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Research Article

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

Wang and Song et al. Association of Obesity with Boys

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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-COVID-19 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 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 (NOS). Statistical analyses were performed using R software (version 4.4.2), with odds ratios (ORs) and 95% confidence intervals (CIs) calculated.
Results: A total of 15,452 studies were initially identified, of which 6 high-quality studies (n=64,485) met the inclusion criteria after screening. The analysis revealed that obesity (defined by 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 demonstrates that obesity and overweight are significantly associated with an increased risk of early puberty in boys. Childhood obesity represents 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.

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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 particularly following the COVID-19 pandemic [3–5]. The etiology of CPP is multifactorial, encompassing genetic predispositions, environmental influences, and metabolic factors, among which obesity has emerged as a critical contributor [6–8]. 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. This systematic review aims to evaluate the potential association between obesity and CPP in boys, providing clarity on a critical 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.

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," "precocious puberty," "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). 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 utilized 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, 6 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 6 cohort studies was evaluated using the Newcastle-Ottawa Scale (NOS), with scores ranging from 7 to 9, indicating high-quality studies (Table 2). The total sample size across all studies was 64,485 participants.

Obesity, defined by BMI, was significantly associated with an increased risk of testicular enlargement (OR=1.27, 95% CI: 1.19–1.36; Fig. 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 (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 results were not statistically 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 demonstrate a significant association between overweight/obesity and precocious puberty in boys, consistent with trends previously observed in girls. These results are consistent with multiple domestic and international studies [1, 3, 6, 9], further supporting the critical 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 a 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 [13–17].

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 reveals both converging and conflicting evidence regarding this association, underscoring 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. (2021) [13] found that total body fat (β= -0.32 years/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 Li et al.'s (2022) [14] Chinese longitudinal study 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. Mechanistically, Liu et al. (2021) [15] 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 central precocious puberty. These mechanistic insights support a dual-effect paradigm of adiposity in male puberty: while promoting hypothalamic-pituitary-gonadal axis activation leading to earlier pubertal onset (testicular enlargement OR=1.27), obesity may simultaneously impair genital development as evidenced by 10% reduced penile growth in obese boys [16]. This paradoxical effect likely contributes to the observed heterogeneity across studies

Contradictory Findings and Methodological Considerations However, several studies challenge this consensus. Li et al. (2018) [17] reported no significant association between prepubertal obesity and earlier voice breaking in Chinese boys (β=0.08 years, p=0.42), despite strong correlations in girls. Similarly, Aghaee et al. (2022) [18] found race/ethnicity modified this relationship, with obesity accelerating pubertal timing in Hispanic boys (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. (2022) [19] 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. (2023) [20] documented a 23% surge in idiopathic precocious puberty cases across Asian

and European centers, paralleled by accelerated weight gain during lockdowns. This trend intersects with biological plausibility: adipose tissue aromatase activity converting androgens to estrogens may lower the hypothalamic-pituitary-gonadal axis activation threshold [21]. Nevertheless, Rosenfield et al. (2020) [22] 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—such as inconsistent puberty markers (e.g., testicular volume vs. pubic hair) and adiposity indices (e.g., BMI vs. central fat)—which hinders direct comparisons across studies [13, 14, 19]. Moreover, many studies focus primarily on concurrent associations rather than exploring critical prepubertal periods that may have a stronger influence on pubertal timing [16, 18]. 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 [23]. These methodological gaps underscore the need for more standardized assessments, longitudinal designs, and rigorous control of confounding variables in future investigations. Third, 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 central obesity effects.

Conclusion

Although accumulating evidence suggests adiposity promotes earlier pubertal onset in boys—particularly in high-obesity populations—this association demonstrates greater context-dependency than observed in girls. Clinicians should recognize the non-linear nature of this relationship, where extreme obesity may paradoxically delay specific pubertal markers, 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 [19] necessitate dynamic monitoring to inform precision prevention strategies.

Implications

These findings underscore the importance of recognizing obesity as a 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 elucidate underlying mechanisms and inform preventive strategies.

