İSTANBUL UNIVERSITY PRESS

Çocuk Dergisi–Journal of Child,
JCHILD 2025, 25 (3): 180–186

https://doi.org/10.26650/jchild.2025.1717390

Submitted: 11.06.2025
Revision Requested: 14.07.2025
Last Revision Received: 18.07.2025
Accepted: 22.07.2025

Çocuk Dergisi
Journal of Child

Review Article

Open Access

Adolescent Obesity: A Global Problem And Solutions Adolescent Obesity

Eren Güzeloğlu [1], Belen Ateş [1], Mustafa Özdemir [1], Hüseyin Dağ [1] & Vefik Arıca [1]

[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye

Abstract Obesity during adolescence is an increasingly prevalent issue both globally and within our country, posing a significant threat to public health. Far from being merely an esthetic concern, obesity can have lasting adverse effects on the physical and psychosocial well-being of children. This review presents a comprehensive synthesis aimed at understanding adolescent obesity, examining its underlying causes, assessing its impact, and proposing potential solutions. A search was conducted on PubMed with the keyword "Adolescent obesity" for the period between 2020 and 2025. The search yielded 16,479 studies on the topic, of which 2,233 were reviews or systematic reviews. From this body of literature, studies deemed current and relevant to the scope of this review were selected for inclusion. Adolescent obesity is a preventable and treatable condition when addressed through early intervention. In adolescents diagnosed with obesity, the etiology should first be identified, followed by the development and implementation of personalized intervention strategies. Promoting healthy eating habits and an active lifestyle remains the most effective approach in combating obesity.

Keywords Adolescent · Obesity · Diagnosis · Prevention · Management

Citation: Güzeloğlu, E., Ateş, B., Özdemir, M., Dağ, H. & Arıca, V. Adolescent Obesity: A Global Problem And Solutions Adolescent Obesity. Çocuk Dergisi–Journal of Child 2025; 25(3): 180-186. DOI: 10.26650/jchild.2025.1717390

Corresponding author: Eren Güzeloğlu dr.erenguzeloglu@gmail.com

INTRODUCTION

In recent years, adolescent obesity has been rising rapidly both in our country and globally, emerging as a widespread public health concern. Obesity represents a clinical condition associated with high lifelong morbidity—including cardiovascular diseases, type 2 diabetes mellitus, and metabolic syndrome—as well as increased mortality. Therefore, its prevention and management are of critical importance for public health. (1-4).

Adolescent obesity is not merely a concern for teenagers; it can result in lifelong health complications. It poses substantial long-term challenges for individuals, families, and caregivers, while also placing a considerable economic burden on the government. Effective prevention and control of adolescent obesity are essential steps toward fostering healthier future generations. (1-4).

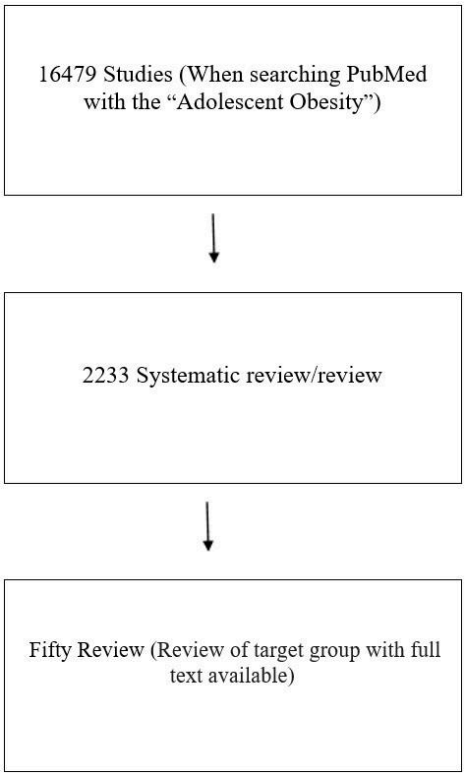
In recent years, the prevalence of obesity during adolescence has been rising both globally and within our country. This trend is also evident in our clinical practice, where an increasing number of adolescents are presenting with obesity. In response to this growing concern, a review was conducted based on studies published on PubMed over the past five years, including reviews, systematic reviews, meta-analyses, clinical trials, and intervention studies. A PubMed search using the keyword "adolescent obesity" for the period between 2020 and 2025 identified 16,479 studies on the topic, 2,233 of which were classified as reviews or systematic reviews. From this literature, studies deemed current and relevant to the scope of this review were selected for inclusion.

