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Association of Obesity and Overweight with Early Puberty in Boys: A Meta Analysis

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What is already known on this topic?

While obesity is a well-documented risk factor for early puberty in girls, its association with male pubertal timing remains controversial. Existing studies show conflicting results, ranging from earlier onset to no effect or even delayed puberty in severe obesity. The post-coronavirus disease-2019 era has seen a global rise in idiopathic cases coinciding with increasing childhood obesity rates, though underlying mechanisms remain unclear. Emerging evidence suggests potential adverse effects on male genital development.

What this study adds?

While obesity is an established risk factor for precocious puberty in females, its role in male pubertal development remains controversial. Our meta-analysis confirms that childhood obesity significantly increases the risk of early puberty in males. Notably, JCEM studies indicate obesity may reduce pubertal penile growth by approximately 10% while lowering testosterone levels. These findings collectively suggest a dual-effect paradigm of adiposity in male development: obesity appears to both accelerate sexual maturation while potentially compromising optimal genital development.

ABSTRACT

Objective: To evaluate the published associations between obesity, overweight, and central obesity and the risk of early puberty in boys.

Methods: A comprehensive systematic search was conducted in accordance with PRISMA guidelines using the Web of Science and PubMed databases up to December 31, 2024. Study quality was assessed using the Newcastle-Ottawa Scale. Statistical analyses were performed using R software (version 4.4.2), with odds ratios (ORs) and 95% confidence intervals (CIs) calculated.

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Results: A total of 15,452 studies were initially identified, of which six high-quality studies (n=64,485) met the inclusion criteria after screening. The analysis found that obesity [defined by body mass index (BMI)] was significantly associated with an increased risk of testicular enlargement (OR=1.27, 95% CI: 1.19-1.36). Overweight also increased the risk of testicular enlargement (OR=1.20, 95% CI: 1.11-1.29). Obesity was significantly associated with an increased risk of pubarche (OR=1.37, 95% CI: 1.23-1.53). Funnel plots and sensitivity analyses indicated no significant publication bias, and the results remained robust.

Conclusion: This study demonstrated that obesity and overweight are reported to be associated with an increased risk of early puberty in boys. Childhood obesity appears to be an important determinant of earlier pubertal onset, though the relationship may follow a non-linear pattern at extreme BMI levels. The potential implications for adult reproductive health warrant further investigation.

Keywords: Obesity, overweight, central obesity, early puberty, testicular enlargement, pubarche, meta-analysis

Introduction

Central precocious puberty (CPP) is defined as the onset of puberty before the age of 8 years in girls and 9 years in boys, resulting from premature activation of the hypothalamic-pituitary-gonadal axis (1,2). In recent decades, the incidence of CPP has shown a significant upward trend globally, with a notable surge observed following the coronavirus disease-2019 (COVID-19) pandemic (3,4,5). The etiology of CPP is multifactorial, encompassing genetic predispositions, environmental influences, and metabolic factors, among which obesity has emerged as an important contributor (6,7,8). An earlier onset of puberty in children has been documented in many countries, with precocious puberty representing a prevalent endocrine disorder in childhood (1). The concurrent trends of declining age at puberty onset and increasing prevalence of obesity have generated interest in the association between obesity and pubertal timing (5).

Epidemiological cross-sectional and longitudinal studies have consistently demonstrated that overweight and obesity are strongly associated with earlier puberty onset and menarche in girls (4,6,7,9). A meta-analysis has further identified obesity as a significant risk factor for the early onset of puberty in girls (10). However, the relationship between obesity and CPP in boys remains poorly understood, with limited and inconsistent evidence available (8,11,12). The incidence of precocious puberty differs markedly between boys and girls, with girls exhibiting a significantly higher prevalence than boys (8). While previous studies have consistently highlighted a strong association between early puberty in girls and elevated body mass index (BMI) or obesity, the evidence in boys remains inconclusive and subject to debate (8,11,12).

To address this gap, we conducted a comprehensive analysis of the existing literature to determine whether obesity or overweight status is similarly associated with early puberty in boys. The aim of this systematic review was to evaluate the potential association between obesity and CPP in boys, providing clearer evidence about an important and understudied aspect of pubertal development.

