ABSTRACT
Background: tobacco smoking has been considered a risk factor for obstructive airway diseases, insulin resistance and development of type 2 diabetes mellitus (DM).
Aim: to assess the effects of cigarette smoking on lung functions and glucose metabolism in asymptomatic current cigarettes smokers.
Methodology: this case-control study was conducted on 100 asymptomatic current cigarette smokers and 100 age and sex matched lifelong non-smokers. Data regarding age, sex, smoking history including age of starting, smoking index and smoking duration were recorded. Spirometric-indices (VC %, FVC%, FEV1 %, FEV1/FVC ratio, FEF25-75%), serum fasting plasma glucose mg/dl (FPG) and glycated hemoglobin % (HbA1C) were measured.
Results: obstructive ventilatory defect and small airways obstruction were significantly higher in asymptomatic smokers than non-smokers (21% vs. 7% and 42% vs. 10% respectively) (p 0.004 and 0.001 respectively). Among smokers the frequencies of the pre-DM and DM detected by FPG there were higher than non-smokers (41% vs. 9% and 10% vs. 1% respectively). Moreover, there was higher frequencies of pre-DM and DM detected by HbA1C among smokers compared to non-smokers (60% vs. 11% and 10% vs. 1% respectively) (p 0.001). In smokers group both FEV1/FVC and FEF25-75% was positively correlated with age of starting smoking /yrs (p 0.001), and negatively correlated with smoking index, smoking duration, FPG and HbA1C (p 0.001). The HbA1C was positively correlated with smoking index and smoking duration and negatively correlated with age of starting smoking/yrs (p< 0.05). Smokers have 17.1 more risk of developing DM, 3.5 more risk of developing obstructive ventilatory defects and 6.5 more risk of developing small airways obstruction compared to non-smokers.
Conclusion: smoking is a risk factor for development of DM, obstructive ventilatory defect and small airways obstruction. Both reduced ventilatory functions and increased FPG and HbA1C negatively affected each other’s.

Keywords: Smoking, pulmonary function tests, glycosylated hemoglobin, diabetes, small airway obstruction

INTRODUCTION
Smoking as a method of consumption is most frequently used for tobacco, chiefly in the form of burnt tobacco and predominately cigarettes [1]. The WHO stated that tobacco smoking killed 100 million persons world-wide in the 20th century and assumed that it could kill one billion persons around the world in the twenty-one century [2]. Individual research’s revealed prevalence rate of tobacco smoking in Egypt that fluctuated between 19.7% in the Global Adult Tobacco Survey report to 30% in Egyptian population-based studies [3].

Smoking induced inflammatory process that leads to long-lasting changes in the lungs. The airways walls become thicker and mucus production is increased. Damage to the bronchial walls causes emphysema, and the lungs lose their elastic proprieties. Stimulation of irritant receptors in the bronchi constricts bronchial smooth muscles through a cholinergic pass-way via the
vagus nerve [4]. Additionally, accumulation of inflammatory cells e.g. B-cells, CD8+ T-lymphocytes, macrophages and neutrophils in the bronchi with secretion of their mediators, in response to irritants substances found in inhaled smoke produced an inflammatory phase. These continuing inflammation leads to structural and functional alterations in the lungs, which hasten the development of bronchial obstruction with subsequent development of chronic obstructive pulmonary disease (COPD) [5].

The link between cigarette smoking and the increased HbA1C may be due to nicotine, which has been demonstrated to increase plasma levels of catecholamines, which increase hepatic glycolysis and gluconeogenesis. The catecholamines might reduce the number of insulin binding sites as well as decrease the synthesis of glucose transporters. Smoking may directly decrease insulin sensitivity which is one of the crucial factors of glucose tolerance [6]. Additionally, constituents existing in tobacco smoke certainly generate free radical processes, interfere with vascular homeostasis and appropriate functioning of the vascular endothelium, and also increase inflammation/oxidative stress, in addition to directly destructing β-cell function [7]. This study was conducted to assess the effects of cigarette smoking on lung functions and glucose metabolism in asymptomatic current cigarettes smokers.

SUBJECTS AND METHODS

Type, place, and period of the study
This case-control study was performed at chest diseases department faculty of medicine for girls, Al-Azhar University, during the period from January 2020-June 2020.

