BMC Pediatrics

RESEARCH Open Access

The relationship between child overweight/obesity and respiratory muscle strength and lung function considering sex, age, and maturity offset status

Júlio Brugnara Mello[1*], Marcos Filipe da Silva Mello[2], Iransé Oliveira-Silva[2], Vitória Milhomem Mourão[2], Lorena Alves Vieira[2], Maria Sebastiana Silva[3], Pedro Henrique de Almeida Silva[2] and Viviane Soares[2,4]

Abstract

Background Rates of overweight and obesity in children are increasing progressively worldwide, which can negatively impact chest mechanics and lung function. However, children at different stages of growth may be impacted differently by obesity, highlighting the need to assess maturation status related to body growth, as the maturity offset.

Objective To investigate the effect of body mass index (BMI) on respiratory muscle strength (RMS) and lung function in children, considering maturity offset status as a covariate.

Methods This cross-sectional analytical study included 90 children aged 5–14 from two Brazilian public schools. BMI was calculated and converted into percentiles for age and sex to classify children as overweight/obese. RMS, including maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP), was measured through a digital manometer, and spirometry was used to assess lung function. The age-to-peak height velocity (APHV) formula was used to determine the maturity offset status. Data were compared between groups, and linear regression was used to examine the effect of BMI on RMS and spirometric variables, adjusted for sex, age, and maturity offset status.

Results MIP ($\Delta = +14.12$ cmH_2O, $p = 0.015$) was higher in overweight/obese children. Regression models indicated a relationship between %MEP (β: 0.142; CI 95%: -1.163; 1.453) and BMI, considering age, sex, and maturity offset status as covariates.

Conclusion Overweight/obesity was associated with higher inspiratory muscle strength and related to %MEP. This relationship was influenced by the covariates of age, sex, and maturity compensation status, indicating that anthropometric variables need to be considered in future studies.

Keywords Body composition, Pulmonary ventilation, Muscle strength, Child development, Adiposity

*Correspondence:
Júlio Brugnara Mello
julio.mello@pucv.cl
[1]School of Physical Education, Pontificia Universidad Católica de Valparaíso, Valparaíso, Chile
[2]Postgraduate Program in Human Movement and Rehabilitation, Evangelical University of Goiás – UniEVANGÉLICA, Anápolis, Brazil
[3]Physiology, Nutrition and Health Laboratory, Federal University of Goiás – UFG, Goiânia, Brazil
[4]Laboratory of Studies in Cardiorespiratory and Metabolic Health. Evangelical, University of Goiás – UniEVANGÉLICA, Anápolis, Brazil

Introduction

Childhood is a period of growth, pubertal development, neuropsychomotor progress, and emotional and social construction [1]. Nutritional and maturity development relies on an ecological model influenced by various factors at different levels [2]. Several of these interconnected factors can lead to excessive fat accumulation in children, resulting in pediatric obesity [1, 3]. Recent global analyses [4] have demonstrated significant shifts in the prevalence of underweight and obesity over the past three decades, with considerable increases in obesity rates across all age groups, especially in children and adolescents. These trends highlight substantial regional variations, with some areas experiencing sharper increases, underscoring the growing public health challenge [4]. This shift is consistent with the observed doubling of pediatric obesity rates in Brazil between 2000 and 2024 [5, 6].

The rise in pediatric obesity rates has triggered a global burden of early-onset comorbidities such as t-2 diabetes, cardiovascular diseases, mental disorders (including anxiety and depression), and respiratory diseases [3]. Theoretically, obese children naturally experience impaired diaphragmatic movement due to increased stiffness and the burden of excess body mass, which can limit lung expansion and affect the entire respiratory cycle [7]. Additionally, in children with overweight or obesity, a reduction in functional residual capacity may predispose the lungs to alveolar instability or even atelectasis. However, compensatory mechanisms—such as increased diaphragmatic effort—support ventilation. The decreased lung volume, combined with altered chest wall compliance and mechanical loading, contributes to an increased respiratory workload [8]. Regarding respiratory muscle strength (RMS), there is no consensus; however, previous studies suggest no significant differences [9, 10].