Data Availability Statement: The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

Conflict of Interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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References

1. Latronico AC, Brito VN, Carel JC. Causes, diagnosis, and treatment of central precocious puberty. Lancet Diabetes Endocrinol. 2016;4(3):265-274.
2. Carel JC, Léger J. Clinical practice. Precocious puberty. N Engl J Med. 2008;358(22):2366-2377.
3. Kaplowitz PB, Oberfield SE. Reexamination of the age limit for defining when puberty is precocious in girls in the United States: implications for evaluation and treatment. Pediatrics. 1999;104(4 Pt 1):936-941.
4. Kim SH, Huh K, Won S, Lee KW, Park MJ. A significant increase in the incidence of central precocious puberty among Korean girls from 2004 to 2010. PLoS One. 2015;10(11):e0141844.
5. Sørensen K, Mouritsen A, Aksglaede L, Hagen CP, Mogensen SS, Juul A. Recent secular trends in pubertal timing: implications for evaluation and diagnosis of precocious puberty. Horm Res Paediatr. 2012;77(3):137-145.
6. Vazquez MJ, Velasco I, Tena-Sempere M. Novel mechanisms for the metabolic control of puberty: implications for pubertal alterations in early-onset obesity and malnutrition. J Endocrinol. 2019;242(2):R51-R65.
7. Reinehr T, Roth CL. Is there a causal relationship between obesity and puberty? Lancet Child Adolesc Health. 2019;3(1):44-54.
8. Soriano-Guillén L, Argente J. Central precocious puberty, functional and tumor-related. Best Pract Res Clin Endocrinol Metab. 2019;33(3):101262.
9. Abreu AP, Dauber A, Macedo DB, Noel SD, Brito VN, Gill JC, et al. Central precocious puberty caused by mutations in the imprinted gene MKRN3. N Engl J Med. 2013;368(26):2467-2475.
10. Zhou X, Hu Y, Yang Z, Gong Z, Zhang S, Liu X, et al. Overweight/obesity in childhood and the risk of early puberty: a systematic review and meta-analysis. Front Pediatr. 2022;10:795596.
11. Soriano-Guillén L, Corripio R, Labarta JI, Cañete R, Castro-Feijóo L, Espino R, et al. Central precocious puberty in children living in Spain: incidence, prevalence, and influence of adoption and immigration. J Clin Endocrinol Metab. 2010;95(9):4305-4313.
12. Wang Q, Chen Y, Wu W, Ma J, Lu J, Shen Y, et al. Secular trends in pubertal growth acceleration in Chinese boys and girls from 1995 to 2019. Lancet Child Adolesc Health. 2023;7(4):256-265.
13. Pereira A, Busch AS, Solares F, Corvalán C, Mericq V, Ong KK, et al. Total and central adiposity are associated with age at gonadarche and incidence of precocious gonadarche in boys. J Clin Endocrinol Metab. 2021;106(5):1352-1361.
14. Li Y, Ma T, Ma Y, Chen Y, He H, Qi L, et al. Adiposity status, trajectories, and earlier puberty onset: results from a longitudinal cohort study. J Clin Endocrinol Metab. 2022;107(9):2462-2472.
15. Liu G, Guo J, Zhang X, Lu Y, Miao J, Wang H, et al. Obesity is a risk factor for central precocious puberty: a case-control study. BMC Pediatr. 2021;21:Article number.
16. Mancini M, Pecori Giraldi F, Andreassi A, Mantellassi G, Salvioni M, Berra CC, et al. Obesity Is Strongly Associated With Low Testosterone and Reduced Penis Growth During Development. J Clin Endocrinol Metab. 2021;106(11):3151-3159.
17. Li W, Liu Q, Deng X, Chen Y, Liu S, Story M. Association of prepubertal obesity with pubertal development in Chinese girls and boys: a longitudinal study. Am J Hum Biol. 2018;30:e23195.
18. Aghaee S, Deardorff J, Quesenberry CP Jr, Greenspan LC, Yen IH, Hiatt RA. Associations between childhood obesity and pubertal timing stratified by sex and race/ethnicity. Am J Epidemiol. 2022;191(11):2026-2036.
19. Deardorff J, Reeves JW, Hyland C, Tilles SA, Vine S, Rauch SA, et al. Childhood overweight and obesity and pubertal onset among Mexican-American boys and girls in the CHAMACOS longitudinal study. Am J Epidemiol. 2022;191(1):7-16.
20. Wang Q, Chen Y, Wu W, Ma J, Lu J, Shen Y, et al. Secular trends in pubertal growth acceleration in Chinese boys and girls from 1995 to 2019. Lancet Child Adolesc Health. 2023;7(4):256-265.