Study population
The study was conducted on 200 healthy adults classified into two groups:

- **Smokers group**: Included 100 asymptomatic current smokers (serve as case group).
- **Non-smokers group**: Included 100 life-long non-smokers subjects with age, sex and BMI matched with cases (serve as a control group).

Exclusion criteria
Individuals with known chest diseases or DM were excluded from the study. Additionally, anemic patients and patients with chronic diseases (liver or kidney or gum disease, H. pylori infection) were also excluded from the study as they cause elevation of HbA1C.

Ethical consideration
The study protocol was approved by ethical review committee of Faculty of Medicine for Girls, Cairo, Al-Azhar University, Egypt. Participation was voluntary; an informed written consent was taken from each participant before enrolment into the study. Data were unnamed and coded to guarantee privacy of the participants.

Methods
Thorough history was taken with special emphasis on age/yrs, sex, age of starting smoking/yrs., number of cigarettes smoked daily and duration of smoking/yrs.

The smoking index (pack/year) was calculated as a number of packs smoked daily multiplied by number of years of smoking. The body mass index (BMI) was calculated according to the following equation [weight (kg)/height (m)²]. Liver and renal function tests as well as complete blood count were done to exclude participants with chronic diseases that affect HbA1C level.

Spirometry was carried out using SPIROSFIT SP-5000, (Japan). The following measurements were recorded; vital capacity (VC %), forced vital capacity (FVC %), forced expiratory volume in the first second (FEV₁ (%), FEV₁/FVC ratio, forced expiratory flow rate 25-75 (FEF25-75%)%. Spirometric-indices were calculated using the best out of three technically acceptable performances in accordance with the recommendations of the ERS [8]. To assess the frequencies of obstructive ventilatory defects, the study participants were classified based on FEV₁/FVC and FEV₁ % predicted ratio into: 1) obstructive ventilatory defect (FEV₁/FVC <70% and FEV₁ < 80%), 2) No obstructive ventilatory defect (FEV₁/FVC ≥70% and FEV₁ ≥ 80%). They also classified based on FEF 25-75% into: 1) Small airways obstruction (<65), and 2) No small airways obstructions (≥65) [9].

Venous blood sample was taken after an 8 hour overnight fasting for HbA1C % and fasting plasma glucose mg/dl (FPG) measurement. Studied Participants were categorized into three groups based on either FPG mg/dl [No DM (< 100 mg/dl), pre-DM (100-125 mg/dl) and DM (≥ 126 mg/dl)] and/or HbA1C [no DM (< 5.7%), pre-DM (5.7- 6.4%), AND DM (≥ 6.5%)] [10].

Statistical analysis
The studied data were analyzed by Statistical Program for Social Science (SPSS) version 24. Parametric variables were presented as mean± standard deviation (SD). Non-parametric variables were presented as numbers and percentages. The Independent-samples t-test was used to compare between two means (for normally distributed data). Chi-square test was used to compare non-parametric data. Pearson’s correlation coefficient (r) was used to assess the relationship between the two parametric variables in the same group. The Odds ratio (OR), ratio of odds of event occurring in exposed vs. unexposed group. OR are used to estimate how strongly a variable is associated with the outcome of interest. Probability was determined as: P-value < 0.05 was considered significant, P-value < 0.001 was considered highly significant, and P-value > 0.05 was considered insignificant (95% confidence interval).
RESULTS
Table (1) shows no statistically significant difference between smokers’ group and non-smokers group regarding age, sex, and BMI (p > 0.05). The FEV\textsubscript{1}/FVC ratio was statistically significantly decreased in smokers compared to non-smokers (p 0.001). Moreover, FPG mg/dl and HbA\textsubscript{1C} were highly statistically significantly increased in smokers’ group than non-smokers group (p 0.001). Table (2) shows that the obstructive ventilatory defect and small airways obstruction were more frequent among smokers than non-smokers (p 0.004 and 0.001 respectively). The frequencies of pre-DM and DM detected by either FPG or HbA\textsubscript{1C} were more common among smokers than non-smokers (p 0.001 each). Moreover, there was concordance of DM frequency detected by either FPG or HbA\textsubscript{1C} methods among both groups, with discordance of the frequency of pre DM detected by either FPG or HbA\textsubscript{1C} methods among both groups, with discordance of the frequency of pre DM detected by either FPG or HbA\textsubscript{1C} methods among both groups, with discordance of the frequency of pre DM detected by either FPG or HbA\textsubscript{1C} methods among both groups.

