Body Composition Analysis and Changes in Airways Function in Obese Adults After Hypocaloric Diet*

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Study objectives: To determine the relationship between weight-loss and pulmonary function indexes, focusing on forced expiratory flows (ie, FEV₁, forced expiratory flow at 50% of vital capacity [FEF₅₀], forced expiratory flow at 75% of vital capacity, and forced expiratory flow at 25 to 75% of vital capacity [FEF₂₅–₇₅]). Specifically, to determine the effect of losses in total and segmental fat mass (FM) and of modifications in lean body mass, after restricted hypocaloric diet, on pulmonary function among obese adults.

Design: Cross-sectional, observational.

Settings: Human Physiology Division, Faculty of Medicine and Surgery, “Tor Vergata” University, Rome, Italy.

Patients: Thirty obese adults (mean [± SD] baseline body mass index [BMI], 32.25 ± 3.99 kg/m²), without significant obstructive airway disease, were selected from among participants in a weight-loss program.

Measurements and results: Anthropometric, body composition (BC), and respiratory parameters of all participants were measured before and after weight loss. Total and segmental lean body and FM were obtained by dual-energy x-ray absorptiometry. Dynamic spirometric tests and maximum voluntary ventilation (MVV) were performed. The BC parameters (ie, body weight [BW], BMI, the sum skinfold thicknesses, thoracic inhalation circumference, thoracic expiration circumference, total FM, and trunk FM [FMtrunk]) were significantly decreased (p < 0.0001) after a hypocaloric diet. The mean vital capacity, FEV₁, FEF₅₀, FEF₂₅–₇₅, expiratory reserve volume, and MVV significantly increased (p < 0.05) with weight loss. The correlation coefficient for ΔFEF₂₅–₇₅ (r = 0.20) was numerically higher than ΔFEF₅₀ and ΔFEV₁ (r = 0.14 and r = 0.08, respectively) for the BW loss. Moreover, the correlation coefficient for ΔFEF₂₅–₇₅ (r = 0.45) was significantly higher (p ≤ 0.02) than those for ΔFEF₅₀ and ΔFEV₁ (r = 0.38 and r = 0.15, respectively) for FMtrunk loss.

Conclusions: This study shows that a decrease in total and upper body fat obtained by restricted diet was not accompanied by a decrease in ventilatory muscle mass. FMtrunk loss was found to have improved airflow limitation, which can be correlated to peripheral airways function.

(CHEST 2001; 119:1409–1415)

Key words: dual-energy radiograph absorptiometry; fat distribution; forced expiratory flows; pulmonary function; weight loss

Abbreviations: BC = body composition; BMI = body mass index; BW = body weight; DXA = dual-energy radiograph absorptiometry; ERV = expiratory reserve volume; FEF₅₀ = forced expiratory flow at 50% of vital capacity; FEF₂₅–₇₅ = forced expiratory flow at 25 to 75% of vital capacity; FM = fat mass; FMtot = total fat mass; FMtrunk = trunk fat mass; LBM = lean body mass; LBMtot = total lean body mass; TorExp = thoracic expiration circumference; TorInh = thoracic inhalation circumference; VC = vital capacity

Obesity, which can be defined as an accumulation of excess body fat and which is the most common nutritional disorder in humans, is a major cause of mortality and morbidity for associated metabolic disorders and cardiovascular disease. With regard to the risk of developing obesity-related disorders in general, extensive research¹,² has shown that the location of body fat deposits is a more important determinant than the size of these deposits. It also

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From: http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21962/ on 06/26/2017
has been shown that the presence of intra-abdominal visceral fat in the mesentery and omentum is a better predictor of coronary heart disease than the body mass index (BMI) and that neck and visceral fat accumulation is a risk factor for obstructive sleep apnea syndrome.4

Many studies also have demonstrated an association between excess weight or weight gain and pulmonary dysfunction.5–9 Specifically, weight gain tends to be accompanied by a decrease in vital capacity (VC) and FEV1 among adults, which is possibly attributable to the effect of obesity on chest-wall compliance or inactivity.10 Furthermore, expiratory reserve volume (ERV), which is related to abdominal and chest fat accumulation, is decreased in obese patients,11 and respiratory and airway resistance increases significantly with the level of obesity.12–15

