

**THE EFFECT OF OBESITY MARKERS ON PEAK EXPIRATORY FLOW RATE IN
YOUNG SAUDI ADULTS**

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Abstract

Background: Obesity is one of the major health issues worldwide and so is in Saudi Arabia. It has enormously contributed to the increase in incidence of diabetes and cardiopulmonary problems. However, very few studies are available where the effects of obesity on pulmonary functions have been investigated in young healthy Saudis.

The aim of this study was to examine the lung function in the form of Peak Expiratory Flow Rate (PEFR) in healthy adults grouped into non-obese, pre-obese and obese. Moreover, Study groups were analyzed for the PEFR changes in association with different obesity markers other than BMI. This was to identify the obesity markers that may have better association with lung function impairment than Body mass index (BMI).

Methods: Young healthy students' ages 18-19 years were selected for this study. Their height, weight, waist & hip circumference and PEFR were measured. On the basis of BMI subjects were divided in non-obese 18.5-24.9, pre-obese 25-29.9 and obese > 30. All the analysis were performed using Graphpad Prism version 4 Mac (Graphpad software).

Results: Average values of all the morphometric parameters except height obtained from both pre-obese and obese subjects when compared with non-obese showed significantly higher values. Both the pre-obese and obese subjects showed reduction in PEFR compared to the non-obese. However, this reduction was found significant only in obese subjects. Further, PEFR showed a significant negative correlation with BMI, Waist circumference (WC), waist hip ratio (WHR) and body fat percentage.

Conclusion: Obesity produces significant deterioration in the PEFR in young healthy subjects and this deterioration is found to have significant negative correlation with all the different obesity markers. This study also reveals that about 50% young students belong to either pre-obese or obese group and this rising trend needs to be addressed.

Key words: Body mass index, peak expiratory flow rate, obesity and lung function

Introduction

Obesity is characterized by excess deposition of fat. This is a serious issue involving the people of both developing as well as developed countries of the world¹. The problem is recognized internationally² because of its increasing incidence and its association with cardiovascular diseases³, Stroke, Type2 Diabetes⁴, Hypertension, Cancers⁵, Osteoarthritis⁶, Respiratory problems including Asthma⁷, Depression⁸ as well as reduction in the ability to perform physical activities⁹. Further, incidence of obesity is not confined to any particular age group or socioeconomic class but is found in people of all ages and socioeconomic classes mainly because of sedentary life style and excess energy intake. Moreover, obesity is found to increase the chances of respiratory symptoms, like breathlessness particularly during exercise¹⁰ and recognized as an important risk factor in the development of respiratory diseases like obstructive sleep apnoea (OSA)¹¹ and obesity hypoventilation syndrome (OHS)¹². Wheezing and bronchial hyper-responsiveness¹³ often associated with asthma are increasingly observed in overweight and obese individuals. Recently, Steele et al¹⁴ reported an inverse relation of lung function with obesity and body fat in young adults.

Obesity induced deterioration in lung function is demonstrated by measuring lung volume and capacities (spirometry). However, many researchers for this purpose used measurement of PEFr because of its simplicity, convenience and cost-effective advantage¹⁵. Decrease in PEFr indicates a restrictive pulmonary defect because of mechanical limitation to the chest expansion due to accumulation of excess fat that interferes with movement of chest and descent of diaphragm¹⁶. PEFr is influenced by many factors such as age, sex, posture, obesity, environmental and racial factors¹⁷. Various studies have reported that, the markers of obesity like body mass index (BMI), waist circumference (WC) and waist

hip ratio (WHR) showed different correlation with PEFr^{18,19}. Even though height, weight and BMI are accepted as reliable tool for the identification of obesity in epidemiological studies, they have some limitations as it can't distinguish between fat mass and lean body mass and both of them are reported to have opposite effects on lung function²⁰. Another disadvantage of using BMI is that, it provides no information about the body fat distribution whether central or lower abdominal²¹. In fact a widely accepted opinion is that, abdominal height is a better marker of obesity as it is largely involved in reducing lung function by restricting the descent of the diaphragm compared to the central obesity which will compress the chest wall²². Effect of obesity on lung function has been reported in different parts of the world however; there is scarcity of literature available regarding these effects in the Saudi population where obesity is a rising problem. Moreover, there are few studies with stratification of younger age subjects in non-obese, pre-obese and obese categories. Therefore, the aim of this study was to find out the effects of obesity on lung function in young Saudi males and also to examine a better marker of deteriorated lung function in obese people other than body mass index (BMI).

