# Clinical and Metabolic Predictors of Obesity Related Complications in School Age Children

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**ABSTRACT**

Childhood obesity is one of the most important pediatric public health problems because it is strongly associated with metabolic, cardiovascular, hepatic, respiratory, orthopedic and psychosocial complications. School age represents a critical period for identifying children at increased risk because many obesity related abnormalities, including insulin resistance, dyslipidemia, hypertension, metabolic dysfunction associated steatotic liver disease and impaired glucose metabolism, may already be present before adolescence. This review summarizes current evidence on clinical and metabolic predictors of obesity related complications in school age children. Particular attention is given to body mass index z score, waist circumference, waist to height ratio, duration and severity of obesity, pubertal status, blood pressure, acanthosis nigricans, family history, birth history, physical inactivity, sleep disturbance and dietary patterns. The review also discusses laboratory markers including fasting glucose, fasting insulin, HOMA IR, glycated hemoglobin, triglycerides, HDL cholesterol, alanine aminotransferase, uric acid, inflammatory markers and emerging adipokines. Evidence indicates that central adiposity, insulin resistance, atherogenic dyslipidemia, elevated blood pressure and hepatic enzyme abnormalities are among the strongest predictors of early cardiometabolic risk. However, no single marker is sufficient for comprehensive risk stratification. A combined clinical and biochemical approach is necessary, especially in children with severe obesity, early onset obesity, family history of type 2 diabetes or cardiovascular disease, and signs of metabolic syndrome. Early identification of high risk children can support targeted lifestyle intervention, rational screening and prevention of long term complications.

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## Introduction

Obesity in school age children is no longer viewed only as excessive body weight. It is now recognized as a chronic, relapsing and biologically complex disease that affects multiple organ systems. The clinical importance of pediatric obesity is determined not only by the degree of adiposity but also by the presence of metabolic, inflammatory, cardiovascular, hepatic and psychosocial complications. These complications may begin silently during childhood and later track into adolescence and adulthood, increasing the risk of type 2 diabetes, hypertension, atherosclerotic cardiovascular disease, metabolic dysfunction associated steatotic liver disease, obstructive sleep apnea and reduced quality of life [1,2].

School age children, generally defined as children between 6 and 12 years, represent a particularly important group for research and prevention. This period is characterized by rapid changes in body composition, eating behavior, physical activity, sleep patterns, family autonomy and exposure to school food environments. It also precedes or overlaps with early puberty, a stage associated with physiological changes in insulin sensitivity. Therefore, metabolic abnormalities detected during this period may represent either early disease, transient pubertal adaptation or the beginning of persistent cardiometabolic risk [2,3].

The main clinical problem is that children with similar body mass index may have different metabolic risk profiles. Some children with obesity show relatively preserved glucose and lipid metabolism, while others develop insulin resistance, dyslipidemia, elevated blood pressure and fatty liver at a young age. This heterogeneity has led to increasing interest in predictors that can identify which children are most likely to develop complications. These predictors include anthropometric indices, such as BMI z score and waist to height ratio; clinical signs, such as acanthosis nigricans and hypertension; family and perinatal history; behavioral factors; and biochemical markers of glucose, lipid, hepatic and inflammatory metabolism [4,5].

The purpose of this review is to summarize current knowledge about clinical and metabolic predictors of obesity related complications in school age children. The review focuses on predictors that are practical for pediatric internal medicine, school health programs and clinical research.

## Definition and classification of obesity in school age children

The classification of obesity in children differs from adults because BMI changes with age and sex. Therefore, BMI must be interpreted using age and sex specific reference standards. The World Health Organization growth reference for children and adolescents aged 5–19 years defines overweight and obesity using BMI for age z scores, while other systems, such as the International Obesity Task Force cut offs, provide age and sex specific BMI thresholds corresponding to adult BMI values of 25 and 30 kg/m² [3,4].

