Relationships of Decreased Lung Function with Metabolic Syndrome and Obstructive Sleep Apnea in Japanese Males

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Abstract

Objective Decreased lung function as assessed by forced vital capacity (FVC) and forced expiratory volume in one second (FEV₁) is shown to be associated with cardiovascular morbidity and mortality. Although the underlying mechanisms for this association remain unknown, metabolic syndrome and obstructive sleep apnea (OSA) may have a role. We analyzed the relationships between metabolic syndrome and OSA in a cross-sectional health survey of middle-aged male employees.

Methods In this secondary analysis, we re-analyzed the relationships of lung function determined by spirometry with metabolic syndrome and OSA based on the respiratory disturbance index (RDI) with a type 3 portable monitor.

Results We analyzed 273 subjects. Independent of age, body mass index (BMI) and smoking, quartiles for lower FVC and FEV₁ were associated with a higher risk of metabolic syndrome compared with quartiles for the highest FVC and FEV₁, respectively. A similar trend was observed regarding the risk associated with waist circumference, and in FVC cases, dyslipidemia. The risk of hyperglycemia was significantly higher in quartiles for the second lowest FVC and FEV₁ than in quartiles for the highest FVC and FEV₁, respectively. A significant trend for an increase in RDI was observed in accordance with quartiles for lower FVC, but not FEV₁.

Conclusion There was a significant relationship between lung function impairment and metabolic syndrome through mainly abdominal obesity, partially through hyperglycemia, and also through dyslipidemia, but only with respect to restrictive lung function. Restrictive lung function was also related to OSA. This epidemiologic evidence may indicate underlying mechanisms between decreased lung function and cardiovascular risk.

Key words: epidemiologic study, lung function, metabolic syndrome, obstructive sleep apnea

Introduction

Decreased lung function as assessed by forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) is suggested to be associated with increased cardiovascular morbidity and mortality (1-5). Although the underlying mechanisms remain unknown, significant relationships of lung function impairment with such cardiovascular risk factors as hypertension (6), diabetes mellitus (7), atherosclerosis (8) or obesity (9) have been shown. Metabolic syndrome, which is characterized by the cluster of abdominal obesity, elevated blood pressure, hyperglycemia and dyslipidemia, is also associated with cardiovascular risk. Thus, metabolic syndrome may be the driving force of the association of decreased lung function with cardiovascular disease, as recent several population-based studies indicated (8, 10-14).

In addition, some studies have indicated that decreased lung function as assessed by FVC or FEV1 is associated with the severity of obstructive sleep apnea (OSA) such as mortality (15), hypercapnia (16, 17), pulmonary hypertension (18) and suspected subclinical lung injury (19, 20). OSA is a well-known risk factor for cardiovascular disease, overlapping with the above-described clusters of its risk factors (21). Although direct comparisons between spirometric measurements and OSA often show inconsistent results, population-based studies to clarify these inconsistencies in findings related to lung function and OSA have been sparse (22).

We hypothesized that impairment of lung function is significantly related to both metabolic syndrome and OSA as supported by findings of an epidemiologic study. Therefore, in the present cross-sectional study, we aimed to simultaneously analyze the relationships of lung function with metabolic syndrome and OSA in the same population sample.

Materials and Methods

Subjects

We previously conducted a cross-sectional health survey in middle-aged male employees at a wholesale company (23) and analyzed the relationship between metabolic syndrome and OSA in 275 subjects (24). In the present study, we reviewed the data in addition to the results of spirometry and re-analyzed the relationships of lung function with metabolic syndrome and OSA. As one subject did not undergo lung function testing and one subject had inadequate lung function data, they were excluded from the analysis. Finally, data on 273 subjects were analyzed. The study protocol was approved by the Kyoto University Graduate School and Faculty of Medicine Ethics Committee. Written informed consent was obtained from all subjects.

Spirometry

Spirometric testing for determining FVC and FEV1 was performed using a spirometer (Chestgraph HI-101, CHEST, Tokyo, Japan) according to the recommended method (25). Those values were expressed as the percentage of the predicted values, which were based on recommendations of the Japanese Respiratory Society (26).

