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Original Research Article

ANTHROPOMETRY, ADIPOSITY INDICES AND PEFR DETERMINANTS IN A YOUNG ADULT CROSS-SECTIONAL STUDY

Neeru Garg[1], Vaishnavi Sharma[2], Yafi Munir[3], Madhav Aryan Goyal[4], Arush Goyal[5], Himani Muniyal Jheetay[6], Sanket Jheetay[7]

[1]*Professor & HOD, Department of Physiology, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[2]*MBBS Student First Year, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[3]*MBBS Student First Year, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[4]*Tutor, Department of Anatomy, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[5]*Junior Resident, Department of Medicine, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[6]*Assistant Professor, Department of Biochemistry, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*
[7]*Assistant Professor, Department of Physiology, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.*

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Corresponding Author:
Dr. Yafi Munir,
MBBS Student First Year, Graphic Era Institute of Medical Sciences, Dehradun, Uttarakhand, India.
Email: yafimunir@gmail.com

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ABSTRACT

Background: Obesity related lung changes are often subtle in youth, and BMI alone may miss distribution effects. We evaluated multiple adiposity indices against spirometry and peak flow in apparently healthy young adults.

Materials and Methods: This analytical cross-sectional study included 139 young adults (17–22 years) after screening. Anthropometry (height, weight, waist, hip) was recorded and adiposity indices were derived (BMI, waist circumference, WHR, WHtR, ABSI, conicity index, BAI). Pulmonary measures included FEV1, FVC, FEV1/FVC, and PEFR. Associations were tested using Spearman correlation. Multivariable linear regression was fitted with PEFR as outcome and BMI, WHtR, height, age, and sex as predictors.

Results: Mean age was 19.1 years, females 64.7%. Mean FEV1 was 2.95 L, FVC 3.20 L, with a high mean FEV1/FVC of 92.6%, suggesting no cohort-level obstruction. BAI showed consistent inverse correlations with FEV1 (rho −0.308, p=0.0001) and PEFR (rho −0.319). Waist circumference correlated positively with FEV1 and FVC, but negatively with FEV1/FVC. ABSI and conicity index had weak non-significant associations. In regression, height (β 337, p<0.001) and male sex (β 97.4, p<0.001) were strong independent predictors of PEFR, while BMI and WHtR were not significant. Model R^2 was 0.543 (adjusted 0.526).

Conclusion: In young adults, lung flow is primarily scaled by height and sex, while BAI may capture early adiposity-related functional reduction.

Keywords: Young adults, spirometry, PEFR, body adiposity index, waist-height ratio, ABSI, central obesity.

INTRODUCTION

Adiposity and lung function are linked but the relation is not as one straight line where volumes and flow depend strongly on height, sex and age.[1] Still excess central fat can alter respiratory mechanics by reducing chest wall compliance and diaphragmatic excursion and by changing airway calibre by inflammatory and metabolic pathways.[2] Recent work is moving beyond BMI alone and is testing abdominal and shape indices (waist circumference, WHR, WHtR, ABSI, BRI etc) against spirometry outputs like FEV1, FVC, FEV1/FVC and PEFR.[3]

The datasets and reviews show that abdominal obesity measures tend to track impaired pulmonary function more consistently than BMI the direction can vary by age group and baseline body size. In healthy adults most included studies found abdominal obesity to be inversely associated with spirometric parameters suggesting central fat is a relevant driver even when people do not have diagnosed lung disease.[4] The association can be non-linear and differs for ratios versus absolute

volumes so interpreting FEV1/FVC separate from FEV1 or FVC becomes important.[3] Longitudinal evidence also supports central obesity as a stronger signal than total adiposity. In a middle-aged Asian cohorts worsening adiposity over time was associated with lung function deterioration with central obesity measures acting as key contributors.[5] Related metabolic clustering matters too meta-analytic evidence suggests metabolic syndrome is associated with poorer pulmonary function reinforces adiposity is not only "weight" but a systemic state.[6] At the same time body composition indices can behave differently across subgroups. In adult athletes, body composition parameters showed measurable relationships with spirometry reminding that muscle mass training status and body build can confound simple obesity lung interpretations.[7] But in older adults obesity and body composition associations with FEV1, FVC and their ratio are again present and often show that obstruction type patterns (lower FEV1/FVC) can coexist with relatively preserved volumes depending on phenotype.[8]

Given this background studying young adults with multiple adiposity indices alongside spirometry is useful for the data. This age group has fewer smoking and chronic disease confounders so the signal of body size, fat distribution and sex related lung growth can be seen cleaner. The present work therefore evaluates standard lung function parameters and a panel of adiposity indices and then tests independent predictors of PEFR while accounting for major physiological drivers like height and sex.

