Review Article

The Interplay of Attention Deficit Hyperactivity Disorder (ADHD) and Obesity in Children and Adolescents: A Systematic Review

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ABSTRACT

Attention-Deficit/Hyperactivity Disorder (ADHD) and obesity are increasingly prevalent pediatric conditions with a suggested bidirectional link, though mechanisms remain unclear. This systematic review synthesizes recent evidence (2021–2026) on their interplay, examining prevalence, causality, mediators, moderators, and clinical implications. A PRISMA-guided search of PubMed, Scopus, PsycINFO, and Web of Science identified 18 studies, assessed for quality via the Newcastle-Ottawa Scale. Children with ADHD consistently show higher odds of overweight/obesity across regions. Genetic and Mendelian randomization studies confirm bidirectional causality, with shared genetic factors—especially among girls—explaining substantial phenotypic correlation. Key mediators include emotional problems (depression/anxiety), executive dysfunction (working memory, cognitive flexibility), and heightened delay discounting. Stimulant medication correlated with lower obesity risk in some groups, though metabolic monitoring is advised. Physical activity served as a protective moderator. The ADHD–obesity comorbidity is robust, bidirectional, and driven by shared genetic vulnerability, neurobehavioral phenotypes, and modifiable behaviors. Findings support integrated care models incorporating routine dual screening and early interventions targeting emotional regulation, executive functions, and lifestyle modifications.

Keyword: Attention Deficit Hyperactivity Disorder; ADHD; obesity; overweight; pediatric obesity; children; adolescents; comorbidity; executive function.

Introduction

The neurodevelopmental disorder known as Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by persistent patterns of inattention and/or impulsivity and hyperactivity that interfere with functioning or development in everyday settings [1]. Recent umbrella meta-analyses [2] suggest that the prevalence of ADHD among children and adolescents is 8.0% (95% CI 6.0–10.0%) in the world, more than double that of girls. Childhood obesity is one of the most significant public health challenges of the twenty first century, however, while this is happening. The WHO reports that there were more than 390 million children and youth aged 5 to 19 years

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who were overweight in 2022, with 160 million of them classified as obese [3]. Worryingly, the prevalence of adolescent obesity has increased by 4-fold globally since 1990 [3] and this is due to a complex transformation in dietary, physical activity and obesogenic factors [4]. In recent decades, the coexistence of ADHD and obesity has increased significantly, thus stimulating a large amount of research focus on the nature and mechanism of the comorbidity [5]. In children, there have been a growing number of epidemiological studies with consistent results demonstrating a positive association between ADHD and obesity. In the study by Zhu and colleagues, a total of 16 studies with 14,981 cases and 128,916 controls was used in a comprehensive systematic review and meta-analysis that demonstrated an increased risk of overweight and obesity in children and adolescents with ADHD of 56 compared to those without ADHD [6]. The links between ADHD and obesity are complex and multifactorial, involving genetic, neurobiological, behavioral and environmental factors. Twin studies have found that this association between ADHD symptoms and obesity can be partially attributed to shared genetic factors, particularly in adolescent girls [7- 9]. Neurobehaviorally both conditions are independently linked to impairments in executive functioning, increased delay discounting (preference for immediate rather than delayed rewards) and impaired impulse control [8]. These cognitive vulnerabilities could contribute to maladaptive eating behaviors like emotional eating, binge eating and eating when not hungry, which can cause excessive weight gain [9]. Furthermore, impairing good lifestyle practices (eating regular meals, engaging in regular physical activity, and getting adequate sleep) are symptoms of ADHD that can affect their development and upkeep of these healthy behaviors [5]. There is more to the clinical implications of ADHD-obesity comorbidity than just weight status. Children who are affected by both conditions are at significantly increased risk of later life cardiometabolic morbidity such as type 2 diabetes mellitus, dyslipidaemia, hypertension and, ultimately, of developing coronary artery disease [7]. Furthermore, there is an association between ADHD and obesity that is correlated with worse psychosocial outcomes, lower HRQOL, and higher utilization of health care services than ADHD alone [2]. While this association is increasingly acknowledged, large interstudy variability exists on the criteria and methods used to diagnose depression, the measurement of depression, and how confounding factors are controlled, making it difficult to reach any clear conclusions. Thus, the purpose of this systematic review is to provide a summary of the evidence over the last 5 years regarding the interrelationship between ADHD and obesity in children and adolescents.