Given that losses in body weight (BW) account for significant improvements in pulmonary function,16–19 dietary or surgical treatment of morbid obesity can be used to sufficiently improve lung-specific parameters and respiratory muscle function.16–18 In this context, many authors have investigated the relationship between body composition (BC) and respiratory functions. Specifically, lean body mass (LBM) has been found to be positively associated with respiratory functions, whereas fat mass (FM) in severely obese persons has a negative association.16,20,21

With regard to the effects of modifications in body fat distribution on pulmonary function, the studies20,21 conducted to date have shown conflicting results, thus the potential association remains unclear.8 It has been suggested22 that among severely obese persons, pulmonary function is impaired to a greater extent among those with upper body obesity, compared to persons with lower body obesity. It also has been suggested often that cardiopulmonary failure occurs only in overweight persons with predominately upper body obesity.

To study BC and fat distribution in humans, De Lorenzo et al16 proposed using dual-energy x-ray absorptiometry (DXA).23 Although DXA determines fat distribution indirectly, it produces the most accurate results, is the only technique capable of determining specific sites of fat deposition, and is preferred over anthropometric indexes for estimating fat distribution (eg, BMI, waist-hip ratio, and skinfold thickness). Furthermore, DXA produces accurate and detailed assessments of soft tissue (ie, FM and LBM) topographies by direct measurements of trunk, abdomen, thigh, and leg.24

The objective of the present study was to establish the relationship between loss of BW and pulmonary-function indexes, focusing on FEV1 and forced expiratory flows in the mid- and lower-half of VC (ie, forced expiratory flow at 50% of vital capacity [FEF50]), forced expiratory flow at 75% of vital capacity, and forced expiratory flow at 25 to 75% of vital capacity [FEF25–75%]). Specifically, we evaluated the effect of the loss of total FM (FMtot) and segmental FM and of changes in LBM, after a restricted hypocaloric diet, on pulmonary functions in obese adults.

**Materials and Methods**

The study was conducted at the Divisions of Human Physiology and Pulmonary Diseases of the “Tor Vergata” University (Rome, Italy). Participants underwent the study voluntarily and provided written informed consent prior to inclusion in the study, and the study protocol was approved by the ethics committee of the University.

The study comprised 30 mildly obese adults (7 men and 23 women; mean (± SD) age, 42 ± 12 years; mean height, 1.64 ± 0.11 m) who were randomly selected from among participants in a weight-loss program at the “Tor Vergata” University. In general, approximately 70 to 80% of program participants were women and 20 to 30% were men (“Tor Vergata” University internal departmental statistics). The program is based on a hypocaloric Mediterranean diet, which has proven to be effective in weight-loss programs.12,13,16 Diets were individualized based on resting energy expenditure, excess BW, height, age, gender, and level of engagement in sports activities (DietoSystem, version 1.25; Terapia Alimentare; Milan, Italy). Diet protein intake levels were calculated as 1 g/kg of BW, as described elsewhere.25 Both current and ex-smokers were included in the study. During the study period, no changes in lifestyle or smoking habits were observed.

**Exclusion Criteria**

Following routine clinical examinations, we excluded from this analysis persons with respiratory symptoms and/or an FEV1/FVC ratio < 76% of the predicted value. We also excluded persons who, at the end of the weight-loss program, showed stationary weight or weight gain; none of the persons excluded for stationary weight or weight gain underwent follow-up tests for respiratory function.

**Measurements**

All of the patients were examined at enrollment into the program (baseline survey) and on its completion (follow-up survey) 6 months later (ie, immediately after the acute phase of BW loss and before the occurrence of any lean tissue repletion).

Anthropometric and BC parameters were measured for all participants. Specifically, BW (in kilograms; measured with participants clothed in underwear with bare feet) was measured to the nearest 0.01 kg using a digital scale (Body Master; Rowenta, Germany). Height was measured using a stadiometer. BMI was calculated as weight/height2 (in kg/m2).

We measured skinfold thickness and circumference parameters, in accordance with standard methods,26 using Holtain calipers (Bryherian, UK). A sum of four skinfolds was calculated (ie, biceps, triceps, subscapular, and suprailiac skinfolds). Thoracic inhalation circumference (TorInh) was measured at total lung capacity, and thoracic expiration circumference (TorExp)
was measured at residual volume using a standard tape measure. Total LBM (LBMtot), total FM (FMtot), trunk LBM (LB-Mtrunk), and trunk FM (FMtrunk) were measured using DXA total body scans of relatively low energy (Lunar DPX densitometer, version 3.6; Lunar Radiation Corp, Madison, WI). At both baseline and follow-up, patients were investigated under standard conditions. BW loss was determined by subtracting their weight at follow-up from that at baseline.

