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ORIGINAL ARTICLE

IMPACT OF OBESITY AND BODY FAT DISTRIBUTION ON PULMONARY FUNCTION OF EGYPTIAN CHILDREN

By

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Background: *Childhood obesity is an emerging global public health challenge. That is because the prevalence of obesity among children and adolescents has increased greatly in all parts of the world.(1) Overweight and obese children are at increased risk of a wide range of health conditions including respiratory diseases. A number of studies have reported an inverse relation between respiratory function and various indices of obesity or fat distribution.(2)*

The aim of this work: *was to study the impact of obesity on pulmonary function and to assess the correlation between lung function impairment, degree of obesity and fat distribution in Egyptian children with simple obesity.*

Patients and method: *This study was conducted on thirty children. They were divided into two groups. Group I included 20 children with simple obesity. Group II included 10 healthy normal children as a control group.*

All children were subjected to full history taking, thorough clinical examination, anthropometric assessment, plane x-ray left hand for bone age assessment, plane x-ray chest and pulmonary function testing including: FVC, FEV1, FEV1 / FVC or FEV1%, PEF maximum, PEF25%, 50 % and 75 %,: FEF25-75%, MVV.

Result: *Obese children had statistically significant higher rate of chest symptoms suggestive of bronchial asthma than the control group. They had also statistically significant reductions in FVC, FEV1, PEF, and MVV. They showed also lower values of FEV1/FVC ratio FEF 25%, 50%, 75% and FEF 25-75% when compared with control group but the difference did not reach significant level. BMI had significant negative correlation with FVC, FEV1, PEF max, flow rates (FEF25 %, FEF50 %, and FEF25-75 %) and MVV. Triceps skin fold thickness had significant negative correlation with, FEV_1, FEF25 %, FEF50 %, PEF max, and MVV. MAC was inversely correlated with FEV_1, FEF25 %, FEF50 %, FEF75 %, PEF max, and MVV. There was no statistically significant correlation between waist circumference or WHR and all the parameters of pulmonary functions.*

Conclusion: *Obese children have more respiratory symptoms than their normal weight peers. They have significant restrictive pulmonary defect, evident small airways obstruction and a defect in respiratory musculature, week effort and coordination, with increased airway resistance. BMI was inversely correlated with most of pulmonary function abnormalities. So, BMI is recommended to be used as a predictor of pulmonary function in assessment of obese children in epidemiological studies.*

INTRODUCTION

There has been a worldwide increase in obesity among people of all ages. As many as 250 million people, or about 7% of the current world population, are obese. Two to three times more people are overweight.[3] According to the World Health Organization, the number of overweight and obese people worldwide will increase to 1.5 billion by 2015 if current trends continue. Clearly, overweight and obesity place a large public health burden on society.[4]

Childhood obesity is an emerging global public health challenge. That is because the prevalence of obesity among children and adolescents has increased greatly in all parts of the world. There is no doubt that the percentages are even greater nowadays because of physical inactivity and westernization in diet1. In Egypt, Salem et al. (2002)[5] found that the prevalence of obesity among children and adolescent was 14.7% and 15.08% for males and females respectively.

Overweight and obese children are at increased risk of a range of medical conditions affecting cardiovascular health (hypercholesterolemia, dyslipidemia, hypertension), the endocrine system (hyperinsulinism, insulin resistance, impaired glucose tolerance, type 2 diabetes mellitus, menstrual irregularity), the pulmonary system, the musculoskeletal system, and mental health (depression, low self-esteem, distorted body image, eating disorders). In addition, obese children are at high risk for adult obesity.[6] Obesity is now recognized as an important risk factor in the development of several respiratory diseases. Of these respiratory diseases, it has already been well established that obesity can lead to obstructive sleep apnea (OSA) and obesity-hypoventilation syndrome (OHS). More recent data suggest that the prevalence of wheezing and bronchial hyper-responsiveness, two symptoms often associated with asthma, are increased in overweight and obese individual. Indeed, epidemiological studies have reported that obesity is a risk factor for the development of asthma.[7] Obese children have more respiratory symptoms than their normal weight peers and respiratory related pathology increases with increasing weight. Breathlessness, wheeze, and cough are not related to increased airway responsiveness and may respond more to weight loss than bronchodilator therapy.[8] Obesity is inversely associated with lung function in adults.[9] Similar data in pediatric population are however, limited and conflicting.

