ORIGINAL ARTICLE

Relationship between degree of central adiposity, inflammatory status and risk of sarcopenia in obese children

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ABSTRACT

Introduction: Childhood obesity is a most prevalent condition worldwide. The accumulation of adipose leads to its infiltration into various organs, including skeletal muscle, which called sarcopenia. Children with sarcopenic-obesity are at an increased risk of developing cardiovascular or metabolic diseases. This study aims to investigate the association between central adipose accumulation and the degree of chronic inflammation on muscle performance in obese children, a topic that has not been studied in Indonesia.

Materials and Methods: This study employed a cross-sectional design with consecutive sampling involving obese primary school children aged 7–13 years from Semarang and its surrounding areas who met the inclusion and exclusion criteria. Data collection included anthropometric measurements (waist and hip circumferences, waist-to-hip ratio, muscle mass assessed by bioelectrical impedance analysis/BIA), handgrip strength, three-day dietary recall, and blood biomarkers (CRP and IL-6). Muscle mass was classified as low when skeletal muscle mass adjusted for age and sex (SMMa z-score) was less than −2 SD. Reduced muscle strength was defined as handgrip strength below the 15th percentile (age/sex-specific). Central adiposity was defined as waist-to-height ratio (WHtR) ≥ 0.50 or waist-to-hip ratio (WHR) above the 90th percentile for age and sex. Elevated inflammatory markers were defined as CRP ≥ 3 mg/L and IL-6 > 5 pg/mL. Associations between variables were analyzed using chi-square or Fisher's exact tests, and logistic regression models were applied with a significance level set at α=0.05.

Results: Of the 86 children, 84.8% had low muscle mass, 38.4% had reduced muscle strength, and 69.8% demonstrated impaired physical performance. High waist-to-height ratio (WHtR) was significantly associated with low muscle mass (p = 0.016) and reduced muscle strength (p = 0.007). High C-reactive protein (CRP) levels were also associated with low muscle mass (p = 0.013). In addition, protein intake was significantly related to muscle mass (p = 0.016). In logistic regression analysis, WHtR was an independent predictor of both reduced strength (OR = 5.324, p = 0.021, 95% CI: 1.280–22.148) and low muscle mass (OR = 0.163, p = 0.011, 95% CI: 0.037–0.714). Although interleukin-6 (IL-6) was elevated in the majority of children, it did not show a significant association with sarcopenia outcomes.

Conclusion: Central adiposity, particularly as measured by WHtR, is a key predictor of sarcopenia in obese children, with significant associations observed with reduced muscle mass and strength. Elevated CRP levels further underscore the role of systemic inflammation in sarcopenia. These findings highlight the need for early identification and targeted interventions to mitigate the adverse effects of sarcopenic obesity in children.

KEYWORDS:
Central adiposity, children, obese, sarcopenia, WHtR

INTRODUCTION

Obesity is a clinical condition that arises from an imbalance between high energy intake and low energy expenditure, leading to high fat accumulation in body tissues. Obesity is a significant public health concern due to its increasing prevalence worldwide, particularly among children and adolescents.[1] In 2018, the prevalence of overweight and obesity in Indonesian children aged 5–12 years and adolescents aged 13–15 years was reported at 20% and 16%, respectively.[2] One of the critical aspects of obesity is the fat distribution pattern in the body, which can be generalized or central. Central obesity is characterized by fat accumulation in the abdominal region and is commonly assessed through anthropometric measurements such as waist circumference (WC), waist-to-hip ratio (WHR), or waist-to-height ratio (WHtR). Unlike generalized obesity, central obesity is strongly associated with a range of metabolic disorders, primarily due to the unique properties of visceral fat.[3,4] Adipose tissue, particularly visceral fat, is now recognized as a dynamic endocrine and metabolic organ that secretes various bioactive molecules, including adipokines and inflammatory mediators. These metabolic signals originating from adipose cells contribute to chronic low-grade inflammation, a hallmark of obesity, which plays a pivotal role in the pathogenesis of metabolic and cardiovascular complications. Central obesity has far-reaching health implications, significantly increasing the risk of chronic diseases such as type 2 diabetes mellitus, hypertension, dyslipidemia, and cardiovascular diseases. The inflammatory state induced by visceral fat not only affects metabolic homeostasis but also exacerbates insulin resistance, impairs vascular function, and promotes atherogenesis. Additionally, the interplay between adiposity and inflammation extends beyond metabolic complications, influencing musculoskeletal health.[4]