Methods

All methods used in this systematic review and meta-analysis were conducted in accordance with the PRISMA guidelines (13).

Search Strategy

A comprehensive literature search was conducted on Web of Science and PubMed. The search strategy included key terms related to obesity, such as “Obesity”, “obese”, “adiposity”, “overweight”, “bodyweight”, “BMI”, “body mass index”, “body fat”, or “body fat mass”, combined with terms related to early puberty, including “pubertal timing”, “puberty timing”, “sexual precocity”, “sexual prematurity”, “premature pubarche”, “first spermatorrhea”, “gonadarche”, or “precocious puberty”. The search was executed on both Web of Science and PubMed databases to ensure a thorough retrieval of relevant studies.

Selection Criteria

This systematic review and meta-analysis included studies that met the following criteria: (i) cohort or case-control studies focusing on children; (ii) an exposure group comprising children classified as obese by the study authors, compared with a control group of children with normal weight; (iii) the primary outcome measured was the onset of secondary sexual characteristics, specifically including testicular enlargement, first ejaculation, and the initial appearance of pubic hair.

Quality Assessment of Literature

The methodological rigor of the included cohort studies was evaluated using the Newcastle-Ottawa Scale (NOS), which assigns a maximum score of 9 points. Studies were categorized as low (0-3 points), medium (4-6 points), or high quality (7-9 points) (14). Two independent researchers (AA, BB) performed the quality assessments. Any disagreements were resolved through discussion or adjudication by a senior author (AA).

Statistical Analysis

All statistical analyses were conducted using R software, version 4.4.2. The odds ratio (OR) was used as the primary measure of effect size for count data, accompanied by a 95% confidence interval (CI). Heterogeneity among studies was assessed using the chi-square test, with the p value and I^2 statistic providing measures of heterogeneity. A fixed-effects model was employed when the studies exhibited low heterogeneity ($p \geq 0.05$, $I^2 \leq 50\%$), while a random-effects model was used in the presence of significant heterogeneity ($p < 0.05$, $I^2 > 50\%$). Publication bias was assessed through funnel plots and Egger's test. Sensitivity analysis was performed to evaluate the robustness of the meta-analysis results by systematically excluding each study and analyzing its influence on the overall effect, with statistical significance set at $p < 0.05$.

Results

The systematic search of databases identified 15,452 potential studies (Figure 1). Following the removal of duplicates and an initial screening of titles and abstracts, 132 full-text articles were selected for detailed eligibility assessment. Studies were excluded based on predefined criteria: 40 were excluded for not focusing on precocious puberty, 65 were excluded due to the absence of both obesity exposure and control groups, and 21 were excluded for not adhering to case-control or cohort study designs. Ultimately, six studies meeting all inclusion criteria were included in the quantitative analysis, as summarized in Table 1. These studies were conducted in Chile, the United States, and China, and included participants of Asian, Black, White, and Caucasian race/ethnicity. The methodological quality of the six cohort studies was evaluated using the NOS, with scores ranging from 7 to 9, indicating high-quality studies (Table 2). The total sample size across all studies combined was 64,485 participants.

BMI-defined obesity was significantly associated with an increased risk of testicular enlargement (≥4 mL, OR=1.27, 95% CI: 1.19-1.36; Figure 2). However, substantial heterogeneity was observed across studies (I^2=54.8%, p=0.0237). Overweight, also defined by BMI, similarly increased the risk of testicular enlargement (≥4 mL, OR=1.20, 95% CI: 1.11-1.29; Figure 3), with high heterogeneity (I^2=58%, p=0.0266). Two studies examined the association between central obesity, defined by waist circumference, and testicular enlargement, but the

