The effect of gender on the relationship between body fat distribution and lung function

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Abstract

Although abdominal obesity, as measured by waist-to-hip ratio (WHR), has long been recognized as a risk factor for metabolic and cardiovascular diseases, little is known about the effect of WHR on pulmonary function, especially in women. In this study of 1094 men and 540 women (18–102 years) from the Baltimore Longitudinal Study of Aging (BLSA), we examined the effect of WHR on forced expiratory volume in 1 s (FEV\textsubscript{1}). Cross-sectional analyses, after accounting for body mass index (BMI) and other variables, showed a strong inverse association of WHR with FEV\textsubscript{1} in men ($\beta = -1.338$, $P = .0001$) but not in women. Furthermore, larger values of WHR were associated with greater reductions of forced vital capacity (FVC) in men ($\beta = -1.383$, $P = .0005$) compared to women ($\beta = -0.679$, $P = .02$). Thus, body fat distribution has independent effects on lung function that are more prominent in men than women. © 2001 Elsevier Science Inc. All rights reserved.

Keywords: WHR; FEV\textsubscript{1}; FVC; Lung function; Body fat distribution; Obesity

1. Introduction

Obesity is associated with reductions in lung volumes, particularly vital capacity (VC) and forced expiratory volume in 1 s (FEV\textsubscript{1}) [1–4]. The mechanism for this reduction in vital capacity has usually been ascribed to the mechanical effects of obesity on the rib cage and abdomen [1,5]. In recent years, however, it has been recognized that the pattern of obesity is an important predictor of adverse health effects such as diabetes [6–8], hypertension [8], hyperlipidemia [7], and coronary events [9,10]. Specifically, a pattern of central i.e., abdominal, obesity, as measured by waist-to-hip ratio (WHR), is associated with greater health risk than lower body obesity.

Most previous studies of the relationship of obesity to lung function have used BMI as an indicator of overall obesity [2,11,12]. Little is known about the effect of WHR on lung function \textit{per se}, particularly in women. Recently, two studies have drawn attention to the effect of fat distribution on pulmonary function in men [5,13]. Additionally, findings from the Lung Health Study showed that even moderate weight gain after smoking cessation had an adverse effect on FEV\textsubscript{1} and FVC, but that the effect was most prominent in men [14]. While that study did not measure the pattern of fat distribution, the investigators hypothesized that the gender difference was the result of differential abdominal deposition of fat in men compared with women. In men, the predominant pattern of abdominal obesity would be expected to have a greater impact on FEV\textsubscript{1} and FVC than peripheral obesity, which is more prominent in women.

The Baltimore Longitudinal Study on Aging (BLSA) provides an ideal population to examine the relationship between patterns of obesity and pulmonary function. This is a well-characterized group of generally health community-dwelling individuals who have undergone high-quality anthropometric and spirometric measurements. Therefore, the present study was undertaken to answer the following questions:

1. Does fat distribution, assessed by WHR, predict lung function in men and women even after accounting for overall obesity.
2. Can the gender difference in the relationship between obesity and lung function be accounted for by different patterns of fat distribution.
2. Methods

2.1. Population

The BLSA is described in detail in other publications [15–17]. Briefly, the BLSA is a long-term study of normal human aging conducted by the National Institute on Aging. The participants are community-dwelling men and women 18–90+-year-old volunteers who are predominantly recruited from the Baltimore–Washington area. Participants return approximately every 2 years for 2 days of clinical, physiological, and psychological tests for the general purpose of characterizing the normal aging process.

2.2. Selection of participants

The BLSA sample for this study population consisted of 1227 men and 645 women who had undergone spirometry and anthropometric testing and had completed smoking and clinical assessments. Ninety men and 57 women were excluded because of nonreproducible spiromgrams. The current analysis was confined to caucasians because the small number of noncaucasians (43 men and 48 women) did not allow valid subgroup analyses to be performed. Thus, the study population consisted of 1094 men and 540 women ranging in age between 18 and 102 years. Data for this cross-sectional study were derived from the earliest BLSA examination that included the relevant study information.

Even though most of the participants were generally healthy at the time of entry into the BLSA, some developed illnesses, including heart and lung diseases, at the time the healthy at the time of entry into the BLSA, some developed illnesses, including heart and lung diseases, at the time the participants were currently smoking pipes or cigars at the time of the examination. Those who were still smoking less than daily within the past 2 years and who had smoke less than 200 cigarettes, 75 cigars, or 5 packages of pipe tobacco in their lifetime. Current smokers were defined as those who had smoke less than daily within the past 2 years and who had quit smoking less than 2 years before the visit date. Occasional smokers were defined as those who did not meet the definition of a never-smoker. Former smokers were defined as those who had smoked less than daily within the past 2 years and who did not meet the definition of a never-smoker. Former smokers were those who had quit smoking more than 2 years prior to the visit. Pipe and cigar smokers were those who were currently smoking pipes or cigars at the time of the examination.