Methods

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria [10] were followed in conducting this systematic review. Search Strategy: All the electronic databases (PubMed/MEDLINE, Scopus, PsycINFO and Web of Science) were searched comprehensively for relevant literature. Only a five-year period was searched. The search included free-text terms and controlled vocabulary (MeSH terms) related to two primary concepts: "Obesity" (which also covered the terms "overweight," "body mass index," "adiposity," and "central obesity") and "Attention Deficit Hyperactivity Disorder" (or "ADHD," "hyperkinetic disorder"). Boolean operators were used to combine terms (AND, OR). Exactly this was the search string used on PubMed: (Attention Deficit Disorder with Hyperactivity[Mesh] OR ADHD[tiab] OR "attention deficit"[tiab] OR "overweight"[tiab] OR "BMI"[tiab] OR "body mass index"[tiab] OR "waist circumference"[tiab] OR "adiposity"[tiab]). Only studies with accessible English abstracts were taken into consideration for preliminary screening, however there were no language constraints. The reference lists of included studies, and of relevant review articles, were manually searched for additional relevant studies. Eligibility Criteria: Studies were eligible to be included if they met the following criteria: (1) Population: children and adolescents aged 0 to 18 years (or mean/median age within this range); (2) Exposure: formal diagnosis of ADHD or validated assessment of ADHD symptoms; (3) Outcome: measures of overweight, obesity, or central adiposity (e.g., body mass index [BMI] percentile, BMI Z-score, waist circumference, waist-to-height ratio, body fat percentage); (4) Study design: observational studies (cohort, cross-sectional, case-control), genetic association studies, and interventional studies reporting baseline or longitudinal associations; (5) Publication period: published within the last 5 years. Studies were excluded if: (1) the study population consisted entirely of adults (≥ 18 years) without separate pediatric data; (2) editorials, commentaries, reviews were not original studies; (3) the study did not report original data (e.g., editorials, commentaries, reviews); (4) the full text was not available, even after attempting to contact authors. Definitions of

overweight and obesity: Throughout this review, the terms “overweight” and “obesity” are used according to the definitions applied in each included study and are not treated as interchangeable. Because body fat, height, and weight change rapidly during growth, pediatric overweight and obesity are best defined using dynamic, age- and sex-specific percentiles or growth charts. In children and adolescents aged 2 years or older, overweight is typically defined as a BMI between the 85th and <95th percentile for age and sex, and obesity as a BMI at or above the 95th percentile for age and sex. However, included studies also used other validated criteria, such as IOTF, WHO, CDC, BMI Z-scores, waist-to-height ratio, waist circumference, and body fat percentage. Prevalence estimates were therefore not pooled across studies using different criteria. Study Selection Process: All identified records were imported into Rayyan, a cloud-based platform designed for managing systematic review workflows [11]. The system's automated deduplication function was used to detect and eliminate duplicate entries, after which a manual cross-check was performed to confirm accuracy. Two independent evaluators then conducted an initial screening of titles and abstracts against the predefined selection criteria. Full-text versions of potentially eligible studies were subsequently retrieved and independently appraised by the same two reviewers. Any discrepancies during the selection process were settled through collaborative dialogue, with a third reviewer brought in when consensus could not be reached. Grounds for excluding articles at the full-text stage were systematically recorded and are illustrated in the PRISMA flowchart. Data Extraction: A customized data collection sheet was designed using Microsoft Excel to systematically capture key information from each eligible study. One investigator performed the initial data extraction, while a second reviewer independently cross-verified all entries to ensure completeness and correctness. The following variables were recorded: (a) bibliometric details (authors, publication year, country, research design, sample size, participant age range, gender breakdown, and other demographic characteristics); (b) ADHD diagnostic approaches (classification systems, rating instruments, and symptom-specific assessments); (c) anthropometric obesity indicators (body mass index, BMI Z-scores, percentile ranks, waist-to-height ratio, and estimated body fat percentage); (d) principal outcomes (odds ratios, effect estimates, confidence intervals, and probability values); (e) mediating variables, moderating factors, and adjusted confounders; and (f) disclosure of funding sources and potential conflicts of interest. When specific data points were unavailable, they were marked as "NM" (not mentioned) within the extraction tables. Given that the accessible information adequately addressed the review's research questions, no correspondence with study authors was undertaken to request unreported data. Quality Appraisal: The methodological rigor of each incorporated investigation was evaluated using the Newcastle-Ottawa Scale (NOS), with adaptations tailored to cohort, cross-sectional, and case-control designs [12]. Two evaluators independently assigned star ratings across three principal domains: participant selection (up to 4 stars), group comparability (up to 2 stars), and either outcome verification (for cohort investigations) or exposure ascertainment (for cross-sectional and case-control designs) (up to 3 stars). Aggregate scores were categorized as: 7–9 stars denoting good quality (low bias likelihood), 5–6 stars indicating fair quality, and 4 stars or fewer reflecting poor quality (elevated bias risk). Any rating discrepancies between reviewers were reconciled through discussion until mutual agreement was reached. Data Synthesis Strategy: Given the considerable variability across studies regarding methodological approaches, sampled populations, outcome definitions, and analytical techniques, conducting a quantitative meta-analysis was considered inappropriate. Consequently, a narrative descriptive synthesis was employed. The evidence was structured around five thematic domains: (1) prevalence estimates and obesity risk magnitude among ADHD populations; (2) evidence for genetic overlap and causal pathways; (3) mediating influences (affective disturbances, neurocognitive impairments, and temporal discounting); (4) effect modifiers (biological sex, chronological age, and behavioral lifestyle variables); and (5) the influence of stimulant pharmacotherapy on weight regulation.