BMI is useful for population screening and routine clinical practice, but it has limitations. It does not directly measure fat mass, visceral adiposity or metabolic health. A muscular child may have a high BMI without excess fat, while a child with normal BMI may have increased central adiposity and metabolic risk. For this reason, BMI should be considered the starting point rather than the complete assessment [2,4].

Severe obesity is especially important because complication risk rises with increasing BMI category. Children with severe obesity are more likely to have insulin resistance, hypertension, dyslipidemia, fatty liver disease, sleep apnea and orthopedic complications than children with mild obesity [1,6]. Duration of obesity also matters. A child who has had obesity since early childhood may have longer metabolic exposure than a child with recent weight gain, even if their current BMI values are similar.

In clinical research, BMI z score, BMI percentile, percentage of the 95th percentile and absolute BMI may all be used. However, in children with severe obesity, BMI z score may become less sensitive at the upper range. Therefore, percentage of the 95th percentile or class based definitions may better represent severity in some studies [1,6].

## Pathophysiological basis of obesity related complications

Obesity related complications are driven by complex interactions among adipose tissue expansion, insulin resistance, chronic low grade inflammation, neuroendocrine adaptation, ectopic lipid accumulation and environmental factors. Adipose tissue is not simply an energy storage organ; it is metabolically active and secretes adipokines, cytokines and inflammatory mediators. When adipose tissue expands beyond its healthy storage capacity, adipocyte hypertrophy, hypoxia, macrophage infiltration and inflammatory signaling increase [5,7].

Insulin resistance is one of the central mechanisms linking obesity with metabolic complications. In children with excess adiposity, especially visceral adiposity, insulin mediated glucose uptake decreases in muscle and liver, while compensatory hyperinsulinemia develops. Initially, pancreatic beta cells may compensate by producing more insulin. Over time, however, beta cell stress may contribute to impaired glucose tolerance and type 2 diabetes in genetically susceptible children [7,8].

Dyslipidemia in pediatric obesity is often characterized by elevated triglycerides, low HDL cholesterol and increased small dense LDL particles. This pattern is closely linked to insulin resistance and hepatic overproduction of very low density lipoprotein. It is considered atherogenic even when total cholesterol is not markedly elevated [9,10].

The liver is another key metabolic organ affected by obesity. Excess delivery of free fatty acids to the liver, de novo lipogenesis, insulin resistance and inflammatory signaling contribute to metabolic dysfunction associated steatotic liver disease, previously known as nonalcoholic fatty liver disease. Pediatric fatty liver disease is strongly associated with obesity, central adiposity, dyslipidemia and impaired glucose metabolism [11,12].

Blood pressure increases through multiple mechanisms, including sympathetic nervous system activation, renal sodium retention, insulin resistance, vascular dysfunction, inflammation and sleep disordered breathing. Thus, hypertension in a child with obesity is not an isolated finding; it often reflects broader cardiometabolic dysfunction [13].

## Anthropometric predictors

BMI remains the most widely used anthropometric predictor of obesity related complications. Higher BMI category is associated with increased probability of insulin resistance, dyslipidemia, hypertension, hepatic steatosis, sleep apnea and orthopedic problems [1,2,6]. BMI is useful because it is inexpensive, reproducible and easy to calculate in school and clinic settings.

However, BMI alone is insufficient for individual risk prediction. It does not distinguish subcutaneous from visceral fat and does not reflect fat distribution. Two children with identical BMI may have different cardiometabolic risk if one has predominantly peripheral subcutaneous fat and the other has central adiposity. Therefore, BMI should be combined with waist circumference, blood pressure, clinical examination and laboratory markers [5,14].

Central adiposity is more closely linked to metabolic risk than general adiposity. Waist circumference reflects abdominal fat, including visceral adipose tissue, and is associated with insulin resistance, triglycerides, HDL cholesterol, blood pressure and inflammatory markers [14]. However, waist circumference changes with age, sex and ethnicity, and standardized pediatric cut offs are not universally accepted.