MATERIALS AND METHODS

This analytical crosssectional observational study was conducted at the GEIMS, Dehradun in a sample of young adults aged 17 -22 years. Institutional Ethics Committee approval was obtained before recruitment and written informed consent was taken from each participant prior to measurements. Data collection was done in a single visit, with anthropometry and pulmonary function testing performed on the same day using a structured proforma.

Apparently healthy young adults were included if they had no acute illness at the time of testing and were fit physically, mentally and socially to participate and agreed to undergo anthropometry and spirometry. Participants were excluded if they had a history or current diagnosis of bronchial asthma, chronic obstructive pulmonary disease, pulmonary tuberculosis, or any other acute or chronic respiratory illness. Those with current or past smoking history or alcohol consumption (as per screening history) were excluded. Additional exclusions included any condition likely to interfere with proper spirometry performance (example recent thoracic or abdominal surgery) and inability or unwillingness to follow spirometry instructions. A total of 150 individuals were assessed and 139 were included in the final analysis after basic data quality screening.

Age was recorded in completed years. Height was measured in centimetres using a calibrated stadiometer with the participant barefoot and standing upright and converted to metres for modelling. Weight was measured in kilograms using a digital weighing scale with light clothing and barefoot. Body mass index, Waist circumference and hip circumference were calculated with standard formula and measurements.[9,10] Adiposity indices including waist hip ratio (WHR), waist height ratio (WHtR), A Body Shape Index (ABSI), conicity index and body adiposity index (BAI) were computed using standard published equations.[9,10,11,12,13] Circumference units were standardised before deriving indices and conversions were applied where required based on the raw datasheet entry format.

Pulmonary function assessment included forced expiratory volume in 1 second (FEV1), forced vital capacity (FVC), FEV1/FVC ratio and peak expiratory flow rate (PEFR). Spirometry was performed using a Vyaire spirometry device with disposable mouthpieces following standard acceptability practices. Participants were seated comfortably, the procedure was explained and repeated forced expiratory manoeuvres were taken to obtain acceptable efforts with a strong start and satisfactory completion the best acceptable values were used for analysis. PEFR was additionally measured in L/min using a Mini-Wright peak flow meter with the participant standing upright; three blows were recorded and the highest value was retained.

All analyses were performed in R. Continuous variables were summarised as mean ± standard deviation and categorical variables as frequency and percentage. Associations between adiposity indices and lung function parameters were assessed using Spearman correlation coefficients with p values. Multivariable linear regression was fitted with PEFR as the dependent variable and BMI, WHtR, height (m), age (years) and sex as predictors; beta coefficients with 95% confidence intervals were reported. Model fit was summarised using R^2 and adjusted R^2, with standard residual diagnostics and collinearity checks performed as routine. A p value <0.05 was taken as statistically significant.

RESULTS

Table 1: Baseline characteristics of study participants

Variable	Overall Values
Number of participants	139
Female n (%)	90 (64.7)
Male n (%)	49 (35.3)
Age (years) Mean	19.1
Height (cm) Mean	166.1
Weight (kg) Mean	68.8

Table 1 shows the total 139 participants. Females were the majority 90 (64.7%), males 49 (35.3%). Age was of younger individuals overall with a mean of 19.1 years. Height and weight averages were 166.1 cm and 68.8 kg.

Table 2: Pulmonary function parameters

Parameter	Mean (SD)
FEV1 (L)	2.95 (0.84)
FVC (L)	3.20 (0.93)
FEV1/FVC (%)	92.6 (6.4)
PEFR	427.5 (110.6)

In Table 2 mean FEV1 was 2.95 L and FVC 3.20 L. The FEV1/FVC ratio was high around 92.6%, so most of the sample was non obstructive on average. PEFR had broader spread (427.5 with SD of 110.6).