As for lung function, obese children have higher airway resistance (both central and peripheral), but lung and thoracic cage compliance are not substantially altered [11]. Moreover, the phenomenon of dysanapsis suggests that obese children experience a disproportionate growth between airways (specifically their caliber) and lungs [12, 13]. Spirometric parameters, including forced vital capacity (FVC) and forced expiratory volume in one second (FEV1), tend to be higher, while the FEV1/FVC ratio is reduced in obese children, although these data are inconclusive [14–16].

Part of this lack of understanding about the effect of obesity on lung function in children may be due to the constant growth of the organs due to the maturation process. We hypothesize that maturity offset, which refers to the estimated time before or after peak height velocity (PVH) during adolescence, may play a significant role in mediating the effects of obesity on lung function and respiratory force in children [17, 18] but to date, the moment there are no studies with include this outcome in the analyses. We know that during critical periods of growth and development, particularly around PVH, physiological changes are pronounced, and the body undergoes rapid growth spurts [19]. Obesity during this period may exacerbate or attenuate these changes, depending on the maturity offset [20]. Children who are pre-PHV or in the early stages of maturity offset may experience more pronounced adverse effects of obesity on lung function due to the combination of increased adiposity and continued development of respiratory structures [16]. Additional weight may impose mechanical constraints on the chest wall and diaphragm, reducing lung volumes and respiratory force [21]. Conversely, those who are post-PHV may have developed more robust compensatory mechanisms, potentially buffering some of the negative impacts of obesity [16]. In this sense, we believe that including analyses on maturity offset is a path that can help health professionals who work directly with children with obesity in their practice. In this context, the objective of this study was to investigate the effect of body mass index (BMI) on RMS and lung function in children, considering maturity offset status as a covariate.

Methods

Design and ethical aspects

This is an analytical cross-sectional study, approved by the Research Ethics Committee of the Evangelical University of Goiás under protocol number 5.971.901, following the guidelines of Resolution 466/12 of the National Health Council and the Declaration of Helsinki. All parents/guardians signed the Informed Consent Form, and the children provided assent. The invitation to participate was sent through the child's school agenda along with the Informed Consent Form. Additionally, an explanatory video was shared in a parents' group, providing information about the data collection days.

Participants

The study included children and adolescents aged 5–14 years who were regularly enrolled in two public schools in a Brazilian city. Children with acute respiratory diseases (such as flu or asthma exacerbation), chronic respiratory or cardiac diseases (congenital), and cognitive impairments were excluded from the data analysis. The sample size calculation was based on the statistical test to be conducted (multiple linear regression – one predictor and three adjustment variables), an effect size (f^2) of 0.15, a statistical power of 80%, and a significance level of 5%, requiring a sample of 85 children. Recruitment was conducted via an invitation sent in the child's school agenda.

Procedures

A form was filled out with information on gender, school grade, age, presence of comorbidities, and use of continuous medication. In this study, the procedures suggested by Projeto Esporte Brasil [22] for anthropometric assessment were adopted. The children undertook the measurements wearing light clothes (e.g. physical education class clothes) and without shoes. For the measurement of body mass, we used a portable scale with a resolution of 100 g (Welmy, LED 200 kg model, São Paulo, Brazil). During the assessment, the children and adolescents remained standing with their elbows extended and close to the body. The measurement was recorded in kilograms to one decimal place. For the measurement of height, we used a portable stadiometer (Sanny®, São Paulo, Brazil) with a resolution of 2 mm. To read the height, the same evaluator made all assessments. Height measurement was recorded in centimetres to two decimal places and converted to meters for later analysis.