Different factors could explain this association. Obesity is capable of reducing pulmonary compliance, lung volumes, and the diameter of peripheral respiratory airways as well as affecting the volume of blood in the lungs and the ventilation-perfusion relationship. Furthermore, the increase in the normal functioning of adipose tissue in obese subjects leads to a systemic proinflammatory state, which produces a rise in the serum concentrations of several cytokines, the soluble fractions of their receptors, and chemokines. Many of these mediators are synthesized and secreted by cells from adipose tissue and receive the generic name of adipokines, including IL-6, IL-10, eotaxin, tumor necrosis factor-alpha, transforming growth factors-beta1, C-reactive protein, leptin, and adiponectin. Finally, specific regions of the human genome related to both asthma and obesity have been identified.[10] Recent investigations toward elucidating a shared genetic basis for these two disorders have identified polymorphisms in specific regions of chromosomes 5q, 6p, 11q13, and 12q, each of which contains one or more genes encoding receptors relevant to asthma, inflammation, and metabolic disorders, including the beta(2)-adrenergic receptor gene ADRB2 and the glucocorticoid receptor gene NR3C1.[11]

The aim of this work was to study the impact of obesity on pulmonary function and to assess the correlation between lung function impairment, degree of obesity and fat distribution in Egyptian children with simple obesity.

PATIENTS AND METHOD

This study was conducted on 30 children, 15 males and 15 females. Their ages ranged between 6 and 16 years (mean 11.45±2.93 SD). They were selected

from the Outpatients Clinic, Children's Hospitals, Ain Shams University in the period between August 2007 to April 2008. They were divided into two groups:

Group I (obese children): This group included 20 children with simple obesity, 10 males and 10 females. Their ages ranged between 6 and 16 years (mean 10.78 ± 2.79).The diagnosis of obesity was based on the body mass index (BMI) which equals: weight (kg) / height2 (m). The children were considered obese when their BMI was above the age and sex specific 95th percentile according to the Egyptian percentiles, developed by Cairo University, Diabetic, Endocrine and Metabolic Pediatric Unit and The National Research Center, 2003. Inclusion criteria included; age ranged between 6 and 18 years, BMI > the 95th of the age and sex specific percentile, exogenous (simple) obesity. While exclusion criteria included; endogenous obesity in which obesity is a part of a recognized genetic defect, clinical syndrome or acquired pathological condition, history of current upper respiratory tract infection, recent hospitalization due to chest problem, any bony cage deformity which can affect pulmonary functions or, history of cardiac problems.

Group II (control children): It included 10 healthy normal children 5 males and 5 females. Their ages ranged between 8 and 15 years (mean 12.8± 2.86). All cases and controls were subjected to the following:

I. Full history taking with emphasizes on symptoms suggestive of chest diseases e.g. asthmatic attacks in previous year, occasional wheezes, persistent wheezes or cough.

II. Thorough clinical examination with emphasis on chest examination

III. Anthropometric measurements:

1. Weight assessment (kg): the child was wearing light clothes using balance with 140kg capacity. The nearest 0.5kg was taken.
2. Standing height assessment (cm): All children were measured without shoes. The child stands with his heels together stretching upward to full extent, the back is straight as possible. It was taken to the nearest 0.5 cm.
3. Body mass index (BMI): was calculated from the previous weight and height measurements. (kg)/ht^2 (cm).
4. Skin fold thickness: triceps skin fold was measured using skin fold device (Harpenden), over the posterior surface of the triceps of the left arm in right handed people and the caliper was applied at a point mid way between the acromion and the olecranon processes as the arm hanged vertically in relaxed fashion at the child's side. Three measurements were taken, the mean value of them was used in all analysis.(12)
5. Waist circumference: was measured at the narrowest point between the rib cage and iliac crest and the hip circumference was measured at the widest point for the buttocks. Waist to hip ratio (w/h) in cm was calculated.(13)
6. Mid arm circumference: was measured at the midpoint between the acromion and olecranon process while the arm was hanging loosely at the side of the standing child. The taps was wrapped around the arm gently with no compression nor leave a gap and repeated for 3 times then the mean value was calculated and used in analysis.(13)

IV. Investigations: plane X-ray on the left hand for assessment of bone age, chest X-ray: postro-anterior and lateral views, pulmonary function testing using Med graphics 1070 series 2E/105 spirometer. The following steps were performed:

1. Calibration of the apparatus
2. Explanation of the maneuver to the patient.
3. Application of the nasal clip to the patient who would breathe by mouth through a

certain plastic tube connected to the system.