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Sarcopenia, a condition characterized by the progressive loss of skeletal muscle mass, strength, and function, is an emerging concern in the context of obesity. Sarcopenic obesity is a phenotype wherein individuals exhibit both high fat mass and low muscle mass, reflecting a complex interaction between excessive adiposity and muscle degeneration.[5] Although the precise mechanisms underlying sarcopenic obesity remain under investigation, it is hypothesized that chronic inflammation driven by visceral fat plays a central role. Inflammatory mediators such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α), secreted by adipose tissue, contribute to insulin resistance and disrupt anabolic signaling pathways in skeletal muscle. These disruptions lead to impaired protein synthesis, increased protein degradation, and ultimately muscle atrophy and weakness.[6]

The implications of sarcopenic obesity are profound, particularly in children, as the combination of high fat mass and reduced muscle strength may compromise physical performance, increase the risk of falls, and contribute to a cycle of decreased physical activity and further adiposity.[7] Despite the growing recognition of sarcopenic obesity as a significant clinical concern, most research to date has focused on adult populations, leaving a critical knowledge gap regarding its prevalence, pathophysiology, and implications in pediatric populations.

In children, the interplay between central adiposity, inflammation, and sarcopenia is particularly complex and underexplored. Obese children with central adiposity may be at heightened risk of developing systemic inflammation, which could have detrimental effects on skeletal muscle growth and development during critical periods. Moreover, the long-term consequences of sarcopenic obesity in childhood, including the potential progression to chronic diseases and functional impairments in adulthood, remain poorly understood.[8] Given the limited research in this area, particularly in pediatric populations, it is crucial to elucidate the relationship between central adiposity, inflammatory markers, and the risk of sarcopenia in obese children.

This study aims to fill this knowledge gap by investigating the degree of central adiposity, markers of inflammatory status, and their association with the risk of sarcopenia in obese children. Understanding these relationships will not only advance the scientific understanding of sarcopenic obesity in pediatric populations but also inform the development of targeted interventions to mitigate its adverse health consequences.

MATERIALS AND METHODS

This cross-sectional study was conducted in public elementary schools within Semarang City and surrounding districts, with venous blood sample analyses performed at the GAKI Laboratory, Faculty of Medicine, Universitas Diponegoro, under internal quality-control procedures. Recruitment and data collection were coordinated with school administrations and carried out during school hours by a trained pediatric research team.

Consecutive sampling was applied to identify eligible participants. The study enrolled children aged 7–13 years, with both sexes eligible; girls were restricted to pre-menarcheal status to minimize maturational confounding. Obesity was defined as a BMI-for-age z-score greater than +2 SD based on WHO BMI-for-age reference standards for 5–19 years. Children classified as overweight only (z-score > +1 to ≤ +2 SD) were not included. Exclusion criteria were syndromic or genetic obesity diagnosed clinically, use of systemic corticosteroids, cytostatics, or other drugs affecting body weight within the past three months, participation in structured weight management programs, known chronic liver disease, or conditions that interfered with anthropometric or functional assessments. On the day of phlebotomy, children were screened for acute illness, including fever or signs of intercurrent infection; blood sampling was postponed when present. In total, 86 children met all eligibility criteria and completed the full protocol.

The study protocol was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Diponegoro, and Dr. Kariadi General Hospital (No. 303/EC/KEPK/FK-UNDIP/VIII/2021). Parents/guardians received written study information sheets, and both parental informed consent and child assent were obtained before participation.

All measurements followed standardized procedures conducted by trained staff. Weight and body composition were measured with a Tanita BC 545N body composition analyzer. Height was measured to the nearest 0.1 cm using a stadiometer, with participants barefoot, heels together, and the head positioned in the Frankfort plane. Waist circumference was measured at the midpoint between the lowest rib and the iliac crest at end-expiration with a non-stretch tape. Hip circumference was measured at the maximal buttock protuberance with the tape horizontal and non-compressive.