Body Mass Index (BMI) was calculated by dividing body mass by height squared (kg/m^2), with children classified as eutrophic if their BMI percentile was < 85 and overweight if their BMI percentile was ≥ 85 [6].

RMS was measured using a digital manometer (MVD-300, Globalmed, Porto Alegre, Brazil). To estimate RMS, static pressures were used: maximum inspiratory pressure (MIP) and maximum expiratory pressure (MEP), obtained from the child's residual volume and total lung capacity, respectively [23]. The manoeuvres were performed with the children seated and attached to a nose clip. Three to seven acceptable manoeuvres were performed without air escaping around the lips, without coughing, and with at least one second of maintenance of maximum effort [24]. The highest value between manoeuvres was chosen if the variation was < 10%. Five children in the sample did not meet the reproducibility criteria and therefore their data were excluded from the study. Reference values were calculated using the equations proposed by Wilson et al. for 7–17-year-olds [25]. The RMS of children aged 5–6 years (n = 4) was not analyzed.

Spirometry was performed using a portable device (MIR, MiniSpir, Rome, Italy) and disposable mouthpieces specifically designed for children, according to the specifications of the European Respiratory Society and the American Thoracic Society [26]. The child performed the test seated, with the head in a neutral and fixed position, attached to a nose clip. The following markers, using the predicted values, were analyzed: FVC, FEV_1, FEV_1/FVC; forced expiratory flow (FEF) between 25 and 75% of the manoeuvre and total expiratory time. The predicted values were derived from the Global Lung Function Initiative [26].

The age-to-peak height velocity (APHV) formula was used to assess maturity offset status, which relates to the child's rate of biological development. Although there are no validated equations for the Brazilian population, the equations proposed by Moore et al. [27] were used:

$$\text{. For girls, } \mathrm{MO} = -7.709133 + 0.0042232 \cdot \mathrm{CA} \cdot \text{height}$$
$$\text{. For boys, } \mathrm{MO} = -7.999994 + 0.0036124 \cdot \mathrm{CA} \cdot \text{height}$$
$$\text{(Height in cm and CA = chronological age)}.$$

Data analysis

Descriptive analysis was presented as mean, standard deviation, frequencies, and percentages. Data normality was assessed using the Kolmogorov-Smirnov (for total sample distribution) and Shapiro-Wilk tests (for group distribution, for example, overweight/obesity and normal weight). Continuous variables were compared between normal weight and overweight/obesity groups using the Student's t-test for independent samples, and the Cohen´s d effect size was calculated. We used a commonly used interpretation to refer to effect sizes as small (d = 0.2), medium (d = 0.5), and large (d = 0.8) based on benchmarks suggested by Cohen [28]. The categorical variable (percentage of comorbidities) was tested using the Chi-square test. Multiple linear regression models were constructed with BMI as the independent variable, respiratory function as the dependent variable, and adjustments for age, sex, and maturity offset status. Regression coefficients (β) and confidence intervals (95% CI) were calculated. An alpha value of < 0.05 was considered significant for all analyses. The effect size was calculated using Cohen's f^2 for the regression models. The collinearity between covariates was tested using the Variance Inflation Factor. Data analysis was performed using the Statistical Package for the Social Sciences (SPSS, IBM, version 23.0, Armonk, NY, USA).

Results

All data showed normal distribution, presenting all variables as means and standard deviations. Table 1 describes the children's characteristics. A total of 85 children were evaluated (49% female). Male children were further from reaching maturity offset status (Δ = −1.12 years), and this was the only significant difference in the anthropometric variables. Regarding the use of continuous medication, boys used medicines for attention deficit (1), anxiolytics (1), and antihistamines (1).