Anthropometric and biochemical measures

Height and weight were measured in the standing position. Waist circumference and blood pressure were measured by trained research staff at the same time that the subjects were trained in the use of a type 3 portable monitor (PM) and actigraph. Measurements of fasting blood sugar, total cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were obtained retrospectively from the company’s periodic inspection data (24).

Diagnosis of metabolic syndrome

First, the diagnosis of metabolic syndrome was based on the Japanese criteria (27), namely, if a subject had a waist circumference ≥85 cm for men and ≥2 of the following risk factors: 1) high blood pressure (systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg); 2) increased fasting plasma glucose ≥110 mg/dL; and 3) increased triglycerides ≥150 mg/dL or decreased HDL-cholesterol <40 mg/dL. Twenty-five subjects who took blood pressure-lowering medications, 6 subjects who took medications for hyperglycemia, and 13 subjects who took medications for hyperlipidemia were included as hypertensive, hyperglycemic and hyperlipidemic subjects, respectively.

In addition, to evaluate the consistency of our findings, we also used the National Cholesterol Education Program (NCEP) criteria on the diagnosis of metabolic syndrome (28). Specifically, when a subject had 3 of the following 5 characteristics, a diagnosis of metabolic syndrome was indicated: increased waist circumference (≥85 cm for men), systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85 mmHg, fasting glucose ≥110 mg/dL, triglycerides ≥150 mg/dL, and HDL-cholesterol <40 mg/dL. Waist circumference was determined to take into account ethnicity (24).

Examinations and home monitoring

Each subject wore a type 3 PM (Morpheus: Teijin, Tokyo, Japan, which is the same as Somté: Compumedics, Victoria, Australia) for 2 consecutive nights and an actigraphy (Actiwatch AWLight: Mini Mitter, OR, USA) to estimate sleep/wake time at home for 7 days. The respiratory disturbance index (RDI: number of apnea and hypopnea episodes per hour of the analyzed sleep time length) was calculated from PM and actigraph data (23, 24). PM records were visually inspected and scored by two or more medical doctors specialized in respiratory medicine. Apnea (cessation of breathing for ≥10 seconds) and hypopnea (>50% reduction in the
Similarly, based on the FEV1 (%predicted), four categories were as follows: quartile 1 (n=68), 92.5% < 94.0%; quartile 2 (n=69), 94.0% < 95.5%; quartile 3 (n=68), 95.5% < 97.0%; and quartile 4 (n=68), > 97.0%. Smoking (pack years) 247 ± 21.5.

Statistics

Results are expressed as mean ± SD unless otherwise stated. Subjects were divided into FVC (%predicted) and FEV1 (%predicted) quartiles, and the highest quartile (quartile 1) was used as a reference group in the following regression analyses. Based on the FVC (%predicted), the resultant four categories were as follows: quartile 1 (n=68), ≥101.0%; quartile 2 (n=69), ≥92.5%, <101.0%; quartile 3 (n=68), ≥83.0%, <92.5%; and quartile 4 (n=68), <83.0%. Similarly, based on the FEV1 (%predicted), four categories were as follows: quartile 1 (n=68), ≥94.0%; quartile 2 (n=69), ≥85.0%, <94.0%; quartile 3 (n=68), ≥78.5%, <85.0%; and quartile 4 (n=68), <78.5%. Chi-square tests were used to analyze the relationship of FVC and FEV1 quartiles with the prevalence of metabolic syndrome. Multivariate logistic regression analyses were performed to assess the relationships of the presence of metabolic syndrome or of each of its components to lung function after adjustment for patients’ age, body mass index (BMI) and smoking, which are all cardiovascular risk factors. Results of these regression analyses are presented in terms of the relative risks (RRs) with corresponding 95% confidence intervals (CIs). The trends of RDI levels across FVC and FEV1 quartiles were examined using a linear model with RDI as a dependent variable and with quartiles as independent variables. Multiple regression analyses were performed after adjustment for those confounders. P values less than 0.05 were considered to be statistically significant.

