg [20]. Socioeconomic status was categorized by monthly income into low, middle, and high.

Body weight and height were measured in subjects wearing light clothing without shoes. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Waist girth was measured from the narrowest point between the lower borders of the rib cage and the iliac crest [23]. Blood pressure was measured in a sitting position after a 10 minute rest period. Two readings were taken at 5-minute intervals and averaged for data analysis.

### 2.3. Data analysis

Clinical characteristics were compared between normal, restrictive lung function, and obstructive lung function using analysis of variance for continuous variables and the  $\chi^2$  test and Fisher exact test for categorical variables. Because TG was highly skewed, this variable was natural-log-transformed for all analyses. To investigate individual associations of each metabolic component with pulmonary function (FVC, FEV<sub>1</sub>, FEV<sub>1</sub>/FVC), we performed multiple linear regression analysis adjusted for covariates and then computed effect size for each variable in a regression model.

Multiple logistic regression was used to examine adjusted odds ratios (ORs) for restrictive lung disease in metabolic syndrome. Adjusted variables for model 1 were sex, age, and

smoking; those for model 2 were waist to height ratio, sex, age, and smoking; and those for model 3 were waist to height ratio, sex, age, smoking, physical activity, alcohol, and socioeconomic status. All statistical analyses were performed using STATA version 10 (StataCorp, College Station, TX).

### 3. Results

The study population included 4001 subjects aged at least 18 years who performed spirometry at least twice. Although there were differences in age and sex between those who performed spirometry at least twice (performers) and who did not (nonperformers), the age and sex distribution patterns of performers were similar to patterns seen in the entire subject group, indicating that the spirometry data were representative [19].

Clinical characteristics of study participants belonging to the normal population, with reference to restrictive and obstructive lung function, are presented in Table 1. Subjects (both men and women) in the restrictive and obstructive lung

function group were more likely to be older; to have more metabolic syndrome; to have higher waist girth, systolic BP, diastolic BP, fasting glucose level, and TG concentration; and to have lower levels of HDL-C. Men with obstructive lung function were likely to be current or former smokers and to drink more and exercise less. In women, none of BMI, smoking status, or alcohol intake differed between groups. When restrictive and obstructive lung functions were compared, metabolic syndrome prevalence was similar in men, but was higher in women with restrictive lung function.

#### 3.1. Relationship between metabolic components and pulmonary functions

Tables 2–4 summarized the regression coefficients for metabolic risk components entered individually into regression models exploring predicted FVC percentage, predicted FEV<sub>1</sub> percentage, and FEV<sub>1</sub>/FVC ratio.

When components were separately entered into models (model 1), waist girth ( $P = .009$ ), systolic BP ( $P < .001$ ), fasting glucose level ( $P = .021$ ), and number of metabolic