Waist to height ratio is a practical alternative because it adjusts waist size for height and can be used across a wider age range. Many studies suggest that waist to height ratio is useful for identifying children with increased cardiometabolic risk. A commonly used public health

message is that waist circumference should be less than half of height, although the optimal cut off may differ by age, sex and population [14].

For research in school age children, waist to height ratio may be especially useful because it is simple, low cost and more directly related to central adiposity than BMI alone. Combining BMI category with waist to height ratio can improve identification of children who require metabolic screening.

The trajectory of weight gain is an important predictor. Rapid weight gain in early childhood, persistent obesity from preschool age and early adiposity rebound are associated with higher risk of later obesity and metabolic complications [2,5]. Early onset severe obesity should raise suspicion for genetic, endocrine or syndromic causes, particularly when accompanied by hyperphagia, developmental delay, short stature or dysmorphic features [5].

In school age children, a history of obesity beginning before age five suggests longer metabolic exposure. Duration of obesity may influence insulin resistance, vascular changes and fatty liver risk. Therefore, clinical history should include age at onset, pattern of weight gain, previous BMI trajectory and family history.

## Clinical predictors

Family history is one of the strongest clinical predictors of obesity related metabolic complications. A family history of type 2 diabetes, hypertension, dyslipidemia, premature cardiovascular disease, fatty liver disease or severe obesity increases the probability that a child with obesity will develop similar complications [1,5,8]. Family history reflects shared genes, household eating patterns, physical activity habits, sleep routines and socioeconomic conditions.

For clinical practice, family history should not be limited to parental obesity. It should include grandparents and siblings, age at diagnosis of diabetes or cardiovascular disease, use of antihypertensive or lipid lowering therapy, and history of bariatric surgery or liver disease. In research, family history can be converted into categorical variables and used in multivariable prediction models.

Acanthosis nigricans is a visible clinical sign strongly associated with hyperinsulinemia and insulin resistance. It appears as dark, velvety thickening of the skin, commonly on the neck, axillae or other flexural areas. Although it is not a diagnostic test, its presence in a child with obesity should prompt evaluation for insulin resistance, impaired glucose metabolism and other metabolic risk factors [5,8].

Acanthosis nigricans is particularly useful in low resource settings because it can be detected during physical examination without laboratory testing. However, its absence does not exclude insulin resistance, and its severity may vary by skin type and ethnicity. Therefore, it should be interpreted as one part of a broader clinical assessment.

Elevated blood pressure is both a complication and a predictor of broader cardiometabolic risk. Children with obesity should have blood pressure measured accurately using an appropriate cuff size and interpreted according to age, sex and height percentiles or adolescent thresholds. Repeated measurements are required because isolated elevated values may reflect anxiety, incorrect technique or temporary illness [13].

Hypertension in a child with obesity is often associated with insulin resistance, dyslipidemia, fatty liver disease and sleep disordered breathing. Therefore, elevated blood pressure should prompt a wider metabolic evaluation. Ambulatory blood pressure monitoring may be useful when white coat hypertension or masked hypertension is suspected [13].

Puberty is associated with physiological insulin resistance, especially during mid puberty. In children with obesity, this normal pubertal reduction in insulin sensitivity may amplify existing metabolic risk. Therefore, pubertal status should be considered when interpreting insulin, glucose and HOMA IR values [5,7].

Girls with obesity may also be at increased risk of early puberty, menstrual irregularity and polycystic ovary syndrome during adolescence. Although PCOS is usually diagnosed after menarche, school age girls with central obesity, insulin resistance and family history may already show risk patterns requiring follow up [6,8].

Sleep duration and quality are important predictors of obesity complications. Short sleep, irregular sleep schedules and obstructive sleep apnea are associated with insulin resistance, hypertension, inflammation, daytime fatigue and reduced physical activity. Children with obesity who snore, have witnessed apneas, morning headaches, poor concentration or excessive daytime sleepiness should be evaluated for sleep disordered breathing [6].