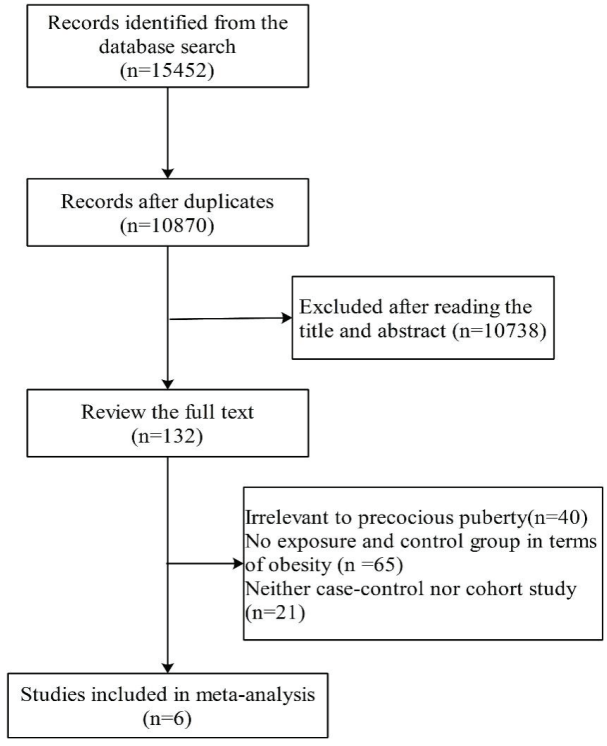

Figure 1. PRISMA flow diagram

Table 1. Characteristics of studies included in the meta-analysis

Study, year	Country	Ethnicity	Design	Sample size	Obesity	Outcome
Pereira et al. (15), 2021	Chile	White	Cohort	345	BMI>+2SD WC>90th	Gonadarche
Li et al. (19), 2018	China	Asian	Cohort	644	BMI>27.9	Gonadarche
Li et al. (16), 2022	China	Asian	Cohort	645	BMI>27.9 WC≥90th	Gonadarche
Liu et al. (17), 2021	China	Asian	Case-control	525	BMI>P95th	Central precocious puberty
Aghaee et al. (20), 2022	California	White, Black, Hispanic, Asian	Cohort	62190	BMI≥P95th	Gonadarche Pubarchec
Deardorff et al. (21), 2021	California	White	Cohort	136	BMI≥P95	Gonadarche Pubarche

Table 2. Newcastle-Ottawa scale for assessing the methodological quality of cohort studies

Study	Selection				Comparability	Outcome		
	Representativeness of the expose cohort	Selection of the non-exposed cohort	Ascertainment of exposure	Demonstration that outcome of interest was not present at start of study	Comparability of cohorts on the basis of the design or analysis	Assessment of outcome	Was follow-up long enough for outcomes to occur	Adequacy of follow up of cohorts	Score
Pereira et al. (15), 2021	1	1	1	1	1	1	1	0	7
Li et al. (19), 2018	1	1	1	1	1	1	1	1	8
Li et al. (16), 2022	1	1	1	1	2	1	1	1	9
Aghaee et al. (20), 2022	1	1	1	1	1	1	1	1	8
Deardorff et al. (21), 2021	1	1	1	1	2	1	1	1	9

results were not significant (OR=2.38, 95% CI: 0.92-6.19; Figure 4). Obesity was significantly associated with an increased risk of pubarche (OR=1.37, 95% CI: 1.23-1.53; Figure 5), with high heterogeneity (I^2=75.9%, p=0.0023). Overweight also increased the risk of pubarche (OR=1.26, 95% CI: 1.18-1.36; Figure 6), with moderate heterogeneity (I^2=55.8%, p=0.06). Funnel plots indicated no significant publication bias, and the adjusted effect size remained statistically significant (OR=1.24, 95% CI: 1.17-1.32; Figure 7). Sensitivity analyses confirmed the robustness of the results, as the exclusion of any single study did not alter the significance of the findings (Figure 8).

Discussion

Our systematic review ultimately included six high-quality studies that met the predefined eligibility criteria. The findings of this analysis demonstrated a significant reported association between overweight/obesity and precocious puberty in boys, consistent with trends previously observed in girls. These results are also consistent with multiple domestic and international studies (1,3,6,9), further supporting the important role of obesity in the onset of precocious puberty.