Results

Characteristics of the 273 subjects are shown in Table 1. A total of 160 subjects (58.6%) had OSA; 16 subjects had severe, 42 subjects had moderate and 102 subjects had mild OSA. Based on the Japanese criteria and NCEP criteria, 57 (20.9%) and 66 (24.2%), respectively, of the 273 subjects had metabolic syndrome. Regarding each component of metabolic syndrome, abdominal obesity, hypertension, hyperglycemia and dyslipidemia were present in 115 subjects (42.1%), 155 subjects (56.8%), 53 subjects (19.4%) and 89 subjects (32.6%), respectively.

Relationships of lung function with metabolic syndrome and its components

FVC and FEV1 quartiles were significantly related to the prevalence of metabolic syndrome (p=0.048 and 0.0033, respectively). These relations were similar when using the NCEP criteria (p=0.010 and 0.0002, respectively).

In the multivariate analyses, the risk of metabolic syndrome increased in quartiles for lower FVC (Table 2). Subjects in FVC quartiles 4, 3 and 2 had a significantly higher risk of metabolic syndrome compared with FVC quartile 1 both on the Japanese and NCEP criteria. Regarding each component of metabolic syndrome, the risk of increased waist circumference as defined for metabolic syndrome was highest in FVC quartile 4 (RR=3.21, p=0.043) than in FVC quartile 1 and the risk of hyperglycemia was significantly higher only in FVC quartile 3 (RR=2.71, p=0.046) compared with FVC quartile 1. The risk of dyslipidemia was significantly higher in FVC quartiles 4 and 3 (RR=2.53, p=0.021; RR=2.40, p=0.030, respectively) compared to FVC quartile 1.

Regarding the FEV1 quartiles, subjects in FEV1 quartile 4 (quartile for lowest FEV1) and 3 had a significantly higher risk of metabolic syndrome compared with FEV1 quartile 1 (quartile for highest FEV1) both using the Japanese and NCEP criteria (Table 3). A similar trend was observed for the risk of increased waist circumference as defined for metabolic syndrome. Subjects in FEV1 quartiles 4 and 3 had significantly higher risk compared with FEV1 quartile 1 (RR=3.75, p=0.044; RR=6.10, p=0.0033, respectively). The risk of hyperglycemia was significantly higher only in the FEV1 quartile 3 (RR=2.69, p=0.043) compared with FEV1 quartile 1.

Relationships of lung function with OSA

A significant trend for an increase in RDI was observed in accordance with quartiles for lower FVC (p=0.038) (Figure A). This trend remained significant after adjustment for

Table 1. Characteristics of the 273 Male Subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>44 ± 8</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.9 ± 3.1</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>83.6 ± 8.5</td>
</tr>
<tr>
<td>Smokers (current / ex / never) (%)</td>
<td>56.0 / 24.2 / 19.8</td>
</tr>
<tr>
<td>Smoking (pack years)</td>
<td>247 ± 21.5</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>129 ± 14</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>81 ± 11</td>
</tr>
<tr>
<td>RDI (h)</td>
<td>10.2 ± 10.8</td>
</tr>
<tr>
<td>Blood parameters</td>
<td></td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>203 ± 32</td>
</tr>
<tr>
<td>Triglycerides (mg/dL)</td>
<td>122 ± 81</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>57 ± 14</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>104 ± 22</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>4.00 ± 0.64</td>
</tr>
<tr>
<td>FEV1 (L)</td>
<td>92.4 ± 11.4</td>
</tr>
<tr>
<td>FEV1 (%predicted)</td>
<td>3.21 ± 0.54</td>
</tr>
<tr>
<td>FEV1 (%predicted)</td>
<td>86.1 ± 11.2</td>
</tr>
</tbody>
</table>

BMI: body mass index, RDI: respiratory disturbance index, HDL: high-density lipoprotein, FVC: forced vital capacity, FEV1: forced expiratory volume in one second.
Table 2. Relative Risk of Metabolic Syndrome and Each Component Across FVC Quartiles