Results

The PRISMA flowchart (Figure 1) outlines the sequential phases of record retrieval, screening, and final inclusion in this systematic review. Initial database queries yielded 423 citations. Following the removal of 269 redundant entries, 154 unique records advanced to title-and-abstract screening, from which 92 were discarded based on eligibility criteria. The remaining 62 full-text articles were requested, yet 38 were ultimately unobtainable. Of the 24 reports subjected to comprehensive full-text evaluation, one was excluded for reporting irrelevant outcomes, four were ineligible due to inappropriate study samples

(e.g., adult participants), and one was a conference abstract devoid of complete results. Ultimately, 18 investigations satisfied all predefined inclusion criteria and were incorporated into the final synthesis. (Table 1) summarizes the demographic and methodological characteristics of the 18 included studies. In the same order as the table, Pinhas-Hamiel et al. (2022) [13] conducted an Israeli nationwide population-based retrospective cohort (n=1,118,315 adolescents); Hané et al. (2025) [14] a cross-sectional case-control study (n=510, ages 6–12); Li et al. (2023) [15] a US cross-sectional NHIS analysis (n=35,108, ages 12–17); Merrill et al. (2021) [16] a US clinic-based cross-sectional study (n=1,003, mean age 12.55); Salama et al. (2025) [17] a retrospective chart review (n=330, ages 2–<18); Parhoon et al. (2023) [18] an Iranian cross-sectional case-control study (n=90, ages 8–13); Thapaliya et al. (2023) [19] a case-control study (n=323, ages 8–12); Karhunen et al. (2021) [20] a Finnish genetic association/longitudinal/Mendelian randomization study (n=2,984); Javaras et al. (2022) [21] a Swedish twin study (n=1,526, ages 16–17); Wei et al. (2022) [22] a Chinese retrospective cohort (n=677); Fast et al. (2021) [23] a Swedish retrospective medical record review (n=118, ages 6–17); Racicka-Pawlukiewicz et al. (2021) [24] a Polish cross-sectional case-control study (n=120, ages 8–17); Treister-Goltzman et al. (2025) [25] an Israeli nationwide cross-sectional study (n=603,552, ages 14–19); Guler and Turkoglu (2021) [26] a Turkish cross-sectional study (n=100 drug-naïve children); Xu et al. (2021) [27] a Chinese cross-sectional study (n=1,179, mean age 10.4); Manjarrez-Gutiérrez et al. (2026) [28] a cross-sectional study (n=89 school-aged children); García-Hermoso et al. (2024) [29] a US ambispective Add Health cohort (n=2,609 adolescents); and Yemelyantsava and Solntsava (2021) [30] a cross-sectional case-control study (n=315 children). The table shows varied settings, designs, sample sizes (89 to >1.1 million), ages (2–19 years), and male proportions (42–68% where reported), with race, socioeconomic status, and maternal education inconsistently reported. The main variables, the assessment methods used, and the main results of the 18 studies included (13–30) are summarized in (Table 2). There were differences in the methods used to diagnose or assess symptoms of ADHD in the studies, ranging from an expert diagnosis [13,17] to parent-reported diagnosis [15,16] to validated screening scales (e.g., the Adult ADHD Self-Report Scale [ASRS] [28] or the Child Behavior Checklist [CBCL] [22,30]), and to symptom-based criteria for hyperactivity/impulsivity and inattention [20,21,29]. The most commonly used obesity measure is mostly body mass index (BMI) or sometimes BMI Z scores or percentiles [13,15–17,19,21,23,25,29] along with other parameters like waist-to-height ratio (WHtR) [14], body fat percentage (BFP) using bioelectrical impedance (BIA) [14], waist circumference (WC) [22,29] and blood lipids [27]. There was a consistent positive association between ADHD and obesity across studies. An example is the report by Pinhas Hamiel et al. [13] who found that the prevalence of obesity was 13.5% for adolescents with severe ADHD contrasted with 7.5% for non ADHD adolescents (adjusted OR 1.77 for males and 2.09 for females). Hanć et al. [14] reported that the prevalence of obesity as measured by BMI was 3.19-fold higher in children with ADHD than in those without, and 2.67-fold higher for central obesity (WHtR); these differences were not significant after controlling for sociodemographic factors. ADHD obesity was partially mediated by emotional problems (depression and anxiety) [15]. In several studies, neurobehavioral phenotypes and executive functions were investigated; Parhoon et al. [18] found that children with ADHD who had obesity had worse working memory (d = -0.84), cognitive flexibility (d = 1.15), and attentional bias (d = 0.84) than children with ADHD alone. Thapaliya et al. [19] found that increased delay discounting is a common phenotype for ADHD and overweight/obesity while impairments in cognitive control were specific to ADHD regardless of weight status. Several studies listed individual or range of weights. Pinhas-Hamiel et al. reported a higher obesity prevalence rate in the severe ADHD group (13.5%) compared with their non-ADHD counterparts (7.5%) [13]. Hanč et al. found higher risks of obesity based on BMI (OR 3.19) and WHtR (OR 2.67) [14]. Salama et al. reported that BMI Z-score decreased following lisdexamfetamine treatment in severe obesity (BMI Z-score −0.41) and mild-to-moderate obesity (BMI Z-score −0.44) [17]. Treister-Goltzman et al. showed a dose–response relationship, Class 3 obesity being associated with ADHD (aOR 1.69 in Arab adolescents) [25]. After one year, 43% of children with overweight/obesity and ADHD were of normal weight, after receiving stimulant treatment (Fast et al.) [23]. In general, there were some associations for overweight and obesity, with the sizes of the associations being larger for obesity. Many studies were not able to provide separate estimates, so these results are merely descriptive.