Table (3) and figures (1–4) show that among smokers group the FEV\textsubscript{1}/FVC ratio was positively correlated with age of starting smoking/yrs, FEV\textsubscript{1}%, FVC %, VC %, and FEF25-75% (p 0.001 each), while it was negatively correlated with smoking index, smoking duration/yrs and FPG mg/dl (p 0.001). Among smokers the FEF25-75% was positively with age of starting smoking/yrs. Pre-BD FEV\textsubscript{1}/FVC ratio, FEV\textsubscript{1}%, FVC%, VC%, and Post-BD FEV\textsubscript{1}/FVC ratio (p <0.05), while it was negatively correlated with age/yrs, smoking index, smoking duration/yrs, FPG mg/dl and HbA\textsubscript{1C} (p <0.05). The HbA\textsubscript{1C} was positively correlated with age/yrs, smoking index, smoking duration/yrs, and FPG mg/dl, while it was negatively correlated with age of starting smoking/yrs, pre-BD FEV\textsubscript{1}/FVC ratio, FEV1%, FVC%, VC%, FEF25-75%, and post-BD FEV\textsubscript{1}/FVC ratio (p <0.05). Table (4) shows that smokers have 17.1 more risk for developing DM (p 0.001), and 3.5 more risk for developing obstructive ventilatory defect (p 0.006) and 6.5 more risk for developing small airways obstruction compared to age, sex and BMI matched non-smokers (p 0.001).

<table>
<thead>
<tr>
<th>Studied variables</th>
<th>Smokers (N = 100)</th>
<th>Non-smokers (N = 100)</th>
<th>Test</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive ventilatory defect (FEV\textsubscript{1}/FVC &lt; 70 and FEV\textsubscript{1} &lt;80%)</td>
<td>No</td>
<td>79 (79%)</td>
<td>93 (93%)</td>
<td>8.1*</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>21 (21%)</td>
<td>7 (7%)</td>
<td></td>
</tr>
<tr>
<td>Small airway obstruction (FEF25-75% &lt; 65</td>
<td>No</td>
<td>58 (58%)</td>
<td>90 (90%)</td>
<td>26.6*</td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>42 (42%)</td>
<td>10 (10%)</td>
<td></td>
</tr>
<tr>
<td>DM (HbA\textsubscript{1C})</td>
<td>No DM</td>
<td>30 (30%)</td>
<td>88 (88%)</td>
<td>69.7*</td>
</tr>
<tr>
<td></td>
<td>Pre DM</td>
<td>60 (60%)</td>
<td>11 (11%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DM</td>
<td>10 (10%)</td>
<td>1 (1%)</td>
<td></td>
</tr>
<tr>
<td>DM (FPG)</td>
<td>No DM</td>
<td>49 (49%)</td>
<td>90 (90%)</td>
<td>39.9*</td>
</tr>
<tr>
<td></td>
<td>Pre DM</td>
<td>41 (41%)</td>
<td>9 (9%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DM</td>
<td>10 (10%)</td>
<td>1(1%)</td>
<td></td>
</tr>
</tbody>
</table>

*: Chi-square test, *: significant test
Table (3): Correlation study of HbA1C%, FEF 25-75%, FEV1/FVC ratio, with other studied variables in smokers’ group

<table>
<thead>
<tr>
<th>Studied variables</th>
<th>Smokers group</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HbA1C</td>
<td>FEF 25-75%</td>
<td>FEV1/FVC ratio</td>
<td></td>
</tr>
<tr>
<td></td>
<td>r</td>
<td>P</td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>Age/yrs</td>
<td>0.36</td>
<td>0.001*</td>
<td>-0.25</td>
<td>0.011*</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>0.11</td>
<td>0.29</td>
<td>-0.007</td>
<td>0.94</td>
</tr>
<tr>
<td>Smoking index (pack/yr.)</td>
<td>0.56</td>
<td>0.001*</td>
<td>-0.97</td>
<td>0.001*</td>
</tr>
<tr>
<td>Smoking duration/yr.</td>
<td>0.54</td>
<td>0.002*</td>
<td>-0.97</td>
<td>0.001*</td>
</tr>
<tr>
<td>Age of starting smoking/yr.</td>
<td>-0.46</td>
<td>0.001*</td>
<td>0.95</td>
<td>0.001*</td>
</tr>
<tr>
<td>Pre-BD FEV1/FVC ratio</td>
<td>-0.58</td>
<td>0.001*</td>
<td>0.97</td>
<td>0.001*</td>
</tr>
<tr>
<td>FVC%</td>
<td>-0.58</td>
<td>0.001*</td>
<td>0.98</td>
<td>0.001*</td>
</tr>
<tr>
<td>VC%</td>
<td>-0.54</td>
<td>0.002*</td>
<td>0.97</td>
<td>0.001*</td>
</tr>
<tr>
<td>FEF 25-75%</td>
<td>-0.62</td>
<td>0.003*</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>FPG mg/dl</td>
<td>0.75</td>
<td>0.001*</td>
<td>-0.88</td>
<td>0.003*</td>
</tr>
</tbody>
</table>