In this review, we aimed to reveal the genetics and risk factors, comorbidity, and prevention and management of adolescent obesity.

The Obesity Epidemic

Adolescent obesity is a serious public health problem that is common worldwide and has high mortality and morbidity rates. In the United States, approximately 21% of adolescents aged 12 to 18 are classified as obese. Globally, as of 2022, 43% of individuals over the age of 18 were considered overweight, and 16% were living with obesity. The global prevalence of obesity among children and adolescents aged 5 to 19 years has increased from 1.9% in 1990 to 8.2% in 2022. In the same year, 37 million children under the age of five were overweight worldwide; 390 million children and adolescents aged 5 to 19 were overweight, and 160 million were living with obesity (1–4). According to the Türkiye Nutrition and Health Survey, the prevalence of overweight and obesity among individuals over the age of 15 in our country was reported as 34% and 31.5%, respectively (5).

Figure 1. Review Selection



Genetics

Syndromic obesity is observed in conditions such as Prader-Willi Syndrome and Bardet-Biedl Syndrome. In non-syndromic obesity, it is thought that monogenic or multigenic inheritance may play a role. Several genetic loci have been implicated in the pathogenesis of obesity, including MC4R (melanocortin 4 receptor), BDNF (brain-derived neurotrophic factor), SH2B1, POMC (proopiomelanocortin), LEP (leptin), LEPR, NPY, SIM1 (single-minded homolog 1), NTRK2, and PCSK1. The fat-mass and obesity-associated gene (FTO) is thought to be particularly effective in polygenic and epigenetic obesity. (6-8).

Risk Factors

The development of adolescent obesity is affected by the duration of exclusive breastfeeding, foods given in complementary feeding, parenting style, nutritional behavior (such as skipping breakfast and eating only dinner), heavy consumption of sugary drinks and fast foods, food selection that will strengthen the intestinal microbiota (prebiotics, probiotics and synbiotics, etc.), physical activity and a sedentary lifestyle (9). Encouraging children to consume

vegetables and fruits in particular may be effective in preventing adolescent obesity (10).

Leptin, a satiety hormone produced by fat cells, holds promise as a valuable biomarker for predicting obesity in both adolescence and adulthood. Leptin concentrations are elevated in individuals with obesity. High serum leptin levels are believed to reduce soluble leptin receptor (sOB-R) concentrations and are associated with leptin resistance. In the development of obesity, the interaction of hormones such as adiponectin, ghrelin, and insulin, as well as the leptin hormone, plays a decisive role (11-13).

Adverse childhood experiences (ACEs) are associated with various physical and mental health problems in adolescents and adults. Studies have shown that ACEs are associated with adolescent obesity. Girls are considered more susceptible to the obesity-related effects of ACEs than boys, and sexual abuse appears to have a greater impact on adolescent obesity than other ACEs. The occurrence of multiple ACEs together may be associated with a higher risk of adolescent obesity. The effects of ACEs on the development of adolescent obesity may take 2 to 5 years to manifest. The data suggest a positive relationship between the ACE score and BMI. Individuals with severe obesity are more likely to have a high ACE risk, with approximately 50% affected, compared to 24%–25% among those without severe obesity (14-16).

In addition to the immediate negative outcomes, adolescent sexual abuse is associated with lifelong harmful mental and physical health outcomes. The biological embedding of adolescent sexual abuse often triggers a series of interrelated conditions that lead to the failure of weight suppression efforts and ultimately obesity. Such biological embedding involves pathways such as inflammation, allostatic load, reward sensitivity, activation of the hypothalamic-pituitary-adrenal axis, epigenetics, and structural and functional changes in the brain. Severe abuse was associated with a higher adult BMI than no abuse, corresponding to a 46% increased risk of obesity (17-20). A study conducted with mothers and babies from 12 countries found that babies who were exclusively breastfed were less likely to be obese. As the duration of breastfeeding (up to 12 months) increased, the likelihood of obesity in babies decreased (21).

Comorbidites

Adolescent obesity can lead to morbidities such as type 2 diabetes mellitus, cardiovascular diseases, and metabolic syndrome in adulthood and can cause early-onset type 2 diabetes mellitus in adolescence. Adolescents living with obesity are at risk of lifelong cardiovascular diseases (22-23).

The association between adolescent obesity and central precocious puberty has long been confirmed, but the mechanisms underlying this association remain unclear. Obesity during adolescence is also associated with menstrual irregularity and polycystic ovary syndrome (PCOS), which can cause infrequent or absent menstrual periods and heavy menstrual bleeding (24-25).