Central adiposity was evaluated using two indices. Waist-to-height ratio (WHtR = waist [cm] / height [cm]) was classified as high when ≥0.50, an established cut-off that is age- and sex-independent in pediatric populations. Waist-to-hip ratio (WHR = waist [cm] / hip [cm]) was categorized as high when at or above the 90th percentile for age and sex. Since there is currently no universally accepted WHR cut-off for children, as noted in our reference studies, we applied a percentile-based approach consistent with the methodology used for waist circumference.[9]

Muscle mass was measured using a TANITA body composition scale at the same time as body weight assessment. The measurement was based on the bioelectrical impedance analysis (BIA) method. Muscle mass values were then categorized into low muscle mass and normal muscle mass according to sex- and age-specific Skeletal Muscle Mass (SMMa) reference curves. Low muscle mass was defined < –2 SD, while normal muscle mass was defined ≥–2 SD.[10]

Maximal isometric handgrip strength was assessed with a handheld dynamometer. Participants held the device in the dominant hand, elbow flexed at 90°, and squeezed maximally for 3–5 seconds. Three trials were performed with

Table I: Demographic, Clinical Characteristics, and Sarcopenia Prevalence in Obese Children

Variable	Category	Frequency (n)	Percentage (%)
Demography			
Age	7 years	18	20.9
	8 years	6	7.0
	9 years	9	10.5
	10 years	26	30.2
	11 years	14	16.3
	12 years	12	14.0
	13 years	1	1.2
Gender	Male	52	60.5
	Female	34	39.5
Central Adiposity			
WHR	Normal	55	64.0
	High	31	36.0
WHtR	Normal	12	14.0
	High	74	86.0
Inflammatory Marker			
IL-6	Normal	2	2.3
	High	84	97.7
CRP	Low	1	1.2
	Normal	69	80.2
	High	16	18.6
Sarcopenia Criteria			
Muscle Mass	Low Muscle Mass	73	84.8
	Normal Muscle Mass	12	15.2
Muscle Strength	Reduced Strength	33	38.4
	Normal Strength	53	61.6
Physical Performance	Low Performance	60	69.8
	Normal Performance	26	30.2
Confounding Variable			
Protein Intake	Insufficient	25	29.1
	Adequate	61	70.9
Carbohydrate Intake	Insufficient	68	79.1
	Adequate	18	20.9
Fat Intake	Insufficient	38	44.2
	Adequate	48	55.8
Physical Activity	Inactive	47	54.7
	Active	39	45.3

at least 30 seconds of rest; the highest value was used for analysis. Reduced muscle strength was defined as <15th percentile for age and sex based on normative pediatric curve of Relative Hand Grip Strength (RHGS).[11]

Physical performance was assessed at school with age-appropriate standardized field tests, which is 10-meter walking speed, results were categorized according to pediatric reference thresholds, with performance below cut-off (normal values as follows males: 1.07–1.45; females: 1.06–1.46 s was considered normal) defined as low.[12]

Dietary intake was assessed with a structured 3-day food recall conducted by trained interviewers with parental assistance. Household measures were converted into grams, and nutrient intakes were calculated using the Indonesian food composition database. Intakes ≥90% of national recommended ranges were considered adequate.[13]

Physical activity was measured using child/parent-assisted recall of the prior week, capturing time spent in moderate-to-vigorous physical activity (MVPA). Children were categorized as active if they achieved ≥60 minutes/day of MVPA on most days, in line with WHO guidelines.[14]

Fasting venous blood samples were collected in the morning when feasible. Prior to phlebotomy, children were screened for fever or intercurrent infection to avoid confounding of inflammatory markers. CRP was quantified with a validated immunoassay and reported in mg/L; low CRP was defined as <0.2 mg/L, normal CRP was defined as 0.2 – 3 mg/L; high CRP was defined as ≥3 mg/L according to pediatric standards. Interleukin-6 (IL-6) was measured with a quantitative immunoassay and reported in pg/mL. Because IL-6 levels are influenced by multiple biological and environmental factors, elevated IL-6 was defined as >5 pg/mL, based on the GAKI Laboratory reference cut-off and consistent with prior pediatric studies applying similar thresholds.[15]