Table (4): Odd ratio for smoking as risk factors for DM, obstructive ventilatory defect and small airways obstruction

<table>
<thead>
<tr>
<th>Studied variables</th>
<th>Smokers (n = 100)</th>
<th>Non-smokers (n = 100)</th>
<th>OR</th>
<th>95% CL</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Increased</td>
<td>30 (30%)</td>
<td>88 (88%)</td>
<td>12 (12%)</td>
</tr>
<tr>
<td>HbA1C %</td>
<td>Normal</td>
<td>Increased</td>
<td>70 (70%)</td>
<td>12 (12%)</td>
<td>17.1</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Decreased</td>
<td>79 (79%)</td>
<td>93 (93%)</td>
<td>7 (7%)</td>
</tr>
<tr>
<td>FEV1/FVC ratio</td>
<td>Normal</td>
<td>Decreased</td>
<td>21 (21%)</td>
<td>7 (7%)</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Decreased</td>
<td>58 (58%)</td>
<td>90 (90%)</td>
<td>10 (10%)</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Decreased</td>
<td>42 (42%)</td>
<td>10 (10%)</td>
<td>6.5</td>
</tr>
</tbody>
</table>

FEV1/FVC: forced expiratory volume in first second/forced vital capacity, FEF25-75: forced expiratory flow at 25-75 of vital capacity, HbA1C: Glycated hemoglobin

Figure (1): Correlations of FEV1/FVC ratio with smoking index, age of starting smoking and smoking duration in smokers’ group. It shows that in smokers group the FEV1/FVC ratio was positively correlated with age of starting smoking, while it was negatively correlated with smoking index and smoking duration. Pre BD: pre bronchodilator FEV1/FVC: forced expiratory volume in first second/forced vital capacity

Figure (2): Correlation of FEF25-75% with smoking index, age of starting smoking and smoking duration in smokers’ group. It shows that among smokers the FEF25-75% was positively correlated with age of starting smoking/yrs, while it was negatively correlated with smoking index and smoking duration/yrs. FEF25-75: forced expiratory flow at 25-75 of vital capacity
DISCUSSION

Systemic inflammation is a common characteristic of both COPD and T2DM, which produce insulin resistance, atherosclerosis, and many extra-pulmonary manifestations of COPD [11]. Accordingly, this study was conducted to assess the effects of cigarette smoking on lung functions and glucose metabolism in asymptomatic current cigarette smokers. Hence, age was independent variable for spirometric-indices, and adiposity limit the normal movements of diaphragm and chest [12], we select smokers and non-smokers subjects’ matched regarding age, sex and BMI to avoid the synergistic effects of these confounders with smoking on airways function and HbA1C level.

In our study the FEV1/FVC ratio was the only spirometric-indices that significantly decreased in smokers than non-smokers. This finding points out that a decreased FEV1/FVC may be the earliest discriminative index for airway obstruction in asymptomatic smokers. Similarly, Wafy et al. [13] reported that the means of all spirometric-indices were significantly reduced in smokers than non-smokers, although, they are within-normal range. The decreased pulmonary function tests (PFT) in smokers were established to be statistically significant for FEF 25-75% with no significant difference in other values. Mistry et al. [14] they reported that at every age group, smokers had decreased FEV1/FVC ratio making them prone to develop COPD in the future. Nawaffeh et al. [15] and Manikandan et al. [4] reported that the FEV1%, FVC%, FEV1/FVC% and PEFR were reduced in smokers than non-smokers, in all age groups. Additionally, active smoking by healthy adults has been documented to cause declines in PFT evidenced by the quicker decline of FEV1 [16] and reductions in FEV1/FVC ratio and FEF25-75% [17]. Karia et al. [18] reported that the spirometric-indices are significantly reduced in smokers than non-smokers. Different results reported by Hasan and Sulaiman [19] as there was no significant difference in FEV1/FVC ratio and FEV1 between smokers and nonsmokers. Dugral and Balkanc [20] reported that smokers exhibited better FVC and FEV1 values. However, they exhibited significantly lower FEV1/FVC ratios than nonsmokers. The authors concluded that smoking improves lung function in young adults; these are “healthy smokers.” This difference may be attributed to that they study students and exclude subjects with evidence of airways obstruction (FEV1/FVC < 80%).