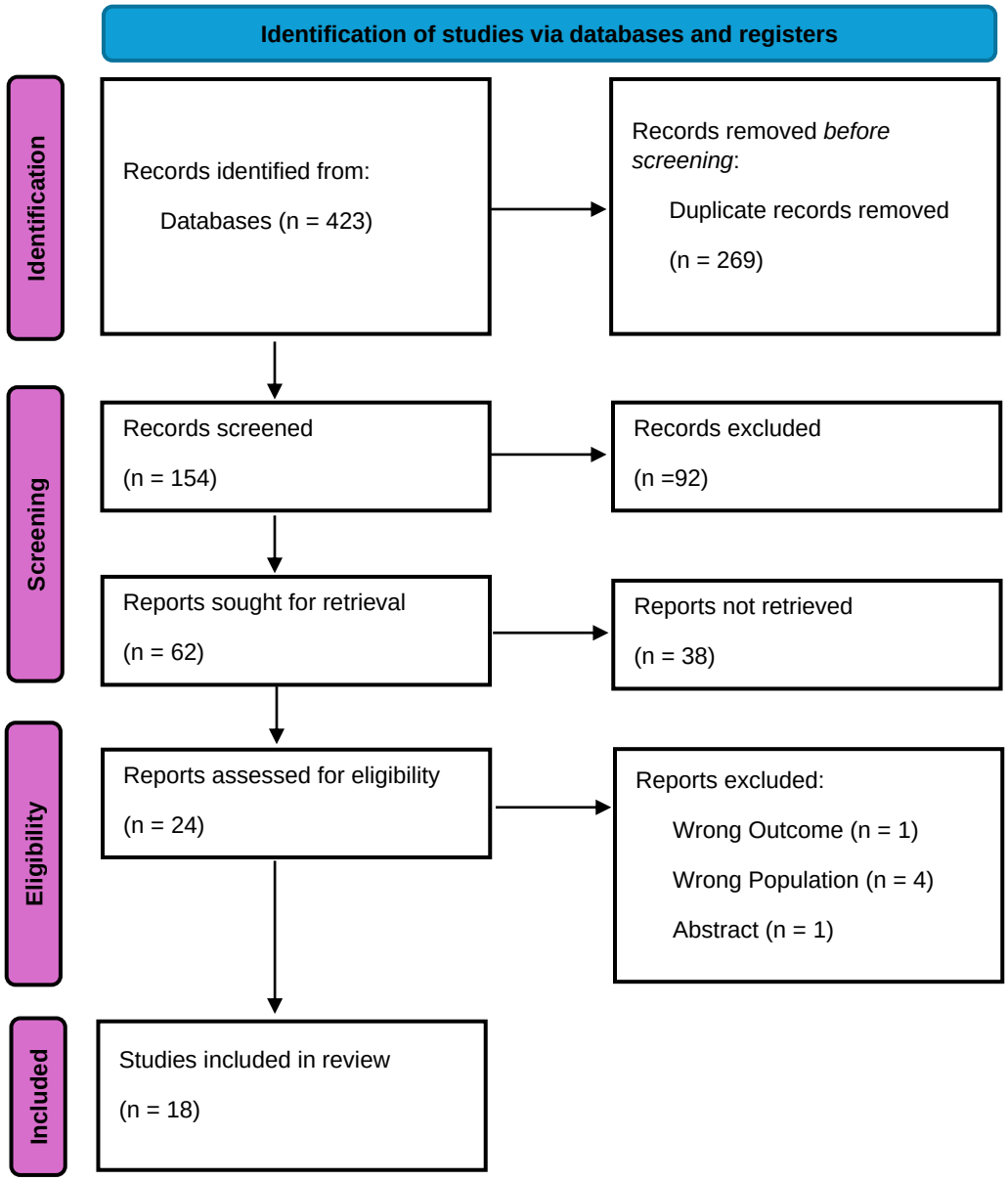

Figure 1:PRISMA Flow Diagram of Study Selection Process.

Table 1: Demographic and Methodological Characteristics of Included Studies (n=18).