Nonalcoholic fatty liver disease (NAFLD) occurs in approximately 36.1% of cases of obesity in adolescents. This rate is expected to increase with the global obesity epidemic (26-27).

Adolescent obesity has been linked to various factors, including air pollution, exposure to metabolism-disrupting chemicals, excessive consumption of ultra-processed foods, alterations in the gut microbiota, and a sedentary lifestyle. These factors are associated with an increased risk of obesity, insulin resistance, type 2 diabetes, and disruptions in lipid metabolism. (28-29).

Cao et al. found a causal relationship between obesity and osteoarthritis. The prevalence of osteoarthritis in the population will rise in parallel with the increasing rates of obesity. (30).

Obesity and asthma are both common diseases with a high population burden worldwide. Recent genetic association studies have shown that obesity is associated with asthma in adults. The relationship between obesity and asthma and the underlying mechanisms linking obesity to asthma have not yet been elucidated (31-32).

Pourghazi et al. reported a significant positive association between higher BMI levels and a higher risk of kidney disease later in life. The findings from several sections suggest a positive association between obesity in early-life kidney disease in later life (33).

The risk of obstructive sleep apnea syndrome (OSAS) is associated with body mass index (BMI), where OSAS increases progressively with increases in BMI and is likely related to upper airway narrowing due to excess adipose tissue. Because of this association, the countries with the highest incidence of OSAS are those with high rates of obesity; therefore, the incidence of OSAS increases as the obesity levels increase (34).

Depression is considered one of the most significant factors contributing to the development of obesity. Conversely, obesity may also contribute to the onset of depression. Numerous studies have highlighted a positive association between obesity and depression; however, it remains unclear whether obesity leads to depression or if depression leads to obesity. (35).

Obesity causes increased secretion of proinflammatory cytokines such as interferon-γ (IFN-γ), interleukin 6 (IL-6), and tumor necrosis factor-α (TNF-α) and infiltration of inflammatory immune cells into adipose tissue. Epidemiological studies have shown a strong association between children with a higher body mass index (BMI) during adolescence and an increased risk for malignancies in adulthood, including leukemia, Hodgkin disease, colorectal cancer, and breast cancer (36-37).

Prevention and Management

Lifestyle changes, a healthy diet, and increased physical activity are the foundation of treatment for adolescent obesity. In some severe cases, medication and bariatric surgery may be necessary. Social and policy changes regarding appropriate dietary recommendations and age-appropriate physical activities for adolescents can protect and prevent the increasing obesity epidemic (38).

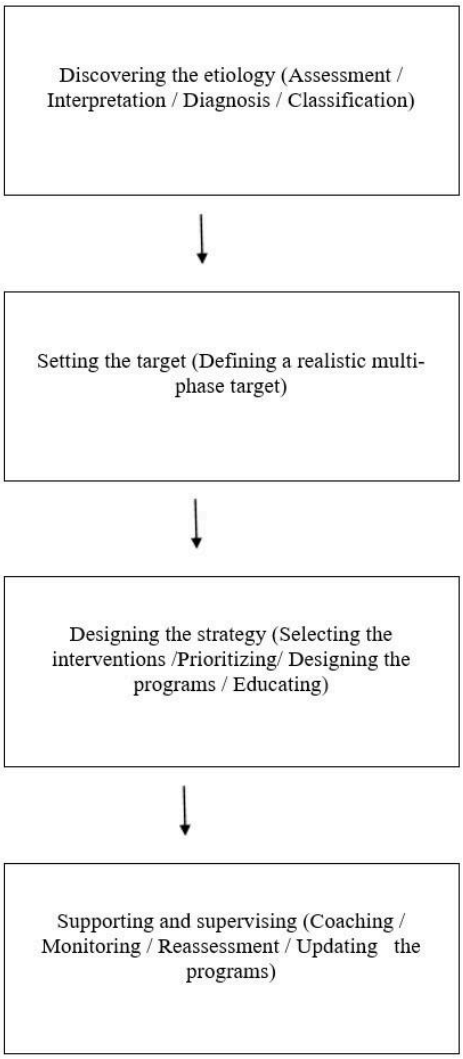
In an intervention study conducted by Flynn and colleagues, various settings were utilized, including schools/nurseries, communities, hospitals, e-health platforms, and mixed environments. The findings indicated that interventions implemented in the home environment and those involving parents or families were more effective in preventing adolescent obesity (39).