Table 1  
Characteristics of participants

	Men (n = 1836)				Women (n = 2165)			
	Normal (n = 1502)	Restrictive lung function (n = 96)	Obstructive lung function (n = 238)	P	Normal (n = 1952)	Restrictive lung function (n = 121)	Obstructive lung function (n = 92)	P
Age (y)	40.4 ± 12.8	52.3 ± 15.1	59.3 ± 12.4	<.001	42.0 ± 13.4	49.7 ± 17.6	56.3 ± 15.9	<.001
BMI (kg/m <sup>2</sup> )	23.7 ± 3.0	24.9 ± 3.6	23.3 ± 2.9	<.001	23.4 ± 3.3	23.8 ± 3.9	23.3 ± 3.2	.44
FEV <sub>1</sub> (% predicted)	98.5 ± 10.0	76.5 ± 9.5	78.3 ± 19.1	<.001	100.2 ± 11.3	75.8 ± 8.1	80.5 ± 14.7	<.001
FVC (% predicted)	99.7 ± 9.8	73.9 ± 6.5	94.0 ± 16.6	<.001	99.8 ± 10.4	73.8 ± 5.8	97.8 ± 16.0	<.001
FEV <sub>1</sub> /FVC ratio	0.82 ± 0.06	0.80 ± 0.06	0.62 ± 0.08	<.001	0.84 ± 0.06	0.83 ± 0.07	0.65 ± 0.05	<.001
Smoking (%)				<.001 <sup>a</sup>				.065 <sup>b</sup>
Current smoking	58.2	52.1	59.7		3.7	6.6	8.7	
Exsmoker	17.9	22.9	28.6		0.8	0.0	1.1	
None	23.9	25.0	11.8		95.5	93.4	90.2	
Alcohol drinking (%)				<.001 <sup>a</sup>				.39 <sup>b</sup>
None	35.4	41.5	36.4		82.7	86.8	78.3	
Current drinker	17.9	5.3	12.6		8.8	9.1	8.7	
Risk drinker	27.0	22.3	20.8		6.7	4.1	9.8	
High-risk drinker	19.7	30.9	30.3		1.7	0.0	3.3	
Physical activity (%)				.001 <sup>a</sup>				.010 <sup>b</sup>
None	61.0	58.3	71.5		73.1	83.5	88.0	
Low exercise	17.5	18.8	9.4		10.3	5.0	4.3	
Medium exercise	17.9	15.6	12.3		14.2	9.9	7.6	
High exercise	3.6	7.3	6.8		2.4	1.7	0.0	
Metabolic syndrome <sup>a</sup> (%)	18.0	30.5	29.9	<.001	17.8	40.5	33.3	<.001
Waist girth (cm)	83.5 ± 8.1	87.9 ± 10.1	85.8 ± 8.1	<.001	77.5 ± 9.2	80.8 ± 11.4	80.6 ± 10.4	<.001
Systolic BP (mm Hg)	122.8 ± 15.4	132.2 ± 20.4	130.1 ± 20.0	<.001	115.7 ± 17.3	125.6 ± 22.3	122.7 ± 22.2	<.001
Diastolic BP (mm Hg)	79.0 ± 10.9	83.5 ± 12.6	79.7 ± 10.4	.003	73.1 ± 10.8	76.2 ± 11.7	74.8 ± 11.8	.010
Fasting glucose (mg/dL)	95.1 ± 22.5	95.9 ± 26.1	98.3 ± 20.4	.036	94.7 ± 19.0	100.2 ± 23.9	95.0 ± 19.4	.010
TG <sup>c</sup> (mg/dL)	135.7 ± 1.70	148.4 ± 1.70	142.4 ± 1.70	<.001	102.2 ± 1.70	124.6 ± 1.74	119.3 ± 1.84	<.001
HDL-C (mg/dL)	44.2 ± 10.1	42.3 ± 10.7	44.3 ± 11.5	.010	48.6 ± 10.2	46.3 ± 10.0	47.9 ± 11.3	.047
Medication								
Hypertension <sup>a</sup> (%)	4.1	18.8	13.5	<.001	6.1	12.6	16.5	<.001
Diabetes <sup>a</sup> (%)	2.3	8.3	3.8	.012	2.0	11.8	7.7	<.001

Values are expressed as means ± SDs unless indicated. Data were calculated using analysis of variance.

<sup>a</sup>  $\chi^2$  test.

<sup>b</sup> Fisher exact test.

<sup>c</sup> Geometric means ± SDs.

Table 2

Regression coefficients of each metabolic components individually entered into separate models predicting FVC (percentage) (model 1) and all metabolic components entered into model (model 2)

	FVC (%)				
	Model 1		Model 2		Effect size ( $\eta^2$ , % change)
	$\beta$	P	$\beta$	P	
Waist girth	−0.13	.009	−0.074	.001	7.93
Systolic BP	−0.065	<.001	−0.055	.009	4.77
Diastolic BP	−0.026	.13		NE	
ln TG	−0.023	.13	−0.021	.054	2.57
Glucose	−0.039	.021	−0.011	.16	1.34
HDL-C	0.009	.58	0.002	.29	0.76
No. of metabolic syndrome components	−0.11	<.001		NE	