Material and Methods

A total number of 152 first year healthy medical students aged between 18-19 years were included in this study. All participants are from Makkah region. They are non-smokers and have no history of regular physical training, sports activity or respiratory diseases like, Bronchitis, Pneumonia, tuberculosis and Asthma during last three years. None of the subjects had cough, wheeze, dyspnoea or nasal catarrh at the time of study. An informed verbal consent was obtained prior to participation in the study. Anthropometric measurements such as; Weight, Height, WC and Hip circumference were measured using standard clinical protocol²³. BMI of each

participant was calculated using the formula; body weight (kilograms)/height (meter)². On the basis of BMI participants were categorized as non-obese 18.5-24.9, Pre-obese 25.0-29.9 and obese 30. Dividing the value of waist circumference by the hip circumference gives us the value of WHR. BF % was estimated from the BMI along with taking the age and gender into account²⁴. The following formula was used for this purpose. Current BMI, age, and gender:

Adult Body Fat % = (1.20 × BMI) + (0.23 × Age) - (10.8 × gender) - 5.4 [Gender values for male =1, female = 0]²⁴.

PEFR was measured in standing position with standard range FERRARIS Pocket Peak flow meter” (Ferraris Medical Ltd. London) as used in other studies²⁵. It was ensured that the subjects were relaxed before performing the test. The procedure was explained to the subjects and a demonstration of manoeuvre was given to them. After proper rest, subjects were requested to take a deep breath and exhale as forcefully as possible in one single blow into the instrument with their nose closed. Three satisfactory reading were taken and best of the three was accepted in accordance with American thoracic society recommendations²⁶. Close watch was made to ensure that a tight seal was maintained between lips and

mouthpiece of the peak flow meter. To maintain uniformity and to avoid any diurnal variation in the tone of the bronchial tree all measurements of PEFR were carried out between 9-11am.

Statistical Analysis

The data was analyzed by student t-test to determine the statistical significance between two groups. For normally distributed data, a P value of <0.05 was considered significant.

To identify the best marker that affect the PEFR among the different groups the correlation coefficient was measured by Pearson correlation. The confidence interval was kept 95% with two tailed P values. All the analysis was performed using Graphpad Prism version 4 Mac (Graphpad software)

Results

The young healthy males of same age group were categorized on the bases of their BMI into three groups Non Obese, Pre Obese and Obese. It was interesting to witness the proportions of these young Saudis as 50% were Non Obese, 30% were Pre Obese and there were about 20% Obese among the students as well. We then observed the PEFR among these different groups. As expected there was declining trend seen in the PEFR with increase in the BMI (Figure 1)

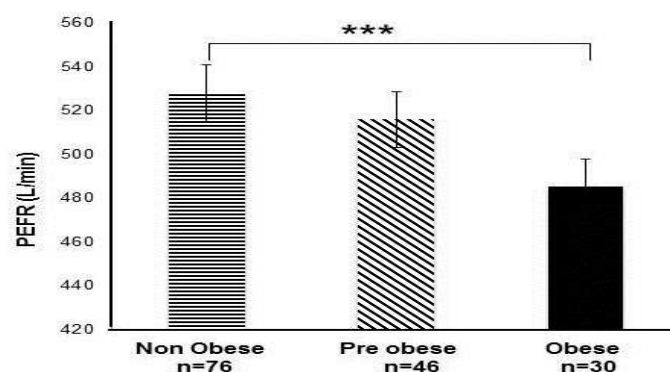


Figure 1 Comparison of mean PEFR

The mean of PEFR measured from three different groups was compared. The Non Obese (n=76) showed maximum mean. Whereas, the lowest mean was seen among the Obese subjects (n=30).

Data are presented as mean±SD. *p<0.05 compared with Non obese.