Obstructive sleep apnea may worsen metabolic health independently of BMI through intermittent hypoxia, sympathetic activation and inflammation. It may also impair school performance and behavior, which can indirectly reduce adherence to lifestyle interventions.

Low physical activity and excessive sedentary behavior contribute to cardiometabolic risk through reduced energy expenditure, reduced muscle insulin sensitivity, lower cardiorespiratory fitness and unfavorable lipid profiles. Screen time is often associated with snacking, sleep disruption and exposure to food marketing [2,5].

Cardiorespiratory fitness may predict metabolic health beyond BMI. Some children with obesity who maintain higher physical fitness have better insulin sensitivity and blood pressure than less active peers. Therefore, assessment of activity level should include not only sports participation but also daily walking, active transport, school physical education and sedentary time.

Dietary predictors include frequent consumption of sugar sweetened beverages, ultra processed foods, energy dense

snacks, fast food, low fiber intake, low fruit and vegetable intake and irregular meal patterns. High fructose intake may contribute to hepatic de novo lipogenesis and fatty liver risk, while low fiber diets may worsen satiety and glycemic control [2,11].

However, dietary assessment in children is challenging because intake is influenced by parents, school meals, cultural habits and reporting bias. In research, repeated dietary recalls, food frequency questionnaires and household food environment assessment can improve accuracy.

### Metabolic predictors

Fasting glucose and glycated hemoglobin are commonly used to screen for prediabetes and diabetes. In children with obesity, abnormal glucose values indicate increased risk but may miss early insulin resistance. Some children have normal fasting glucose despite marked hyperinsulinemia because pancreatic beta cells are still compensating [8,15].

HbA1c is convenient because it does not require fasting, but its sensitivity for detecting early dysglycemia in children may be limited. It can also be affected by anemia, hemoglobinopathies and ethnicity. Therefore, fasting glucose, HbA1c and oral glucose tolerance testing may provide complementary information in selected high risk children [15].

Fasting insulin and the homeostatic model assessment of insulin resistance are widely used in pediatric obesity research. Higher fasting insulin and HOMA IR are associated with central obesity, acanthosis nigricans, dyslipidemia, fatty liver disease and future glucose abnormalities [7,8].

Nevertheless, insulin assays are not standardized across laboratories, and there is no universally accepted pediatric HOMA IR cut off. Puberty, sex, ethnicity and body composition affect insulin values. Therefore, HOMA IR is more useful for research and risk stratification than for making a single clinical diagnosis. In a review article or dissertation, it is scientifically appropriate to present HOMA IR as a predictor rather than a definitive diagnostic criterion.

Atherogenic dyslipidemia is one of the most common metabolic abnormalities in children with obesity. The typical pattern includes high triglycerides, low HDL cholesterol and sometimes elevated non HDL cholesterol. This pattern reflects insulin resistance and increased hepatic lipid production [9,10].

Triglyceride to HDL cholesterol ratio has been proposed as a simple marker of insulin resistance and cardiometabolic risk, although cut offs vary by population. Non HDL cholesterol is also useful because it includes all atherogenic apolipoprotein B containing particles and can be calculated from a non fasting lipid profile [9].

Children with obesity and abnormal lipid profile should be evaluated for diet quality, family history, diabetes risk, thyroid disease and liver disease. Persistent dyslipidemia may require specialist management, especially when LDL cholesterol is markedly elevated or familial hypercholesterolemia is suspected.

Alanine aminotransferase is the most commonly used screening marker for pediatric fatty liver disease. Elevated ALT in a child with obesity, especially when persistent, suggests possible metabolic dysfunction associated steatotic liver disease and warrants further assessment [11,12]. However, ALT may be normal in some children with hepatic steatosis, and mild elevation does not always correlate with histological severity.