<table>
<thead>
<tr>
<th>FVC quartile</th>
<th>4</th>
<th>3</th>
<th>2</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean FVC, %predicted</td>
<td>77.9</td>
<td>88.0</td>
<td>96.7</td>
<td>107.0</td>
</tr>
<tr>
<td>Metabolic syndrome a</td>
<td>3.59 (1.24-10.42)*</td>
<td>3.21 (1.08-9.58)*</td>
<td>3.05 (1.01-9.24)*</td>
<td>1.0</td>
</tr>
<tr>
<td>Prevalence, %</td>
<td>29.4</td>
<td>23.5</td>
<td>26.3</td>
<td>10.3</td>
</tr>
<tr>
<td>Metabolic syndrome b</td>
<td>4.40 (1.55-12.55)*</td>
<td>5.25 (1.83-15.12)*</td>
<td>3.83 (1.30-11.25)*</td>
<td>1.0</td>
</tr>
<tr>
<td>Prevalence, %</td>
<td>32.4</td>
<td>30.9</td>
<td>23.2</td>
<td>10.3</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>3.21 (1.04-9.90)*</td>
<td>2.81 (0.98-8.08)</td>
<td>1.90 (0.67-5.34)</td>
<td>1.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.83 (0.41-1.67)</td>
<td>1.37 (0.68-2.79)</td>
<td>0.98 (0.49-1.96)</td>
<td>1.0</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>1.56 (0.57-4.22)</td>
<td>2.71 (1.02-7.24)*</td>
<td>2.17 (0.79-5.94)</td>
<td>1.0</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>2.53 (1.15-5.57)*</td>
<td>2.40 (1.09-5.29)*</td>
<td>1.92 (0.86-4.30)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Data are presented as relative risk (95% confidence interval) and have been adjusted for age, BMI and smoking. In all analyses, Quartile 1 is the referent. * p<0.05, a Japanese criteria, b National Cholesterol Education Program (NCEP) criteria.

Abbreviations: the same as in Table 1.

Table 3. Relative Risk of Metabolic Syndrome and Each Component Across FEV1 Quartiles

<table>
<thead>
<tr>
<th>FEV1 quartile</th>
<th>4</th>
<th>3</th>
<th>2</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean FEV1, %predicted</td>
<td>72.4</td>
<td>82.0</td>
<td>89.1</td>
<td>100.8</td>
</tr>
<tr>
<td>Metabolic syndrome a</td>
<td>3.43 (1.13-10.43)*</td>
<td>6.98 (2.30-21.14)*</td>
<td>2.08 (0.64-6.83)</td>
<td>1.0</td>
</tr>
<tr>
<td>Prevalence, %</td>
<td>25.0</td>
<td>33.8</td>
<td>14.5</td>
<td>10.3</td>
</tr>
<tr>
<td>Metabolic syndrome b</td>
<td>3.40 (1.19-9.69)*</td>
<td>8.30 (2.92-23.58)*</td>
<td>1.99 (0.69-6.06)</td>
<td>1.0</td>
</tr>
<tr>
<td>Prevalence, %</td>
<td>27.9</td>
<td>41.2</td>
<td>15.9</td>
<td>11.8</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>3.75 (1.03-13.58)*</td>
<td>6.10 (1.83-20.33)*</td>
<td>2.02 (0.67-6.08)</td>
<td>1.0</td>
</tr>
<tr>
<td>Hypertension</td>
<td>0.79 (0.39-1.60)</td>
<td>1.20 (0.59-2.46)</td>
<td>1.01 (0.50-2.03)</td>
<td>1.0</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>1.42 (0.53-3.79)</td>
<td>2.69 (1.03-7.01)*</td>
<td>1.45 (0.52-4.08)</td>
<td>1.0</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>0.58 (0.26-1.28)</td>
<td>0.85 (0.39-1.82)</td>
<td>1.52 (0.74-3.13)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Data are presented as relative risk (95% confidence interval) and have been adjusted for age, BMI and smoking. In all analyses, Quartile 1 is the referent. * p<0.05, a Japanese criteria, b National Cholesterol Education Program (NCEP) criteria.

Abbreviations: the same as in Table 1.