Field teams were trained and standardized before data collection. Equipment was calibrated daily. Duplicate anthropometric measures were recorded; outliers were rechecked immediately. Data entry was double-checked and cross-validated. Statistical analyses were performed using SPSS (IBM Corp., Armonk, NY). Descriptive statistics summarized participant characteristics. Associations between categorical exposures (central adiposity indices, inflammatory markers, dietary adequacy, physical activity) and sarcopenia outcomes (low muscle mass, reduced

Table II: Relationships Between Variables and Outcomes

Variable	Category	Low Muscle Mass (n,%)	Normal Muscle Mass (n,%)	p	Low Muscle Strength (n,%)	Normal Muscle Strength (n,%)	p	Low Physical Performance (n,%)	Normal Physical Performance (n,%)	p
Central Adiposity										
WHR	Normal	44 (60.3)	11 (84.6)	0.122¥	23 (69.7)	32 (60.4)	0.519¥	38 (63.3)	17 (65.4)	1.000¥
	High	29 (39.7)	2 (15.4)		10 (30.3)	21 (39.6)		22 (36.7)	9 (34.6)	
WHtR	Normal	7 (9.6)	5 (38.5)	0.016£*	9 (27.3)	3 (5.7)	0.007£*	7 (11.7)	5 (19.2)	0.271£
	High	66 (90.4)	8 (61.5)		24 (72.7)	50 (94.3)		53 (88.3)	21 (80.8)	
Inflammatory Marker										
IL-6	Normal	2 (2.7)	0 (0)	1.000£	0 (0)	2 (3.8)	0.377£	1 (1.7)	1 (3.8)	0.516£
	High	71 (97.3)	13 (100)		33 (100)	51 (96.2)		59 (98.3)	25 (96.2)	
CRP	Low	0 (0)	1 (7.7)	0.013¶*	0 (0)	1 (1.9)	0.414¶	0 (0)	1 (3.8)	0.074¶
	Normal	57 (78.1)	12 (92.3)		26 (78.8)	43 (81.1)		46 (76.7)	23 (88.5)	
	High	16 (21.9)	0 (0)		7 (21.2)	9 (17)		14 (23.3)	2 (7.7)	
Confounding Variable										
Protein Intake	Insufficient	25 (34.2)	0 (0)	0.016£*	8 (24.2)	17 (32.1)	0.594¥	19 (31.7)	6 (23.1)	0.584¥
	Adequate	48 (65.8)	13 (100)		25 (75.8)	36 (67.9)		41 (68.3)	20 (76.9)	
Carbohydrate	Insufficient	58 (79.5)	10 (76.9)	1.000£	22 (66.7)	46 (86.8)	0.050¥	46 (76.7)		
Intake	Adequate	15 (20.5)	3 (23.1)		11 (33.3)	7 (13.2)		14 (23.3)	4 (15.4)	
Fat Intake	Insufficient	31 (42.5)	7 (53.8)	0.549¥	13 (39.4)	25 (47.2)	0.629¥	24 (40)	14 (53.8)	0.342¥
	Adequate	42 (57.5)	6 (46.2)		20 (60.6)	28 (52.8)		36 (60)	12 (46.2)	
Physical Activity	Inactive	42 (57.5)	5 (38.5)	0.238¥	21 (63.6)	26 (49.1)	0.272¥	36 (60)	11 (42.3)	0.201¥
	Active	31 (42.5)	8 (61.5)		12 (36.4)	27 (50.9)		24 (40)	15 (57.7)	

Note: * Significant ($p < 0.05$); ¥ Continuity Correction; £ Fisher's exact; ¶ Fisher's exact (alternative χ^2)

Table III: Logistic Regression Analysis for Physical Performance, Muscle Strength, and Muscle Mass