Table 2 shows the raw and predictive values for RMS and the predictive values for forced expiratory maneuver. The MIP presents a significant difference between the groups ($p < 0.05$) with a medium effect size (d = 0.58). The results for all predictive values demonstrate that it is not possible to identify a significant difference between the variances of the normal weight and overweight/obese

Table 1 Participants' characterization ($n = 85$)

Variables	Total ($n = 85$)	Male ($n = 43$)	Female ($n = 42$)	*p*-value
	x̄±SD	x̄±SD	x̄±SD	
Age (years)	9.23 ± 1.50	9.81 ± 1.41	9.64 ± 1.60	0.538
Body weight (kg)	37.16 ± 10.53	39.08 ± 11.16	36.54 ± 10.51	0.267
Height (m)	1.41 ± 0.12	1.42 ± 0.23	1.42 ± 0.56	0.870
BMI (kg/m^2)	18.35 ± 4.37	19.14 ± 4.65	17.76 ± 3.78	0.191
MO (APHV)	−2.63 ± 1.22	−3.13 ± 1.04	−2.12 ± 1.20	**< 0.001**
Comorbidities	**n (%)**	**n (%)**	**n (%)**	
No comorbidities	69 (79.6)	32 (74.3)	35 (84.8)	
Anxiety symptoms	11 (12.6)	07 (16.1)	04 (8.7)	
Gastrointestinal disorders	01 (2.2)	01 (2.1)	02 (4.3)	0.574
Others	04 (5.6)	03 (7.5)	01 (2.2)	

BMI Body Mass Index, *Kg* kilograms, *m* meters, *MO* maturity offset, *APHV* Age-to-Peak Height Velocity

Boldface are significant values

groups for all outcomes ($p > 0.05$ for all outcomes). However, the difference between groups for %MEP %FEC, and % $FEF_{25-75\%}$ has a small effect size, indicating a need to investigate with covariates.

The collinearity analysis indicated that the regression models could include sex, age, and maturity offset status (VIF < 10). In this sense, Table 3 shows the results of the crude (bivariate) and multiple regression analyses (each outcome analyzed in a model adjusted for sex, age, and maturity offset). The crude analyses demonstrated that not all variables are directly associated with BMI. When we tested the adjusted models, the %MEP is associated with BMI (β: 1.120; 95% CI: 0.042; 2.202; $p = 0.041$), showing a positive but weak relationship.

Discussion

Children with overweight/obesity had higher MIP, and linear regression models showed a relationship between BMI and %MEP when models were adjusted for sex, age,

Table 2 Comparison of respiratory muscle strength and pulmonary function variables by BMI classification ($n = 85$)

	Nutritional Status				
	Total ($n = 81$)	**Normal weight ($n = 68$)**	**Overweight/Obesity ($n = 13$)**	**Cohen´s d Effect size**	***p*-value**
Respiratory Muscle Strength**					
MIP (cmH_2O)	66.83 ± 18.64	63.74 ± 16.69	77.92 ± 20.17	0.58	**0.014**
%MIP	98.72 ± 25.73	98.82 ± 24.67	101.00 ± 26.97	0.08	0.156
MEP (cmH_2O)	71.90 ± 18.06	70.68 ± 17.55	78.29 ± 20.21	0.39	0.132
% MEP	93.14 ± 25.64	91.52 ± 23.58	101.16 ± 28.46	0.35	0.256
	Total (n = 85)	**Normal weight (n = 71)**	**Overweight/Obesity (n = 14)**	**Cohen´s d Effect size**	**p-value**
Forced Expiratory Maneuver					
%FVC	95.86 ± 9.79	95.45 ± 9.74	97.65 ± 9.65	0.21	0.467
$\%FEV_1$	94.90 ± 10.47	94.67 ± 10.63	96.26 ± 8.34	0.16	0.610
% FEV_1/FVC	98.60 ± 6.79	98.54 ± 7.17	98.17 ± 5.56	0.05	0.835
% $FEF_{25-75\%}$	74.78 ± 17.80	75.48 ± 18.80	71.94 ± 11.66	0.21	0.506