The relationship between obesity/overweight and earlier pubertal initiation in girls has been well-documented in prior research (4,7,9). In contrast, studies focusing on boys remain limited, likely due to the lower incidence of precocious puberty in males compared to females, as well as the historical emphasis on pathological etiologies in male cases. However, emerging evidence suggests a rising trend in idiopathic male precocious puberty globally, particularly following the COVID-19 pandemic. This trend parallels observations reported in China and other regions (15,16,17,18,19).

The relationship between childhood obesity and precocious puberty in boys has attracted increasing attention amid global trends of declining pubertal age and increasing pediatric adiposity. This systematic analysis of six contemporary studies revealed both converging and conflicting evidence regarding this association, highlighting the need for nuanced interpretation of biological and environmental interactions.

Supportive Evidence for Obesity-Puberty Link

Multiple longitudinal studies demonstrate measurable associations between adiposity and earlier gonadarche in boys. Pereira et al. (15) found that total body fat [β=-0.32 years/standard deviation (SD), p<0.01] and central adiposity (waist-height ratio β=-0.41 years/SD, p<0.001) independently predicted earlier testicular enlargement in a multiethnic cohort, with obese boys experiencing gonadarche 1.1 years earlier than lean peers. These findings align with the Chinese longitudinal study of Li et al. (16), showing boys in the highest adiposity trajectory had 2.3-fold increased risk of precocious pubarche (95% CI: 1.4-3.8) compared to normal-weight counterparts.

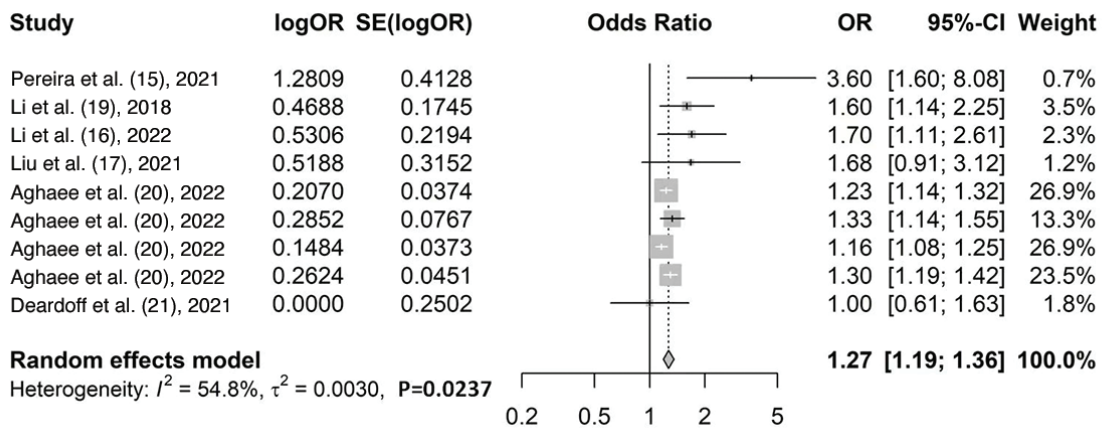

Figure 2. Forest plot of obesity (BMI-defined) and testicular enlargement
OR: odds ratio, CI: confidence interval, SE: standard error, BMI: body mass index

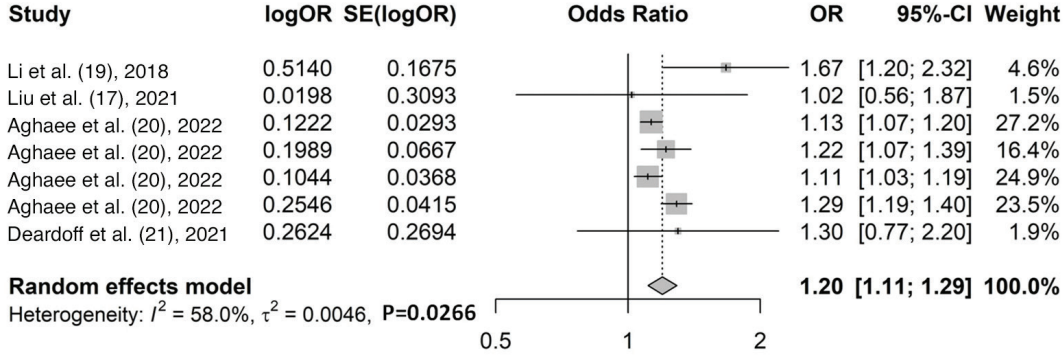

Figure 3. Forest plot of overweight (BMI-defined) and testicular enlargement
OR: odds ratio, CI: confidence interval, SE: standard error, BMI: body mass index

