Original Article

Waist circumference is an important predictor of pulmonary function and lung age in young adult smokers

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Abstract

Background and Aim: Despite the multitude of evidence on the deteriorating effect of smoking on lung function and lung age, less attention was devoted to the use of such effects as an effective strategy to quit smoking. Therefore, in this study, we aim to determine the predictability of waist circumference (WC) and body mass index (BMI) for pulmonary function and lung age in normal weight young adult smokers and nonsmokers.

Methods: One hundred and thirteen smokers and 95 nonsmoker control male students of Taif University were recruited. Pulmonary function tests including forced vital capacity (FVC), forced expiratory volume in 1 s (FEV1), FEV1/FVC ratio, and forced expiratory flow 25–75% (FEF25–75%) were performed by each student to measure the lung age. Anthropometric measurements included WC and BMI were performed.

Results: We found a significant lower mean value of FVC, FEV1/FVC ratio, and FEF25–75% in smokers as compared to mean values of age-matched nonsmoker students. Furthermore, the results revealed a significant increase in lung age of smokers as compared to that of nonsmoker students. In smoker subjects, FEV1, FVC, and FEF25–75% are correlated negatively with WC of the subject while their lung age correlated positively with WC.

Conclusion: There is a significant deteriorating effect of smoking on lung function on lung age. WC appears to be a better predictor of pulmonary function and lung age than BMI in normal weight young adults.

Key words: Body mass index, lung age, spirometry, waist circumference

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INTRODUCTION

Evaluation of pulmonary function by spirometry is the most important tool for clinical assessment of respiratory functions.[1,2] For maintaining the reliability of pulmonary function evaluation, population-specific reference values are required.[3,4] There is a significant difference in these values due to heritable, environmental, and ethnic differences of the studied population.[5,6] Smoking has been linked to a number of respiratory diseases including chronic obstructive pulmonary disease, lung cancer or cancers of the airways, and bronchial asthma. Smoking is the main cause of lung cancer which is considered the first leading cause of cancer death in men and the second leading cause of cancer death in women.[7] Furthermore, chronic obstructive pulmonary disease which is the fourth most common cause of death worldwide is largely caused by smoking.[8]

These diseases and their associated lung damage will deteriorate the lung function leading to premature lung aging. Estimation of "lung age" helps the patients

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to understand how low their lung function is. Hence, comparing both smoker's spirometric results and their lung age with the predicted values of nonsmoker normal subjects can be used as a potential psychological tool to show smokers the deterioration of their lung function, and the apparent premature aging of their lungs as an incentive to quit smoking, especially in those with most damage.[9]

Numerous studies have examined the association between body mass index (BMI) and waist circumference (WC) to pulmonary function testing (PTF) variables, and the associations vary in different subpopulations.[10-14] Nevertheless, whether the adiposity is linked to pulmonary function and lung age is not known and also there is a paucity of data on predictability of pulmonary function by adiposity indices in young adult smokers. Hence, the objective of the study was to determine the predictability of WC and BMI for pulmonary function and lung age in smoker and nonsmoker normal weight young adults.

MATERIALS AND METHODS

Subjects

This study was approved by the Ethics Committee of Taif University, Saudi Arabia. A total of 208 male students (113 smokers with average age of 20.6 ± 1.8 years and 95 nonsmoker controls with average age of 20.4 ± 1.7 years) were enrolled in this study. All subjects were informed about the experiments before giving their informed consent. In addition to age, subjects included in this study were nearly similar with respect to weight and WC.

Exclusion criteria

Subjects with asthma, respiratory illness, musculoskeletal disorders and taking treatment interfering with respiratory function, and obese and overweight persons were excluded from the study.

Spirometric measurements

Spirometric parameters (forced vital capacity [FVC], forced expiratory volume 1 [FEV1], FEV1/FVC ratio, and forced expiratory flow 25–75% [FEF25–75%]) were measured using Spirolab III (Medical International Research, Roma, Italy) according to the guidelines of the American Thorax Society and European Respiratory Society.[15] Before each measurement, the spirometer was calibrated. Subjects rested for 15 min before measurements and were briefed about the procedure. After appropriate placement of mouthpiece and nose clip, a powerful, quick, forced expiration was performed following maximum forced inhalation. By doing at least three technically appropriate measurements, the highest value was recorded as the baseline value. All volumes were reported in body temperature and pressure saturated.[15] All tests were done by an experienced technician.