Dynamic spirometric tests were performed using a portable open circuit spirometer (KIT-Cosmed spirometer; Cosmed; Rome, Italy) in the standing position, in accordance with the guidelines of the American Thoracic Society. At both baseline and follow-up, forced expiratory blows were performed three times, and the best value obtained from the maximum expiratory flow-volume curve was considered in the analysis of FVC, VC, FEV1, FEF50, and FEF25–75. Maximum voluntary ventilation (MVV) was determined by fast, deep breathing for 12 s.

To study changes in respiratory functions at different degrees of weight loss, participants were divided into three subgroups based on the extent of loss in BW and FMtrunk. The three levels were defined by visually inspecting scatter plots (Fig 1) of losses. For BW, the three subgroups were as follows: < 3.49 kg (n = 5); 3.50 to 6.49 kg (n = 10); and > 6.50 kg (n = 13). For FMtrunk, the three subgroups were as follows: < 1.49 kg (n = 5); 1.50 to 2.99 kg (n = 10); and > 3.00 kg (n = 13).

Statistical Analysis

Statistical analyses were carried out using a commercially available statistical software package (StatView, version 5.0; SAS Institute Inc; Cary, NC). Paired Student’s t tests of significance were used to compare different parameters at both baseline and follow-up. The significance level was defined as p ≤ 0.05. Simple regression analysis and analysis of variance were performed to establish different correlation coefficients between spirometric parameters and losses in BW and FMtrunk.

RESULTS

At baseline, the study participants had a mean (± SD) weight of 87.26 ± 14.39 kg and a mean height of 1.64 ± 0.11 m. These results, together with those regarding the other anthropometric and BC parameters before and after weight loss, are shown in Table 1. The follow-up survey revealed significant decreases (p ≤ 0.0001) in the mean values for BC parameters (ie, BW, BMI, sum of skinfolds, TorInh, TorExp, FMtot, and FMtrunk), whereas no significant differences were observed for LBMtot or LB-Mtrunk.

Total BW loss (ie, baseline weight minus follow-up weight) ranged from 0.50 to 11.20 kg (mean loss, 6.47 ± 2.65 kg), and segmental FMtrunk loss ranged from 0.17 to 6.55 kg (mean loss, 2.83 ± 1.61 kg). Figure 1 shows the scatter plots of BW loss against FMtrunk loss.

With regard to respiratory parameters, the mean VC, FEV1, FEF50, FEF25–75, ERV, and MVV significantly increased (p ≤ 0.05) with weight loss (Table 2), whereas no significant changes were found for FVC, peak expiratory flow (PEF), FEV1/VC ratio, FEV1/FVC ratio, or FEF25–75.

Figure 2 shows the correlation coefficients between changes in airways function parameters (ΔFEV1, ΔFEF50, and ΔFEF25–75) and losses in BW and FMtrunk. Changes were calculated as the difference between follow-up and baseline values. When considering BW loss, the correlation coefficient was higher for ΔFEF25–75 (r = 0.20) compared to ΔFEV1 (r = 0.08) and ΔFEF50 (r = 0.14). Similar results were obtained when considering FMtrunk loss (ΔFEF25–75, r = 0.45, p ≤ 0.02; ΔFEV1, r = 0.15; and ΔFEF50, r = 0.38; p ≤ 0.05).

To evaluate the effect of BW loss alone on changes in respiratory parameters, we divided BW loss into three subgroups (< 3.49 kg, 3.50 to 6.49 kg, and > 6.50 kg). As shown in Figure 3, the correlation coefficients for ΔFEV1 and ΔVC progressively de-
increased with increasing BW loss, whereas the coefficient for $\Delta$FEF$_{25-75}$ showed an initial increase by BW loss (from < 3.49 kg to 3.50 to 6.49 kg), followed by a decrease (from 3.50 to 6.49 kg to > 6.50 kg).