MIP Maximum Inspiratory Pressure, *MEP* Maximum Expiratory Pressure, *PEF* Peak Expiratory Flow, *FVC* Forced Vital Capacity, FEV_1 Expiratory Volume in the First Second, $FEF_{25-75\%}$ Forced Expiratory Flow at Mid-Range, *FET* Forced Expiratory Time, Data presented as mean ± standard deviation

Boldface are significant values

** Data analyzed for children between 7–14 years old ($n = 81$)

Table 3 Multiple linear regression models of respiratory variables (Dependent variables) with BMI measures (Independent Variable) ($n = 81$)

	BMI (independent outcome)			
	β (CI 95%)*	***p*-value***	**β (CI 95%)****	**Adjusted *p*-value****
% MIP	0.007 (−1.262; 1.278)	0.991	0.142 (−1.163; 1.453)	0.826
% MEP	0.903 (−0.330; 2.137)	0.148	1.120 (0.042; 2.202)	**0.041**
%FVC	0.263 (−0.207; 0.733)	0.270	0.308 (−0.175; 0.793)	0.208
$\%FEV_1$	0.307 (−0.194; 0.810)	0.227	0.269 (−0.247; 0.785)	0.306
% FEV_1/FVC	0.041 (−0.292; 0.375)	0.804	−0.041 (−0.382; 0.303)	0.819
% $FEF_{25-75\%}$	−0.050 (−0.913; 0.812)	0.908	−0.372 (−1.204; 0.460)	0.380

MIP Maximum Inspiratory Pressure, *MEP* Maximum Expiratory Pressure, *PEF* Peak Expiratory Flow, *FVC* Forced Vital Capacity, FEV_1 Expiratory Volume in the First Second, $FEF_{25-75\%}$ Forced Expiratory Flow at Mid-Range

Boldface are significant values

*Crude analysis in the regression models

** adjusted model by age, sex, and maturity offset status

and maturity offset status of the children. It is worth noting that boys are further from reaching maturity offset when assessed by height.

RMS is crucial in investigating respiratory insufficiencies and whether certain comorbidities alter muscle fiber recruitment patterns and, consequently, strength for generating maximum respiratory volumes [29]. In children with elevated BMI, lower MIP and MEP are expected, but there is no consensus in the literature about the reduction and the structural and functional mechanisms involved [9, 10]. In the present study, MIP was higher in children with overweight/obesity. For MIP, the predicted values are based on the child's body mass, whereas MEP takes into account the child's age. Therefore, the values would be higher in overweight/obese children. In fact, this is what the results showed and was a limitation of the study. Furthermore, multiple linear regression showed an association between BMI and %MEP. Studies with children of the same age range did not show differences in raw values or predicted percentages between normal weight and overweight/obese children [9, 10, 30], although one study indicated a negative correlation (r= −0.34) between BMI and %MEP [9].

Obesity may impair diaphragmatic movement in terms of flattening during inspiration due to increased thoracic load and the need for greater force to push abdominal contents downward and forward [31]. In obese children, the RMS may be greater than in normal-weight children [32]. However, these changes in the inspiratory phase may not occur. This paradox could be explained by greater thoracic compliance (with ribs not yet consolidated), which enhances "bucket handle" movement and muscle fiber adaptations ("training") due to increased load, leading to maintained or even increased RMS.

Another aspect to consider is that measurements of MIP and MEP depend not only on RMS but also on the lung volume at which measurements were taken and the corresponding value of elastic recoil pressure of the respiratory system [33]. This elastic recoil pressure results from the sum of the elastic recoil pressures of the lungs and thorax [34]. Some body composition measures are also related to RMS. Dassios et al. [35] showed that muscle strength is directly related to height in healthy children. However, Costa Júnior et al. [10] showed that body fat percentage and lean mass are not predictors.