Risk is higher in children with central obesity, insulin resistance, high triglycerides, low HDL cholesterol, impaired glucose metabolism and family history of fatty liver disease. Boys and children with severe obesity appear to be at higher risk in many cohorts [11,12]. Ultrasound can detect steatosis but has limited sensitivity for mild disease and cannot reliably stage fibrosis. Advanced imaging and fibrosis assessment may be needed in specialized settings.

Serum uric acid has gained attention as a marker associated with obesity, insulin resistance, hypertension and fatty liver disease. Hyperuricemia may reflect high fructose intake, renal handling changes, oxidative stress and metabolic dysfunction. Although uric acid is not universally included in obesity screening guidelines, it may be useful in research models evaluating cardiometabolic risk [6,7].

The interpretation of uric acid should consider age, sex, pubertal stage, renal function and diet. It is best used as an additional marker rather than a stand alone predictor.

Low grade inflammation is a key feature of obesity related metabolic dysfunction. High sensitivity C reactive protein, interleukin 6, tumor necrosis factor alpha, leptin, adiponectin and other adipokines have been studied as predictors of complications [5,7]. Low adiponectin and high leptin levels are often associated with insulin resistance and fatty liver risk.

However, these markers are not routinely used in general pediatric practice because of cost, variability and limited standardized cut offs. They may be valuable in research, especially when studying mechanisms or constructing predictive models. For most clinical settings, anthropometry, blood pressure, glucose, lipid profile and ALT remain more practical.

## Major obesity related complications and their predictors

Metabolic syndrome describes clustering of central obesity, dyslipidemia, elevated blood pressure and abnormal glucose metabolism. In children, definitions vary, and this creates difficulty comparing studies. The International Diabetes Federation definition provides one standardized approach, especially for children aged 10 years and older, but younger school age children may not fit neatly into these criteria [16].

Predictors of metabolic syndrome include high waist circumference, severe obesity, insulin resistance, high triglycerides, low HDL cholesterol, elevated blood pressure, family history of type 2 diabetes and low physical activity [16]. Because metabolic syndrome components cluster, children with one abnormality should be assessed for others. For example, high triglycerides in a child with obesity should prompt evaluation of blood pressure, glucose metabolism and liver enzymes.

Type 2 diabetes in youth is strongly associated with obesity, family history, pubertal insulin resistance, sedentary behavior and certain ethnic backgrounds. Clinical predictors include acanthosis nigricans, severe obesity, central adiposity and maternal history of gestational diabetes. Metabolic predictors include elevated fasting glucose, impaired glucose tolerance, elevated HbA1c, hyperinsulinemia and dyslipidemia [8,15].

An important point is that type 2 diabetes in youth often progresses more aggressively than adult onset disease. Therefore, early identification of children with prediabetes or marked insulin resistance is clinically important. Screening should be targeted to children with obesity plus additional risk factors rather than applied without clinical reasoning.

Obesity related dyslipidemia contributes to early vascular risk. The combination of elevated triglycerides, low HDL cholesterol, elevated non HDL cholesterol and hypertension is particularly concerning. Childhood lipid abnormalities can track into adulthood, especially when obesity persists [9,10].

Clinical predictors include central obesity, high intake of refined carbohydrates, family history of dyslipidemia, low physical activity and fatty liver disease. Metabolic predictors include insulin resistance, high ALT, high uric acid and elevated inflammatory markers. Non HDL cholesterol and triglyceride to HDL ratio are useful research variables because they reflect atherogenic risk beyond LDL cholesterol alone [9].

Hypertension is increasingly detected among children with obesity. Predictors include higher BMI category, central adiposity, family history of hypertension, high sodium intake, low physical activity, sleep apnea and insulin resistance [13]. Blood pressure should be measured at every clinical visit in children with obesity, using correct technique and cuff size.

Metabolic clustering is common. A child with obesity and hypertension should be assessed for dyslipidemia, impaired glucose metabolism and fatty liver disease. This integrated approach is necessary because hypertension may be an early visible sign of systemic cardiometabolic risk.