For FMtrunk loss (subgroups: < 1.49 kg, 1.50 to 2.99 kg, and > 3.00 kg) as shown in Fig 4, the correlation coefficients for $\Delta$FEV$_1$ and $\Delta$VC initially decreased with increasing loss (from < 1.49 kg to 1.50 to 2.99 kg); they then increased (from 1.50 to 2.99 kg to > 3.00 kg). The correlation coefficient for $\Delta$FEF$_{25-75}$ progressively increased with increasing loss (loss of > 3.00 kg, $r = 0.68$ and $p = 0.02$; loss of 1.5 to 2.99 kg, $r = 0.68$ and $p = 0.02$; loss of 1.5 to 2.99 kg, $r = 0.36$; and < 1.49 kg, $r = 0.25$).

**Discussion**

The findings of other studies that obese patients may have airflow limitation, although their FEV$_1$/FVC ratio is normal, led us to study the effect of total BW loss on airways function. One explanation for this finding could be that smaller airways are constrained to a greater extent by visceral fat than are larger airways. In our patients, BW loss manifested as a highly significant decrease in FMtot reduction ($p \leq 0.0001$) with no significant changes in LBMtot, and it was associated with improved pulmonary function (ie, significant improvements in VC, ERV, FEV$_1$, FEF$_{50}$, and FEF$_{25-75}$). This finding is consistent with reports of an association between improvements in VC and PEF and unchanged LBMtot in weight-loss programs.

We measured total and segmental FM and LBM using DXA. FMtrunk has been shown to be indicative of truncal obesity, which is associated with poor respiratory function. The association between forced expiratory flow rates over low- and mid-volume ranges and small airways function is known. These rates have been shown to be reduced in some obese individuals who had no evidence of obstructive airway disease. By contrast, the mechanisms underlying the decreased peripheral airway caliber in obese persons are not well known. It is believed that pulmonary blood volume is increased in obese patients, possibly leading to congestion of bronchial vessels in the airway submucosa, thickening of the airway wall, and decrease in airway size. Altered lipid metabolism of obese patients may amplify these effects; the presence of very low-density lipoproteins has been found to be related to the release of histamine, which is an effective mediator of vascular permeability and smooth muscle contraction in the airway.

**Table 1—Anthropometric Parameters of Obese Adults (n = 30) Before and After Weight Loss**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before Weight Loss</th>
<th>After Weight Loss</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW, kg</td>
<td>87.26 ± 14.39</td>
<td>80.79 ± 14.26</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>32.25 ± 3.99</td>
<td>29.84 ± 4.06</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>TorInh, cm</td>
<td>107.16 ± 8.80</td>
<td>104.20 ± 8.64</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>TorEsp, cm</td>
<td>103.47 ± 9.23</td>
<td>100.30 ± 9.04</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Sum SF, mm</td>
<td>56.91 ± 22.70</td>
<td>71.08 ± 22.49</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>FMtot, kg</td>
<td>36.58 ± 9.76</td>
<td>31.32 ± 11.07</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>FMtrunk, kg</td>
<td>17.74 ± 4.32</td>
<td>15.02 ± 5.11</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>LBMtot, kg</td>
<td>46.65 ± 11.48</td>
<td>46.50 ± 11.70</td>
<td>NS</td>
</tr>
<tr>
<td>LBMtrunk, kg</td>
<td>22.48 ± 4.99</td>
<td>22.59 ± 5.04</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. Sum SF = sum skinfolds; NS = not significant.

