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From Prenatal Exposure to Lifestyle Behaviors: Risk Factors for Childhood Obesity

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ABSTRACT

Background. In recent years, there has been an increase in the prevalence of childhood obesity. To identify early on children who are at increased risk of obesity, it is necessary to have a thorough understanding of the factors that contribute to obesity in this age group. Such an understanding will enable timely delivery of appropriate care and the introduction of effective preventive and treatment interventions as early as possible in the course of obesity development. **Aim.** This review analyzes current evidence regarding the principal risk factors contributing to the development of obesity in the pediatric population. **Materials and methods.** A narrative literature review was carried out using the PubMed and Google Scholar databases. The search included English-language articles published from January 2019 to January 2026. **Results.** The results show that several factors influence the development of childhood obesity, including genetic predisposition, the course of fetal and early childhood development, and lifestyle factors. The behavior of parents has a strong effect on a child's lifestyle. Common causes of excess body fat accumulation include poor diet, inadequate physical activity, and sleep disturbances. Environmental aspects also affect this process. **Conclusions.** The development of childhood obesity is modulated by genetic predisposition and environmental factors. Mechanisms such as metabolic and appetite dysregulation are involved. Lifestyle-related factors, which are largely modifiable, play the most important role. Effective prevention of childhood obesity demands caregiver education, the establishment of healthy dietary habits, and frequent physical exercise.

Keywords: childhood obesity, pediatric obesity, prevention, lifestyle factors, risk factors

1. INTRODUCTION

Obesity is a chronic condition characterized by excessive adipose tissue accumulation and negatively impacts health (Młynarska et al., 2025). There has been an increase in the rate of childhood obesity in recent years (Chung et al., 2023;

Brambilla et al., 2022), so obesity among children has become one of the biggest challenges to public health. In 2022, the number of children and adolescents between the ages of 5 and 19 who were obese exceeded 159 million. This represents an increase of about 127.3 million people when compared with 1990. The prevalence of obesity rose from 1.7% in 1990 to 6.9% in 2022 among girls and from 2.1% to 9.3% among boys (NCD-RisC, 2024).

Dealing with childhood obesity is difficult and takes a lot of time, and in most cases it is necessary to use an approach that involves more than one discipline. If treatment is inadequate or omitted over time, comorbidities will develop. Excessive body weight negatively affects many organs and body systems. Hypertension and dyslipidemia, which are associated with obesity, raise the risk of suffering cardiovascular events. At the same time, the risk of metabolic complications, including insulin resistance and diabetes, increases, especially in instances of central obesity. It has also been shown that excess body weight adversely affects the development of the musculoskeletal and respiratory systems. Moreover, obesity can have a negative impact on a child's mental health. Also, childhood obesity usually continues into adulthood (Daley and Balasundaram, 2025).

A full understanding of the modifiable risk factors associated with childhood obesity might allow their effects to be reduced at an early stage. It is important to identify children who are at a particularly high risk of becoming obese so that they can receive targeted care and appropriate prevention and treatment measures can be put into place early on, before any complications arise. For this reason, the present review seeks to examine and synthesize existing evidence on the main risk factors for the development of childhood obesity, with a special focus on modifiable determinants that could be addressed in preventive initiatives.

