Patients with COPD and bronchial asthma have been reported to have an increase in the energy cost of breathing which is reflected by changes in their basal metabolic rates (BMR).

**Aim:** To assess the changes that may occur in basal metabolic rates in patients with bronchial asthma and those with chronic obstructive pulmonary disease.

**Patients and methods:** We used indirect calorimetry using oro-nasal mask to assess the BMR in 35 patients with COPD, 20 patients with bronchial asthma and 10 healthy volunteers (control group).

**Results:** There was marked increase in basal metabolic rates in patients with COPD compared to patients with bronchial asthma (p value < 0.001) and to the control group (p value < 0.001), also between asthmatic patients and control group (p value < 0.05). The BMR also showed a highly significant negative correlation (p value < 0.001) with the level of hypoxemia and pulmonary functions (the severity of the disease) in both COPD and asthmatic patients.

**Conclusion:** Patients with COPD and bronchial asthma have increased metabolic rates. These increased rates are directly correlated to the severity of the diseases and the impairment in the respiratory function.

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shown that these patients often have seemingly adequate caloric intake suggesting that the weight loss may be due to an increase in energy expenditure [4].

Resting energy expenditure (REE) is often increased and may contribute towards energy imbalance in patients with chronic pulmonary diseases. Energy expenditure may be elevated due to increased basal metabolic rate, increased activity expenditure, or an increase in nutrient-induced thermogenesis [5].

The REE is typically divided into basal metabolic rate, which is normally the largest component of REE (>60% of REE), thermogenesis (mainly dietary induced thermogenesis, which accounts for about 10% of energy expenditure) and physical activity energy expenditure (PAEE) [6].

Patients with COPD have been reported to have a raised BMR, partly because of an increase in the energy cost of breathing. However, there is no universal agreement on this issue. Traditionally, it has been explained on the basis of an increased oxygen consumption of the respiratory muscles due to the increased work of breathing that characterizes the disease. However, it has been recently shown that skeletal non-respiratory muscle oxygen consumption is higher at any given load in patients with COPD than in age-matched healthy controls, indicating that bioenergetic abnormalities are also present in non-respiratory muscles, and that these abnormalities probably contribute to the increased metabolic rate in patients with COPD [7].

Several mechanisms could conceivably contribute to the increased metabolic rate in bronchial asthma and COPD. First, drugs commonly used in the treatment (e.g. B2-agonists) can increase metabolic rate. Second, systemic inflammation could also play a significant role. Third, tissue hypoxia which may also make a contribution, since other diseases characterized by tissue hypoxia, such as congestive heart failure, show increased metabolic rate [2].

Aim

The aim of the current study was to assess the changes that may occur in basal metabolic rates in patients with bronchial asthma and those with chronic obstructive pulmonary disease.

Subject and methods

The present study included 55 patients (35 patients with COPD and 20 patients had bronchial asthma) admitted to chest department, Menoufiya University hospitals in the period from February 2012 to August 2012. We also included 10 healthy non smoker subjects who volunteered as a control group.

Criteria of exclusion

Any patient suffering from other medical conditions, metabolic or endocriinal disorders was excluded from the study.

After having an informed consent from the patients, they underwent history taking, clinical examination, and radiological examination of the chest (P-A view).

Pulmonary function tests were done for all studied patients. Patients with COPD were classified according to GOLD 2010 [8] classification into mild, moderate, severe and very severe.

Arterial blood gases (partial pressure of oxygen (P\textsubscript{a}O\textsubscript{2}) and partial pressure of carbon dioxide (P\textsubscript{a}CO\textsubscript{2}) were measured in all patients.

The weight and height of the subjects were measured and used to calculate the body mass index (BMI). BMI was calculated as weight (kg) divided by height\textsuperscript{2} (m\textsuperscript{2}).

BMR measurement using indirect calorimetry

BMR was measured by indirect calorimetry using oronasal mask. The equipment used was a Quark BMR (COSMED, Chicago, USA). Before starting test the device and the analyzers should be calibrated according to the manufacturer’s instructions before each measurement [9].

- The subjects were informed not to eat for at least 12 h or smoke for at least 2 h before the test.
- The subjects were instructed to limit their physical activity the evening before the measurement.
- The measurements were performed in an environmental temperature between 22 and 23 °C.
- After a 30 min rest in the supine position, BMR was measured during 30 min when the subjects were awake in the supine position.
- The first 5 min of data acquisition was discarded when performing the test. The presented mean BMR for each patient is based on the last 25 min of the measurement.
- A steady state during the test was established when a 5 min of average oxygen uptake through the lung (VO\textsubscript{2}) and the amount of carbon dioxide expelled to atmosphere (VCO\textsubscript{2}) changes by less than 10% and the average respiratory Quotient (RQ) changes by less than 5%.