2.3. Anthropometric measurements

Anthropometric measurements were obtained at each visit. Measurements of height were made using a clinical sta-

2.4. Spirometry

Forced expiratory spirometry was started in 1962 for men and in 1978 for women, and continued until 1994. Testing was performed with a volume-displacement water-sealed spirometer that met accuracy criteria of the American Thoracic Society (ATS) [20]. The testing procedure was performed as previously described [16,17]. After 1987, measurements were digitally recorded to permit automated determination of quality and reproducibility. Spirometry tests that were acquired before 1987 were digitized to allow computerized assessment of quality and reproducibility. Only those participants who demonstrated two acceptable quality maneuvers that were reproducible within 5% of the largest value were included in the present study. Spirometry tests that did not extend for a full 6 s were not included in the analyses of FVC. For this reason, the number of subjects used in the FVC analysis was smaller than that in the FEV1 analysis. Percent of predicted FEV1 and FVC were calculated using values derived from the BLSA-derived prediction equations, which were based on gender, age, and height.

2.5. Smoking status

Smoking status was defined in accordance with standard definitions used by the BLSA [16,17]. Never-smokers were defined as those who had not smoked more than 200 cigarettes, 75 cigars, or 5 packages of pipe tobacco in their lifetime. Current smokers were defined as those who smoked cigarettes every day, or who had quit smoking less than 2 years before the visit date. Occasional smokers were defined as those who had smoked less than daily within the past 2 years and who did not meet the definition of a never-smoker. Former smokers were those who had quit smoking more than 2 years prior to the visit. Pipe and cigar smokers were those who were currently smoking pipes or cigars at the time of the examination.

2.6. Coronary heart disease

Participants were considered to have definite or possible coronary heart disease (CHD) if they ever had one of the following: angina pectoris, history of myocardial infarction, pathologic Q-waves on rest ECG (Minnesota code 1:1 or 1:2), or ischemic ST segment depression ≥ 1 mm on treadmill exercise testing.
2.7. Pulmonary disease

Pulmonary disease was considered to be present if one of the following conditions was present: asthma, bronchitis, emphysema, chronic airway obstruction, bronchiectasis, chronic cough, abnormal sputum, abnormal chest radiograph or lung scans, dyspnea and respiratory abnormalities such as shortness of breath, respiratory distress or insufficiency.

2.8. Data analysis

To examine the effects of age and gender on measures of obesity, descriptive statistics for WHR and BMI were calculated separately for men and women at each decade of age (Fig. 1). The effects of WHR on FEV\(_1\) and FVC were next examined by computing the mean FEV\(_1\) percent predicted and FVC percent predicted for each quartile of WHR (Fig. 2). The relative contributions of BMI and WHR to spirometric measures were determined from cross-sectional regression analyses. Stepwise multiple regression models were constructed using either FEV\(_1\), FEV\(_1\) percent predicted, FVC, or FVC percent predicted as dependent variables. Independent variables that were included in the full model were: age, height, WHR, BMI, and coded variables for the presence of cardiac disease, pulmonary disease, and smoking status (1 = never-smoker, 0 = ever-smoker). Analyses were conducted for each gender separately, and also for the subgroup of individuals who did not have evidence of cardiac or pulmonary disease. The most parsimonious multiple regression models were constructed by backward elimination of the nonsignificant variables (\(P > .1\)). Statistical significance was inferred for \(P < .05\). All statistical analyses were performed using SAS software (SAS Institute, Cary, NC) [21].

3. Results

3.1. Participant characteristics

The characteristics of the sample are given in Table 1. The men ranged in age from 18 to 102 years (mean: 53.7 years), whereas the women ranged from 19 to 93 years (mean: 52.5 years). On average, the men had larger BMI and WHR. Approximately 43% of the men and 21% of the women had BMI between 25 and 30; 8% of men and 10% of the women had BMI > 30; only three men and one woman had BMI > 40. Only a small proportion of subjects were current smokers or had evidence of pulmonary or coronary heart disease.