Lung age calculation

Spirometry determines the lung age (another way to look at the effect of smoking on lung function),[9] which is computed by matching an individual's FEV1 value with the age at which that FEV1 value is considered normal based on predicted values. If a 6-foot, a 47-year-old man has an FEV1 of 2.2, for example, his lung age is 72 because 2.2 is the FEVI measurement considered normal for a 72-year-old man of that height. Calculating a patient's lung age can be a motivator in smoking cessation.[9]

Calculation of lung age was based on estimates developed by Morris and Temple.[9]

Lung age (men) $= 2.87 \times$ height (in inches) $- (31.25 \times$ observed FEV1 [liters]) $- 39.375$

Anthropometry

The body weight (in kg) was measured on digital weight scales (made in Germany) to the nearest 100 g with the subjects wearing light indoor clothes and no shoes after more than 8 h fasting. Height (in cm) was measured by stadiometer to the nearest 0.1 cm while students stood on a hard surface, wearing no shoes. After estimation of weight and height, BMI was measured (in kg/m^2) using the following equation (BMI = body weight/height^2 [kg/m^2]).[16]

WC, which is a measure of central obesity, was measured with a measuring tape in centimeters as the average of measurements made after inspiration and after expiration at the midpoint between the lowest rib and the iliac crest. Waist-to-hip ratio was determined as the ratio of WC and the circumference of the hips at the trochanter major.[17]

Smoking status

Smokers were defined as participants who currently smoke cigarettes daily and who had smoked at least 100 cigarettes before the date of spirometry. Nonsmokers were defined as participants who had not smoked at all or who had smoked fewer than 100 cigarettes during their lifetime.[18]

Statistical analysis of data

SPSS version 22 (SPSS Software Inc., Chicago, IL, USA) was used for data analysis. The data were presented as a mean ± standard deviation. The normality test was performed using Kolmogorov–Smirnov test. Independent *t*-test was applied to compare the measured general characteristics and respiratory values between control and smokers. Pearson's correlation test was used to determine the relationship between the pulmonary functions and lung age and study variables. The relationship between pulmonary functions and lung age (as dependent variables) and other variables (as the independent variables) was analyzed using the linear regression analysis. Stepwise multiple regression analysis in the whole study sample to assess the effect of smoking as a predictor determining lung age values was used. Level of significance was set at $P < 0.05$.

RESULTS

General characteristics of the participants

Table 1 shows the comparison of mean values for anthropometric and smoking characteristics between smoker and nonsmoker subjects. Among different anthropometric parameters, mean values for age, weight, height, BMI, and WC were nonsignificantly ($P > 0.05$) different.

Respiratory parameters and lung age in smokers and nonsmoker controls

The comparison of the mean values for lung age and pulmonary function characteristics between smoker and nonsmoker subjects are shown in Table 2. Among different pulmonary function parameters, FVC, FEV1/FVC ratio, and FEF25–75% mean values were found to be significantly ($P < 0.05$) lower in smoker subjects than their nonsmoker counterpart. On the other hand, lung age in smokers was significantly ($P < 0.05$) higher than in nonsmokers.

Table 1: Characteristics of the participating students

Parameters	Mean±SD		P Significant
	Nonsmokers (*n*=95)	Smokers (*n*=113)	
Age (years)	20.4±1.7	20.6±1.8	0.790
Weight	77.6±10	78.01±12	0.790
Height	1.72±0.05	1.74±0.06	0.016
BMI	24.8±3.24	24.5±3.36	0.566
WC	90.99±16.09	91.86±17.57	0.132
Smoking duration (years)	-	4.33±2.03	-
Cigarette number (cig/day)	-	22.7±5.71	-

Values are expressed as mean±SD. *P* value is of *t*-test. $P<0.05$ is significant. BMI: Body mass index, WC: Waist circumference, SD: Standard deviation

Table 2: Respiratory parameters and lung age in smokers and nonsmokers

Parameters	Mean±SD		P Significant
	Nonsmokers (*n*=95)	Smokers (*n*=113)	
FEV1	3.83±0.58	3.94±0.57	0.194
FVC	4.31±0.84	4.08±0.64	0.024
FEV1/FVC	94.23±6.07	92.27±7.45	0.038
FEF 25-75%	5.70±1.41	5.32±1.24	0.040
Lung age	30.01±18	66.12±29	0.000

Values are expressed as mean±SD. *P* value is of *t*-test. $P<0.05$ is significant. FEV1: Forced expiratory volume 1, FVC: Forced vital capacity, FEF 25-75%: Forced expiratory flow 25-75%, SD: Standard deviation

In smoker subjects, FEV1, FVC, and FEF25–75% are correlated negatively with WC of the subject while their lung age correlated positively with WC. No significant correlation between these parameters and BMI was seen in this group. Furthermore, among the nonsmoker group, there was no significant correlation between lung age and PTFs and WC and BMI [Table 3].