Statistical analysis

Data were collected, tabulated, statistically analyzed by computer using SPSS version 16.

Descriptive statistics

Quantitative data are expressed to measure the central tendency of data and diversion around the mean, mean (x) and standard deviation (SD).

Qualitative data expressed in number and percentage.

Analytic statistics

- Students T test was used for comparison of two quantitative variables.
- Kruskal Wallis test was used for comparison of more than two groups, LSD post hoc test was used to detect the inter-group differences.
- Pearson correlation (r) was used to detect association between quantitative variables.
- Chi square test was used for comparison of qualitative variables.
- p value > 0.05 was considered statistically non significant.
- p value ≤ 0.05 was considered statistically significant.
- p value ≤ 0.01 was considered statistically highly significant.
Results

There was marked increase in basal metabolic rates in patients with COPD compared to patients with bronchial asthma and to the control group, also between asthmatic patients and control group. The BMR also showed a highly significant negative correlation with the level of hypoxemia and pulmonary functions (the severity of the disease) in both COPD and asthmatic patients.

Tables 1–5 show results of this study.

Discussion

Nutritional abnormalities, including alterations in caloric intake, basal metabolic rate, intermediate metabolism, and body composition, are common in COPD. It is unlikely that these abnormalities are due to decreased caloric intake, which does not appear to be prominent in these patients except during episodes of exacerbation of the disease. In contrast, most patients with COPD have changes in basal metabolic rate, which often result in weight loss [5]. A few studies related to the changes that also may occur in patients with bronchial asthma are present, these studies showed changes in basal metabolic rate either increased especially during the attacks or decreased mostly due to associated hormonal abnormalities [10–12].

So, the aim of this work was to assess changes in basal metabolic rates that occur in patients with bronchial asthma and those with COPD.

In the present study there was a highly significant difference ($p$ value $<0.001$) between the ages of COPD patients and those of both bronchial asthma and the control group while the difference between the ages of patients with bronchial asthma and the control group was non significant ($p$ value $>0.05$) (Table 1). According to GOLD guidelines, COPD should be suspected in any patient aged 40 years or more with symptoms of cough, sputum production, or breathlessness and/or a history of exposure to risk factors, in particular smoking [13]. Fletcher and Peto [14] reported that COPD is characterized by an accelerated rate of decline of FEV1 with age. According to this, one might expect patients with severe COPD to be older. Although bronchial asthma is not rare in the elderly population, it has been considered a disease of childhood and young adults [10].

In the present work, there was a highly significant difference between COPD and asthmatic patients regarding the smoking habits ($p$ value $<0.001$). This is in agreement with the study of Laniado-Laborin [15], who stated that smoking is the most important causative factor for development of COPD and that lifelong smokers have a 50% probability of developing COPD during their lifetime.

In the present study, there was a highly significant increase in BMR in COPD patients and to a lesser extent in the asthmatic patients. There was a highly significant difference ($p$ value $<0.001$) between COPD and both asthmatic patients and control group while there was a significant difference ($p$ value $<0.05$) between asthmatic and control group (Table 1).

These results are in agreement with the results of Goldstein et al. [16] who found that energy expenditure was greater than predicted (116%) in patients with COPD and less than predicted (90%) in the control group.

Also, Hugli et al. [17] in their study showed that patients with stable COPD are characterized by a normal daily energy expenditure in controlled conditions in spite of an increased basal metabolic rate. They concluded that COPD patients appear to save energy by reducing their spontaneous level of physical activity. They found that BMR increased (137–145% of predicted) in their studied group.

Creutzberg et al. [7] supported the previous findings and concluded that hypermetabolism commonly occurs in COPD. They suggested contribution of an elevated oxygen cost of breathing (OCB) to hypermetabolism in COPD.

Slinde et al. [18] found that measured BMR was higher in the COPD group than the controllers. The median values for BMR was 103% of predicted in patients with COPD versus 99.7% of predicted in control group. They concluded in their study that patients with moderate-to-severe COPD tend to have higher BMR than normal subjects matched for age and habits. These results were explained by the influence of COPD associated mediators, chronic low-grade inflammation, hypoxia, hormonal alterations, medications and physical activity.

Table 1: Statistical comparison between COPD, bronchial asthma and control groups in different parameters.