In the present study, the pulmonary function variables of children did not differ. Recent literature indicates an increase in FVC and FEV_1 parameters in obese children due to the phenomenon of dysanapsis, which refers to the imbalance between the rapid growth of airway length and lung volume and a slower rate of increase in airway cross-sectional area [13]. Our results suggest that obese children did not exhibit this phenomenon, as it is influenced by sex and body composition, and when compared by height (data not shown), they did not show differences in somatic maturity since it depends on the child's age and height [27].

Regarding the covariates, the age, sex, and maturity offset status make the association between BMI and %MEP significant statically, suggesting that differences between sexes, age, and the approach to peak height velocity influence the relationship between BMI and RMS´s expiratory component. In addition, our results showed that girls were closer to reaching the APHV than boys, and there is evidence that lung volumes in girls increase until menarche, while in boys, they continue through puberty until reaching adult height [36]. Further studies are needed to consider height to determine if there are impacts on FVC and FEV_1 as well as other pulmonary function parameters and RMS.

Strengths and limitations

Among the strengths of this study is the use of somatic maturity through height to assess its influence on respiratory function, as body composition and height affect muscle strength and pulmonary function in children. It is worth noting that studies solely involving obese children are fewer in number, making it difficult to establish consensus and identify the main mechanisms through which changes occur.

Regarding the limitations encountered during this study, it is worth noting the use of BMI as a marker for overweight/obesity, as it is very nonspecific and does not indicate body fat reserve. However, it is the marker most closely related to gold-standard obesity assessment methods. The challenge in maintaining child concentration and effective verbal command to achieve the recommended maneuver time is also a limitation. As a cross-sectional study, it does not establish causation but helps detect which variables may be influenced by BMI. Lastly, the lack of studies evaluating RMS in obese children limited the discussion.

Conclusion

This study examined the relationship between respiratory muscle strength and pulmonary function indicators in children across different body mass index classifications, accounting for maturity offset as a covariate. Children with overweight or obesity demonstrated higher MIP, with a moderate effect size, suggesting potential adaptations in inspiratory muscle performance.

Although no significant differences were found in predictive pulmonary values between normal-weight and overweight/obese groups, small effect sizes for select variables highlight trends worth further exploration. Notably, after adjusting for sex, age, and maturity offset, BMI was positively but weakly associated with %MEP. These findings suggest that increased body mass

may contribute to compensatory increases in expiratory muscle activity, even in the absence of overt pulmonary dysfunction. Future longitudinal studies are warranted to investigate the trajectory and clinical relevance of these adaptations, considering both physiological and behavioral factors.

Abbreviations

BMI	Body mass index
RMS	Respiratory muscle strength
MIP	Maximal inspiratory pressure
MEP	Maximal expiratory pressure
APHV	Age-to-peak height velocity
RMS	Respiratory muscle strength
FVC	Forced vital capacity
FEV1	Forced expiratory volume in one second
PVH	Peak height velocity
FEF	Forced expiratory flow
95% CI	Confidence intervals

Acknowledgements
We would like to thank the participating schools for facilitating access and development of the research, all parents and guardians for authorizing their children's participation in the study, and the participants who collaborated at all stages of the research.

Authors' contributions
JBM designed and performed the main analysis, interpreted the results, and drafted and revised the manuscript. MFM and IOS contributed to the conceptualization and methodology of the study, collected the data, and contributed to the writing of the manuscript. VMM, LAV, MSS, and PHA contributed to the conceptualization of the study and contributed to the writing of the manuscript. VS interpreted the results, supervised the writing of the manuscript, and coordinated the project. All authors read and approved the final manuscript.

Funding
No external funding was received for this study.

Data availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This research was approved by the Research Ethics Committee of the Evangelical University of Goiás under protocol number 5.971.901, following the guidelines of Resolution 466/12 of the National Health Council and the Declaration of Helsinki.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Received: 16 October 2024 / Accepted: 22 September 2025
Published online: 29 October 2025

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