The current study demonstrated that both obstructive ventilatory defect and small airways obstruction were more frequent among smokers (21% and 42% respectively, p=0.004) than non-smokers (7% and 10% respectively) (p=0.001). These findings suggest that after adjustment of age, sex and BMI, cigarette smoking have negative impacts on airways function. Nicotine was described to be chemotactic for neutrophils, the main cell to be recruited in smoke-induced pulmonary inflammation and could therefore play a significant role in the initiation of the lung response finally leading to bronchial obstruction. The mixture of direct effects of cigarette smoke and indirect injury caused by inflammatory cells leads to a chain of epithelial alterations leading to airway obstruction [21]. Barthwal [22] documented that airway obstruction was seen in 12.6% smokers, of which 68.9% demonstrated mild obstruction and 31% demonstrated moderate obstruction. Sophie et al. [23] reported that among smokers PFT was abnormal in 72% of smokers and in 50% of non-smokers. Abnormal PFT was seen in 69.77% smokers with pack.
years <15 and in 85.6% of smokers with pack years >15, while mixed pattern was the most common abnormality in PFT observed in 35.0%. Manikandan et al. [41] shows that out of 30 cigarette smokers, 13.2% had obstructive ventilatory defect and 3.3% had restrictive pattern and 3.3% had mixed pulmonary impairment. Most of the nonsmokers (96.0%) had normal PFT results. Lower incidence of obstructive PFT was documented by Isah et al. [24] who found obstructive ventilatory defect in six smokers (4%) and two controls (4%). Khalil et al. [25] reported that the FEV1/FVC ratio <70% was more prevalent among smokers compared with non-smokers (17.3% and 6.7%, respectively).

In the early stage of COPD, some indices that reflect large airways functions e.g. FEV1 are generally within normal ranges, although small airways (<2mm) functions are impaired [26]. The higher frequency of small airways obstruction than obstructive ventilatory defect among smokers in our study (21% and 42% respectively) may be attributed to the well-known fact that small airways are devoid of some protective mechanisms present in the large airways as postulated by Bohadana et al. [21] who documented that the large airways are lined with ciliated cells “mucociliary escalator” that aids in eliminating inspired particles from the lungs. On the other hands, bronchi beyond 14 division do not have this defense, being therefore susceptible to the hazards of inhaled particles, particularly those in the respirable size (0.5-5 μm) such as particulate substance of the smoke (0.5 μm).

Additionally, the quantity of deposited particles and the location of deposition may differ based on the physical properties of the smoke particles and the smoking manner (volume and depth of inhalation, etc.). The long-lasting action of cigarette smoke on lung structures make an array of responses that will finally lead to small airways obstruction in the vulnerable smokers. It is reported that 15%-20% of heavy smokers have airways obstruction due to abnormalities in the small airways (<2mm in diameter) [5].

Our study revealed that after adjustment of age, sex and BMI, smokers have 3.5 times increased risk of developing obstructive ventilatory defect (p 0.006) and 6.5-time increased risk of developing small airways obstruction compared to non-smokers. Similar result was reported by Nighute and Avari [27] as the risk of having reduced PFT was 18 times in smokers than non-smokers. Urrutia et al. [28] stated that the risk of pulmonary disorders was directly linked to the number of cigarettes smoked daily.