4. Performance of the following spirometric maneuvers:

a) Forced expiratory maneuver: in which the child takes full inspiration to the total lung capacity followed by rapid, forceful and maximal expiration to residual volume. The display was plotted with volume in liters and flow in liters/second. The following data were obtained: Forced vital capacity FVC (liter), Forced expiratory volume in the first second FEV_1 (liter),Forced expiratory volume in the first second percent FEV_1 / FVC or FEV_1%, Peak expiratory flow maximum, PEF maximum (liter/second), Peak expiratory flow 25%, 50 % and 75 % (liter / second), The forced midexpiratory flow rate: FEF25-75% (liter/sec).

b) Maximal voluntary ventilation: in which the child breathes as hard and as fast as possible for 12-15 second. The total volume is expressed in (Liters/ minute).

All volumes were corrected to body temperature and pressure saturated with water vapor. For every parameter obtained, actual and predicted values for age, sex, height, weight and percentage (%) of the predicated were calculated.

Statistical Analysis: This study was a randomized case-control study. The data were coded, entered and processed on an IBM-PC compatible computer using SPSS (version 15). The level $P < 0.05$ was considered the cut-off value for significance. Student's t-test was used to assess the statistical significance of the difference between two population means in a study involving independent samples. Correlation analysis: assessing the strength of association between two variables. The correlation coefficient denoted symbolically r, defines the strength and direction of the linear relationship between two variables.

RESULTS

The results of this study were illustrated in Tables 1-3. and (Figs. 1,2). There was a statistically significant higher differences as regards mean weight, BMI, Triceps skin fold thickness TSF (mm), Mid arm circumference MAC (cm), Waist circumference (WC), and (Waist/Hip Ratio) WHR in cases when compared to control $P<0.05$ but there was no statistical significant difference between cases and control as regards the mean age and height $P>0.05$ Table 1. Comparison between cases and control showed that 40.0% of obese children had recurrent chest symptoms suggestive of bronchial asthma with a statistically significant higher rate than control $P<0.05$. Table 2. and (Figs. 1,2) showed that there was a statistical significant difference between cases and control as regards the mean value of FVC , FEV_1, PEF max, and MVV (%predicted) $P< 0.05$. Although, FEV_1/FVC %, FEF25 %, FEF50 %, FEF75 %, FEF25-75 %, showed lower mean in cases when compared to controls the difference did not reach significant levels $P>0.05$. Table 3. showed that BMI was inversely correlated (significant negative correlation) with FVC, FEF25 %, FEF50 %, PEF max, FEF25-75 %, and MVV $P<0.05$.There was a highly significant negative correlation between BMI and FEV_1. While there was a negative correlation between BMI and FEV_1/FVC % and but this correlation was statistically not significant ($P>0.05$). Triceps skin fold thickness was inversely correlated (significant negative correlation) with, FEV_1, FEF25 %, FEF50 %, PEF max, and MVV $P<0.05$.While it was not significantly correlated to FVC, FEV_1/FVC %, FEF75 % and FEF25-75 %, $P>0.05$. MAC was inversely correlated (significant negative correlation) with, FEV_1, FEF25 %, FEF50 %, FEF75 %, PEF max, and MVV and $P<0.05$ while, it was not correlated to FVC, FEV_1/FVC %and FEF25-75 %, $P>0.05$. Waist circumference and WHR were not significantly correlated to all parameters of pulmonary function $P>0.05$.

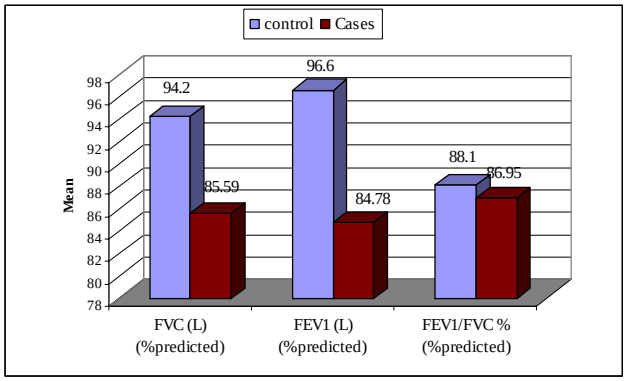

Fig 1. Comparison between cases and control as regards FVC (L), FEV_1 (L) FEV_1/FVC %.