The EPISTCO (Etiology-Based Personalized Intervention Strategy Targeting Childhood Obesity) approach was developed to prevent and manage childhood obesity. This approach consists of four stages (discovering the etiology, setting the target, designing the strategy, supporting and supervising) and includes nutrition, education, behavior, and physical activity intervention strategies directly based on the etiology of obesity and individual characteristics (40).

A study examining school-based interventions involving adolescents (<18 years), published between 2009 and 2021, suggested that interventions in the food sector (e.g., taxation of high-fat/sugar foods, front-of-pack labeling) and in the mass media (e.g., restricting food advertising to children only) may directly or indirectly help manage adolescent obesity. Overall, most intervention studies have not shown consistent effects in changing children's BMI, reporting only small, clinically insignificant weight losses or no effects (41).

In a randomized controlled trial on the prevention of early childhood obesity, a nutritional education intervention implemented in preschool children (3-4 years old) together with their parents improved the BMI of children, especially those with a high BMI for their age. It supported the prevention of overweight and obesity (42). interventions in the first 1000 days of pregnancy in developing countries affect the BMI of children in later life and reduce the risk of obesity (43-44).

Figure 2. Schematic design of the EPISTCO (Etiology-Based Personalized Intervention Strategy Targeting Childhood Obesity) model, which is based on four multi-stage steps(40)



A review covering the period 1949–2020 in China found 14 major national childhood obesity-related interventions: comprehensive interventions (e.g., "Healthy Lifestyle for All Campaign," 2007), diet and nutrition (e.g., "China Rural Compulsory Education Student Nutrition Improvement Program," since 2011), and physical activity (e.g., "Happy 10 Minutes Program," 2006) (45).

In Turkey, the "Turkey Obesity Combat and Control Program (2010-2014)" was prepared under the name of "Turkey Healthy Nutrition and Active Life Program" by the Department of Nutrition and Physical Activities of the General Directorate of Primary Health Care of the Ministry of Health of the Republic of Turkey. A training of trainers meeting was held in Antalya between 04 and 08 October 2010 to introduce the program to healthcare personnel and to increase the knowledge level

of healthcare personnel on combating obesity. The program's action plan, 'Action Plan For Prevention And Control Of Adult And Childhood Obesity And Physical Activity', was revised and reimplemented in October 2019 (46-48).

Clinical presentation, assessment, and diagnosis

The American Academy of Pediatrics (AAP) recommends that clinicians screen all adolescents for overweight and obesity annually. Overweight is defined as a BMI at or above the 85th percentile and below the 95th percentile for children and teens of the same age and sex. Class 1 obesity is defined as BMI being at or above the 95th percentile or less than 120% according to age and gender, Class 2 obesity (≥120% to <140% at the 95th percentile) or BMI ≥ 35 kg/m2 to <40 kg/m2, whichever is lower for age and gender, and Class 3 obesity (≥140% at the 95th percentile) or BMI ≥ 40 kg/m2, whichever is lower for age and gender. Annual laboratory testing for obese adolescents includes type 2 diabetes (hemoglobin A1c, fasting glucose, or oral glucose tolerance test), steatotic liver disease associated with metabolic dysfunction (alanine aminotransferase), and cholesterol (fasting lipid panel) (3).

Treatment

Evidence-based obesity treatment includes lifestyle changes, pharmacotherapy, and metabolic and bariatric surgery. The appropriate treatment should be selected according to the clinical condition of the patient (3).

Lifestyle modification therapy

Lifestyle changes refer to changes in diet, physical activity, sleep, or other daily habits that are obesity risk factors to reduce BMI and improve overall health. These lifestyle recommendations include reducing sugary drinks, getting 60 minutes of moderate to intense physical activity daily, and limiting social media use and overall screen time, although no upper limit is specified for use (3).

Pharmacotherapy

Until 2020, Orlistat was the only drug approved by the FDA for adolescent obesity. However, after randomized controlled trials, drugs such as glucagon-like peptide-1 receptor agonists (liraglutide/semaglutide), phentermine/topiramate, and setmelanotide began to be used in patients with syndromic and/or monogenic obesity over the age of 12 years (3,49).