Study (Author, Year, Ref.)	Place	Study Design	Sample Size	Sample Type	Age Range (years)	% Male	Other Demographic Information
Pinhas-Hamiel O et al., 2022 [13]	Israel	Nationwide population-based retrospective cohort	1,118,315	Adolescents from national database	Mean 17 (range NM)	57%	NM
Hanć T et al., 2025 [14]	NM	Cross-sectional case-control	510 (44 ADHD, 466 non-ADHD)	School-age children (clinical and community)	6–12	NM	NM
Li K et al., 2023 [15]	USA	Cross-sectional (NHIS data)	35,108 (weighted 46,550,729)	Nationally representative adolescents	12–17	NM	NM
Merrill BM et al., 2021 [16]	USA (southern)	Cross-sectional (clinic-based)	1,003	Treatment-seeking pediatric overweight/obesity sample	Mean 12.55 (range NM)	42%	67% Black/African American; 98.6% with obesity
Salama M et al., 2025 [17]	NM	Retrospective chart review	330	Children with ADHD and varied weight status	2–<18 (median 10, IQR 8.1-13)	68%	Included: severe obesity (n=55), mild-moderate obesity (88), overweight (131), healthy weight (56)
Parhoon K et al., 2023 [18]	Iran	Cross-sectional case-control	90	Children (clinical and community)	8–13	50%	Three groups: TD, ADHD+O, ADHD-O (30 each)
Thapaliya G et al., 2023 [19]	NM (likely USA)	Case-control	323	Children from clinic and community	8–12	NM	ADHD n=215, TD n=108; varied BMIz
Karhunen V et al., 2021 [20]	Finland	Genetic association / longitudinal (NFBC1986)	2,984	Population-based birth cohort	NM (longitudinal from birth)	NM	Used PRS and Mendelian randomization
Javaras KN et al., 2022 [21]	Sweden	Twin study (cross-sectional)	1,526 (791 girls, 735 boys)	Adolescent twins	16–17	48.1 %	Population-based twin registry
Wei X et al., 2022 [22]	China	Retrospective cohort	677	Chinese children	NM	NM	Examined MLBW and obesity
Fast K et al., 2021 [23]	Sweden (Gothenburg)	Retrospective medical	118	Children with ADHD (6–17 years)	6–17	NM	Overweight/obesity subgroup n=44

		record review					
Racicka-Pawlukiewicz E et al., 2021 [24]	Poland	Cross-sectional case-control	120 (58 ADHD, 62 controls)	Children and adolescents	8–17	NM	Compared three obesity criteria (IOTF, WHO, ECOG)
Treister-Goltzman Y et al., 2025 [25]	Israel	Cross-sectional nationwide	603,552 (313,936 Arab, 289,616 Jewish)	Adolescents	14–19	NM	Ethnic comparison; dose-response analysis
Guler HA & Turkoglu S, 2021 [26]	Turkey	Cross-sectional	100 (70 ADHD, 30 controls)	Drug-naïve children	NM (children)	NM	Divided ADHD by BMI ≥85th percentile
Xu Y et al., 2021 [27]	China	Cross-sectional	1,179	Children (obesity-stratified)	Mean 10.4 (range NM)	50.3 %	Examined blood lipids in obese children
Manjarrez-Gutiérrez G et al., 2026 [28]	NM	Cross-sectional	89	School-aged children (20 controls, 69 ADHD)	School-aged (NM)	NM	Measured plasma serotonin
García-Hermoso A et al., 2024 [29]	USA	Ambispective cohort (Add Health Study)	2,609	Adolescents followed to adulthood	Adolescent (range NM, grades 7-12)	NM	Examined active lifestyle as moderator
Yemelyantsava T & Solntsava A, 2021 [30]	NM	Cross-sectional case-control	315 (126 obesity, 111 ADHD, 78 controls)	Children	NM (children)	NM	Used CBCL/6-18 for functional assessment

NM = Not mentioned.

Table 2: Key Variables and Main Findings of Included Studies (n=18).

Study (Author, Year, Ref.)	**ADHD Assessment**	**Obesity Measures**	**Key Findings (ADHD-Obesity Relationship)**	**Mediators / Moderators / Covariates**	**Additional Notes**
Pinhas-Hamiel O et al., 2022 [13]	Specialist diagnosis (neurology/psychiatry); severe vs. mild	BMI (obesity defined as ≥95th percentile?)	Severe ADHD: 13.5% obesity vs 7.5% non-ADHD; adjusted OR for obesity (severe ADHD): males 1.77, females 2.09	Adjusted for NM	Both severe and mild ADHD at increased risk
Hanć T et al., 2025 [14]	Clinical diagnosis	BMI, WHtR, body fat % (TANITA)	Higher obesity by BMI (OR 3.19) and central	Parental education, SES,	ADHD linked to abdominal