In our study FPG mg/dl and HbA1C% were significantly increased in smokers than non-smokers (p 0.001). These results indicate that smoking has long-term negative effects on glucose metabolism. Similar result was reported by Khalil et al. [25] as the FPG and HbA1C% were significantly higher among smokers compared to nonsmokers. Other study reported that there is a significant increase in the HbA1C in smokers than non-smokers with no significant difference in FPG between both groups [6]. Another study has stated that current smokers show higher HbA1C than non-smokers, even in people without DM [29]. Soulimane et al. [30] in their meta-analysis involved individual’s from 14 countries recognized that HbA1C are elevated in smokers compared to nonsmokers without known DM. Dissimilar result was reported by McCulloch et al. [31] as smokers and non-smokers did not differ significantly regarding HbA1C level. Therefore, they suggested that smoking does not have a significant effect on HbA1C in patients with T2DM.

The present study revealed that the frequencies of pre-DM and DM detected by HbA1C were more common in smokers (60 and 10 respectively) than non-smokers (11 and 1 respectively) (p 0.001). These results suggest that after age, sex and BMI adjustment, cigarette smoking is an independent risk factor for DM. This agrees with Foy et al. [32] who studied the association between smoking status (never, former, and current) and occurrence of 5-year T2DM, in 906 individuals free of DM at baseline. They found that 25% of current smokers developed DM at five years, compared to 14% of never smokers. Akter et al. [33] conducted study involved 53,930 Japanese employees, aged 15-83 years, who did not have DM at baseline. They stated that through 3.9 years of median follow-up, 4.5% persons developed T2D. This variance in DM occurrence between the studies may be attributed to different susceptibility of populations under concern.

Our study revealed that after adjustment for age, sex and BMI smokers have 17.1 times increased risk for DM compared to non-smokers. Foy et al. [32] documented that smokers have significantly higher risk for T2DM than non-smokers (OR 2.66). Akter et al. [33] reported that the multivariate-adjusted risk ratios for DM were 1, 1.16 and 1.34 for never smokers, former smokers, and current smokers, respectively. Sang-Mo et al. [34] reported that subjects with a history of smoking more likely to have HbA1C between 5.7-6.4% compared to those who have HbA1C below 5.6% (OR1.091, 1.168 respectively). Cho et al. [29] found that ex-smokers and current smokers had a statistically higher risk for T2DM, and the risk increased with the number of cigarettes smoked. Additionally, Śliwińska-Mosson and Milnerowicz [7] stated that smoking is a predictor of the progression of glucose intolerance at both the conversion from normo-glycaemia to impaired glucose tolerance and the increased risk of developing DM.

The negative correlation detected between HbA1C and studied spirometric-indices in our study (p< 0.05), indicate that the well-known fact that the underlying inflammatory state that accompany DM have negative effects on airways function with subsequent development of COPD. On the other hand, the underlying systemic inflammatory response in COPD may alter glucose metabolism with subsequent development of DM. Similarly, Jamatia et al. [35] established that FVC
and FEV\(_1\) were roughly negatively correlated with post prandial blood sugar and HbA\(_1c\). Tina et al. [36] found no correlation between both COPD and small airway diseases in spirometry and duration of DM or level of glycemic control. Litonjua et al. [37] in the Normative Aging Study found that the decline of PFT over time was the same in participants with DM and participants without DM. Korean study, found that the reduced PFT is independently linked to the occurrence of T\(_2\)DM. FVC and FEV\(_1\) were negatively correlated with T\(_2\)DM. Therefore, they proposed that the likelihood that the reduced FEV\(_1\) and FVC may precede the development of T\(_2\)DM [38]. Dissimilar finding was reported by Baba et al. [39] as they found no association between the prevalence of FEV\(_1\)/FVC <70% and DM.

The main strength of the current study is that it is a case-control study thus it confirms the causal relationship between smoking and the outcomes measured (reduced PFT and DM). Additionally, all study participants did not have other risk factors for either reduced PFT or increase HbA\(_1c\) thus the effects of confounders in either airways functions or HbA\(_1c\) are avoided. The limitations of our research are that it was a single center study, so the results cannot be generalized. The smokers differ widely in their smoking manner, making quantitation of the nicotine dose absorbed by an individual smoker and from an individual cigarette are difficult.

CONCLUSIONS
Results of this study show that, current smokers had a greater incidence of non-manifested airways obstruction than those who are life-long non-smokers with matching of other related factors (age, sex, BMI). Also, results of this study add support to the hypothesis that smoking has long-term effects on glucose homeostasis, an association that cannot be clarified by confounding factors as age, sex, and BMI. Smoking was associated with preclinical or non-manifested reduction of spirometric-indices and higher HbA\(_1c\) levels in a sample of Egyptian current smokers who were non-COPD and non-diabetic adult males. Initiation of smoking cessation clinic in every hospital to teach people the importance of not to smoke is recommended. Engaging smoking cessation programs are important strategies for the management of patients with either respiratory diseases or diabetics. Health education campaigns are needed to keep community aware of the risk of smoking and to implement health promotion strategies that help prevent or reduce the incidence of both respiratory diseases and DM among smokers.