Metabolic dysfunction associated steatotic liver disease

Fatty liver disease is one of the most important hepatic complications of pediatric obesity. Predictors include severe obesity, male sex, central adiposity, insulin resistance, high triglycerides, low HDL cholesterol, prediabetes and family history [11,12]. ALT is commonly used for screening, but it should be interpreted carefully.

The clinical significance of pediatric fatty liver disease is that it can progress from simple steatosis to steatohepatitis, fibrosis and, rarely, cirrhosis. It is also associated with systemic cardiometabolic risk. Therefore, children with obesity and elevated ALT should not be managed only as "liver cases"; they require full metabolic assessment.

### Obstructive sleep apnea

Obstructive sleep apnea is associated with obesity severity, enlarged tonsils, craniofacial factors, male sex and family history. Clinical predictors include habitual snoring, witnessed apnea, restless sleep, daytime sleepiness, morning headache, behavioral problems and poor school performance [6]. Metabolic predictors may include insulin resistance, elevated blood pressure and inflammatory markers.

Because sleep apnea can worsen hypertension and insulin resistance, it may act both as a complication and a contributor to further metabolic dysfunction. Screening questions about sleep should therefore be included in obesity assessment.

Musculoskeletal complications include slipped capital femoral epiphysis, Blount disease, knee pain, foot pain and reduced mobility. Predictors include severe obesity, rapid weight gain, pain with walking and abnormal gait [6]. These complications reduce physical activity, creating a cycle that worsens obesity and metabolic risk.

Psychosocial complications include low self esteem, bullying, anxiety, depression and disordered eating. These may not be "metabolic" complications, but they strongly influence adherence, lifestyle change and long term prognosis. School age children are particularly vulnerable to stigma. Therefore, obesity assessment should be nonjudgmental and family centered [1,2].

Integrated clinical risk stratification

No single predictor can reliably identify all children at risk. The best approach is a layered model combining anthropometric, clinical and biochemical data. A practical high risk profile includes severe obesity, central adiposity, family history of type 2 diabetes or premature cardiovascular disease, acanthosis nigricans, elevated blood pressure, short sleep or snoring, low physical activity, high triglycerides, low HDL cholesterol, elevated ALT and abnormal glucose metabolism [1,5,6].

For research, predictive models should include variables that are inexpensive and reproducible. A core model may include age, sex, pubertal stage, BMI z score, waist to height ratio, blood pressure percentile, family history, acanthosis nigricans, fasting glucose, fasting insulin or HOMA IR, triglycerides, HDL cholesterol and ALT. Additional markers such as uric acid, CRP, leptin and adiponectin can be included in advanced models.

Risk stratification is also useful for prioritizing intervention. Children with obesity but no metabolic abnormalities still require prevention and lifestyle support. However, children with multiple abnormalities require more intensive follow up, multidisciplinary care and sometimes referral to endocrinology, cardiology, gastroenterology or sleep medicine.

### Implications for prevention and management

The identification of predictors is clinically meaningful only if it leads to earlier and better intervention. The foundation of management remains family based lifestyle intervention, including nutrition improvement, physical activity, sleep optimization, reduction of sedentary behavior and behavioral support [1,5]. School age children depend heavily on family routines and school environments, so interventions must involve caregivers and, when possible, educational institutions.

Children with insulin resistance, dyslipidemia, hypertension or fatty liver disease require structured follow up. Monitoring should include BMI trajectory, waist measures, blood pressure, lipid profile, glucose metabolism and ALT according to risk level. Treatment should avoid blame and focus on health outcomes, not only weight reduction. Even modest improvement in BMI trajectory, waist circumference, fitness or diet quality can improve metabolic markers [1,2].

Pharmacotherapy and bariatric surgery are generally more relevant to adolescents with severe obesity, but early identification in school age children can prevent progression to that stage. The most important goal in younger children is to detect complications early, reduce risk exposure and support sustainable family level change.