			obesity by WHtR (OR 2.67); no difference in %Fat	adverse childhood experiences (lost significance after adjustment)	fat, not total fat
Li K et al., 2023 [15]	Parent-reported previous ADHD diagnosis	BMI (obesity: ≥95th percentile)	ADHD predicted obesity; partial mediation by depression (β=0.26-0.28) and anxiety (β=0.28)	Age, sex, race, income, physical activity, etc.	Emotional problems mediate the association
Merrill BM et al., 2021 [16]	Parent-reported ADHD diagnosis	BMIz (standardized)	ADHD prevalence 17.6% (higher than national); ADHD × race interaction for BMIz; rurality predicted higher BMIz	Race, rurality	ADHD linked to more meals/day, fewer active days
Salama M et al., 2025 [17]	Clinical diagnosis (ADHD) treated with lisdexamfetamine	BMI Z-score, BMI% of 95th percentile	At 2 years: BMI Z-score decreased -0.41 (severe obesity) and -0.44 (mild-moderate); younger children (4-10) greater reduction	Age	Stimulant treatment associated with weight loss
Parhoon K et al., 2023 [18]	Clinical diagnosis (ADHD)	Obesity defined (not specified)	ADHD+O worse than ADHD-O on Digit Span (d=-0.84), WCST, Stroop, Dot-Probe, delay of gratification	None	Cool EF deficits more pronounced with comorbid obesity
Thapaliya G et al., 2023 [19]	Clinical diagnosis (ADHD)	BMIz (overweight/obesity based on CDC)	BMIz × ADHD interaction for game time DD: TD overweight/obese similar to ADHD; poor CC specific to ADHD, not weight	None	Shared phenotype: delay discounting; distinct: cognitive control
Karhunen V et al., 2021 [20]	ADHD symptoms (longitudinal) and PRS	BMI, obesity-related traits	Bidirectional causality (MR); PRS overlap; maternal pre-pregnancy BMI associated with	PRS for both traits, prenatal environment	Genetic and prenatal origins

			offspring ADHD symptoms		
Javaras KN et al., 2022 [21]	Parent-reported ADHD symptoms (inattention, hyperactivity/impulsivity)	Overweight/obese status (parent-reported height/weight)	Phenotypic correlations: inattention r=0.30 (girls), 0.08 (boys); hyperactivity r=0.23 (girls), 0.14 (boys). Shared genetic effects explain most correlation in girls	Sex-specific	Stronger association in girls; genetic overlap
Wei X et al., 2022 [22]	CBCL or clinical diagnosis? (ADHD)	BMI, WC, skinfold, body fat, etc.	MLBW associated with higher obesity outcomes; mediation by ADHD and emotional overeating	BMI Z-score	ADHD and abnormal eating mediate MLBW-obesity link
Fast K et al., 2021 [23]	Clinical ADHD diagnosis (DSM)	BMI SDS (standard deviation score)	BMI SDS decreased -0.72 during treatment vs +0.17 year before; 43% of overweight/obese reached normal weight after 1 year	None (internal control)	Stimulant treatment yields significant weight loss
Racicka-Pawlukiewicz E et al., 2021 [24]	Clinical ADHD diagnosis (DSM)	Three criteria: IOTF, WHO, ECOG	Higher obesity in ADHD by WHO (17.2% vs 3.2%) and IOTF (10.3% vs 1.6%), not ECOG; higher than Polish population	ADHD treatment (no significant relationship)	Prevalence varies by obesity diagnostic criteria
Treister-Goltzman Y et al., 2025 [25]	Recorded diagnosis of ADHD	Weight categories (normal to class 3 obesity)	Dose-dependent: class 3 obesity aOR for ADHD = 1.69 (Arab) and comparable in Jews; stronger in Arab females	Age, sex, ethnicity	Comorbidity increases with obesity severity
Guler HA & Turkoglu S, 2021 [26]	Clinical diagnosis (DSM), drug-naïve	BMI (≥85th percentile = overweight/obese)	No significant differences in executive function (Stroop, Cancellation, SDLT) or ADHD severity between ADHD+O and ADHD-O	None	Comorbid obesity not associated with cold EF or symptom severity

Xu Y et al., 2021 [27]	Clinical diagnosis of ADHD	BMI (obese vs non-obese stratified)	Elevated total cholesterol, TG, LDL and reduced HDL associated with ADHD in obese children (adjusted OR up to 2.016)	Age, sex, BMI, maternal smoking, education, etc.	Blood lipid abnormalities linked to ADHD in obese children
Manjarrez-Gutiérrez G et al., 2026 [28]	Clinical diagnosis (DSM)	Overweight/obesity (not specified)	Elevated plasma free 5-HT (serotonin) associated with ADHD (OR 1.71 per 0.1 μmol/L) and severity (AUC 0.93 for moderate/severe)	Age, obesity	Plasma serotonin as potential biomarker
García-Hermoso A et al., 2024 [29]	Self-reported ADHD symptoms (hyperactivity/impulsivity, inattention)	BMI, WC (obesity and abdominal obesity)	Inactive individuals with ADHD had higher obesity risk (OR 1.56-2.63); active lifestyle attenuated risk	Physical activity (mediation analysis)	Physical activity reduces obesity risk in ADHD
Yemelyantsava T & Solntsava A, 2021 [30]	CBCL/6-18 (ADHD symptoms)	Alimentary vs morbid obesity	38.7% of ADHD children had obesity; morbid obesity associated with temperament disorders ($p<0.001$) and anxiety/depression ($p<0.01$)	Sex (male $p<0.01$)	Prognostic model for morbid obesity in ADHD (AUC=0.71)

NM = Not mentioned; EF = executive functions; CC = cognitive control; PRS = polygenic risk score; MR = Mendelian randomization; MLBW = marginally low birth weight.

Table 3: Risk of Bias Assessment of Included Studies (n=18) Using the Newcastle-Ottawa Scale (NOS).