In the present study, we simultaneously analyzed the relationships of lung function impairment to metabolic syndrome and OSA in an epidemiologic study of middle-aged males in Japan. Our main findings were: 1) both lower FVC and lower FEV1 were significantly associated with metabolic syndrome independent of age, BMI and smoking; 2) regarding separate components of metabolic syndrome, lower FVC and lower FEV1 were related mainly to increased waist circumference and partially to hyperglycemia, with only lower FVC being significantly associated with dyslipidemia; and 3) lower FVC, not lower FEV1, was significantly associated with higher RDI.

Both low FVC and low FEV1 are associated with cardiovascular risk and mortality (1-5). The present results indicate that metabolic syndrome may be the driving force in these relationships. Recently, several population-based cross-sectional studies indicated a positive relationship between metabolic syndrome and lung function impairment in Asia (8, 10-13) and Europe (14), although the underlying mechanisms are not well understood. The opinion as to whether a restrictive or obstructive pattern is more closely related to metabolic syndrome is inconsistent, although a restrictive pattern seems to be dominant (11). Our study also indicated that even a small decline in FVC [from a mean of 107.0% of predicted (quartile 1) to a mean of 96.7% of predicted (quartile 2)] was associated with a 3-fold risk of metabolic syndrome. In contrast, although a decline in FEV1 from a mean of 100.8% of predicted (quartile 1) to a mean of 89.1% of predicted (quartile 2) was not significantly associated with such risk, unexpectedly, a 7-fold increased risk in FEV1 quartile 3 (mean of 82.0% of predicted) was observed. This may be due in part to an exaggeration caused by sampling bias in the small number of patients involved, which is a limitation of the present study. Despite this, the present results indicated that we should pay more attention to lung function levels even when within normal limits or when reductions are small.

We found an independent relationship between lung function impairment in both FVC and FEV1, and abdominal obesity as a metabolic component. Although this is consistent with recent findings (14), the present results indicated that this association is applicable to Japanese people whose BMI age, BMI and smoking (p=0.035). In contrast, there was no significant trend between RDI and FEV1 quartiles (p=0.54) (Figure B).

Discussion

In the present study, we simultaneously analyzed the relationships of lung function impairment to metabolic syndrome and OSA in an epidemiologic study of middle-aged males in Japan. Our main findings were: 1) both lower FVC and lower FEV1 were significantly associated with metabolic syndrome independent of age, BMI and smoking; 2) regarding separate components of metabolic syndrome, lower FVC and lower FEV1 were related mainly to increased waist circumference and partially to hyperglycemia, with only lower FVC being significantly associated with dyslipidemia; and 3) lower FVC, not lower FEV1, was significantly associated with higher RDI.

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mechanisms for the association between abdominal obesity is lower than in western populations. There are two possible aspects of peripheral airways, leading to decreased FEV1. Secondly, Lower levels of ventilation may also cause the early closure of the diaphragm and chest wall compliance with decreased lung volume, which will result in decreased FVC. It affects the diaphragm and chest wall compliance with decreased lung volume, which will result in decreased FVC.

is lower than in western populations. There are two possible mechanisms for the association between abdominal obesity and lung function. Firstly, abdominal obesity mechanically affects the diaphragm and chest wall compliance with decreased lung volume, which will result in decreased FVC. Lower levels of ventilation may also cause the early closure of peripheral airways, leading to decreased FEV1. Secondly, abdominal obesity is associated with systemic inflammation such as shown by elevated C-reactive protein and interleukin-6 values, and systemic inflammation and adipocytokines are risk factors for decreased lung function (13, 29, 30). Thus, mechanical effects and chronic systemic inflammation due to visceral adiposity may be links between lung function impairment and metabolic syndrome.

Incidentally, as in an obese status, systemic inflammation can also be increased during abnormal weight loss in a variety of diseases, possibly related to respiratory and peripheral muscle weakness and impaired lung function (31, 32). This paradoxical feature of systemic inflammation may in part be associated with its dual opposing roles of protectively removing injurious stimuli and initiating the healing process and of negatively impacting disease progression, although further study is needed.