3.2. Effect of age on BMI and WHR

Fig. 1 shows the effect of age on mean BMI and WHR in men and women of this study group. In men (top panel), BMI initially increased with age, reached a maximum in the fifth decade, then decreased thereafter. In contrast, WHR increased throughout the age span. In women, (bottom panel) BMI also increased with age, peaked around age 55 and then started to decline, while WHR increased throughout life. As the lower panel of Fig. 1 shows, there were two periods of rapid WHR increase in men: the first occurred between ages 25 and 35, and the other between 55 and 65 years of age. It is noteworthy that the rapid BMI increase in women between ages 35 and 55 was accompanied by a much smaller WHR increase, indicating the development of peripheral obesity.

Despite the age-associated increase in WHR and BMI that the women experienced, the highest average WHR values observed in the older women (≥70 years) remained lower than the smallest WHR observed in the younger men (≤30 years). This was not true for BMI, which showed a high degree of overlap between the men and women.

3.3. The effect of WHR on FEV\(_1\)

In order to assess the relationship between WHR and FEV\(_1\), the men and women were divided into quartiles by WHR, and the mean FEV\(_1\) percent predicted for each quartile was determined. Fig. 2 depicts the effect of WHR on FEV\(_1\) percent predicted (top panel) by gender. Men showed a significant inverse association between WHR and FEV\(_1\) percent predicted, whereas no such relationship was observed in women.

Multiple regression analyses were performed separately by gender in order to determine the effect of WHR on FEV\(_1\), when BMI and other variables were accounted for. The results (Table 2) showed that for men, WHR was a significant negative predictor of FEV\(_1\), whereas BMI was not. In contrast, neither WHR nor BMI was significantly associated with FEV\(_1\) in women. Table 2 also shows the other predictors of FEV\(_1\) in men and women. When the interaction term between BMI and WHR was added to the regression model, it was found to be significant only in men, thereby indicating that WHR had its greatest effects in men with the highest BMI. The effect of being a lifetime never-smoker on FEV\(_1\), when pulmonary and coronary artery disease were accounted for, was larger in men than in women.

3.4. The effect of WHR on FVC

The effect of WHR on FVC was examined by selecting FVC reportable spiromgrams from 935 men and 439 women (see Methods). The characteristics of the subjects in the FVC subgroup are shown in Table 3. Although, on average, the men and women of this subgroup were 2–3 years older than the overall group, other anthropometric and clinical characteristics were similar.

The men and women of this subgroup were divided into quartiles by WHR, and the mean FVC percent predicted for each quartile was determined for each gender. Fig. 2 (bottom panel) shows a significant inverse association between WHR and FVC percent predicted in men, whereas in women the association, though significant, appears to be weaker.

By regression analyses, WHR was found to be a highly significant predictor of FVC in men (Table 4). In women,
however, there was only a modest association between WHR and FVC. Table 4 also shows that BMI was a significant negative predictor of FVC in men but not in women. When, in later analyses, we added the interaction term between BMI and WHR to the regression model, we found that the interaction was significant only in women.

A comparison of Tables 2 and 4 shows that, in men, WHR had a similar negative effect on FEV$_1$ as on FVC, whereas in women WHR was correlated only with FVC. BMI had no effect on either spirometric measures in women, whereas in men BMI was associated with FVC but not FEV$_1$.

3.5. The effect of WHR on FEV$_1$ and FVC in healthy subjects

In order to eliminate the effect of lung or coronary heart disease on the relationship between obesity and lung function, we performed subgroup analyses on BLSA participants with no evidence of pulmonary or coronary heart disease. The average ages (± S.D.) of the 772 healthy men and the 423 healthy women in this subgroup were 48.8 ± 17.2 and 48.8 ± 17.2 years, respectively. Multiple regression analysis to assess the cross-sectional relationship between WHR and FEV$_1$ demonstrated that WHR was a significant negative predictor of FEV$_1$ in men but not in women. The magnitude and degree of significance of the WHR coefficient in this healthy subgroup of men (β = −0.755, P = .02) was smaller than that for the total cohort shown in Table 2. BMI was not a significant predictor of FEV$_1$ for either the men or the women of this subgroup.

When healthy participants with FVC reportable spiromgrams were selected, the resulting subgroup of 600 men and 326 women had average ages of 50.4 ± 16.7 and 51.9 ± 16.9 years, respectively. Multiple regression analysis, as described previously, demonstrated that WHR was a negative determinant of FVC in men (β = −1.743, P = .0006), but not in women. Thus, in the subset of healthy men, just as in the overall sample, WHR appears to be more strongly asso-
associated with FVC than with FEV1. In contrast, BMI had no effect on FVC in healthy women, and was only marginally significant in men ($\beta = 0.016, P = .08$).