GLP-1 Receptor Agonists (Liraglutide and Semaglutide)

Glucagon-like peptide-1 receptor agonists (GLP1RA) increase glucose-dependent insulin secretion and reduce glucagon secretion and gastric emptying, which has led to their successful development for the treatment of type 2 diabetes (T2D). These agents also inhibit food intake and reduce body weight. GLP1RA may also be effective in obesity-related conditions such as cardiovascular disease and nonalcoholic steatohepatitis (NASH) (50). In an intervention study in obese adolescents, the weekly administration of semaglutide at a dose of 2.4 mg resulted in significantly greater reductions in BMI compared with the lifestyle intervention alone. The semaglutide group also experienced more pronounced decreases in body weight as improvements in cardiometabolic risk factors. These factors included waist circumference, glycated hemoglobin, lipid profiles excluding high-density lipoprotein cholesterol, and alanine aminotransferase levels compared with the placebo (51).

In a study of adolescents (12 to <18 years) with obesity who were poorly responsive to lifestyle therapy alone, liraglutide (3.0 mg) administered subcutaneously once daily in addition to lifestyle therapy was superior to placebo in change from baseline in BMI standard deviation score at Week 56 (estimate difference, −0.22; 95% confidence interval [CI], −0.37 to −0.08; P = 0.002). In adolescents with obesity, liraglutide (3.0 mg) plus lifestyle therapy resulted in significantly greater reductions in the BMI standard deviation score than placebo plus lifestyle therapy (52).

Phentermine/Topiramate

It is a fixed-dose combination of phentermine and topiramate developed for the treatment of obesity, sleep apnea syndrome, type 2 diabetes mellitus, and nonalcoholic steatohepatitis (NASH). The once-daily formulation of phentermine (a sympathomimetic amine) and topiramate is designed to combat obesity by reducing appetite and increasing feelings of fullness. In July 2022, phentermine/topiramate received initial U.S. approval for chronic weight management in pediatric patients ≥ 12 years of age with an age- and sex-standardized BMI of the 95th percentile or greater, as an adjunct to a reduced-calorie diet and increased physical activity (53).

Setmelanotide

Setmelanotide, a melanocortin-4 receptor agonist, was titrated to a dose of 3.0 mg and administered subcutaneously once daily for 16 weeks. This treatment was given to patients with obesity who had a history of hypothalamic

injury or a diagnosis of a non-malignant tumor affecting the hypothalamus. These patients had previously undergone surgery, chemotherapy, or radiation. The primary endpoint of at least a 5% decrease in BMI from baseline was achieved ($p<0.0001$). The mean decrease in BMI across all patients was 15% (SD 10). These findings support setmelanotide as a novel and effective treatment for hypothalamic obesity (54).

Metabolic and Bariatric Surgery

Pharmacotherapy should be offered only in cases of class 3 obesity or as an adjunct to lifestyle interventions when a life-threatening comorbidity is present. Surgical treatment should be offered to children over the age of 12 years with class 2 or class 3 obesity, with or without associated comorbidities, only after an appropriate trial of intensive lifestyle modifications and pharmacotherapy lasting at least six months has failed (55,56). According to the American Society for Metabolic and Bariatric Surgery (ASMBS), surgical intervention should be considered for individuals with a BMI between 30 and 34.9 kg/m² who have not achieved significant or sustained weight loss or improvement in comorbidities through nonsurgical methods. (57).

CONCLUSION

The prevalence of obesity is increasing globally and in our country. Therefore, countries and institutions must implement preventive measures and interventions, including promoting healthy lifestyles, physical activity, psychosocial support, nutrition, and preventive programs. Furthermore, for children and adolescents with obesity encountered in clinical practice, the underlying etiology should be identified first, and interventions should be tailored to the individual.

Peer Review	Externally peer-reviewed.
Author Contributions	Conception/Design of Study- E.G., H.D.; Data Acquisition- E.G., H.D.; Data Analysis/Interpretation- E.G., M.Ö.; Drafting Manuscript- E.G.; Critical Revision of Manuscript- E.G., H.D. V.A.; Final Approval and Accountability- E.G., H.D. V.A. M.Ö.
Conflict of Interest	Authors declared no conflict of interest.
Financial Disclosure	Authors declared no financial support.

Author Details

Eren Güzeloğlu
[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye
0000-0003-4316-2491 dr.erenguzeloglu@gmail.com

Belen Ateş
[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye
0000-0002-4525-8764

Mustafa Özdemir
[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye
0000-0001-7481-3926

Hüseyin Dağ
[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye
0000-0001-7596-7687

Vefik Arıca
[1] University of Health Sciences, Prof. Dr. Cemil Taşcıoğlu City Hospital, Depatment of Pediatrics, İstanbul, Türkiye
0000-0002-2080-4677

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