Data were calculated using multiple linear regression. Adjustments for model 1 were waist girth to height ratio, sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status. Adjustments for model 2 were sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status. NE indicates not entered in the analysis.

syndrome components ( $P < .001$ ) were strongly related to FVC after adjustment for covariates (Table 2). Next, using multiple regression models, all metabolic components were entered into the model (except diastolic BP, because of interaction with systolic BP [model 2]); and it was observed that all identified relationships were still valid. To explore the degree of attribution to the FVC, we used the effect sizes of metabolic components in a regression model and found that waist girth (7.93%) was more tightly linked to FVC than systolic BP (4.7%), ln TG, (2.57%), or other components.

When separately entered into models (model 1), waist girth ( $P < .001$ ), systolic BP ( $P = .015$ ), fasting glucose level ( $P = .005$ ), and number of metabolic syndrome components

Table 3

Regression coefficients of each metabolic components individually entered into separate models predicting FEV<sub>1</sub> (percentage) (model 1) and all metabolic components entered into model (model 2)

	FEV <sub>1</sub> (%)				
	Model 1		Model 2		Effect size ( $\eta^2$ , % change)
	$\beta$	P	$\beta$	P	
Waist girth	−0.21	<.001	−0.018	.38	0.38
Systolic BP	−0.043	.015	−0.027	.74	0.06
Diastolic BP	−0.011	.51		NE	
ln TG	−0.005	.75	−0.036	.008	3.58
Glucose	−0.047	.005	0.004	.42	0.34
HDL-C	−0.005	.76	−0.015	.11	1.31
No. of metabolic syndrome components	−0.069	<.001		NE	

Data were calculated using multiple linear regression. Adjustments for model 1 were waist girth to height ratio, sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status. Adjustments for model 2 were sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status.

Table 4

Regression coefficients of each metabolic components individually entered into separate models predicting FEV<sub>1</sub>/FVC (model 1) and all metabolic components entered into model (model 2)

	FEV <sub>1</sub> /FVC				
	Model 1		Model 2		Effect size ( $\eta^2$ , % change)
	$\beta$	P	$\beta$	P	
Waist girth	−0.29	<.001	−0.077	<.001	1.21
Systolic BP	0.018	.23	0.022	.032	.22
Diastolic BP	0.020	.16		NE	
ln TG	0.021	.087	−0.007	.72	.006
Glucose	−0.006	.67	0.017	.41	.032
HDL-C	−0.008	.56	−0.014	.75	.005
No. of metabolic syndrome components	0.026	.095		NE	

Data were calculated using multiple linear regression. Adjustments for model 1 were waist girth to height ratio, sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status. Adjustments for model 2 were sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status.

( $P < .001$ ) were all negatively associated with FEV<sub>1</sub> after adjusting for covariates; but only ln TG was associated with FEV<sub>1</sub>, with an effect size of 3.58% (Table 3) when all the metabolic components were entered into model.

Only waist girth was associated with decreased FEV<sub>1</sub>/FVC when separately entered ( $P < .001$ ), and all metabolic components ( $P < .001$ ; effect size, 1.21%) were analyzed (Table 4). There was no association between number of metabolic syndrome components and FEV<sub>1</sub>/FVC. Decreased FVC ( $\beta = -0.11$ ) was more associated with metabolic abnormalities than was decreased FEV<sub>1</sub> ( $\beta = -0.069$ ), as shown by regression analysis.

### 3.2. Relationship between metabolic syndrome and restrictive lung disease

The ORs of metabolic syndrome for restrictive lung disease were 1.49 (95% confidence interval [CI], 1.07–2.08)

Table 5

Odds ratios for restrictive lung disease (FVC <80%, FEV<sub>1</sub>/FVC >0.7) and obstructive lung disease (FEV<sub>1</sub>/FVC <0.7) in metabolic syndrome

		Metabolic syndrome		P
		OR	95% CI	
Restrictive lung disease	Model 1 <sup>a</sup>	1.49	1.07–2.08	.018
	Model 2 <sup>b</sup>	1.42	1.01–2.00	.043
	Model 3 <sup>c</sup>	1.40	1.01–1.98	.049
Obstructive lung disease	Model 1 <sup>a</sup>	0.86	0.65–1.15	.31
	Model 2 <sup>b</sup>	0.93	0.67–1.28	.65
	Model 3 <sup>c</sup>	0.93	0.67–1.28	.65

Data were calculated using multiple logistic regression.