Outcome	Variable	p	OR	95% CI	Significant
Physical Performance	CRP	0.059	0.228	0.049 – 1.058	No
	Physical Activity	0.190	1.895	0.729 – 4.929	No
Muscle Strength	WHtR	0.021*	5.324	1.280 – 22.148	Yes
	Carbohydrate Intake	0.081	0.366	0.119 – 1.131	No
Muscle Mass	WHtR	0.011*	0.163	0.037 – 0.714	Yes
	CRP	0.998	0.000	–	No
	Protein Intake	0.252	3.574	0.408 – 31.308	No

Note: * Significant ($p < 0.05$); ¥ Continuity Correction; £ Fisher's exact; ¶ Fisher's exact (alternative χ^2)

strength, low physical performance) were examined using chi-square tests; Fisher's exact test or continuity correction was used when expected cell counts were <5. Variables with p <0.10 in bivariate analysis were entered into logistic regression to estimate adjusted odds ratios (OR) with 95% confidence intervals (CI). Statistical significance was set at p <0.05 (two-sided).

RESULTS

The study examined 86 obese children aged 7–13 years to assess the prevalence of sarcopenia and associated factors. Cutoffs for WHR, WHtR, CRP, IL-6, muscle mass, muscle strength, physical performance, nutrient intake, and physical activity were applied as described in the Methods section. Demographically, the majority were aged 10 years (30.2%), with a higher proportion of males (60.5%). Central adiposity was prevalent, with 36.0% having a high WHR and 86.0% a high WHtR. Inflammatory markers revealed elevated IL-6 in 97.7% and high CRP in 18.6% of participants. Sarcopenia criteria showed that 84.8% had low muscle mass, 38.4% had reduced muscle strength, and 69.8% exhibited low physical performance, highlighting a significant burden. Confounding variables included insufficient protein intake in 29.1%, inadequate carbohydrate intake in 79.1%, and low physical activity in 54.7%, underscoring the need for targeted nutritional and physical interventions to mitigate sarcopenia risk in this population.

The analysis reveals critical associations between central adiposity, inflammatory markers, dietary intake, and sarcopenia-related outcomes in obese children. Central adiposity, specifically a high WHtR, was significantly linked to low muscle mass (p=0.016) and reduced muscle strength (p=0.007), underscoring its role in sarcopenia development. While WHR showed no significant relationship with these outcomes, the overall CRP level demonstrated a significant association with low muscle mass (p=0.013), indicating that systemic inflammation may contribute to sarcopenia in obese children. Protein intake adequacy was strongly associated with normal muscle mass (p=0.016), highlighting the importance of sufficient protein consumption. Other factors, such as fat, carbohydrate intake and physical activity, did not exhibit significant relationships but remain critical considerations in comprehensive interventions.

The logistic regression analysis highlights key factors influencing physical performance, muscle strength, and muscle mass in obese children. WHtR was significantly associated with muscle strength (p=0.021, OR=5.324) and muscle mass (p=0.011, OR=0.163), underscoring its critical role in sarcopenia development. Although CRP and physical activity were not statistically significant for physical performance, CRP showed a notable trend toward association (p=0.059, OR=0.228), indicating inflammation as a potential contributor. Carbohydrate or protein intake, WHR, and physical activity were not significant predictors of muscle strength or mass, respectively, but remain relevant variables for further exploration.

DISCUSSION

This study examined 86 obese children aged 7–13 years to assess the prevalence of sarcopenia and its associated factors. The demographic data (Table I) provide valuable insights into the characteristics of the study population and highlight significant health concerns that merit detailed discussion.

Demographic Characteristics

The majority of participants were aged 10 years (30.2%), with the age distribution reflecting a critical period of growth and development. A higher proportion of males (60.5%) compared to females (39.5%) participated in the study. This gender difference aligns with some research suggesting that boys may have a higher prevalence of obesity due to differences in physical activity levels and dietary habits.[8] However, other studies have found varying gender distributions depending on cultural and socioeconomic factors.[16]

Central adiposity was highly prevalent, with 36.0% of the children having a high WHR and a striking 86.0% having a high WHtR. High WHtR, defined as ≥0.50, is widely recognized as a superior measure of abdominal fat distribution and cardiovascular risk in children, being age- and sex-independent.[9,17] By contrast, WHR thresholds for children are less established; hence, this study used the percentile 90th of the sample distribution, a limitation that should be noted. The significant associations of WHtR with both low muscle mass and reduced strength reinforce its utility over WHR for identifying sarcopenia risk in pediatric obesity.