4. Discussion

A few previous studies have examined the relationship of fat distribution and pulmonary function in men [5,13,30], but no previous studies have been conducted in women. To our knowledge, this is the first time that the effect of body fat distribution, assessed by WHR, on lung function has been examined in a relatively large group of well-characterized, community-dwelling men and women with a wide age range.

The main finding of this cross-sectional study is that WHR is a more important determinant of pulmonary function in men than in women. WHR is a significant predictor of FEV1 in men only, and appears to have a greater impact on FVC in men than in women. This finding supports the hypothesis that the gender difference in the pattern of fat distribution is one mechanism for the sex difference in lung function impairment due to weight gain [11,14]. Using multiple regression analysis, it is also apparent that WHR has a greater effect on lung function than commonly used measures of general obesity such as BMI.

Several possiblities could account for the gender difference that we observed. First, it is possible that there is a difference in the way that distribution of fat affects the thoracic mechanics in men compared to women. Second, and more likely, is that there is a threshold below which WHR does not affect lung function. In a study of men only, Collins et al. [13] reported that men with WHR greater than 0.95 had a lower FEV1 and FVC than those with lower WHR. In accordance with previous studies, we found that there was little overlap in the distributions of WHR between men and women, with men having greater WHR at all ages [18,19].

Fig. 2. The relationship between WHR and FEV1 percent predicted in 1094 men and 540 women (top panel), and FVC percent predicted in 935 men and 439 women (bottom panel). Results are shown as mean ± S.E. of FEV1 percent predicted or FVC percent predicted versus mean WHR within quartiles of WHR. For men FEV1 percent predicted was significantly associated with WHR ($P = .0001$). For women, there was no significant association between WHR and FEV1 percent predicted. In men FVC percent predicted was significantly associated with WHR ($P = .0002$), and BMI ($P = .0001$). In women, FVC percent predicted was significantly associated with WHR ($P = .0031$).
Thus, it is possible that abdominal fat deposition in women was not high enough to adversely affect lung function. In men however, abdominal obesity impaired ventilatory function. Since our study population did not include substantial numbers of morbidly obese women, we cannot say whether this gender difference would pertain when WHR was similar for both men and women.

It is noteworthy that gender differences in the distribution of fat have been postulated to account for the male–female difference in sleep apnea syndrome [22]. Premenopausal women who develop sleep apnea are substantially heavier than men. Previous studies have also shown that the increase in body weight that occurs following smoking cessation has greater impact on lung function in men than in women [14]. This may also be accounted for by the difference in fat distribution between men and women.

The adverse effect of abdominal obesity on lung function is likely mediated through direct effects on the rib cage or thoracic compliance. It is also possible that compression of the abdominal viscera by local fat redistributes blood to the thoracic compartment, thus reducing vital capacity. This is the explanation that has been given for the fall in vital capacity that occurs in the supine position [23]. It has also been shown that increased abdominal fat deposition is associated with increased visceral fat in the abdomen [24]. The accumulation of fat in the epicardium may also reduce vital capacity.

We speculate that the body fat distribution may explain the link between low FEV1 and increased coronary risk. High WHR is associated with impaired glucose metabolism, hypertension, and hyperlipidemia, all coronary risk factors. In addition, it has been well established in general population samples that measures of lung function such as FEV1 and FVC are predictors of overall mortality as well as coronary mortality, and that this association cannot be explained entirely by cigarette smoking status [16,25–27]. However, the link between abnormal lung function and coronary risk is not known. If central obesity is a marker for multiple coronary risk factors and also has a specific adverse effect on spirometry, then aberrant spirometry could be merely an epiphenomenon and not a direct cause of coronary death. In other words, it is possible that low lung function per se does not increase coronary risk, but is merely a marker for abdominal fat distribution and the associated coronary risk factors. This speculation, however, must be tempered by the fact that low levels of lung function appear to be important predictors of coronary death in women as well as men [24].

A potential confounding factor in the relationship between BMI, WHR and lung function is smoking status. Smokers tend to have a lower BMI but higher WHR than nonsmokers [28]. Thus, it is possible that smoking status, which has a detrimental effect on lung function, accounted for the better association of lung function with WHR than with BMI. However, even after adjustment for smoking status in the multiple regression analysis, the strong relationship between WHR and lung function in men persisted. Further investigation is needed to determine if the association between WHR and lung function is independent of smoking status.