Future directions
- A further larger population-based study is needed to confirm these results.

REFERENCES
الملخص العربي

تقييم آثار تدخين السجائر على وظائف الرئة واستقلاب الجلوكوز في مدخنين السجائر الحاليين الذين لا يعانون من أعراض

رانيا عبدالكريم عبد الرؤوف، سميرة محمد ابوبكر، هبة حامد الطراوي، إيناس حسن أحمد

قسم الأمراض الصدرية، كلية طب البنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية.
قسم الدماء والأيض، كلية طب البنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية.

ملخص الحث
الخلفية: اعتبر تدخين التبغ عامل خطر لأمراض ضيق الشعب الهوائية، ومقاومة الأنسولين والإصابة بالسكري من النوع الثاني.

الهدف: تقييم آثار تدخين السجائر على وظائف الرئة واستقلاب الجلوكوز في مدخنين السجائر الحاليين الذين لا يعانون من أعراض.

الطريقة: أجريت دراسة حالات والشواهد هذه على 100 مدخن من مدخنين السجائر الحاليين الذين لا يعانون من أعراض و 100 غير المدخنين متوازئين من حيث العمر والجنس وتاريخ التدخين بما في ذلك سن بدء التدخين ومؤشر التدخين وما بعد التدخين. تم قياس مؤشرات قياس التنفس (السعة الحيوية للرئة والسعة الحيوية القصوى للرئة وأقصى معدل للزفير في الثانية الأولى) و نسب عنصر ما بين أقصى معدل للزفير في الثانية الأولى / السعة الحيوية للرئة و أقصى معدل للزفير في النسبة ما بين 25 و 75 من السعة الحيوية. كما تم قياس الجلوكوز الصائم في البلازما و الهيموجلوبين السكري.

النتائج: كان نمط ضيق الشعب الهوائية و ضيق الشعب الهوائية الصغرى أعلى إحصائياً في المدخنين الذين لا يعانون من أعراض مقارنة غير المدخنين (21% مقابل 7% و 42% مقابل 10% على التوالي). وجد أن نسبة ما قبل السكري و السكري المكتشفة بواسطة الجلوكوز الصائم في البلازما أعلى في المدخنين مقارنة غير المدخنين (41% مقابل 9% و 10% مقابل 1% على التوالي). كما كانت نسبة ما قبل السكري و السكري المكتشفة بواسطة الهيموجلوبين السكري أعلى في المدخنين مقارنة غير المدخنين (60% مقابل 11% و 10% مقابل 1% على التوالي). أرتبط كل من النسبة بين أقصى معدل للزفير في الثانية الأولى / السعة الحيوية للرئة وأقصى معدل للزفير في النسبة ما بين 25 و 75 من السعة الحيوية ارتباطاً إيجابياً بعمر الشخص عند بدء التدخين، بينما ارتبط سلبياً بمؤشر التدخين وما بعد التدخين. وجد أن المدخنين لديهم 17.1 خطر أكبر للإصابة بالسكري و 3.5 خطر أكبر للإصابة برض Civic ضيق الشعب الهوائية و 6.5 خطر أكبر للإصابة برض Civic ضيق الشعب الهوائية الصغرى مقارنة بغير المدخنين.

الاستنتاجات: التدخين عامل خطر للإصابة برض Civic ضيق الشعب الهوائية و ضيق الشعب الهوائية الصغرى و مرض السكري. كلاً من وظائف الرئة المنخفضة وزيادة الجلوكوز الصائم في البلازما / حجم الهيموجلوبين السكري تؤثر سلبية على بعضها البعض.

الكلمات المفتاحية: التدخين، اختبارات وظائف الرئة، الهيموجلوبين الغليكوزيليات، السكري، إنسداد مجرى الهواء الصغير.

الباحث الرئيسي

الأسم: رانيا عبد الكريم عبد الرؤوف

الهاتف: 01060289024

البريد الإلكتروني: ramyel76@gmail.com