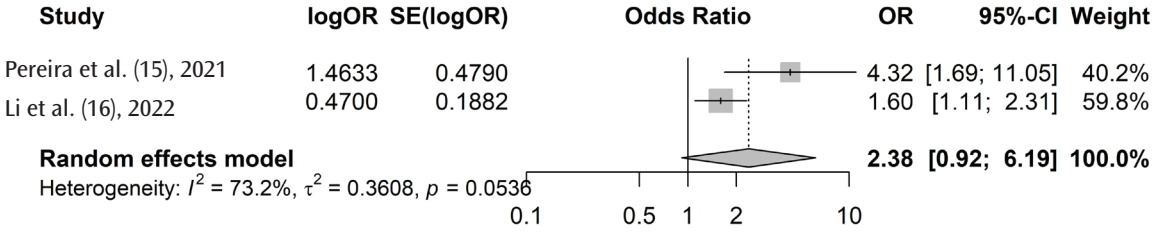

Figure 4. Forest plot of central obesity (waist circumference-defined) and testicular enlargement
OR: odds ratio, CI: confidence interval, SE: standard error

Mechanistically, Liu et al. (17) identified elevated leptin levels (OR=1.8, p=0.02) and leptin-to-adiponectin ratios (OR=2.1, p=0.01) as potential mediators in their case-control analysis of CPP. These mechanistic insights suggest a dual-effect model of adiposity in male puberty. Obesity may promote hypothalamic-pituitary-gonadal axis activation leading to earlier pubertal onset (testicular enlargement OR=1.27). Paradoxically, obesity may simultaneously impair genital development as shown by

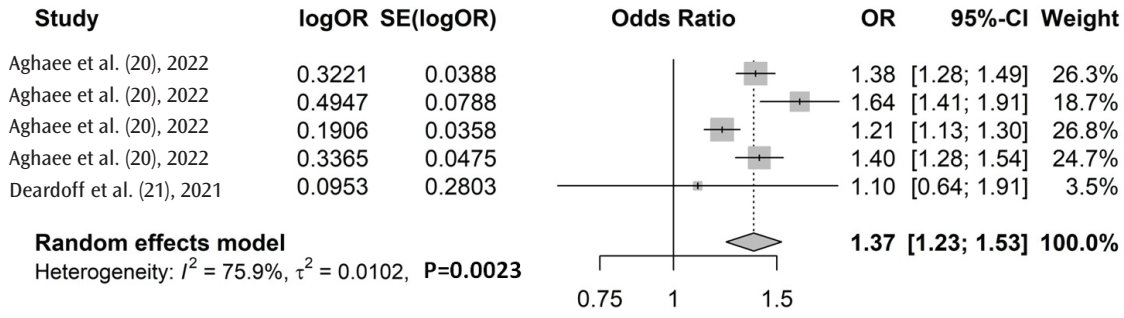

Figure 5. Forest plot of obesity (BMI-defined) and pubarche
OR: odds ratio, CI: confidence interval, SE: standard error, BMI: body mass index