<table>
<thead>
<tr>
<th>Variable</th>
<th>COPD $n=35$</th>
<th>Bronchial asthma $n=20$</th>
<th>Control group $n=10$</th>
<th>$p$ value</th>
<th>COPD vs. bronchial asthma</th>
<th>COPD vs. control</th>
<th>Bronchial asthma vs. control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Mean ± SD</td>
<td>60.54 ± 7.29</td>
<td>39.75 ± 10.51</td>
<td>41.7 ± 14.99</td>
<td>≤0.001**</td>
<td>≤0.001**</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>Male</td>
<td>25</td>
<td>8</td>
<td></td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Female</td>
<td>10</td>
<td>12</td>
<td></td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Smoking habit</td>
<td></td>
<td>Active smoker</td>
<td>22</td>
<td>7</td>
<td></td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Passive smoker</td>
<td>8</td>
<td>6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking index</td>
<td>Mean ± SD</td>
<td>297.14 ± 252.91</td>
<td>105 ± 152.95</td>
<td>22.4 ± 1.26</td>
<td>≤0.001**</td>
<td>&gt;0.05</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BMI</td>
<td>Mean ± SD</td>
<td>20.89 ± 3.92</td>
<td>22.25 ± 3.23</td>
<td>66.9 ± 13.17</td>
<td>&gt;0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV1</td>
<td>Mean ± SD</td>
<td>58.74 ± 19.08</td>
<td>75.05 ± 9.73</td>
<td>89.4 ± 1.51</td>
<td>≤0.001**</td>
<td>≤0.001**</td>
<td>≤0.05</td>
</tr>
<tr>
<td>PO2</td>
<td>Mean ± SD</td>
<td>66.83 ± 14.23</td>
<td>75.05 ± 9.73</td>
<td>89.4 ± 1.51</td>
<td>≤0.001**</td>
<td>≤0.001**</td>
<td>≤0.05</td>
</tr>
<tr>
<td>BMR</td>
<td>Mean ± SD</td>
<td>114.06 ± 15.23</td>
<td>102.1 ± 5.58</td>
<td>99.7 ± 1.51</td>
<td>≤0.001**</td>
<td>≤0.001**</td>
<td>≤0.05</td>
</tr>
</tbody>
</table>

* means statistically significant.
** means statistically highly significant.
In the present work, we found a significant difference ($p$-value $\leq 0.05$) between BMR in patients with bronchial asthma and the control group. These findings are in accordance with that of Zeitlin et al. [10] who found that the mean BMR of the asthmatic group was significantly greater than controls. Also, Maffei et al. [19] measured energy intake (EI) and total energy expenditure (TEE) of asthmatic males, they found that resting energy expenditure (REE) adjusted for fat-free mass was higher in asthmatic than in non asthmatic males. They concluded that males with mild-to-moderate asthma had a higher metabolic activity per unit fat-free mass than non asthmatic males.

The studies which disagreed with this work and showed decreased BMR in asthmatic patients explained their findings by the increased numbers of patients with hypothyroidism in their selected asthmatic patients group [11,12]. Moreover, there are conflicting data on the relation between BMI and bronchial asthma severity. Tavasoli et al. [20] found that with increasing asthma severity, they observed higher occurrence of obesity in adults. They concluded that the association of asthma severity with obesity suggests that obesity may be a potentially modifiable risk factor for asthma or asthma exacerbation.

On the other hand, Nadi et al. [21], in their study on asthmatic patients, observed negative association between BMI and asthma severity. Regarding the parameters affecting the BMR in all studied persons, we found that there was a highly significant correlation ($p$ value $\leq 0.001$) between BMR and the age and BMI (Table 2).

Starvation and weight loss have long been associated with a decrease in metabolic rate. However, patients with respiratory disease have an elevated metabolic rate even when malnourished [2].

The present work showed that BMR differed significantly between different grades of COPD severity, with a significant increase in BMR with increasing severity of the disease (Table 3). Also, there was a significant positive correlation ($p$ value $\leq 0.05$) regarding COPD patients between BMR and age, and a highly significant positive correlation ($p$ value $\leq 0.001$) between the BMR and smoking index, while there was a highly significant negative correlation between BMR with the BMI, FEV1 and level of hypoxemia (Table 4).

Nordenson et al. [22] stated that COPD patients who are underweight or are losing weight involuntarily have a higher mortality rate than other patients with COPD. Among patients with FEV1 of less than 35% predicted, 50% were undernourished. Thus, severity of airway obstruction increases the risk of undernutrition. Almost 50% of all COPD patients become underweight, and a low body mass index (BMI) is a major mortality risk factor.

Moreover, Donahoe et al. [23] studied 10 malnourished and 9 normally nourished patients with COPD and 7 control subjects. They found that BMR was elevated ($>120\%$ normal) in malnourished patients with COPD. Also, in malnourished patients, the oxygen cost of breathing was significantly higher than in the normally nourished and control group subjects. Therefore, it has been postulated that increased REE in undernourished patients with COPD is in part due to increased oxygen cost of breathing. Other possible mechanisms of the increased metabolic rate in patients with COPD regarding the severity may include; increased ventilatory requirements, inefficient use of the respiratory muscles, and possibly the effects of sympathomimetics such as theophylline and beta agonists.