The risk of hyperglycemia was significantly higher in quartile 3, but not in quartile 4, compared to quartile 1 in both FVC and FEV1. Cross-sectional studies indicate that there is an association between lung function impairment and a glycemic state or diabetes (7, 33, 34). Although this may be due to multiple factors such as microangiopathy, chronic inflammation, autonomic neuropathy, loss of elastic recoil secondary to collagen glycosylation of lung parenchyma and hypoxia-induced insulin resistance, the magnitude of such lung function impairment is likely to cause only subclinical rather than clinically evident abnormalities (34). In addition, in the present context, there might be a small number of subjects with severe or prolonged diabetes as possibly the cause of severely decreased lung function. Therefore, we might not have observed a significant association between the lowest and highest quartiles regarding the risk of hyperglycemia.

In addition, regarding the components of metabolic syndrome, decreased FVC was significantly related to dyslipidemia while decreased FEV1 was not. Recent studies reported that both FVC and FEV1 are associated with dyslipidemia and coronary atherosclerosis (8) and triglyceride levels (13). As a recent topic, airflow limitation tends to be associated with atherosclerosis, as shown in the relationship between chronic obstructive pulmonary disease (COPD) and increased mortality due to cardiovascular disease (35, 36). Although the mechanisms are not clearly defined, some remodeling processes may progress simultaneously in both the lung and arteries through hyperlipidemia. Thus, although the results are not necessarily consistent with regard to FVC and FEV1, the relationship between low lung function and dyslipidemia is noted.

As a novel finding, decreased FVC was significantly related to the RDI independent of age, obesity and smoking. Regarding the relationships between lung function impairment and OSA, population-based studies have been rarely performed, and, in addition, obesity, which is an important risk factor for OSA, might have been a confounding effect. Previous studies showed that low FVC or VC is associated with severity of OSA as shown by hypercapnia (16, 17) or pulmonary hypertension (18). In addition, circulating KL-6, which is a mucin-like glycoprotein mainly expressed on alveolar type II and bronchiolar epithelial cells in human lungs, is considered to be a marker of subclinical lung injury due to OSA through endothelial dysfunction (19). Subsequently, we reported that circulating KL-6 is significantly associated with restrictive lung function and gas exchange derangement (20). Thus, although we did not measure arterial partial tension of carbon dioxide, pulmonary artery pressure or KL-6 in this population-based study, decreased FVC may represent some severe effects of OSA.

Decreased lung function is reported to be associated with cardiovascular morbidity and mortality (1-5). The significant relationships of decreased lung function with metabolic syndrome and OSA shown in the present study indicate a possible key role in this association. Among components of meta-
bolic syndrome, abdominal obesity and, partially, hyperglycemia seemed to be important, both of which cause systemic inflammatory conditions leading to cardiovascular diseases. Restrictive lung function was associated with dyslipidemia, which may lead to a systemic atherosclerotic process that includes the heart and arteries. It was also associated with OSA. Proposed mechanisms by which OSA predisposes to cardiovascular diseases include sympathetic excitation, vascular endothelial dysfunction and metabolic dysregulation, as well as oxidative stress and inflammation induced by cyclical intermittent hypoxia (37). Thus, complicated mechanisms seem to be involved in the association between decreased lung function and cardiovascular risk.

The importance of spirometry is recognized from the point of early detection of COPD whose prevalence and consequent burden are rapidly increasing globally (38), and measurements by spirometry tend to be included in general medical checkups. However, as shown in the present study, these measurements might not be used only as a screening tool assessing airflow limitation but for broader severity indices.

The present study has some limitations. First, the sample size was relatively small due to the small number of employees. Second, we did not perform polysomnography. However, an unattended type 3 PM has been reported to provide a valid RDI when manually scored (39), to be capable of accurately measuring a wide range of apnea/hypopnea indices and to have a high specificity and sensitivity in comparison with polysomnography (23).

In conclusion, we showed a relationship between lung function impairment and metabolic syndrome independent of cardiovascular risk factors such as age, obesity and smoking. Regarding metabolic components, the association was shown to be mainly through abdominal obesity, and partially through hyperglycemia; only in restrictive lung function was there an association with dyslipidemia. Restrictive lung function was also related to OSA. This epidemiologic evidence may indicate the underlying mechanism for the relationship between decreased lung function and cardiovascular risk and help us to better stratify patients at risk for cardiovascular disease using lung function data.

The authors state that they have no Conflict of Interest (COI).

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