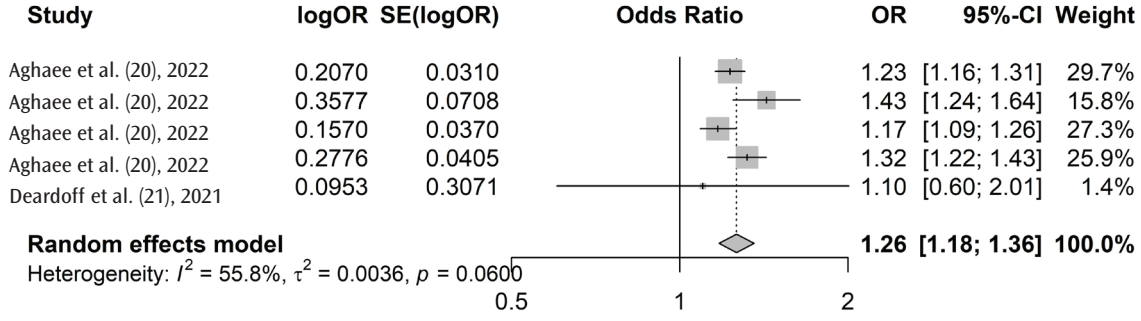

Figure 6. Forest plot of overweight (BMI-defined) and pubarche
OR: odds ratio, CI: confidence interval, SE: standard error, BMI: body mass index

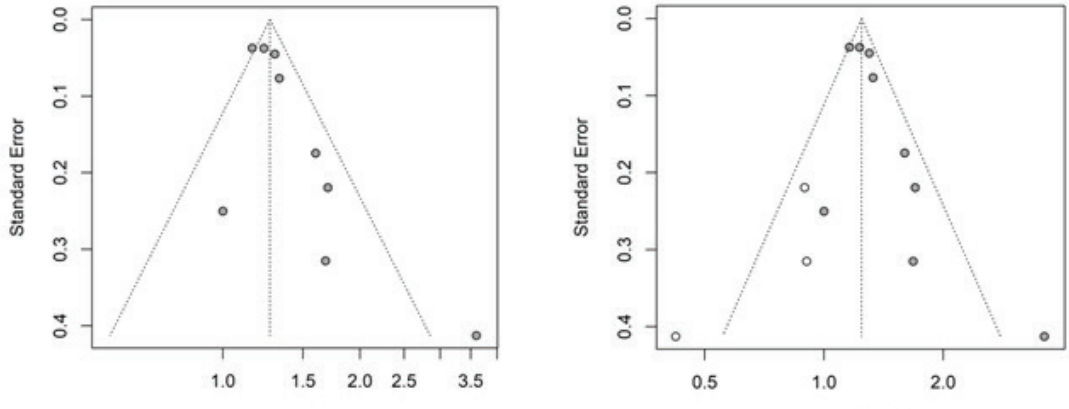

Figure 7. Funnel plot for publication bias assessment

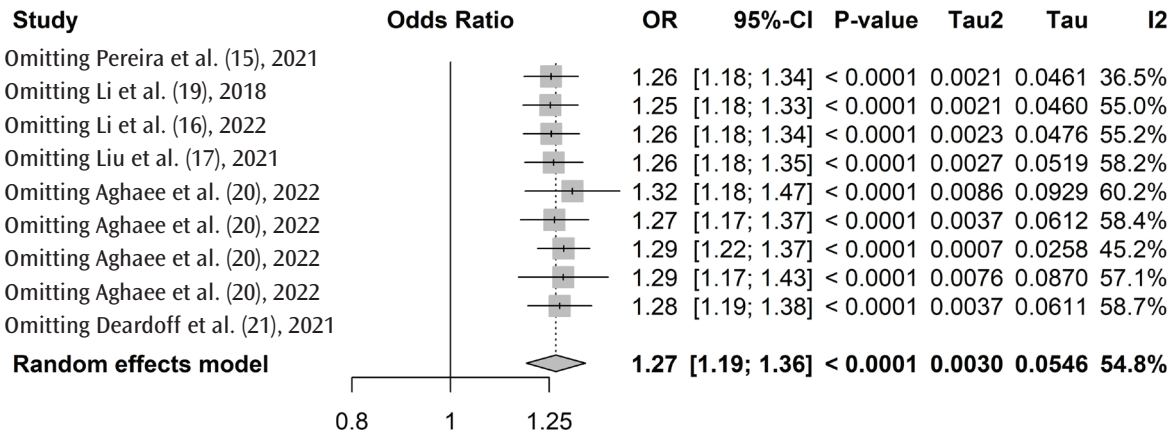

Figure 8. Sensitivity analysis (sequential exclusion of each study followed by re-analysis)
OR: odds ratio, CI: confidence interval

10% reduced penile growth in obese boys (18). This effect likely contributes to the observed heterogeneity across studies.