The previous findings are supported by Ramires et al. [24] who found that the COPD group had lower BMI than control, and elderly patients with COPD had higher BMR.

Choi and Pai [25] investigated the association between respiratory functions and BMR. Pulmonary function indices and BMR were measured in 251 elderly persons. They concluded that respiratory function is more closely associated with BMR in elderly persons.

The present study showed that regarding asthmatic patients, there was a non significant correlation ($p$ value $>0.05$) between BMR and the age of patients, their BMI or their smoking index (Table 5), this may explained by the large number of asthmatic patients who were young and non smok-

### Table 2 Pearson correlation between BMR and different parameters in all studied persons ($n = 65$).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild COPD (I) $n = 7$</th>
<th>Moderate COPD (II) $n = 13$</th>
<th>Severe COPD (III) $n = 9$</th>
<th>Very severe COPD (IV) $n = 6$</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>58.7 ± 7.4</td>
<td>57.1 ± 6.5</td>
<td>64.9 ± 6.2</td>
<td>63.7 ± 7.1</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Gender</td>
<td>Male</td>
<td>4</td>
<td>6</td>
<td>9</td>
<td>&gt;0.05 &gt;0.05</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>3</td>
<td>7</td>
<td>0</td>
<td>&gt;0.05 ≤0.05</td>
</tr>
<tr>
<td>Smoking habit</td>
<td>Active</td>
<td>2</td>
<td>5</td>
<td>9</td>
<td>≤0.05</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>≤0.05</td>
</tr>
<tr>
<td>Smoking index (mean ± SD)</td>
<td>135.7 ± 232.2</td>
<td>134.6 ± 190.8</td>
<td>555.6 ± 84.6</td>
<td>450 ± 134.2</td>
<td>&gt;0.05 ≤0.05</td>
</tr>
<tr>
<td>BMI (mean ± SD)</td>
<td>20.4 ± 1.8</td>
<td>24.4 ± 4.1</td>
<td>18.3 ± 1.3</td>
<td>17.7 ± 1.2</td>
<td>≤0.01 ≤0.05</td>
</tr>
<tr>
<td>PO2 (mean ± SD)</td>
<td>78.6 ± 7.1</td>
<td>76 ± 5.3</td>
<td>52.7 ± 7.5</td>
<td>54.5 ± 14.3</td>
<td>&gt;0.05 &lt;0.001</td>
</tr>
<tr>
<td>BMR (mean ± SD)</td>
<td>106 ± 3.1</td>
<td>102.9 ± 7.2</td>
<td>127.9 ± 8.4</td>
<td>127 ± 19.2</td>
<td>&gt;0.05 &lt;0.001</td>
</tr>
</tbody>
</table>

** means statistically highly significant.
ers. But, there was a highly significant correlation ($p$ value $<0.001$) between BMR and both $\text{PO}_2$ and FEV$_1$.

To our knowledge, BMR was not extensively studied in asthmatic patients, however it could be postulated that severe degree of bronchial asthma especially if affecting oxygenation would affect metabolic rates by the same mechanisms as in COPD patients.

**Conclusion**

Basal metabolic rate increases in patients with COPD and bronchial asthma but with marked increase in the COPD patients.

In COPD patients, BMR levels are directly correlated to the severity of the disease including level of arterial oxygen tensions and pulmonary functions. Smoking increases the BMR in COPD patients either through its direct effects or through increasing the severity of the disease.

Special interest should be directed towards assessment of nutritional and metabolic status in chronic respiratory disease patients especially COPD, and special nutritional interventions should be planned to improve general condition and performance in such patients.

More studies should be done for more evaluation of changes in BMR in bronchial asthma.

**References**


**Table 4** Pearson correlation between BMR and different parameters in COPD patients ($n=35$).

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Smoking index</th>
<th>BMI</th>
<th>FEV1</th>
<th>$\text{PaO}_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$p$ value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMR</td>
<td>0.36</td>
<td>$&lt;0.05^*$</td>
<td>0.52</td>
<td>$&lt;0.001^{**}$</td>
<td>$-0.62$</td>
</tr>
</tbody>
</table>

* means statistically significant.

**Table 5** Pearson correlation between BMR and different parameters in bronchial asthma patients ($n=20$).

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Smoking index</th>
<th>BMI</th>
<th>FEV1</th>
<th>$\text{PaO}_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$R$</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>$p$ value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMR</td>
<td>0.13</td>
<td>$&gt;0.05$</td>
<td>$-0.14$</td>
<td>$&gt;0.05$</td>
<td>$-0.44$</td>
</tr>
</tbody>
</table>

$^{**}$ means statistically highly significant.