### Research gaps

Several important gaps remain. First, there is no universally accepted pediatric definition of metabolic syndrome, especially for younger school age children. Second, insulin resistance markers are limited by assay variability and lack of standardized cut offs. Third, many studies are cross sectional, making it difficult to determine whether predictors truly precede complications or simply coexist with them.

Fourth, more region specific studies are needed. Ethnicity, diet, physical activity, socioeconomic factors, urbanization and healthcare access influence both obesity prevalence and metabolic risk. Therefore, prediction models developed in one population may not perform well in another. Fifth, more research should evaluate combined clinical and metabolic scores that are feasible in primary care and school health settings.

Future studies should prioritize longitudinal designs, standardized anthropometric measurements, pubertal staging, family history, objective activity assessment and clinically accessible biomarkers. Research in Central Asian and other underrepresented populations would be valuable because local data are limited and may differ from Western cohorts.

## Conclusions

Obesity related complications in school age children are predictable to some extent when clinical and metabolic markers are assessed together. The strongest practical predictors include severe and early onset obesity, central adiposity, family history, acanthosis nigricans, elevated blood pressure, low physical activity, sleep disturbance, insulin resistance, atherogenic dyslipidemia and elevated ALT. BMI remains essential for screening, but it should not be used alone. Waist to height ratio, blood pressure, lipid profile, glucose metabolism and hepatic markers provide additional risk stratification. The most scientifically justified approach is integrated assessment, because obesity related complications cluster and share common mechanisms. Early identification of high risk children allows timely lifestyle intervention, targeted laboratory screening and prevention of long term cardiometabolic disease.

## References

1. Hampl, S. E., Hassink, S. G., Skinner, A. C., Armstrong, S. C., Barlow, S. E., Bolling, C. F., Avila Edwards, K. C., Eneli, I., Hamre, R., Joseph, M. M., Lunsford, D., Mendonca, E., Michalsky, M. P., Mirza, N., Ochoa, E. R., Sharifi, M., Staiano, A. E., Weedn, A. E., Flinn, S. K., ... Okechukwu, K. (2023). Clinical practice guideline for the evaluation and treatment of children and adolescents with obesity. Pediatrics, 151(2), e2022060640. https://doi.org/10.1542/peds.2022-060640

2. Jebeile, H., Kelly, A. S., O'Malley, G., & Baur, L. A. (2022). Obesity in children and adolescents: Epidemiology, causes, assessment, and management. The Lancet Diabetes & Endocrinology, 10(5), 351–365. https://doi.org/10.1016/S2213-8587(22)00047-X

3. de Onis, M., Onyango, A. W., Borghi, E., Siyam, A., Nishida, C., & Siekmann, J. (2007). Development of a WHO growth reference for school-aged children and adolescents. Bulletin of the World Health Organization, 85(9), 660–667. https://doi.org/10.2471/BLT.07.043497

4. Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: International survey. BMJ, 320(7244), 1240–1243. https://doi.org/10.1136/bmj.320.7244.1240

5. Styne, D. M., Arslanian, S. A., Connor, E. L., Farooqi, I. S., Murad, M. H., Silverstein, J. H., & Yanovski, J. A. (2017). Pediatric obesity—Assessment, treatment, and prevention: An Endocrine Society clinical practice guideline. The Journal of Clinical Endocrinology & Metabolism, 102(3), 709–757. https://doi.org/10.1210/jc.2016-2573

6. Vajravelu, M. E., Tas, E., & Arslanian, S. (2023). Pediatric obesity: Complications and current day management. Life, 13(7), 1591. https://doi.org/10.3390/life13071591

7. Park, H., Choi, J. E., Jun, S., Lee, H., Kim, H. S., Lee, H. A., & Park, H. (2024). Metabolic complications of obesity in children and adolescents. Clinical and Experimental Pediatrics, 67(7), 328–355. https://doi.org/10.3345/cep.2023.00892