In smoker group, WC was negatively associated with FEV1, FVC, and FEF25–75%. The association of WC with FEV1/FVC was not significant ($P = 0.784$). In addition, WC was positively associated with lung age in smoker subjects. BMI was not significantly associated with lung age or with PTF parameters in neither smoker nor nonsmoker groups [Table 4].

Stepwise multiple regression analysis in the whole study sample revealed that smoking is a strong predictor determining lung age values in the entire sample. Smoking alone in model 1, accounts for 59.2% ($R = 592$, $F = 61.201$, $P = 0.000$) of the change in lung age [Table 5].

DISCUSSION

The results of our study demonstrate the deterioration of respiratory function and premature lung aging in smokers when compared to that of nonsmoker control students. There was a significant decrease in the FVC, FEV1/FVC ratio, and FEF25–75% and significant increase in lung age in smoker students' values as compared to the predicted normal values of nonsmoker students. Smoking has been identified to be the most important determinant of respiratory impairment.[19] This study showed nonsignificant differences between the mean values of FEV1in smoker students when compared with the nonsmoker controls.

These significant differences between smoker's spirometric values and that of nonsmokers were supported by a study done by Jawed *et al*.[20] They observed the association of smoking with deterioration of lung functions which is directly proportionate with the number of cigarettes smoked/day. Urrutia *et al*.[21] showed a dose-dependent association of cigarette smoking with the deterioration in FEV1/FVC ratio and the onset of respiratory complaints.

Jannet and Jeyanthi[22] showed a significant deterioration in the mean spirometric values of FEV1/FVC ratio of the smokers, and they described a positive correlation between smoking period and the reduction in pulmonary

Table 3: Pearson correlation of lung age and pulmonary function tests with waist circumference and body mass index in the entire study population

Parameters	WC				BMI			
	Nonsmokers (*n*=95)		Smokers (*n*=113)		Nonsmokers (*n*=95)		Smokers (*n*=113)	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
FEV1	0.005	0.962	−0.246	0.009	−0.016	0.879	0.092	0.334
FVC	0.005	0.961	−0.684	0.000	−0.019	0.851	0.009	0.926
Ratio	−0.008	0.939	−0.026	0.784	0.008	0.942	0.002	0.979
FEF 25-75%	−0.005	0.964	−0.582	0.000	−0.026	0.805	−0.027	0.775
Lung age	0.060	0.561	0.775	0.000	0.086	0.410	0.087	0.360

Statistical analysis was done by Pearsons correlation analysis. $P<0.05$ was considered significant. BMI: Body mass index, WC: Waist circumference, FEV1: Forced expiratory volume 1, FVC: Forced vital capacity, FEF 25-75%: Forced expiratory flow 25-75%

Table 4: Regression of lung age and pulmonary function tests with waist circumference and body mass index in the entire study population

Parameters	WC				BMI			
	Nonsmokers (*n*=95)		Smokers (*n*=113)		Nonsmokers (*n*=95)		Smokers (*n*=113)	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*
FEV1	0.005	0.962	0.246	0.009	0.016	0.879	0.092	0.334
FVC	0.005	0.961	0.684	0.000	0.019	0.851	0.009	0.926
Ratio	0.008	0.939	0.026	0.784	0.008	0.942	0.002	0.979
FEF 25-75%	0.005	0.964	0.582	0.000	0.026	0.805	0.027	0.775
Lung age	0.060	0.561	0.775	0.000	0.086	0.410	0.087	0.360

Statistical analysis was done by linear regression. $P<0.05$ was considered significant. BMI: Body mass index, WC: Waist circumference, FEV1: Forced expiratory volume 1, FVC: Forced vital capacity, FEF 25-75%: Forced expiratory flow 25-75%