Elevated inflammatory markers were observed, with 97.7% of participants showing high IL-6 levels and 18.6% exhibiting high CRP levels. Obesity, particularly central obesity, is associated with a chronic low-grade inflammatory state due to increased secretion of pro-inflammatory cytokines from adipose tissue. Elevated IL-6 and CRP levels have been linked to insulin resistance and endothelial dysfunction.[18] In comparison, a study by Dayal et al. (19) reported elevated CRP levels in obese children, which correlated with other cardiovascular risk factors.[19] The high prevalence of elevated IL-6 in this study underscores the potential for early development of inflammatory-related complications in obese children.

Sarcopenia criteria revealed that 84.8% of the children had low muscle mass, 38.4% had reduced muscle strength, and 69.8% exhibited low physical performance. These findings indicate a significant burden of sarcopenia among obese children, which is concerning given that sarcopenia is typically associated with aging populations.[20] The concept of sarcopenic obesity in children is emerging, with evidence suggesting that excess adiposity can coexist with diminished muscle mass and function. Park, et al (21) demonstrated that sarcopenic obesity in children was linked to increased metabolic risk factors than normal demographic.[21]

The study found that 29.1% of participants had insufficient protein intake, and a significant 79.1% had inadequate carbohydrate intake. Adequate protein intake is essential for muscle protein synthesis and growth, particularly during

childhood and adolescence.[22] Insufficient protein consumption may contribute to the high prevalence of low muscle mass observed. Inadequate carbohydrate intake can lead to decreased energy availability, potentially affecting growth, physical activity, and muscle function.[23] The high rate of insufficient carbohydrate intake suggests dietary patterns that may not support optimal muscle development and overall health.

Low physical activity levels were reported in 54.7% of the participants. Physical inactivity is a well-known risk factor for obesity and is associated with decreased muscle mass and strength.[24] Regular physical activity is crucial for developing and maintaining muscle mass, enhancing metabolic health, and preventing obesity-related complications. Physical inactivity among children is associated with higher adiposity and lower fitness levels. The combination of low physical activity and poor dietary intake may synergistically contribute to the development of sarcopenic obesity.[25]

The co-occurrence of high central adiposity, elevated inflammatory markers, low muscle mass, and poor dietary and physical activity behaviors in this study underscores the complex interplay between these factors in obese children. Other studies have similarly reported associations between central obesity, inflammation, and sarcopenia. For example, Axelrod et al. (26) discussed the concept of sarcopenic obesity and its implications for health outcomes. They emphasized that excess adiposity, particularly visceral fat, can promote inflammation and insulin resistance, leading to muscle catabolism.[26] Furthermore, Pena et al. (27) found that higher levels of IL-6 were associated with reduced muscle mass and strength in obese adolescents.[27] This supports the notion that chronic inflammation may contribute to muscle degradation in obese youth. The dietary findings align with research indicating that obese children often have diets high in energy-dense, nutrient-poor foods but low in essential nutrients like protein and complex carbohydrates.[28] Such dietary patterns may not provide sufficient nutrients for muscle growth and may contribute to adiposity and inflammation.

Central Adiposity and Sarcopenia Risk

Our results indicate that a high WHtR is significantly associated with low muscle mass (p=0.016) and reduced muscle strength (p=0.007) in obese children. This aligns with previous research suggesting that central adiposity adversely affects muscle health. For instance, Khaleghi et al. (29) reported that increased visceral fat is inversely related to muscle mass and strength in adolescents, potentially due to adipose tissue's endocrine function influencing muscle metabolism. Moreover, the lack of significant association between waist-to-hip ratio and sarcopenia risk in our study may reflect the limitations of WHR as a measure of central adiposity in children.[29] WHtR has been proposed as a more sensitive indicator of central fat distribution and associated metabolic risks in pediatric populations.[30] Therefore, WHtR may be a more appropriate anthropometric measure for assessing sarcopenia risk related to central adiposity in obese children.