21. Lee JM, Appugliese D, Kaciroti N, Corwyn RF, Bradley RH, Lumeng JC. Age of puberty in overweight vs. obese boys. J Pediatr. 2007;151(6):618-622.
22. Rosenfield RL, Cooke DW, Radovick S. Puberty and its disorders in the female. N Engl J Med. 2020;382(10):943-952.
23. Herman-Giddens ME, Slora EJ, Wasserman RC, Bourdony CJ, Bhapkar MV, Koch GG, et al. Secondary sexual characteristics and menses in young girls seen in office practice: a study from the Pediatric Research in Office Settings network. Pediatrics. 1997;99(4):505-512.

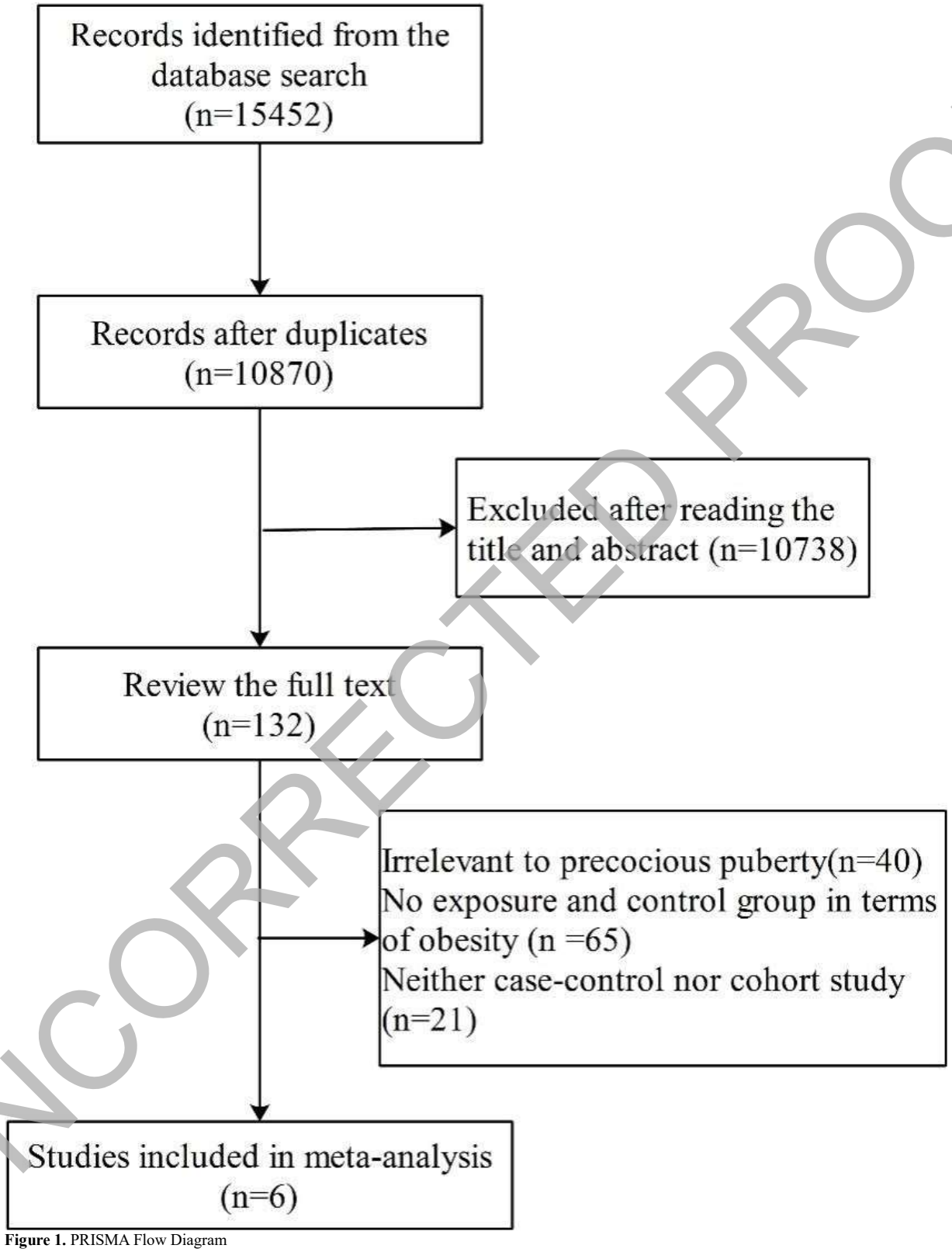

Figure 1. PRISMA Flow Diagram

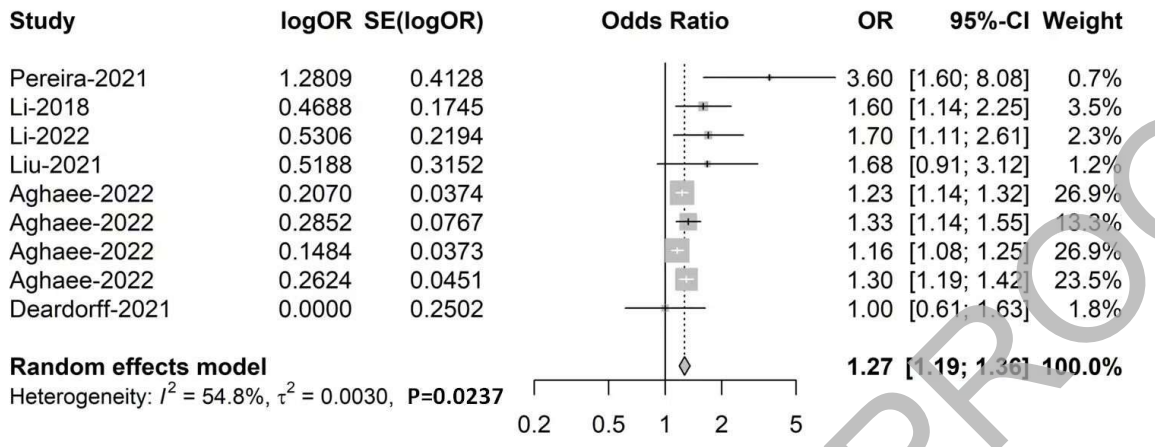

Figure 2. Forest plot of obesity (BMI-defined) and testicular enlargement

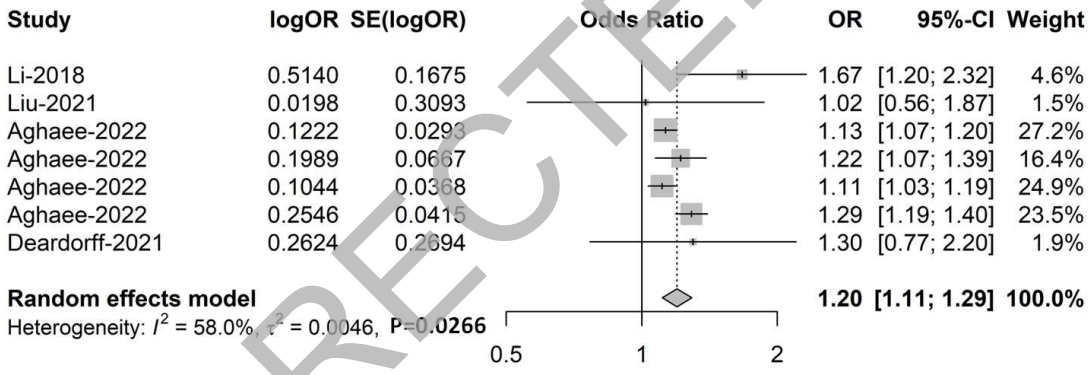

Figure 3. Forest plot of overweight (BMI-defined) and testicular enlargement

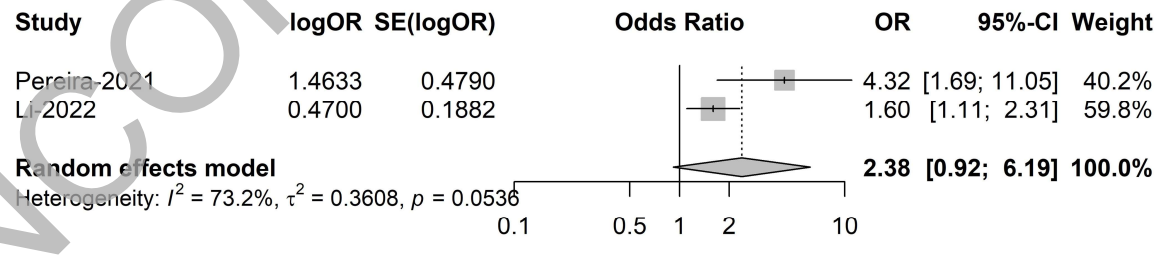

Figure 4. Forest plot of central obesity (waist circumference-defined) and testicular enlargement