Obesity is known to reduce lung expansion and hence the peak expiratory flow rate. It was tempting to examine the correlation between PEFR and obesity markers. We

noticed a significant correlation of PEFR with different obesity markers as shown in (Table1).

Table1 Correlation Coefficient of obesity markers with PEFR

Parameters	BMI (kg/m ²)	WHR	Waist circumference	Body fat %age
Pearson r	-0.161	-0.26	-0.237	-0.16
P value	0.043*	0.001**	0.003**	0.046*
R squared	0.02	0.068	0.056	0.02

Variables are expressed as mean \pm SD * \leq P<0.05
BMI=body mass index, WHR=waist hip ratio

Average values of morphometric parameters, BMI, WC, WHR and BF% obtained from healthy subjects (Table.2&3) have shown that there are three distinct groups among them. Moreover, the PEFR measured has an inverse correlation with these obesity markers. We then explored the differences in Obesity markers and PEFR between our three categories on the basis of

BMI. Initially a comparison was performed between the obesity markers of Non Obese vs. pre obese group. Despite the fact that we observed a declining trend in the PEFR as the BMI increases, statistically non-significant difference was witnessed. Whereas, all the obesity markers showed a significant difference in pre-obese compared with non-obese as shown in (Table 2).

Table 2 Comparison of Mean Anthropometric data between Non-Obese and Pre-Obese

Parameters	Non Obese (n=76)	Pre Obese (n=46)
Height (cm)	170.5 \pm 0.6302	170.2 \pm 0.862
Weight (kg)	62.27 \pm 0.982	79.48 \pm 0.9588***
W/H ratio	0.8238 \pm 0.0062	0.8741 \pm 0.0076***
Waist Circumference (inch)	31.03 \pm 0.3271	37.01 \pm 0.468***
B. Fat %age	13.16 \pm 0.3166	20.86 \pm 0.2816***
BMI (Kg/m ²)	21.02 \pm 0.26	27.43 \pm 0.234***
PEFR (L/min)	528.6 \pm 6.968	516.8 \pm 13.38

Variables are expressed as mean \pm SD * \leq P<0.05
PEFR= Peak expiratory flow rate

Thereafter, the healthy Non Obese so to say our control group was compared with the obese subjects. Interestingly, we have a statistically significant difference in the

PEFR. Perhaps more body fat limited the lung expansion sufficiently, which in turn affected the PEFR in this group (Table3).

Table 3 Comparison of Mean Anthropometric data between Non-Obese and Obese

Parameters	Non Obese (n=76)	Obese (n=30)
Height (cm)	170.5 ± 0.6302	170.5 ± 1.053
Weight (kg)	62.27 ± 0.982	102.5 ± 3.7***
W/H ratio	0.8238 ± 0.0062	0.8951 ± 0.009***
Waist Circumference (inch)	31.03 ± 0.3271	43.03 ± 0.93 ***
B. Fat %age	13.16 ± 0.3166	30.01 ± 1.121 ***
BMI (Kg/m ²)	21.02 ± 0.26	35.05 ± 0.934 ***
PEFR (L/min)	528.6 ± 6.968	485 ± 12.87***

Variables are expressed as mean ± SD *P<0.05
PEFR= Peak expiratory flow rate

Discussion

The significant reduction in PEFR in obese subjects may be explained on the basis of mass load of adipose tissue around the rib cage, abdomen and in the visceral cavity that results in a shift in the balance of inflationary and deflationary pressure on the lungs as reported by J.T. Sharp et al²⁷. These obese subjects may also have limited lung expansion and air flow because of the restricted downward movement of the diaphragm due to increase abdominal adipose tissue leading to significantly reduced PEFR²⁸. Reduction was observed in total lung capacity (TLC) with an increase in the body weight. This possibility is also supported by the results of Thomas and Milledge²⁹ who observed an increase in TLC by reduction in the weight in obese and morbidly obese subjects. We are of the opinion that in our pre-obese and obese subjects an increase abdominal fat as indicated by significantly higher values of WC and WHR is responsible for restricted