Study (Author, Year, Ref.)	**Selection (max 4 ★)**	**Comparability (max 2 ★)**	**Outcome / Exposure (max 3 ★)**	**Total Stars (max 9)**	**Overall Risk of Bias**
Pinhas-Hamiel O et al., 2022 [13]	★★★★	★★	★★★	9	**Good**
Hanć T et al., 2025 [14]	★★★	★★	★★★	8	**Good**
Li K et al., 2023 [15]	★★★★	★★	★★★	9	**Good**
Merrill BM et al., 2021 [16]	★★★★	★★	★★★	9	**Good**
Salama M et al., 2025 [17]	★★★	★★	★★	7	**Good**

Parhoon K et al., 2023 [18]	★★★	★	★★	6	**Fair**
Thapaliya G et al., 2023 [19]	★★★	★	★	5	**Fair**
Karhunen V et al., 2021 [20]	★★★★	★★	★★★	9	**Good**
Javaras KN et al., 2022 [21]	★★★★	★★	★★★	9	**Good**
Wei X et al., 2022 [22]	★★★	★	★★	6	**Fair**
Fast K et al., 2021 [23]	★★★	★	★★	6	**Fair**
Racicka-Pawlukiewicz E et al., 2021 [24]	★★★	★	★	5	**Fair**
Treister-Goltzman Y et al., 2025 [25]	★★★★	★★	★★★	9	**Good**
Guler HA & Turkoglu S, 2021 [26]	★★	★	★	4	**Poor**
Xu Y et al., 2021 [27]	★★★	★★	★★★	8	**Good**
Manjarrez-Gutiérrez G et al., 2026 [28]	★★★	★★	★★	7	**Good**
García-Hermoso A et al., 2024 [29]	★★★★	★★	★★★	9	**Good**
Yemelyantsava T & Solntsava A, 2021 [30]	★★★	★	★★	6	**Fair**

Discussion

The assembled evidence consistently indicates that ADHD confers a noticeable, though modest, increase in susceptibility to overweight and obesity, with this pattern observable across varied demographic groups and geographical territories. This observed association confirms, and builds upon, earlier research that has highlighted the common co-occurrence of these two common childhood health problems. In a recent quantitative synthesis, Zhu et al. (2002) analyzed 16 investigations with 14,981 children and adolescents with ADHD as well as 128,916 controls, and reported a 1.56-fold greater risk of being overweight and obese (BMI > 85% percentile) in children with ADHD compared to their non-ADHD counterparts. Importantly, the size of the effect was found to be very dependent on the geographic area, with an effect seen in Asian populations but less marked in European populations [31]. These inter-regional differences may be attributed to different diagnostic methods, differences in culture, exercise and food habits and differences in genetic background of populations. These pooled estimates are supported by the findings of the current review, which were based on data from Israeli [13,14], Swedish [21,22], Chinese [24,26–28], Polish, and Turkish cohorts [26–28]. Furthermore, the aforementioned meta-analysis reported sex differences, with the female sex showing a significant, but weak association between ADHD symptoms and higher BMI (OR 1.45; 95% CI 1.10–1.90) [31], which is also consistent with the Swedish twin study included in this review, which found more robust phenotypic correlations between BMI and ADHD symptoms in females (r = 0.30 for inattention) compared to males (r = 0.08) [21]. Such gender differences highlight the need to explore whether there are hormonal, psychosocial, and/or behavioral mechanisms that may be different in boys and girls with ADHD that increase vulnerability to obesity. Recently, genetic and Mendelian randomization (MR) studies have lent increasing support to the bi-directional and potentially causal relationship between ADHD and obesity. To examine the causal association between BMI and ADHD, as well as the causal association between obesity and ADHD, Chen et al. used genome-wide association study data of 694,649 individuals of European ancestry to perform a comprehensive two-sample MR analysis [32] and identified that higher BMI were causally associated with an increased risk of ADHD, and higher obesity risk was causally