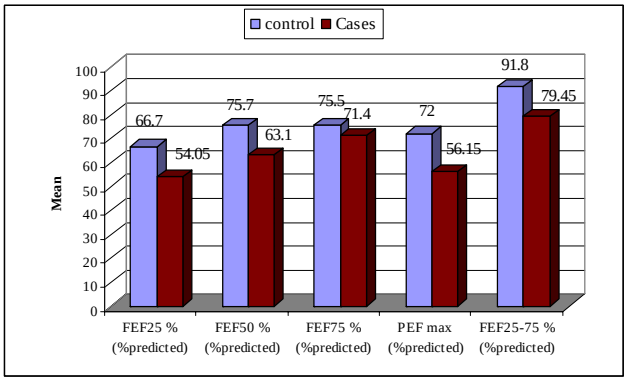

Fig 2. Comparison between cases and control as regards FEF25 %, FEF50 %, FEF75 %, PEF max, FEF25-75 %, ISVC (L) and MVV.

Table 1. Comparison between cases and controls as regard age, weight (kg), height (cm), BMI (kg/m2), triceps skin fold thickness (TS) (mm), mid arm circumference (MAC) (cm), waist circumference (WC) (cm), and waist/hip ratio WHR.

Variant	Control		Cases		T	P	Sig.
	Mean	±SD	Mean	±SD			
Age	12.80	2.86	10.78	2.79	1.86	0.07	NS
Weight (kg)	42.76	8.33	67.67	20.65	3.64	0.001	S
Height (cm)	145.05	11.20	143.10	11.94	0.43	0.67	NS
BMI (kg/m2)	20.19	1.79	32.52	6.07	6.23	<0.0001	HS
Triceps skin fold thickness (mm) (TS)	18.70	4.22	29.85	5.56	5.58	<0.0001	HS
Mid arm circumference (cm) (MAC)	25.65	3.28	34.55	5.12	4.98	<0.0001	HS
Waist circumference (cm)(WC)	77.40	8.52	86.53	13.02	2.00	0.05	S
Waist/Hip Ratio (WHR)	0.85	0.03	1.14	0.21	4.13	<0.0001	HS

Student's t test.

Table 2. Comparison between cases and controls as regards: parameters of pulmonary function.

Variant	Control		Cases		T	P	Sig.
	Mean	±SD	Mean	±SD			
FVC (L) (%predicted)	94.20	9.38	85.59	9.49	2.35	0.03	S
FEV*1* (L) (%predicted)	96.60	11.63	84.78	11.60	2.63	0.01	S
FEV*1*/FVC % (%predicted)	88.10	2.08	86.95	1.50	1.74	0.09	NS
FEF25 % (%predicted)	66.70	19.50	54.05	17.98	1.77	0.09	NS
FEF50 % (%predicted)	75.70	20.63	63.10	17.79	1.73	0.09	NS
FEF75 % (%predicted)	75.50	31.77	71.40	21.10	0.42	0.68	NS
PEF max (%predicted)	72.00	19.28	56.15	18.96	2.15	0.04	S
FEF25-75 % (%predicted)	91.80	20.12	79.45	19.77	1.60	0.12	NS
MVV (%predicted)	96.52	10.95	81.64	13.78	2.97	0.006	S

Student's t test.

Table 3. Correlation between BMI, TSF, MAC, WC, WHR and the parameters of pulmonary function.

	BMI		TSF		MAC		WC		WHR	
	R	P	R	P	R	P	R	P	R	P
FVC (L) (%predicted)	-0.44	0.02	-0.30	0.11	0.01	0.33	0.01	0.94	0.20	0.40
FEV*1* (L) (%predicted)	-0.62	<0.0001	-0.55	0.002	-0.14	0.003	-0.14	0.44	0.05	0.85
FEV*1*/FVC % (%predicted)	-0.20	0.29	-0.20	0.28	-0.12	0.18	-0.12	0.54	0.10	0.67
FEF25 % (%predicted)	-0.50	0.005	-0.45	0.01	-0.16	0.04	-0.16	0.39	0.04	0.88
FEF50 % (%predicted)	-0.50	0.005	-0.43	0.02	-0.18	0.02	-0.18	0.35	-0.06	0.79
FEF75 % (%predicted)	-0.50	0.005	-0.34	0.07	-0.20	0.03	-0.20	0.29	-0.24	0.32
FEF25-75 % (%predicted)	-0.50	0.005	-0.52	0.003	-0.27	0.004	-0.27	0.14	-0.02	0.95
PEF max (%predicted)	-0.59	0.001	-0.30	0.11	0.07	0.36	0.07	0.71	-0.10	0.68
MVV (%predicted)	-0.57	0.001	-0.44	0.01	-0.20	0.02	-0.20	0.29	-0.01	0.98

Pearson correlation coefficient.