8. American Diabetes Association Professional Practice Committee. (2025). 14. Children and adolescents: Standards of care in diabetes—2025. Diabetes Care, 48(Supplement 1), S283–S305. https://doi.org/10.2337/dc25-S014

9. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents, & National Heart, Lung, and Blood Institute. (2011). Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents:

Summary report. Pediatrics, 128(Supplement 5), S213–S256. https://doi.org/10.1542/peds.2009-2107C

10. de Ferranti, S. D., Steinberger, J., Ameduri, R., Baker, A., Gooding, H., Kelly, A. S., Mietus-Snyder, M., Mitsnefes, M. M., Peterson, A. L., St-Pierre, J., Urbina, E. M., Zachariah, J. P., & Zaidi, A. N. (2019). Cardiovascular risk reduction in high-risk pediatric patients: A scientific statement from the American Heart Association. Circulation, 139(13), e603–e634. https://doi.org/10.1161/CIR.0000000000000618

11. Vos, M. B., Abrams, S. H., Barlow, S. E., Caprio, S., Daniels, S. R., Kohli, R., Mouzaki, M., Sathya, P., Schwimmer, J. B., Sundaram, S. S., Xanthakos, S. A., & American Gastroenterological Association Institute. (2017). NASPGHAN clinical practice guideline for the diagnosis and treatment of nonalcoholic fatty liver disease in children. Journal of Pediatric Gastroenterology and Nutrition, 64(2), 319–334. https://doi.org/10.1097/MPG.0000000000001482

12. Scapaticci, S., D'Adamo, E., Mohn, A., & Chiarelli, F. (2021). Non-alcoholic fatty liver disease in obese youth with insulin resistance and type 2 diabetes. Frontiers in Endocrinology, 12, 639548. https://doi.org/10.3389/fendo.2021.639548

13. Flynn, J. T., Kaelber, D. C., Baker-Smith, C. M., Blowey, D., Carroll, A. E., Daniels, S. R., de Ferranti, S. D., Dionne, J. M., Falkner, B., Flinn, S. K., Gidding, S. S., Goodwin, C., Leu, M. G., Powers, M. E., Rea, C., Samuels, J., Simasek, M., Thaker, V. V., Urbina, E. M., & Subcommittee on Screening and Management of High Blood Pressure in Children. (2017). Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics, 140(3), e20171904. https://doi.org/10.1542/peds.2017-1904

14. Ezzatvar, Y., Izquierdo, M., Ramírez-Vélez, R., & García-Hermoso, A. (2022). Accuracy of different cutoffs of the waist-to-height ratio as a screening tool for cardiometabolic risk in children and adolescents: A systematic review and meta-analysis of diagnostic test accuracy studies. Obesity Reviews, 23(2), e13375. https://doi.org/10.1111/obr.13375

15. Zimmet, P., Alberti, K. G. M. M., Kaufman, F., Tajima, N., Silink, M., Arslanian, S., Wong, G., Bennett, P., Shaw, J., Caprio, S., & International Diabetes Federation Task Force on Epidemiology and Prevention of Diabetes. (2007). The metabolic syndrome in children and adolescents—An IDF consensus report. Pediatric Diabetes, 8(5), 299–306. https://doi.org/10.1111/j.1399-5448.2007.00271.x

16. Chung, Y. L., Rhie, Y. J., & Kim, H. S. (2022). Metabolic syndrome in children and adolescents. Endocrinology and Metabolism, 37(5), 745–757. https://doi.org/10.3803/EnM.2022.1588

17. Luo, Y., Liu, M., & Wang, T. (2024). Insulin resistance in pediatric obesity: From mechanisms to clinical implications. International Journal of Molecular Sciences, 25(7), 4000. https://doi.org/10.3390/ijms25074000

18. Ciężki, S., Odyjewska, E., Bossowski, A., & Głowińska-Olszewska, B. (2024). Not only metabolic complications of childhood obesity. Nutrients, 16(4), 539. https://doi.org/10.3390/nu16040539