diaphragmatic movement leading to the reduced PEFR. However, this reduction is found significant only in the obese subjects. This indicates that, the level of adiposity required to produce a significant reduction in PEFR values might not be achieved in pre-obese subjects and therefore they represent non-significant reduction in PEFR. Our results of PEFR in pre-obese subjects are in accordance with another study that reported non-significantly reduced PEFR in 30-40years of age healthy sedentary subjects. This means that not only young males 18-19 year of age but adults in their 30-40years of age if pre-obese won't show significant reduction in PEFR. Reduction in the volume of chest cavity produced as a result of deposition of fat in the sub-plural spaces³⁰ might also be responsible to reduce the PEFR in pre-obese and obese subjects and again the degree of deposition might not be sufficient in pre-obese subjects to produce a significant reduction. It is possible that our obese subjects have an increased

airway resistance that might result in significantly decreased PEFR as observed by Ghobain et al³¹. Our opinion of increased air way resistance as a possible cause of decreased PEFR is further strengthened by the study that showed reduction in alveolar diffusion capacity possibly due to structural alterations of the lung interstice by lipid deposition in obese children and/or a reduction in alveolar surface area³². This was observed in asthmatic and non-asthmatic obese children³². Changes in lipid deposition in the lungs are also reported³³ in another study of diet induced obesity in rats, which may affect surfactant function³⁴. It is also possible that the air way structures could be remodeled by exposure to pro-inflammatory adipokines, or damaged by the continual opening and closing of small air ways throughout the breathing cycle³⁵.

Pearson analysis shows significantly negative correlation of PEFR with all the obesity markers studied i.e. BMI, WC, WHR and BF% across the entire spectrum of non-obese, pre-obese and obese subjects. This negative association of PEFR with obesity markers particularly WC and WHR is representing primary restrictive lung function pattern. This restrictive pattern may be the result of limited diaphragmatic descent or may be because of diminished rib cage movement and thoracic compliance due to fat deposition in the chest wall. Both of these mechanisms lead to restricted respiratory movement³⁶. It is also interesting to note that the results of PEFR obtained from pre-obese and obese subjects as well as the Pearson correlation of PEFR with different obesity markers demonstrated an impairment of the pulmonary function with increasing obesity but not to such an extent that it will be evident clinically. Earlier several researchers also reported negative correlation between measures of obesity markers in general and abdominal obesity in particular and pulmonary function parameters. Yogesh Sexena reported an

inverse correlation of PEFR with all obesity markers but he found significant association only with WHR in 20-40 years of age healthy young males²³. An inverse association of abdominal height and WC with pulmonary function in men and women having BMI more than 25Kg/m² has been established²². In another study an inverse relation between pulmonary function and WHR both in men and women was observed³⁷. However, others observed and reported inverse relation of FEV1 with WHR only in men³⁸. In present study although PEFR showed significantly negative correlation with all the obesity markers but it is found to be stronger with WC and WHR being 0.001 and 0.003 respectively as compared to BMI and BF%. Further, significantly higher values of WHR shown by the pre-obese and obese groups of this study may be the result of decreased physical activity and sedentary life style of these subjects as it is reported that, increased physical activity is related to lower WHR in young adult men and women³⁹. However, this result differed from that of the study by Collins et al²⁰ who reported more strong negative correlation of lung function with BMI than WHR. Further, breathing mechanics involve contraction and descent of diaphragm during inspiration to increase the vertical diameter of thorax and intra-thoracic negativity. In this connection trunk obesity is more important than the overall adipose tissue represented by BMI. WC as a measure of abdominal fat deposition therefore, is reported to have more consistent predictability for pulmonary function than BMI that does not distinguish between fat mass and muscle mass²¹. These researchers further reported that WHR compared to WC is a more conveniently measured and is less likely to be influenced by sex or degree of obesity. Lazarus et al²⁰ also supported this view. Considering the results of this study and several earlier studies it can be stated that, as abdominal obesity plays more effective role in

restricting the lung function than the overall obesity therefore, WC and WHR are better anthropometric measurements that can be used clinically to assess the impact of obesity on pulmonary function rather than that of BMI.