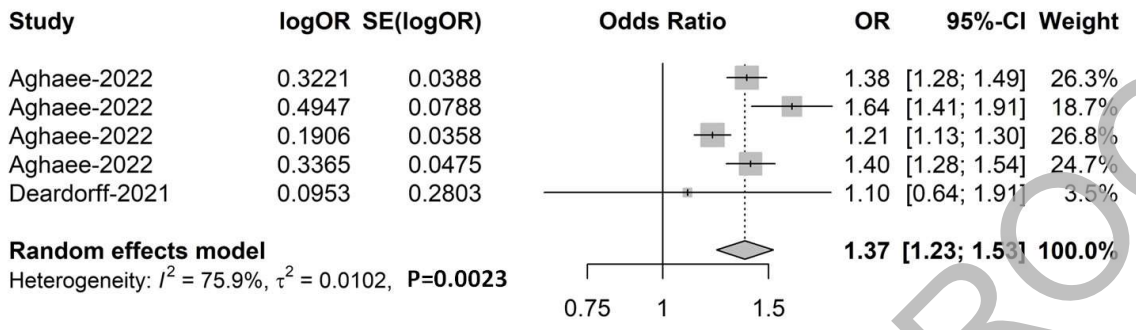

Figure 5. Forest plot of obesity (BMI-defined) and pubarche

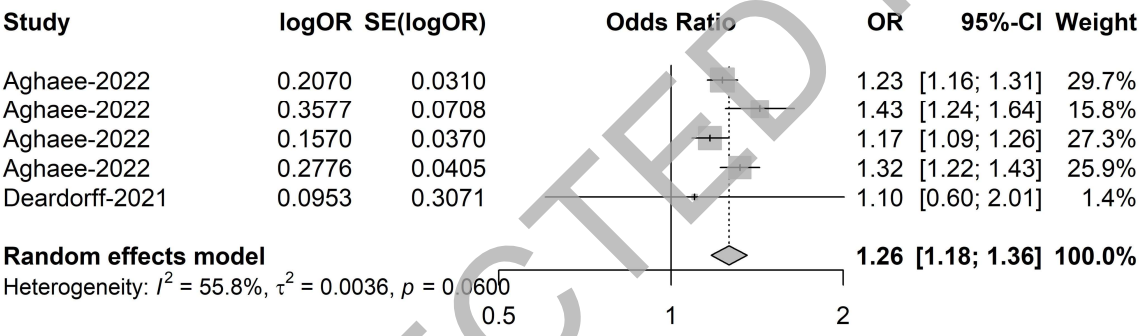

Figure 6. Forest plot of overweight (BMI-defined) and pubarche

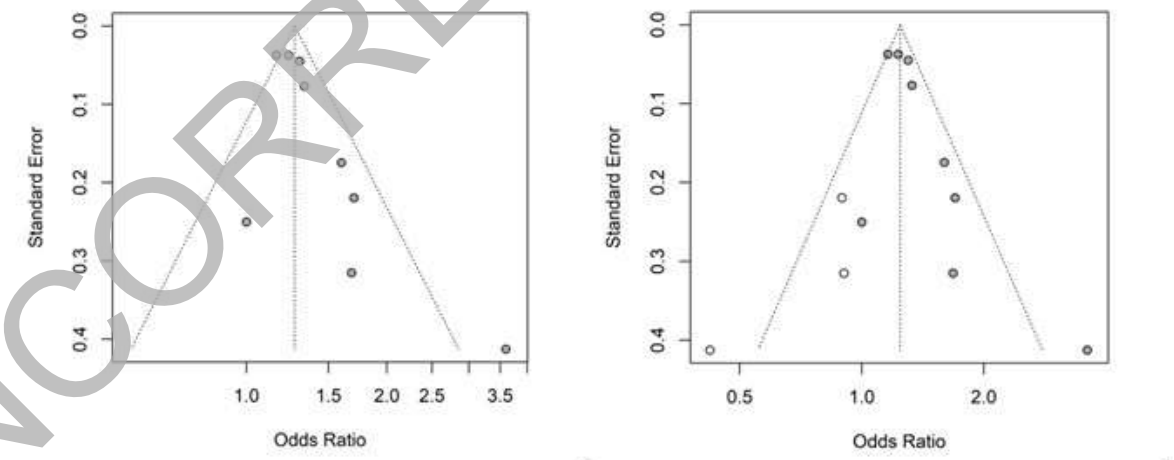

Figure 7. Funnel plot for publication bias assessment

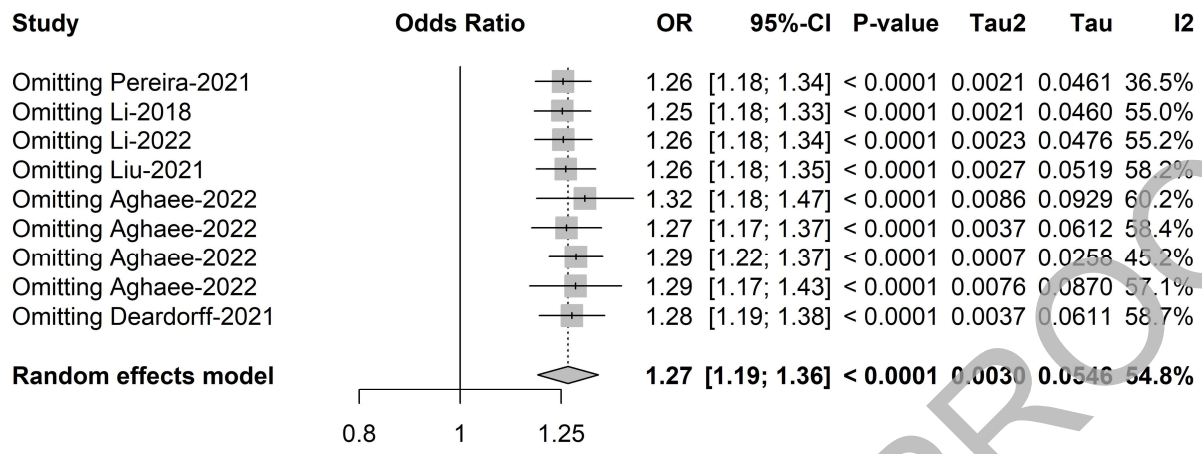

Figure 8. Sensitivity Analysis (Sequential Exclusion of Each Study Followed by Re-analysis)

Table 1.Characteristics of Studies Included in the Meta-Analysis

Study, year	Country	Ethnicity	Design	Sample Size	Obesity	Outcome
Pereira,2021	Chile	White	cohort	345	BMI>+2SD WC > 90th	Gonadarche
Li,2018	China	Asian	cohort	644	BMI>27.9	Gonadarche
Li,2022	China	Asian	cohort	645	BMI>27.9 WC≥90th	Gonadarche
Liu,2021	China	Asian	case-control	525	BMI>P95th	Central precocious puberty
Aghaee,2022	California	White, Black, Hispanic, Asian	cohort	62190	BMI≥P95 th	Gonadarche Pubarchec
Deardorff,2021	California	White	cohort	136	BMI≥P95	Gonadarche Pubarche

Table 2. Newcastle-Ottawa Scale (NOS) for Assessing the Methodological Quality of Cohort Studies

Study	Selection				Comparability	Outcome			Score
	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	
Pereira,2021	1	1	1	1	1	1	1	0	7
Li,2018	1	1	1	1	1	1	1	1	8
Li,2022	1	1	1	1	2	1	1	1	9
Aghaee,2022	1	1	1	1	1	1	1	1	8
Deardorff,2021	1	1	1	1	2	1	1	1	9