Table 3: Distribution of adiposity indices

Adiposity index	Mean ± SD (Median)
BMI (kg /per m2)	24.67 ± 4.08 (24.48)
Waist circumference (cm)	83.12 ± 12.30 (81.28)
Hip circumference (cm)	97.42 ± 12.48 (96.52)
Waist hip ratio	0.855 ± 0.084 (0.857)
Waist height ratio	0.502 ± 0.070 (0.493)
ABSI	0.07641 ± 0.006 (0.0756)
Conicity index	1.194 ± 0.105 (1.177)
BAI	27.86 ± 6.43 (27.12)

Table 3 gives the central obesity picture rather than using BMI alone. Mean BMI 24.67 ± 4.08 with median 24.48. Waist and hip were 83.12 ±12.30 (median 81.28) and 97.42 ±12.48 (median 96.52). Ratios were quite stable mean close to median: WHR 0.855 ±0.084 median 0.857 and WHtR 0.502 ±0.070 median 0.493. ABSI and conicity were in expected band 0.07641 ±0.006 and 1.194 ±0.105, BAI mean 27.86 ±6.43.

Table 4: Multivariable linear regression for PEFR

Predictor	Beta	95 percent CI	p value
BMI	4.73	-0.44 to 9.91	0.073
Waist height ratio	-205	-509 to 99	0.184
Height (m)	337	160 to 515	<0.001
Age (years)	2.84	-9.30 to 15.0	0.645
Male sex	97.4	57.9 to 137	<0.001

Model performance:
R squared 0.543, adjusted R squared 0.526.
Height and sex are the strong drivers for PEFR. Height (m) had a positive beta 337 with CI 160 to 515 and p value below 0.001. Male sex also showed higher PEFR beta 97.4, CI 57.9 to 137, p value below 0.001. BMI and WHtR were not statistically significant here (BMI p 0.073, WHtR p 0.184) so once height and sex are in the model the adiposity associations become weaker. Model fit was decent, R squared 0.543 and adjusted 0.526, so about half the PEFR variability is explained which is ok for clinical field data.

Table 5: Spearman correlation between adiposity indices and lung function

Adiposity index	FEV1 rho (p)	FVC rho (p)	FEV1/FVC rho (p)	PEFR rho (p)
ABSI	0.037 (0.653)	0.068	-0.062 (0.454)	0.007
BAI	-0.308 (0.0001)	-0.352	-0.069 (0.404)	-0.319
BMI	0.084 (0.309)	0.206	-0.211 (0.010)	0.102
Conicity index	0.069 (0.403)	0.130	-0.137 (0.096)	0.052
Waist circumference	0.242 (0.003)	0.354	-0.185 (0.024)	0.220
Waist hip ratio	0.178 (0.030)	0.283	-0.075 (0.365)	0.164
Waist height ratio	-0.056 (0.501)	-0.013	-0.157 (0.057)	-0.070

Correlation pattern in Table 5 was mixed but a few points stood out. BAI had negative rho with FEV1 (-0.308, p 0.0001) and with PEFR (-0.319) also trending similarly so higher BAI tends to go with lower lung volumes and flow. Waist circumference correlated positively with volumes, FEV1 rho 0.242 (p 0.003) and FVC rho 0.354 and it also related to the ratio FEV1/FVC rho -0.185 (p 0.024) suggesting the pattern is more associated with mechanics and size effects. BMI had small positive rho with FVC and PEFR but a negative rho with ratio -0.211 (p 0.010). ABSI and conicity were basically weak and mostly non-significant in this dataset. WHR and WHtR were small correlations with inconsistent direction.

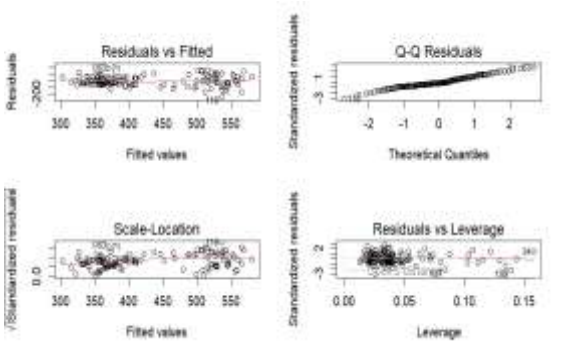

Figure 1: Regression diagnostic plots showing model fit and residual behaviour

DISCUSSION

In this young adult cohort (n=139, mean age 19.1 years, female 64.7%) group-level spirometry looked largely "non-obstructive" with a high mean FEV1/FVC (92.6%). PEFR showed wide scatter (SD of 110) and when we modelled PEFR the dominant independent drivers were height and male sex. Adiposity indices showed mixed crude associations. The most consistent adverse signal was with BAI, which correlated negatively with both FEV1 and PEFR, while waist circumference showed positive crude correlations with FEV1 and FVC but a negative correlation with the FEV1/FVC ratio.