Table 5: Regression coefficients and statistical significance of factors influencing lung age, based on multivariate stepwise linear regression

Model	Unstandardized coefficients		Standardized coefficients	*t*	Significant
	B	SE	Beta		
1					
Constant	−6.080	5.559		−1.094	0.275
Smoking	36.094	3.428	0.592	10.530	0.000

Dependent variable: Lung age. For model 1, R=592, F=61.201, P=0.000. SE: Standard error

functions. Moreover, Viegi *et al*.[23] showed that the prevalence of pathological PTFs is significantly higher in active and/or passive smokers. The deterioration of FVC may be an early marker of the small airways morphological changes in subjects exposed to smoke. The reduction in FVC reflects gas trapping as a result of small airway narrowing determined by loss of elastic load or airway thickening, whereas the decrease in FEV1/FVC ratio reflects smooth muscle contraction in the large airways.[24] Another explanation for deteriorating effect of smoking on respiratory function is that the cigarette smoking is believed to cause oxidative stress leading to apoptosis and lung injury. The mechanisms of smoking-induced oxidative stress include direct damage by oxidants and the smoking-induced inflammatory response.[25] Smoking also disturbs the oxidant-antioxidant balance which induces an oxidative burden leading to cellular damage in the lungs, destruction of the alveolar wall and airway enlargement, and also can trigger proinflammatory cytokines, which are increased in the lungs of smokers.[26] Accelerated decline of smoker's lung function can be further explained on the basis of the smoke-induced inflammatory processes with an increased number of macrophages in the first and second generation respiratory bronchioles.[27] Furthermore, the defensive lung reflex in response to smoking and the resulting airway narrowing and increased airway resistance may explain the abnormal lung function.[28] On the other hand, our results revealed a significant increase in smoker's lung age as compared to the nonsmoker control students. These findings are similar to those of Wada,[29] who assessed the lung age differences between smokers and nonsmokers in Japan. Dockery *et al*.[30] also reported that FEV1 decreased by 7.4 mL/1 pack-year increase in lifetime pack-year for male smokers, which translated to a lung age decrease of 1 year per 3 pack-year increase. These results were also consistent with the previous study by Beck *et al*.,[31] which described an inverse association of cigarette smoking with FEV1, which translated to a lung age.

Many studies conducted in the rest of the world have addressed that the relationship between obesity and spirometry tests demonstrated heterogeneous results, with some studies depicting no effects and other studies depicting positive effects. This discrepancy between studies could be explained by the wide variations in ethnicity of different population in PFT values, or this may be a result of methodological differences in these studies. This study showed that the pulmonary functions are significantly influenced by anthropometric indices such as WC. On analyzing the impact of WC on lung volumes in smoker subjects, FEV1, FVC, and FEF25–75% are correlated negatively with WC of the subject while their lung age correlated positively with WC. Our results were consistent with a previous study that reported WC as a better predictor of pulmonary function than BMI, although the study did not examine the associations in different BMI categories.[32] A recent study suggests that for every copy of the minor allele associated with cigarette consumption (i.e., increasing cigarette per day consumption by approximately one cigarette), WC will be increased by 0.14% if BMI was to remain constant. This suggests a preferential redistribution toward central adiposity associated with higher cigarette consumption.[33]

This study demonstrates the impact of central adiposity on pulmonary functions and is consistent with the results of other studies.[34-36] Abdominal adiposity (central fat distribution) may limit lung expansion and increase the thoracic pressure, leading to restrictive respiratory impairment. In mild obesity, the spirometry results may be normal or may suggest a restrictive process, with a symmetric reduction in FEV1 and FVC.[36] The most important change in pulmonary functions in obesity is a decrease in lung compliance due to the increased weight of chest wall and the higher position of diaphragm in the thoracic cavity resulting in a decrease in the lung expansion which subsequently leads to increase in work of breathing.[37] In addition, the deposition of fat on the chest wall may impede the expansion and excursion of the rib cage, through a direct loading effect or by altering the intercostal muscle function.[38] Furthermore, obesity has been shown to be associated with markers of systemic and vascular inflammation such as the cytokine leptin.[39] These inflammatory factors may exert local effects on lung tissue, leading to subtle reductions in airway diameter.