Inflammation Status and Muscle Health

Overall CRP levels were significantly associated with low muscle mass (p=0.013), with high CRP values found exclusively in participants with low muscle mass, suggesting that systemic inflammation contributes to sarcopenia risk in obese children. Chronic low-grade inflammation is a known consequence of excess adiposity, particularly visceral fat, which secretes pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α).[30] These cytokines can promote muscle protein degradation and inhibit muscle synthesis, leading to muscle atrophy. While IL-6 levels were elevated in 97.7% of participants, no significant association was found with muscle outcomes, likely because IL-6 secretion from adipocytes occurs early and pervasively, reducing variability across the cohort. CRP, in contrast, is synthesized hepatically in response to sustained cytokine signaling and reflects a downstream inflammatory burden that more directly disrupts anabolic signaling pathways while enhancing proteolysis, thereby accelerating muscle loss. This difference also reflects a broader contrast between adults and children: in adults, both IL-6 and CRP are frequently elevated due to obesity, comorbidities, and age-related "inflammaging," making it harder to isolate their roles, whereas in children baseline levels are lower, so widespread IL-6 elevation marks early inflammation, while CRP identifies the subset in whom inflammation has advanced to produce measurable musculoskeletal consequences.[31]

Nutritional Intake and Sarcopenia Risk

Adequate protein intake was strongly associated with normal muscle mass (p=0.016), emphasizing the crucial role of dietary protein in muscle development and maintenance during childhood. Protein provides essential amino acids necessary for muscle protein synthesis, which is vital for growth and muscle repair.[32] Consistent with our findings, Arnesen et al. (33) found that higher protein intake was associated with better muscle mass indices in overweight and obese children.[33] Although carbohydrate intake showed a borderline significant relationship with reduced muscle strength (p=0.050), it remains an important energy source for physical activity and muscle function. Insufficient carbohydrate intake may impair glycogen stores, reducing energy availability for muscle contraction and potentially affecting muscle performance.[34] Fat intake did not exhibit a significant relationship with muscle health indicators in our study. However, dietary fats, particularly omega-3 fatty acids, have been shown to have anti-inflammatory properties and may support muscle protein synthesis.[35] Further research is needed to elucidate the role of specific types of dietary fats in pediatric muscle health.

Physical Activity and Muscle Function

Physical activity was assessed by recall and classified in binary terms: active (≥60 minutes/day moderate-to-vigorous activity, WHO standard) or inactive. In this study, physical activity did not show a significant association with muscle mass or strength in our sample. However, there was a trend indicating that physically active children had better muscle strength and physical performance. Regular physical activity, especially resistance training, is known to stimulate muscle protein synthesis and improve muscle mass and strength in

children and adolescents.[36] The lack of statistical significance may be attributed to the binary approach for screening, while practical, it may mask important gradations of activity (e.g., 15 vs. 45 minutes both classified as inactive), which could attenuate associations. Another possible reason was the self-reported physical activity measures, which can be prone to bias, or the cross-sectional nature of the study, which limits causal inference.

Our findings are consistent with prior studies that have identified central adiposity and inflammation as key factors in sarcopenia development among obese youth. For example, Shuster et al demonstrated that visceral adiposity is associated with metabolic complications and reduced muscle mass in children.[37] Additionally, Rolland et al. (38) highlighted the role of inflammation in muscle catabolism and sarcopenia in obese adolescents. However, some studies have reported differing results regarding the impact of physical activity and dietary factors on muscle health.[38] This variability may stem from differences in study design, population characteristics, and assessment methods. Longitudinal studies with objective measures of physical activity and comprehensive dietary assessments may provide more definitive insights.

A strength of this study is the comprehensive evaluation of factors related to sarcopenia risk, including anthropometric measures, inflammatory markers, dietary intake, and physical activity. However, several limitations should be acknowledged. The cross-sectional design precludes the establishment of causality. The relatively small sample size may limit the generalizability of the findings and reduce the power to detect significant associations for some variables. Additionally, reliance on self-reported dietary intake and physical activity may introduce reporting biases. Another limitation is that the cutoff for waist-to-hip ratio (WHR) was derived linearly from waist circumference, since no established pediatric-specific cutoff was available from previous studies.

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