DISCUSSION

Childhood obesity is an emerging global public health challenge. That is because the prevalence of obesity among children and adolescents has increased greatly in all parts of the world,1. Overweight and obese children are at increased risk of a wide range of health conditions including respiratory diseases. A number of studies have reported an inverse relation between respiratory function and various indices of obesity or fat distribution.[2]

In the present study, we found a highly significant statistical difference between cases and controls as regard history of chest symptoms e.g. asthmatic attacks in previous year, occasional or persistent wheezes and history of persistent cough. We found a statistically significant higher rate among obese children. This suggested that obese children were more susceptible to chest diseases especially bronchial asthma than normal children. This agreed with Deane and Thomson (2006)[8] who found that obese children have more respiratory symptoms than their normal weight peers and respiratory related pathology increases with increasing weight.

In our study, the obese group showed lower values of FVC, FEV_1 when compared with the control group and the difference was statistically significant ($p<0.05$). Low FVC, FEV_1 indicated a restrictive pulmonary defect. This may be due to mechanical limitation of chest expansion as accumulation of excess fat interferes with the movement of the chest wall and the descent of the diaphragm. This may reflect intrinsic changes within the lung in the presence of obesity. Lipid deposition, cellular hyperplasia, alveolar enlargement, and reductions in alveolar surface area relative to lung volume occur (Li, 2003).[2]

Also, we found a statistical significant lower value of PEF in obese children than the control. Low PEF indicated that there was small air ways obstructive defect. In addition, obese children showed lower values of FEV_1/FVC ratio and forced expiratory flow rates FEF 25%, 50%, 75% and FEF 25-75% when compared with control group but the difference was not significant ($p< 0.05$). Low FEV_1/FVC ratio and flow rates are a spirometric signature of obstructive airway diseases. This result suggested that, obesity had also a significant obstructive effect on small airways. And so, obese children are at high risk to develop bronchial asthma. The possible explanation may be through the influence of obesity on airway smooth muscle function. In obese individuals, even in the absence of an overt inflammatory insult, there is chronic, low-grade systemic inflammation characterized by increased circulating leukocytes and increased serum concentrations of cytokines, cytokine receptors, chemokines, and acute-phase proteins that could predispose to airway hyper responsiveness (AHR).[14] In addition, there are also changes in the serum concentrations of hormones and other factors derived from adipose tissue that could affect airway function. These include leptin, adiponectin, plasminogen activator inhibitor
(PAI-1) and tumor necrosis factorα (TNFα).[15]

The results of our study agreed with Eisenmann et al. (2007)16 who found an increase in pulmonary function in normal weight children and a decrease in pulmonary function of obese children. Significant differences between both groups existed for FVC, FEV1, FEV1% and FEF25-75.

Also our study agreed with, Harik-Khan et al. (2001)[17] who found that FVC, FEV1decreased in obese cases than in normal.

Our results were also similar to the results of Zerah et al. (1993)[18] who concluded that in obesity, airway abnormalities involved a predominant increase in proximal airway resistance but only minimal distal obstruction.

In contrast to our results, Li et al. (2003)[2] studied the effects of obesity on pulmonary function in obese Chinese children. They concluded that obesity does not affect spirometric values. The disagreement could be due to the difference in the nature of the study as they did not include a control group, also, racial factors may play a role.

As regards the MVV we noted in our study a

statistically significant difference between obese and normal children as the obese cases showed a lower mean level of MVV (% predicted). This indicated a defect in respiratory musculature, week effort and coordination, and increased airway resistance among obese children. This result can be explained by the deposition of fat between the muscles and the ribs which may decrease chest wall compliance, increasing the metabolic demands and workload of breathing in the obese even when at rest.[16]

Our results were in accordance with Fabris et al. (2007)[19] who examined the respiratory dynamics in obese adult patients. They concluded that respiratory muscle function was markedly impaired in obese patients. Fat excess, particularly visceral obesity, probably interfered with respiratory muscle activity.