### Table 2
**Determinants of FEV1 in BLSA subjects with backward selection**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (n = 1094)</th>
<th>Women (n = 540)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>−0.02851</td>
<td>−0.0252</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>0.0354</td>
<td>0.0307</td>
</tr>
<tr>
<td>WHR</td>
<td>−1.3384</td>
<td>NS</td>
</tr>
<tr>
<td>Pulmonary diseaseb</td>
<td>−0.4708</td>
<td>−0.2607</td>
</tr>
<tr>
<td>R²</td>
<td>.68</td>
<td>.74</td>
</tr>
</tbody>
</table>

WHR = waist to hip ratio; BMI = body mass index (kg/m²); NS = not significant (P > .1).

Parameter coefficients in final model (with FEV1 as dependent variable) were obtained through backward selection. Variables that were not in the final model included BMI and presence of coronary heart disease (NS = P > .1).

*Lifetime never-smoker (0 = no; 1 = yes).

bPresence of pulmonary disease as defined in Methods: (0 = no; 1 = yes).

### Table 3
**Descriptive statistics for BLSA subjects used in FVC analysis (mean ± S.D.)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men (n=935)</th>
<th>Women (n=439)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.0 ± 17.5</td>
<td>55.8 ± 17.5</td>
</tr>
<tr>
<td>FEV1 % pred.*</td>
<td>97.0 ± 15.3</td>
<td>99.2 ± 14.6</td>
</tr>
<tr>
<td>FVC (L)</td>
<td>4.76 ± 0.97</td>
<td>3.34 ± 0.77</td>
</tr>
<tr>
<td>FVC % pred.</td>
<td>98.2 ± 12.9</td>
<td>98.8 ± 13.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>79.6 ± 11.9</td>
<td>64.8 ± 11.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>176.5 ± 6.7</td>
<td>162.7 ± 6.8</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.5 ± 3.2</td>
<td>24.5 ± 4.2</td>
</tr>
<tr>
<td>WHR*</td>
<td>0.920 ± 0.06</td>
<td>0.771 ± 0.073</td>
</tr>
<tr>
<td>% Current smokers*</td>
<td>15.1</td>
<td>10.7</td>
</tr>
<tr>
<td>% Lifetime never-smokers*</td>
<td>33.5</td>
<td>52.6</td>
</tr>
<tr>
<td>% Coronary heart disease</td>
<td>22.8</td>
<td>19.1</td>
</tr>
<tr>
<td>% Pulmonary disease*</td>
<td>17.8</td>
<td>8.7</td>
</tr>
</tbody>
</table>

WHR = waist to hip ratio; BMI = body mass index (kg/m²). Presence of coronary heart disease (CHD) and pulmonary disease as defined in Methods.

*Gender comparison (P < .05).
thermore, when we repeated the analysis separately for lifetime nonsmoking BLSA men we found that higher WHR had a weak, but significant, effect on FEV₁. In nonsmoking BLSA women, WHR was modestly linked only with FVC, whereas BMI did not affect either FEV₁ or FVC, even when WHR was not included in the multiple regression model.

While a number of studies have shown that body weight can adversely affect lung function, the relationship is complex [11,12,29]. In younger people, increasing BMI may be associated with an increase in lung function (muscularity effect), whereas in older people, increasing BMI is associated with a decrease in lung function (adiposity effect). Consequently, the overall impact of BMI on lung function in cross-sectional analyses is small [12]. Of the few previous studies that have examined the relationship of body fat distribution and pulmonary function in men, Marcus et al. [30] found that FEV₁ was negatively associated with body fat distribution as measured by the sum of skinfold thickness in Japanese American men. Lazarus et al. [5] found that greater subscapular skinfold thickness was associated with lower FEV₁ and FVC in men under 60. Neither of these studies, however, directly measured the pattern of obesity. The present study extends these studies by using a more direct measure of fat distribution pattern and by including women in the study group. Because our study population was very well characterized, we were also able to control for confounding illnesses. A potential limitation of the present study is that the population was confined to caucasians, and thus we are unable to confidently extrapolate our results to other racial groups.

The implication of this study is that, in men, the pattern of fat distribution, measured by WHR, is an important determinant of lung function, and is a more direct predictor than BMI. These findings suggest that both cross-sectional and longitudinal studies of lung function in men should consider the potential confounding effect of fat distribution rather than merely weight or weight gain per se. WHR is a well-standardized and simple measure that may be easily included in population studies. In women, however, neither BMI nor WHR are important predictors of lung function and, therefore, it is unlikely that changes in lung function can be attributed to changes in weight over a wide range.

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References