**Table 2—Respiratory Parameters of Obese Adults (n = 30) Before and After Weight Loss**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before Weight Loss</th>
<th>After Weight Loss</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, L</td>
<td>3.71 ± 1.12</td>
<td>3.77 ± 1.37</td>
<td>NS</td>
</tr>
<tr>
<td>VC, L</td>
<td>3.69 ± 1.26</td>
<td>3.9 ± 1.23</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>FEV$_1$, L</td>
<td>2.97 ± 0.91</td>
<td>3.12 ± 0.99</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>PEF, L/s</td>
<td>6.42 ± 2.10</td>
<td>6.98 ± 2.31</td>
<td>NS</td>
</tr>
<tr>
<td>FEV$_1$/VC, %</td>
<td>80.53 ± 6.94</td>
<td>80.35 ± 5.13</td>
<td>NS</td>
</tr>
<tr>
<td>FEV$_2$/FVC, %</td>
<td>81.47 ± 4.12</td>
<td>81.13 ± 4.86</td>
<td>NS</td>
</tr>
<tr>
<td>FEF$_{50}$, L/s</td>
<td>3.65 ± 1.30</td>
<td>4.08 ± 1.45</td>
<td>&lt; 0.005</td>
</tr>
<tr>
<td>FEF$_{75}$, L/s</td>
<td>1.47 ± 0.58</td>
<td>1.75 ± 0.70</td>
<td>NS</td>
</tr>
<tr>
<td>FEF$_{25-75}$, L/s</td>
<td>2.92 ± 0.87</td>
<td>3.36 ± 1.38</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>ERV, L</td>
<td>1.08 ± 0.65</td>
<td>1.34 ± 0.72</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>MVV, L/min</td>
<td>108.91 ± 42.28</td>
<td>121.60 ± 44.77</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. FEF$_{75}$ = forced expiratory flow at 75% of vital capacity. See Table 1 for abbreviations not used in text.
on the elastic recoil pressure of the lung, tends to increase the airway size at high pulmonary volumes and to reduce it at low pulmonary volumes when this pressure diminishes.\textsuperscript{12}

In our study, the correlation coefficient for $\Delta$FEF\textsubscript{25–75} was higher than that for $\Delta$FEV\textsubscript{1}, for both BW loss and FMtrunk loss. This may be due to $\Delta$FEF\textsubscript{25–75} being more sensitive to decreases in airflow limitation induced by BW and FMtrunk losses. FMtrunk loss was found to improve airflow limitation, which might result from improved peripheral airway function. Although smoking may decrease small airways function, this was not evident from our results, in that the number of smokers (ie, <3) was not sufficient for reaching statistical significance. We chose to analyze ERV because it is a marker of pulmonary function that is sensitive to changes in BW. However, the correlation coefficient ($r = 0.26$) between changes in ERV and changes in FMtrunk did not reach statistical significance.

We observed an association between improvements in the respiratory parameters VC and FEV\textsubscript{1} and the initial BW loss, however, the extent of improvement decreased for greater BW loss. Previous studies have shown that dietary treatment and gastroplasty for morbid obesity are sufficient for inducing improvements in lung volume and respiratory muscle performance.\textsuperscript{18,32} By contrast, decreases in FVC and FEV\textsubscript{1} are associated with gains in BW,\textsuperscript{5–8} which is apparently consistent with our finding of improved VC and FEV\textsubscript{1} with greater BW loss. The finding that this improvement was less evident for the highest level of BW loss (ie, >6.50 kg) could be explained by the absence of changes, after moderate weight loss, in LBM\textsubscript{tot} and upper LBM, in addition to decreased muscle work. In
addition, after weight loss, MVV showed a highly significant (p ≤ 0.0001) improvement, which also is related to respiratory muscle function. The finding that the correlation coefficient for ∆FEF25–75 tended to be higher for a BW loss of 3.50 to 6.49 kg, with respect to the other two subgroups, remains unclear.

Regarding FMtrunk loss, the finding that among patients with the greatest loss (ie, > 3.00 kg), the correlation coefficient for ∆FEF25–75 was significantly higher (p < 0.02) than that among those with less extensive loss (ie, < 1.49 kg and 1.50 to 2.99 kg) suggests that losses in upper body fat may be responsible for improving the respiratory parameters of small airways. Although we cannot explain the finding that the ∆FEV1 and ∆VC correlation coefficients initially decreased with increased FMtrunk loss (ie, losses of < 1.49 kg and 1.50 to 2.99 kg), the increase coefficients for the highest loss (> 3.00 kg) is fairly logical.

CONCLUSION

The decrease in total and upper body fat resulting from a hypocaloric Mediterranean diet was not accompanied by a decrease in ventilatory muscle mass. We observed that FMtrunk loss improves airflow limitation, which can be correlated with peripheral airways. To the best of our knowledge, this is the first report of an association between improved airflow limitation for peripheral airways and FMtrunk loss. These results confirm the importance of DXA in assessing obesity. The effect of smoking on small airways function accompanying FMtrunk loss needs to be more thoroughly investigated.

ACKNOWLEDGMENT: We would like to express our thanks to Mr. Mark Kanieff for editorial assistance.

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Figure 4. Correlation coefficients of changes in respiratory parameters for FMtrunk loss, by extent of loss (30 obese adults).
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