<sup>a</sup> Adjustments for model 1 were sex, age, and pack-year.

<sup>b</sup> Adjustments for model 2 were waist girth to height ratio, sex, age, and pack-year.

<sup>c</sup> Adjustments for model 3 were waist girth to height ratio, sex, age, pack-year, physical activity, alcohol consumption, and socioeconomic status.



in model 1, 1.42 (95% CI 1.01–2.00) in model 2, and 1.40 (95% CI, 1.01–1.98) (Table 5) in model 3.

However, the OR of metabolic syndrome for obstructive lung disease were 0.86 (95% CI, 0.65–1.15) in model 1 and 0.93 (95% CI, 0.67–1.28) in models 2 and 3.

#### 4. Discussion

This nationally representative, cross-sectional study demonstrated that impaired lung function, particularly measured by FVC, is inversely associated with each metabolic risk factor and metabolic syndrome. Furthermore, we found restrictive lung disease to be more prevalent in subjects with metabolic syndrome after adjusting for other variables.

Previous studies [24,25] have reported that restrictive lung disease was related with metabolic syndrome, as was also found in the present study. A small-sized cross-sectional study of elderly individuals showed that a restrictive, but not an obstructive, respiratory pattern was associated with metabolic syndrome and insulin resistance [24]. Another study in Taiwan, with a large sample size composed of subjects from 4 nationwide health screening centers, also revealed that obesity and metabolic syndrome were associated with restrictive lung impairment [25].

Our data, however, are representative of the entire Korean population because we selected subjects using a multistage clustered probability sampling method [19] and the survey was performed in conjunction with KNHNS [17]. We used all of FVC, FEV<sub>1</sub>, and FEV<sub>1</sub>/FVC as lung function tests and analyzed the associations of these data with separate metabolic components after adjusting for waist to height ratio, sex, age, smoking, physical activity, alcohol consumption, and socioeconomic status using multiple linear regression analysis. We were able to show an inverse relationship between lung function and metabolic syndrome in healthy adults without overt impaired lung function.

Previous prospective studies have reported that low FVC predicted the onset of diabetes [6,9,26–29], but all other studies except one have failed to confirm this observation [9]. Two previous cross-sectional studies mentioned above have also suggested that restrictive disease defined by predicted FVC less than 80% and FEV<sub>1</sub>/FVC greater than 0.7 was associated with metabolic syndrome, but obstructive disease defined by FEV<sub>1</sub>/FVC less than 0.7 was not so related [24,25]. Contrary to many other studies, one group reported a link between metabolic syndrome and COPD [30]. Our finding that FEV<sub>1</sub>/FVC was not associated with metabolic risk factors is similar to that of most previous studies. The OR for restrictive lung disease in metabolic syndrome subjects was significant (OR = 1.40; CI, 1.01–1.98), whereas the OR for the relationship between obstructive lung disease (FEV<sub>1</sub>/FVC <0.7) and metabolic syndrome was not significant (OR = 0.93; CI, 0.67–1.28) (Table 5).

Cigarette smoking provokes an inflammatory response [31] and is inversely associated with pulmonary function. Insulin resistance can be induced by smoking [32], and smoking is a known risk factor for metabolic syndrome [33]. However, in our study, the link between lower pulmonary function and metabolic risk factors was independent of cigarette exposure; and inverse association of lower pulmonary function and metabolic syndrome was also found in the nonsmoking subgroup (data not shown).