Conclusion

It is an alarming sign that 50% young healthy Saudi male students are either pre-obese or obese and need to address seriously by promoting awareness regarding the adverse effects of obesity, improving the level of physical activity and through changes in the life style. Nevertheless, obesity like in other areas of the world impairs pulmonary function and the same is seen with the Saudi population. However, this reduction in PEFr was significant in the obese subjects and even in this group not to such an extent that become clinically evident. An interesting feature of our study that makes it different from earlier studies is significant negative correlation of PEFr with all the obesity markers. However, like several other researchers we also support the opinion that, WC and WHR are better markers to represent obesity-induced deterioration in the lung function than BMI. In the end we emphasize the need of a study to examine the effect of obesity on various pulmonary function parameters involving both male and female Saudi subjects of different age groups as well as a longitudinal study on university students to find out effects of changes in age, body weight, physical activity and smoking on lung function parameters.

References

1. Racette SB, Deusinger SS, Deusinger RH. Obesity: overview of prevalence, etiology, and treatment. *PhysTher.* 2003.
2. James PT, Leach R, Kalamara E, Shayeghi M. The worldwide obesity epidemic. *Obes Res.* 2001 Oct 31;9Suppl 4:228S–233S.
3. Manson JE, Colditz GA, Stampfer MJ, Willett WC, Rosner B, Monson RR, et al. A prospective study of obesity and risk of coronary heart disease in women. *N Engl J Med.* 1990 Mar 28;322(13):882–9.
4. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N Engl J Med.* 2001 Sep 12;345(11):790–7.
5. Stoll BAB. Perimenopausal weight gain and progression of breast cancer precursors. *Cancer Detect Prev.* 1998 Dec 31;23(1):31–6.
6. Davis MA, Ettinger WH, Neuhaus JM. Obesity and osteoarthritis of the knee: evidence from the National Health and Nutrition Examination Survey (NHANES I). *Semin Arthritis Rheum.* 1990 Nov 30;20(3 Suppl 1):34–41.
7. Chen Y, Dales R, Tang M, Krewski D. Obesity may increase the incidence of asthma in women but not in men: longitudinal observations from the Canadian National Population Health Surveys. *Am J Epidemiol.* 2002 Jan 31;155(3):191–7.
8. Friedman KE, Reichmann SK, Costanzo PR, Musante GJ. Body image partially mediates the relationship between obesity and psychological distress. *Obes Res.* 2002 Jan;10(1):33–41.
9. Tsuritani I, Honda R, Noborisaka Y, Ishida M, Ishizaki M, Yamada Y. Impact of obesity on musculoskeletal pain and difficulty of daily movements in Japanese middle-aged women. *Maturitas.* 2001 Dec 31;42(1):23–30.
10. Sahebjami H. Dyspnea in obese healthy men. *CHEST Journal.* 1998.
11. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med.* 2002 Apr 30;165(9):1217–39.
12. Akashiba T, Akahoshi T, Kawahara S. Clinical characteristics of obesity-hypoventilation syndrome in Japan: a multi-center study. ... (Tokyo. 2005.