Young adults are useful for adiposity lung stuides because major chronic confounders (smoking burden, COPD, long diabetes, long-standing metabolic syndrome) are usually minimal. So one often sees "body size with mechanics" more than irreversible airway remodelling. But the same advantage becomes a limitation too as exposure duration to adiposity is short and the respiratory system has high reserve. So effects can be subtle, non-linear and easier to miss after adjustment.

Large literature supports this non-linear with reserve idea. In NHANES analysis (n=7346) both underweight and obesity were associated with reduced FEV1 and FVC while overweight was not clearly different from normal weight and the BMI lung association was reversed U-shaped (not linear). Obesity also showed a slightly higher FEV1/FVC ratio consistent with a restrictive tendency rather than obstruction.[3]

Our mean FEV1 and FVC are reasonable for this age band and the high FEV1/FVC argues against population level obstruction. A high ratio can occur in normal youthful physiology but it can also happen when FVC is relatively "pulled down" more than FEV1 (a restrictive tendency). We cannot label restriction from spirometry alone without TLC or at least percent predicted with LLN based on reference equations. Obesity related reductions in FEV1 and FVC can coexist with preserved or slightly elevated FEV1/FVC, which clinically reads as restrictive-pattern mechanics more than classic airflow obstruction.[3,14]

Mean BMI 24.7 kg/m² places many participants at the normal overweight border not severe obesity. WHtR mean 0.50 is near the commonly used risk threshold. In this middle band the adiposity lung relationship is often weak or directionally inconsistent as body composition (lean mass against fat mass) and frame size start to matter more than "fat load" alone. This is exactly where crude correlations can mislead. NHANES data show that lung function deficits become clearer at the extremes (underweight and obesity), not necessarily in the borderline zone.[3]

BAI uses hip circumference and height. It tracks adiposity differently than BMI and it is less entangled with thoracic/abdominal cavity size than waist circumference.[13] In our data BAI showed consistent negative correlations with FEV1 and PEFR. That can be interpreted as higher adiposity (captured by hip-based measure) relates to lower flow/volume even in apparently healthy youth. Gluteofemoral size is a mix of subcutaneous fat, pelvic structure and gluteal muscle mass. So BAI may be acting as a proxy for overall mass and adiposity phenotype not "hip fat is harmful". In young adults hip circumference can rise with fat gain and with muscularity too. If muscularity were dominant we would expect better PEFR not worse as our direction is negative adiposity burden is the more possible driver. A longitudinal Taiwanese followup (n=9059 of 4 years) showed that higher values of several obesity indices including BAI were associated with a higher baseline FEV1/FVC but a more rapid decline in FEV1/FVC over follow-up (lower Δ FEV1/FVC). That pattern supports a concept of "baseline reserve but faster deterioration" in higher adiposity.[15] In our cross-sectional snapshot BAI's negative association with PEFR/FEV1 can be framed as an early functional signal that may precede larger volume losses.

Our waist circumference correlated positively with FEV1 and FVC yet negatively with the FEV1/FVC ratio. In young adults waist circumference is not "pure visceral fat". It also scales with overall body size, skeletal frame and sometimes athletic build. Bigger frame do mean larger lungs with higher absolute FEV1 and FVC. That is why crude correlations can flip direction before adjustment. A review in apparently healthy adults (26 studies of 68,024 participants) reported that >88% of studies

found abdominal obesity inversely associated with pulmonary function and proposed mechanisms were mechanical compression and obesity-induced airway inflammation.[4] So our "positive waist against higher volumes" is best explained as a size effect not that central fat improves lung function.

Our multivariable regression shows height and male sex are strong independent predictors of PEFR. That is expected that airway calibre, lung size and expiratory muscle strength scale strongly with height and sex. In a mean age-19 group, lungs are near peak growth, so structural anatomy gives a large respiratory reserve. This reserve can mask mild mechanical crowding effects of adiposity, especially when adiposity range is not extreme. Longitudinal evidence supports that adiposity effects become clearer when you examine decline over time not just cross-sectional baseline. In a Korean analysis increases in adiposity indices were associated with deterioration in lung function, fat mass index and waist/waist-hip metrics showed inverse associations with FVC and FEV1 with stronger decline patterns in men.[5] This supports our narrative that in youth, height and sex dominate baseline PEFR while adiposity may show its harm more as trajectory later.