Our results were similar to Sahebjami and Gartside (1995)[20] who examined pulmonary function test (PFT) profile and respiratory muscle strength in obese subjects with FEV1/FVC ratio greater than 80%. They found that FVC, FEV1, forced expiratory flow at 50% vital capacity and maximum inspiratory flow rate were significantly lower in obese subjects with low MVV compared with those in whom MVV was normal. They concluded that the standard PFTs allow recognition of a subgroup of obese subjects without overt obstructive airway disease who have more severe lung dysfunction, the marker of which is a low MVV. Peripheral airway abnormalities may be responsible for these observations.

In our study, we examined the correlation between the degree of obesity, fat distribution and pulmonary function. General adiposity was marked by BMI, abdominal adiposity by waist circumference (WC) and waist/hip ratio (W/H) and the peripheral adiposity by midarm circumference (MAC) and triceps skin fold thickness which can give idea about obesity on the chest wall. We expressed the correlation between each one and the pulmonary function and we found that, BMI was inversely correlated with most pulmonary function abnormalities. There was a significant negative correlation between BMI and FVC and a highly significant negative correlation between BMI and FEV1. Meaning that as BMI increased, the restrictive pattern of the pulmonary function was increased. Also, BMI was negatively correlated to PEF max, flow rates (FEF25 %, FEF50 %, FEF25-75 %) and MVV. Low FEV1/FVC % was not significantly correlated with increased BMI. We concluded that BMI as a measure of overall obesity had significant effects on ventilatory function. This well-known index has much to recommend it for epidemiologic purposes, being a relatively simple and reliable measure that is largely independent of height. But, it gives no indication of fat distribution.

As regards both triceps skin fold thickness and mid arm circumference MAC, used as markers of peripheral fat distribution in the upper part of the body, we found that both of them were not significantly correlated to FVC, FEV1/FVC % ratio and FEF25-75 %. But we found a significant negative correlation between both of them and FEV1, PEF max, flow rates (FEF25 %, FEF50 %) and MVV.

As regards waist circumference, it was not significantly correlated to any parameter of the pulmonary function $P>0.05$. As regards waist/hip ratio, as a marker of abdominal adiposity, we could not find any significant relation between it and any of the parameters of pulmonary function.

Our findings suggest that both obesity itself and the pattern of body fat distribution have independent effects on ventilatory function. Relative adiposity, as measured by BMI, had a significant effect on ventilatory function. This supports the hypotheses that obesity affects the respiratory function by multiple mechanisms in addition to its direct mechanical effect on the abdominal and chest wall as fat is a metabolically active tissue.

Our results agreed with Harik-Khan et al. (2001)[17] who found that BMI was inversely correlated to the FVC, FEV1.

On the other hand, our study disagreed with

Medarov et al. (2005)[21] who found that FEV_1 was not affected and had no relationship to BMI.

Also, Chen et al. (2007)[22] found that WC (waist circumference) was negatively associated with FVC and FEV_1, and the associations were consistent across sex, age, and BMI categories this was explained by the effect of abdominal adiposity on the diaphragm, causing limitation of its free movement. , also disagreed with Balcom et al. (2006)[23] who found that abdominal adiposity is a better predictor of pulmonary function than weight or BMI. Canoy et al. (2004)[24] also found that both FEV_1 and FVC were linearly and inversely related across the entire range of waist/hip ratio in both men and women.

Lazarus et al. (1997)[25] found that BMI was positively correlated to FEV_1/FVC ratio in adults at all ages and negatively with FVC between 40 and 69 years whereas the ratio of abdominal girth to hip breadth (AG/HB) was negatively associated with FVC and FEV_1 in men aged 50 to 59 years only ($p<0.0004$). So they concluded that Body fat distribution had independent effects on ventilatory function for overall obesity in men and age modifies this association.

Our results also disagreed with Collins et al. (1995)[26] who examined healthy normal or mildly obese men. They found that biceps skin fold thickness had the strongest inverse relationship with total lung capacity. However, comparing pulmonary function tests between patients with a WHR less than 0.950 (lower body fat distribution) and subjects with a WHR of 0.950 or greater (upper body fat distribution) revealed that FVC, FEVI, and TLC were significantly lower in the patients with upper body fat distribution. This suggested that upper body fat distribution may be associated with a modest impairment of lung volumes in normal and mildly obese men. The discrepancy between these results and ours may be due to differences in the age group.

Limitations: the cross-sectional nature of this study is a limitation, so longitudinal studies are needed to investigate the effect of reduction of weight on pulmonary function in children.

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