A possible explanation for the link between impaired lung function and metabolic syndrome is that obesity is likely a cause of pulmonary function decline. In the present study, FVC was associated with waist girth, systolic BP, and TG level, whereas FEV<sub>1</sub> was associated only with TG concentration. Of the metabolic components analyzed, the effect size of waist girth on FVC was higher than that of systolic BP or TG level. In contrast, there was no difference in the effect size of waist girth, compared with other components, when FEV<sub>1</sub> and FEV<sub>1</sub>/FVC were examined. It was thus assumed that the association between metabolic syndrome and FVC was mediated by abdominal obesity because FVC largely reflects lung volume, whereas FEV<sub>1</sub> data are affected by airway flow obstruction. Mechanical limitation of chest expansion during the FVC maneuver may have a role in the negative association seen. Increased intraabdominal fat volume may impede the descent of the diaphragm and increase thoracic pressure. Abdominal adiposity is likely to reduce expiratory reserve volume by compressing the lungs and diaphragm. Many studies have examined the link between BMI and pulmonary function tests [34–37], and some reports used waist girth as an index of abdominal adiposity. These studies showed a negative association between adiposity and both FVC and FEV<sub>1</sub> [15,16,38–40].

Another possible explanation is that ventilatory impairment may be related to insulin resistance. Several prospective studies have shown that parameters of pulmonary function and restrictive lung disease were associated with baseline insulin resistance and were predictive of diabetes onset [6,9,26–29]. Furthermore, metabolic syndrome is known to be related to insulin resistance and the prediabetic state. Thus, insulin resistance might contribute to the association between pulmonary function and other risk factors.

A third possible explanation is that inflammation by metabolic syndrome is implicated in pulmonary disorders. Several studies have shown an inverse association between pulmonary function tests and levels of inflammation-sensitive plasma proteins (fibrinogen,  $\alpha$ 1-antitrypsin, haptoglobin, and ceruloplasmin) [6,41]. The amount of visceral adipose tissue affects the levels of cytokines such as interleukin-6, tumor necrosis factor- $\alpha$ , leptin, and adiponectin [42–45], all of which may act via systemic inflammation to negatively affect pulmonary function; and several systemic inflammatory markers such as C-reactive protein, leukocytes, and fibrinogen are inversely associated with lung function [46]. Some reports demonstrated that individuals with restrictive lung disease [47] and moderate or

severe airflow obstruction [47,48], as well as those without apparent airflow limitation [49], were likely to have increased levels of serum C-reactive protein.

Several strengths and limitations of this study deserve comment. Our data are nationally representative sampling compared with other studies in which data were collected from a local hospital [24,25] and older people [24]. Because this survey was performed with KNHNS [17], the results permit more detailed analyses of the demographic and socioeconomic factors including variables such as a complete smoking history with pack-years and physical activities measured by weekly energy consumption quantitatively, so we could adjust smoking and physical activity more concisely, which may have an influence on pulmonary function and metabolic syndrome. Although, as a confirmatory study, ours showed an inverse association between reduced pulmonary function and restrictive lung disease with metabolic risk factors, the cross-sectional design of the study limited our ability to establish causal relationships among these factors. Another limitation is that our survey had a low participation rate, especially in spirometry. We analyzed only 4001 (48.7%) of 8209 subjects that met the American Thoracic Society criteria [18]. It might be mainly because this was a nationwide lung function study for the first time in Korea and there was no economic attraction to the participants. In a previous study, among subjects older than 45 years, age, sex, and the proportions with chronic sputum production and physician diagnoses of COPD were different between the performers and nonperformers [19]. However, the proportions of smokers were not different between these 2 groups; and the age and sex distribution patterns of subjects who underwent spirometry were similar to patterns seen in the entire subject group, suggesting that our data are representative [19].

In conclusion, lower pulmonary function and restrictive lung disease are negatively associated with metabolic risk factors in the Korean population. The reasons for these associations, however, should be explored.

## Acknowledgment

This work was supported by Wonkwang University in 2009, South Korea.

The study was approved by the Institutional Review Board of Hallym University Sacred Heart Hospital.

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