Contradictory Findings and Methodological Considerations

Despite some studies (16,17,18) suggesting a link between obesity/overweight and earlier puberty in boys, the remaining studies challenged this consensus. Li et al. (19) reported no significant association between prepubertal obesity and earlier voice breaking in Chinese boys (β=0.08 years, p=0.42), despite strong correlation in girls. Similarly, Aghaee et al. (20) found race/ethnicity modified this relationship, with obesity accelerating pubertal timing in Hispanic boys [hazard ratio (HR)=1.4, p=0.03] but not in non-Hispanic white peers (HR=0.9, p=0.61). These discrepancies may stem from varying outcome measures (clinical vs self-reported puberty markers) and population-specific genetic/environmental factors. Notably, Deardorff et al. (21) observed paradoxical associations in Mexican-American boys, where severe obesity (BMI≥99th percentile) correlated with delayed pubic hair development (HR=0.7, p=0.04), suggesting potential threshold effects of adiposity.

Emerging Post-Pandemic Patterns

Recent epidemiological shifts post-COVID-19 warrant special consideration. Wang et al. (12) documented a 23% surge in idiopathic precocious puberty cases across Asian and European centers, paralleling accelerated weight gain during lockdowns. This trend is consistent with biological mechanisms where adipose tissue aromatase activity may convert androgens to estrogens, thus lowering the hypothalamic-pituitary-gonadal axis activation threshold (22). Nevertheless, Rosenfield (23) caution against overattributing idiopathic cases to obesity alone, given the historical predominance of pathological etiologies (e.g., CNS lesions) in male precocious puberty diagnoses.

Study Limitations

Current research on the relationship between adiposity and puberty has notable limitations, including variability in measurement methods, for example inconsistent use of markers of male puberty, such as testicular volume vs. pubic hair, and adiposity indices, including BMI vs. central adiposity, which hinders direct comparisons across studies (15,16,21). Moreover, many studies focus primarily on concurrent associations rather than exploring the likely important prepubertal period that may have a stronger influence on pubertal timing (18,20). Another key limitation is the insufficient adjustment for potential confounders, such as endocrine-disrupting chemicals and socioeconomic factors, both of which are known to independently affect pubertal development (24). These methodological gaps highlight the need for more standardized assessments, longitudinal designs, and rigorous control of confounding variables in future investigations. Finally, while BMI remains the standard adiposity metric, its inability to distinguish fat from muscle mass may obscure true associations. Although we analyzed waist circumference data, the limited available studies precluded definitive conclusions regarding the effect of central obesity.

Conclusion

Although accumulating evidence suggests adiposity promotes earlier pubertal onset in boys, with a notable association in high-obesity populations, this association demonstrates greater context-dependency than has been reported in girls. Clinicians should be aware of the non-linear nature of this relationship, where extreme obesity may paradoxically delay specific pubertal markers while accentuating others, and the need for individualized assessment considering population-specific adiposity patterns. Future research must incorporate

standardized genital morphometry and body composition profiling to elucidate these complex interactions. Furthermore, evolving global childhood adiposity trends in the post-pandemic era (21) necessitate dynamic monitoring to inform precision prevention strategies.

Implications

These findings suggest that it will be important to recognize that obesity is a probable significant risk factor for precocious puberty in boys, mirroring patterns seen in girls. The increasing prevalence of idiopathic male precocious puberty, particularly in the post-pandemic era, highlights the need for further research to confirm the findings and elucidate underlying mechanisms, thus informing preventive strategies.

Ethics

Ethics Committee Approval: Not applicable (since this is a meta-analysis based on published literature).

Informed Consent: Not applicable.

Footnotes

Authorship Contributions

Concept: Ziqin Liu, Design: Ziqin Liu, Data Collection and Processing: Xiou Wang, Analysis or Interpretation: Xiou Wang, Yi Song, Literature Research: Xiou Wang, Yi Song, Ziqin Liu, Writing: Yi Song, Ziqin Liu.

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