An important observation of the current study is that no significant correlation between these parameters and BMI was seen in the smoker group. For two reasons, BMI is not an ideal adiposity predictor of pulmonary function. The first is that normal-weight persons have more muscle mass than fat mass. Second, BMI is calculated from body weight and height, which are correlated with body size, with the larger the body size, the greater the PTF variables.[6]

Limitations of the study

A major limitation of BMI is that it does not distinguish fat mass and muscle mass, while they have different effects on pulmonary function.[40] Moreover, another limitation of estimation of only BMI is that it provides less information on body fat distribution.[6] Further, we have not assessed cytokines related to obesity.

CONCLUSION

In this study, an attempt was made to compare the lung function and lung age between smokers and nonsmokers, and the results clarify the significant deteriorating effect of smoking on lung function and its association with premature lung aging. Furthermore, we demonstrated WC is a better predictor of lung function and lung age than BMI in normal weight smokers. We strongly support the policy of reminding the smokers, their spirometry results expressed as "lung age" as the abnormal lung age is a clear message that the lungs are undergoing accelerated deterioration that would be slowed if the smoker stopped smoking. This is along with counseling about the dangers of continuing to smoke and ways to quit smoking.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Barreiro TJ, Perillo I. An approach to interpreting spirometry. Am Fam Physician 2004;69:1107-14.
2. Ferguson GT, Enright PL, Buist AS, Higgins MW. Office spirometry for lung health assessment in adults: A consensus statement from the National Lung Health Education Program. Respir Care 2000;45:513-30.
3. Budhiraja S, Singh D, Pooni PA, Dhooria GS. Pulmonary functions in normal school children in the age group of 6-15 years in North India. Iran J Pediatr 2010;20:82-90.
4. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, *et al*. Interpretative strategies for lung function tests. Eur Respir J 2005;26:948-68.
5. Trabelsi Y, Tabka Z, Richalet JP, Gharbi N, Bienvenu A, Guenard H, *et al*. Spirometric values in Tunisian children: Relationship with pubertal status. Ann Hum Biol 2007;34:195-205.
6. Chen Y, Rennie D, Cormier YF, Dosman J. Waist circumference is associated with pulmonary function in normal-weight, overweight, and obese subjects. Am J Clin Nutr 2007;85:35-9.
7. AIHW. Australia's Health 2004: The Ninth Biennial Health Report of Australian Institute of Health and Welfare. Canberra: AIHW; 2004.
8. Price D, Duerden M. Chronic obstructive pulmonary disease. BMJ 2003;326:1046-7.
9. Morris JF, Temple W. Spirometric "lung age" estimation for motivating smoking cessation. Prev Med 1985;14:655-62.
10. McKay RT, Levin LS, Lockey JE, Lemasters GK, Medvedovic M, Papes DM, *et al*. Weight change and lung function: Implications for workplace surveillance studies. J Occup Environ Med 1999;41:596-604.
11. Tsai WL, Yang CY, Lin SF, Fang FM. Impact of obesity on medical problems and quality of life in Taiwan. Am J Epidemiol 2004;160:557-65.
12. Carey IM, Cook DG, Strachan DP. The effects of adiposity

and weight change on forced expiratory volume decline in a longitudinal study of adults. Int J Obes Relat Metab Disord 1999;23:979-85.