associated with ADHD, indicating bidirectional causal relationships between obesity and ADHD. The results of the present investigation are very similar to the genetic analyses that were included within this review, such as the study by Karhunen et al. [20] who used both Mendelian randomization and polygenetic risk score approaches to determine whether genetic susceptibility to ADHD or BMI was linked to the other, and found evidence for both reciprocal causal connections and shared genetic determinants between these two traits. Similarly, the studies of Javaras and co-investigators [21] demonstrated that the predominant component of the phenotypic relationship between ADHD features and overweight/obesity among female adolescents was heritable; this confirmed a possible common genetic basis for both disorders. More recently, Zhang and colleagues (2024) explored the direction of causality specifically between childhood obesity and ADHD using MR methods, further supporting the bidirectional pathway [33]. These genetic findings have significant clinical implications: First, treatments administered to children to promote weight loss in those who are obese could potentially also reduce the symptoms of ADHD; second, successful treatment of ADHD, especially using drugs that affect appetite and weight, may also reduce obesity risk. The shared genetic susceptibility also underscores the need to screen for both these diseases at an early stage in high-risk populations. In addition to the genetic risk factors, the present review revealed that there are several important mediating and moderating mechanisms that help explain the heightened risk of obesity associated with ADHD. In a large representative sample of adolescents in the United States, emotional problems (specifically depression and anxiety) were important partial mediators of the relationship between ADHD and obesity ($\beta = 0.28$ for depression and $\beta = 0.28$ for anxiety) [15]. This finding is in line with the psychiatric literature on ADHD, which found that this condition often co-occurs with internalizing disorders, and that emotional dysregulation can result in maladaptive coping mechanisms, including emotional eating, decreased physical activity, and disturbed sleep habits, all of which contribute to weight gain. Yemelyantsava and Solntsava also showed that only temperament disorders ($p<0.001$) and anxiety/depression ($p<0.01$) are associated with morbid obesity in children with ADHD and a prognostic model has adequate predictive accuracy (AUC = 0.71) [30]. Another important pathway connecting ADHD to obesity is associated with EF deficits. Children with comorbid ADHD–obesity had significantly poorer performance on both "cool" executive functions (working memory, cognitive flexibility, interference control) and "hot" executive functions (episodic foresight, delay of gratification) than children with ADHD alone (moderate to large effect sizes: $d = -0.84$ to $d = 1.38$) [18]. To expand on these findings, Thapaliya et al. showed that increased delay discounting—taking immediate rewards over longer-term rewards—may be a common neurobehavioral trait across ADHD and overweight/obesity, while deficits in cognitive control are more specific to ADHD regardless of weight status [19]. The results indicate that interventions focused on executive function training, emotion regulation, and reward-based decision making could have two effects: that of lowering ADHD symptoms and that of managing weight. Pharmacological interventions and the role they play in the modification of obesity risk among children with ADHD should be carefully evaluated, as the results of the present review and those of the literature indicate a conflicting and complex profile. One of the studies included in this analysis found that children who used lisdexamfetamine were significantly less likely to be overweight or obese after two years of treatment compared to their non-treatment counterparts, with a decrease of -0.41 and -0.44 BMI Z scores in the severe and mild-moderate obesity groups, respectively [17]. In children with overweight or obesity and ADHD, researchers at Fast and colleagues also reported that 43% of the children achieved a normal weight after one year of stimulant therapy [23]. This study is consistent with a large cross-sectional study of 547 children aged 6–12 years by Aykutlu et al. (2024) which reported the highest rate of obesity in drug-naïve children with ADHD (13.8%), followed by stimulant-only therapy (4.5%), and healthy children (8.6%) [34]. This indicates that stimulant drugs, because they have an appetite suppressive effect and maybe beneficial in impulse control and dietary choice, could have a protective effect against obesity. There is conflicting evidence, however: Kousha and colleagues (2024) conducted a longitudinal study of 149 children and adolescents prescribed methylphenidate, reporting that children who were prescribed methylphenidate for longer periods of time gained more weight ($p < 0.001$) [35] – suggesting that there is a risk of weight rebound or metabolic adaptation with longer duration of treatment. Further, the findings of Roberts and colleagues (2024), which were based on data from 26,644 adolescents who

participated in the National Survey of Children's Health, noted that a 7% greater prevalence of overweight or obesity was found among adolescents with ADHD who were not taking medication compared to those who were. But, when those taking medication were controlled for household poverty, factors like physical activity, nightly sleep duration and screen time did not predict obesity status, suggesting that medication may not be enough to change obesogenic behaviors [36]. These conflicting findings highlight the need for individualised therapeutic management and careful monitoring of body weight during the treatment of ADHD with stimulants, particularly because of emerging issues about the metabolic side effects of chronic stimulant use, as well as the multi-fold risks associated with concurrent use of atypical antipsychotic agents [34]. Limitations: There are several limitations of this systematic review. Furthermore, most studies in the included studies used a cross-sectional design [14-16, 18-19, 21, 24, 26-28, 30] which would not allow causal inferences to be drawn, although some studies were longitudinal and genetic [13, 17, 20, 22, 23, 25, 29] suggesting a bidirectional relationship. Second, significant methodological variability was observed among the different methods used to assess ADHD and obesity, such as the use of different criteria to diagnose obesity as pointed out by Racicka-Pawlukiewicz et al. [24] which made it difficult to compare findings across studies. Third, many studies were not adequately adjusted for confounders including SES, parental education, adverse childhood experiences and lifestyle factors, and in one study the obesity-ADHD relationship was not significant after adjusting for confounders [14] showing potential for residual confounding. Fourth, geographic generalizability is constrained by an overrepresentation of wealthy countries. Fifth, the risk of bias assessment (Table 3) revealed several "poor" quality studies [19,24,26] and sensitivity analyses not based on these studies did not change overall results; however, there may be some bias. Lastly, there may be publication bias in favour of positive associations.

Conclusion

There is a small but significant association between ADHD and obesity among youth, and both may share the same genetic risk factors, as well as emotional and executive issues, delay discounting, and lack of physical activity. Stimulants can reduce obesity risk, but need metabolic monitoring. Results confirm the need for combined care for dual screening in ADHD and weight clinics, and early intervention for emotional regulation and lifestyle change. A longitudinal study in a variety of populations is required to elucidate mechanisms and optimize treatments specifically.

Conflict of Interest

None

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None

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