13. Deane S, Thomson A. Obesity and the pulmonologist. *Arch Dis Child*. 2006 Jan 31;91(2):188–91.
14. Steele RM, Finucane FM, Griffin SJ, Wareham NJ, Ekelund U. Obesity is associated with altered lung function independently of physical activity and fitness. *Obesity (Silver Spring)*. 2009 Feb 28;17(3):578–84.
15. Collins LC, Hoberty PD, Walker JF, Fletcher EC, Peiris AN. The effect of body fat distribution on pulmonary function tests. *Chest*. 1995 May;107(5):1298–302.
16. Benjaponpitak S, Direkwattanachai C, Kraissarin C, Sasisakulporn C. Peak expiratory flow rate values of students in Bangkok. *J Med Assoc Thai*. 1999 Oct 31;82Suppl 1:S137–43.
17. Lim TOT. Peak Expiratory Flow Rate (PEFR) in elderly Malaysians. *Med J Malaysia*. 1999 Nov 30;54(4):531–4.
18. Carey IM, Cook DG, Strachan DP. The effects of adiposity and weight change on forced expiratory volume decline in a longitudinal study of adults. *CORD Conference Proceedings*. 1999 Aug 31;23(9):979–85.
19. Salome CM, King GG, Berend N. Physiology of obesity and effects on lung function. *J Appl Physiol* (1985). 2009 Dec 31;108(1):206–11.
20. Lazarus R, Sparrow D, Weiss ST. Effects of obesity and fat distribution on ventilatory function: the normative aging study. *Chest*. 1997 Apr;111(4):891–8.
21. 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*. 2006 Dec 31;85(1):35–9.
22. Ochs-Balcom HM, Grant BJB, Muti P, Sempos CT, Freudenheim JL, Trevisan M, et al. Pulmonary function and abdominal adiposity in the general population. *Chest*. 2006 Apr;129(4):853–62.
23. Saxena Y, Purwar B, Upmanyu R. Adiposity: determinant of peak expiratory flow rate in young Indian adults male. *Indian J Chest Dis Allied Sci*. 2011 Jan;53(1):29–33.
24. Deurenberg P, Weststrate JA, Seidell JC. Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. *Br J Nutr*. 1991 Mar;65(2):105–14.
25. Jain P, Kavuru MS, Emerman CL, Ahmad M. Utility of peak expiratory flow monitoring. *Chest*. 1998 Sep;114(3):861–76.
26. Standardization of Spirometry, 1994 Update. American Thoracic Society. *Am J Respir Crit Care Med*. 1995 Sep;152(3):1107–36.
27. sharpjt, henry jp, sweanysk, meadows wr, pietrasrj. effects of mass loading the respiratory system in man. *J Appl Physiol* (1985). 1964 Sep;19:959–66.
28. Guenette JA, Jensen D, O'Donnell DE. Respiratory function and the obesity paradox. *Curr Opin Clin Nutr Metab Care*. 2010 Oct 31;13(6):618–24.
29. Thomas PS, Cowen ER, Hulands G, Milledge JS. Respiratory function in the morbidly obese before and after weight loss. *Thorax*. 1989 Apr 30;44(5):382–6.
30. sargent en, boswellwd, ralls pw, markovitz a. subpleural fat pads in patients exposed to asbestos: distinction from non-calcified pleural plaques. *radiology*. 1984 jul 31;152(2):273–7.
31. Ghobain Al M. The effect of obesity on spirometry tests among healthy non-smoking adults. *BMC Pulm Med*. 2011 Dec 31;12(1):10–0.
32. del Río-Navarro B, Cisneros-Rivero M, Berber-Eslava A, Espínola-Reyna G, Sienna-Monge J. Exercise induced bronchospasm in asthmatic and non-asthmatic obese children. *Allergol Immunopathol (Madr)*. 1999 Dec 31;28(1):5–11.

33. Spathopoulos D, Paraskakis E, Trypsianis G, Tsalkidis A, Arvanitidou V, Emporiadou M, et al. The effect of obesity on pulmonary lung function of school aged children in Greece. *PediatrPulmonol.* 2009 Feb 28;44(3):273–80.
34. Inselman LS, Chander A, Spitzer AR. Diminished lung compliance and elevated surfactant lipids and proteins in nutritionally obese young rats. *Lung.* 2004;182(2):101–17.
35. Milic-Emili J, Torchio R, D'Angelo E. Closing volume: a reappraisal (1967-2007). *Eur J Appl Physiol.* 2007 Mar 31;99(6):567–83.
36. Wannamethee SG, Shaper AG, Whincup PH. Body fat distribution, body composition, and respiratory function in elderly men. *The American Journal of Clinical Nutrition.* 2005 Nov 1;82(5):996–1003.
37. 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 Jun 14;159(12):1140–9.
38. Harik-Khan RI, Wise RA, Fleg JL. The effect of gender on the relationship between body fat distribution and lung function. *J ClinEpidemiol.* 2001 Mar 31;54(4):399–406.
39. Tremblay AA, Després JPI, Leblanc CC, Craig CLC, Ferris BB, Stephens TT, et al. Effect of intensity of physical activity on body fatness and fat distribution. *Am J ClinNutr.* 1990 Jan 31;51(2):153–7.