Our BMI beta for PEFR is positive and near-significant (p=0.073). This is a classic borderline-zone finding. In young adults BMI can reflect lean mass and fitness as much as fat, depending on the cohort. If a subset has higher BMI due to muscularity PEFR can rise due to stronger expiratory muscle effort and larger chest dimensions. Meanwhile true adiposity-related restriction tends to appear after a threshold of fat load or longer exposure duration, which may not be fully present at ages 19. The non-linear NHANES pattern (reversed U-shape) is aligned with this, where the strongest reductions are at the extremes, not in the mid-range.[3] The BMI PEFR association may reflect mixed body composition in this age group and BMI partly captures lean mass and frame size.

Mechanical pathway like adipose deposition around abdomen or chest wall reduces chest wall compliance and limits diaphragm excursion. This reduces lung volumes and can reduce expiratory flows indirectly through lower operating lung volumes and airway closure tendency at low volumes. A longitudinal biobank study discussion describes fat accumulation limiting diaphragm displacement and decreasing thoracic compliance, with downstream muscle inefficiency and weaker respiratory muscles over time.[15]

Inflammatory pathway with adipose tissue produces low grade systemic inflammation that can influence airway reactivity and subclinical airway inflammation. NHANES analysis noted that the obesity lung association increases after additional adjustment for insulin resistance or CRP and obesity-related lower FEV1/FVC patterns were most evident in higher inflammation/insulin resistance strata. That supports inflammatory mediation rather than "fat mass alone".[3] In obesity asthma literature IL-6 is repeatedly discussed as part of the inflammatory signalling axis relevant to obese phenotypes.[16] For our data we did not measure CRP or IL-6 so mechanism should be stated as plausible not proven. BAI may better reflect adiposity load with less thoracic size confounding than waist circumference hence it correlates negatively with lung outcomes even when waist shows size-driven positive crude associations.

PEFR is effort dependent. A wide SD like ours is common in field studies particularly if coaching and repeatability criteria vary slightly between participants. We acknowledge that some of the unexplained variance in PEFR likely reflects manoeuvre technique and transient factors (motivation, learning effect, recent activity) beyond pure anthropometry. Current spirometry standardisation documents emphasise acceptability and repeatability criteria and the need for best-of-three reproducible efforts.[17] Our outcomes are raw values rather than percent-predicted or LLN interpretation is focused on internal association patterns, not diagnostic classification. Multiple adiposity indices were assessed not only BMI. Using both correlation and multivariable modelling helps show how crude associations can change after accounting for body size. The young adult age band reduces major chronic confounding, making the "early physiology" question clearer.

Cross-sectional design cannot infer causality or progression. Sample size is moderate and sex distribution is female predominant, which may influence PEFR distribution and generalisability. PEFR is effort dependent so residual variability is expected. We did not include inflammatory or metabolic biomarkers (CRP, insulin resistance markers), physical activity, or body composition measures (fat mass vs lean mass), so mechanistic inference is indirect. Also raw spirometry values were used without LLN/percent-predicted, restrictive patterns cannot be confirmed.

For young adults, structural size (height, sex) remains the major determinant of PEFR. The consistent negative association of BAI with FEV1 and PEFR suggests adiposity may already exert subtle functional effects before overt obstruction appears. The most valuable next step is adding percent-predicted spirometry with LLN, body composition and a minimal biomarker panel (CRP ± fasting insulin/HOMA-IR). Literature suggests obesity-related lung impairment becomes clearer in strata with higher metabolic inflammation and across follow-up time. A followup design in the same age band would test whether BAI or WHtR predicts early decline signals similar to longitudinal biobank patterns.

CONCLUSION

This young adult cohort showed a high mean FEV1/FVC, so obstruction is unlikely at group level.

PEFR variation was large and was mainly explained by height and male sex, showing lung scaling dominates in this age band. BAI had a consistent negative relationship with FEV1 and PEFR, suggesting adiposity load may already affect function even when values look “normal”. Waist measures showed mixed crude correlations likely reflecting body size and thoracic build more than pure fat effect. ABSI and conicity index did not perform well here, possibly due to limited adiposity range and modest sample size.

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