13. Canoy D, Luben R, Welch A, Bingham S, Wareham N, Day N, *et al.* Abdominal obesity and respiratory function in men and women in the EPIC-Norfolk study, United Kingdom. Am J Epidemiol 2004;159:1140-9.
14. Chen R, Tunstall-Pedoe H, Bolton-Smith C, Hannah MK, Morrison C. Association of dietary antioxidants and waist circumference with pulmonary function and airway obstruction. Am J Epidemiol 2001;153:157-63.
15. Medical International Research (MIR) Spirolab III User Manual; 2007. p. 1-77. Available from: http://www.zonemedical.com.au/productfiles/SpiroLab3UserManual.pdf. [Last accessed on 2011 Jul 08].
16. Carroll JF, Zenebe WJ, Strange TB. Cardiovascular function in a rat model of diet-induced obesity. Hypertension 2006;48:65-72.
17. Osho O, Akinbo S, Osinubi A, Olawale O. Effect of progressive aerobic and resistance exercises on the pulmonary functions of individuals with type 2 diabetes in Nigeria. Int J Endocrinol Metab 2012;10:411-7.
18. Centers for Disease Control and Prevention (CDC). Cigarette smoking among adults – United States, 1992, and changes in the definition of current cigarette smoking. MMWR Morb Mortal Wkly Rep 1994;43:342-6.
19. Peat JK, Woolcock AJ, Cullen K. Decline of lung function and development of chronic airflow limitation: A longitudinal study of non-smokers and smokers in Busselton, Western Australia. Thorax 1990;45:32-7.
20. Jawed S, Ejaz S, Rehman R. Influence of smoking on lung functions in young adults. J Pak Med Assoc 2012;62:772-5.
21. Urrutia I, Capelastegui A, Quintana JM, Muñiozguren N, Basagana X, Sunyer J; Spanish Group of the European Community Respiratory Health Survey (ECRHS-I). Smoking habit, respiratory symptoms and lung function in young adults. Eur J Public Health 2005;15:160-5.
22. Jannet JV, Jeyanthi GP. Pulmonary health status of ginning factory women laborers in Tirupur, India. Indian J Occup Environ Med 2006;10:116-20.
23. Viegi G, Pedreschi M, Pistelli F, Di Pede F, Baldacci S, Carrozzi L, *et al.* Prevalence of airways obstruction in a general population: European Respiratory Society vs American Thoracic Society definition. Chest 2000;117 5 Suppl 2:339S-45S.
24. Corsico A, Milanese M, Baraldo S, Casoni GL, Papi A, Riccio AM, *et al.* Small airway morphology and lung function in the transition from normality to chronic airway obstruction. J Appl Physiol. 2003;95:441-7.
25. Halliwell B, Gutteridge JM. Free Radicals in Biology and Medicine. 4th ed. Oxford: Oxford University Press; 2007.
26. Devereux G. ABC of chronic obstructive pulmonary disease. Definition, epidemiology, and risk factors. BMJ 2006;332:1142-4.
27. Walter R, Gottlieb DJ, O'Connor GT. Environmental and genetic risk factors and gene-environment interactions in the pathogenesis of chronic obstructive lung disease. Environ Health Perspect 2000;108 Suppl 4:733-42.
28. Sterling GM. Mechanism of bronchoconstriction caused by cigarette smoking. Br Med J 1967;3:275-7.
29. Wada T. Lung age in smokers, past smokers anid non second-hand smoke. Rinsho Byori 2009;57:1159-63.
30. Dockery DW, Speizer FE, Ferris BG Jr., Ware JH, Louis TA, Spiro A 3rd. Cumulative and reversible effects of lifetime smoking on simple tests of lung function in adults. Am Rev Respir Dis 1988;137:286-92.
31. Beck GJ, Doyle CA, Schachter EN. Smoking and lung function. Am Rev Respir Dis 1981;123:149-55.
32. Ochs-Balcom HM, Grant BJ, Muti P, Sempos CT, Freudenheim JL, Trevisan M, *et al.* Pulmonary function and abdominal adiposity in the general population. Chest 2006;129:853-62.
33. Morris RW, Taylor AE, Fluharty ME, Bjørngaard JH, Åsvold BO, Elvestad Gabrielsen M, *et al.* Heavier smoking may lead to a relative increase in waist circumference: Evidence for a causal relationship from a Mendelian randomisation meta-analysis. The CARTA consortium. BMJ Open 2015;5:e008808.
34. Canoy D, Luben R, Welch A, Bingham S, Wareham KN, Day N, *et al.* Abdominal obesity and respiratory function in men. JCPSP 2004;17:265-8.
35. Lin WY, Yao CA, Wang HC, Huang KC. Impaired lung function is associated with obesity and metabolic syndrome in adults. Obesity (Silver Spring) 2006;14:1654-61.
36. Collins LC, Hoberty PD, Walker JF, Fletcher EC, Peiris AN. The effect of body fat distribution on pulmonary function tests. Chest 1995;107:1298-302.
37. Jones RL, Nzekwu MM. The effects of body mass index on lung volumes. Chest 2006;130:827-33.
38. Poulain M, Doucet M, Major GC, Drapeau V, Sériès F, Boulet LP, *et al.* The effect of obesity on chronic respiratory diseases: Pathophysiology and therapeutic strategies. CMAJ 2006;174:1293-9.
39. Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. Arch Intern Med 2002;162:1477-81.
40. Alipour B, Hosseini SZ, Sharifi A, Ansarin K. Influence of anthropometric measurements in lung function in patients with asthma. Int J Prev Med 2015;6:50.