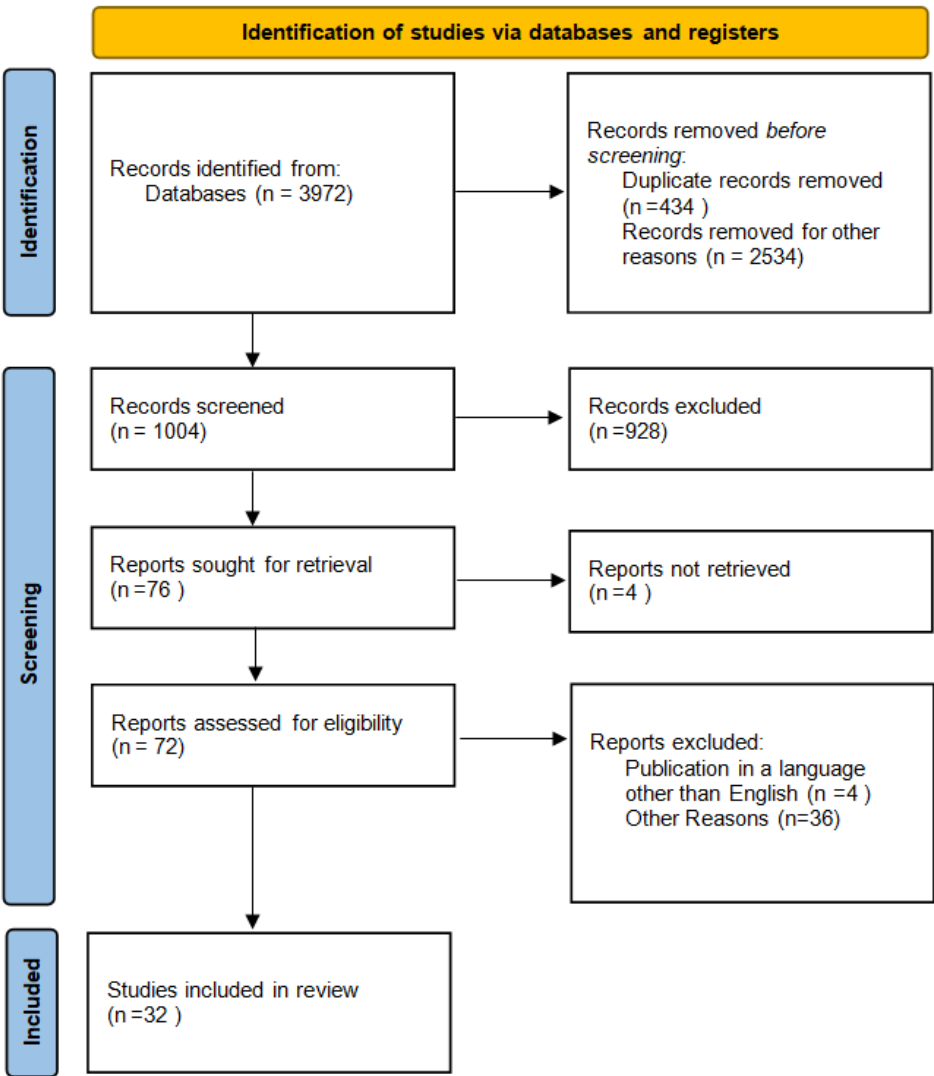


Figure 1. PRISMA flow diagram

2. REVIEW METHODS

A narrative literature review was conducted using the PubMed and Google Scholar databases, with the search covering English-language articles published between January 2019 to January 2026. The keywords used were childhood obesity, pediatric obesity, risk factors, lifestyle, diet, physical activity, sleep, breastfeeding, socioeconomic status, and prenatal factors, as well as their combinations.

Only original research articles, systematic reviews, and meta-analyses that were concerned with the risk factors for obesity in children and adolescents were included; the studies were chosen based on their relevance to the subject, their methodological quality, and the extent to which they contributed to an understanding of both modifiable and non-modifiable determinants of pediatric obesity. Exclusions included conference abstracts, letters to the editor, and animal studies. The study selection process is presented in the PRISMA flow diagram (Figure 1).

3. RESULTS AND DISCUSSION

The main risk factors for the development of childhood obesity are presented in Table 1.

Table 1. Summary of the main risk factors for childhood obesity

Risk Factor	Main Findings
Genetic and epigenetic factors	Genetic causes of obesity include monogenic and polygenic mechanisms, with polygenic mechanisms being more common. They may affect appetite regulation, adipogenesis, and metabolic processes. Epigenetic mechanisms also play an important role.
Prenatal and perinatal factors	Maternal obesity prior to pregnancy, excessive weight gain during pregnancy, gestational diabetes, smoking while pregnant, and exposure to adverse environmental factors are important risk factors during pregnancy and the perinatal period. They can affect the development of the fetus and lead to disturbances in metabolic programming.
Parental body weight	Excessive body weight in children may be associated with parental obesity. Maternal obesity during pregnancy and lactation may affect appetite regulation, energy balance, and metabolism in the developing fetus. Maternal malnutrition during pregnancy may also increase the risk of obesity in the child.
Mode of delivery	Children born by cesarean section may have an increased risk of developing obesity, which may be associated with differences in gut microbiota composition.
Birth weight and early weight Gain	Children with high birth weight and those who experience rapid weight gain during the first months of life have an increased risk of developing obesity.
Early-life feeding	Breastfeeding may reduce the risk of developing obesity.
Sedentary behavior and screen time	Low physical activity and prolonged screen time are associated with increased obesity risk.
Sleep quality	Sleep disturbances and insufficient sleep duration are associated with an increased risk of developing obesity.
Dietary Factors and Processed Foods	Consumption of highly processed foods and products high in sugar, salt, and fat is associated with an increased risk of developing obesity.
Socioeconomic and Environmental Factors	Poverty and low parental education may be associated with limited access to healthy food and chronic stress, which may increase the risk of obesity. Air pollution and exposure to obesogens may also increase this risk.

Genetic and Epigenetic Factors

Genetic factors that contribute to the continued presence of obesity within families involve mutations in genes related to weight regulation. It has been estimated that genetic factors account for 40% to 75% of the variation in body mass index and that of abdominal obesity. Rarely do autosomal-dominant harmful mutations in certain genes cause obesity to appear in heterozygous individuals and to be seen across multiple generations (Młynarska et al., 2025; Wójcik and Zachurzok, 2024).

The most frequent genetically based type of obesity is caused by mutations in the melanocortin 4 receptor (MC4R) gene and occurs in 3–5% of individuals with severe obesity. Such mutations are inherited in an autosomal dominant way. Most monogenic and syndromic forms of obesity are inherited in an autosomal recessive manner, which is the reason why familial obesity is not typically seen in these cases (Mahmoud et al., 2022; Trang and Grant, 2023; Wójcik and Zachurzok, 2024).

A more common situation is one in which polygenic obesity arises from the combined influence of multiple genetic variants associated with appetite control, fat formation, and metabolism. Although each polymorphism has only a small effect on the phenotype, the combined effect of many of them becomes important. A large genome-wide association study involving 340,000 individuals identified 97 loci associated with BMI, accounting for 2.7% of the variation in BMI. The groups of these loci vary across ethnic populations, meaning that obesity risks also differ. In the European population, the long-term stability of access to food across generations has led to the elimination of about 30 genes that raise the risk of cardiometabolic diseases. It has been estimated that the entire genomic variability could account for up to 21 percent of the differences in BMI (Górczyńska-Kosiorz et al., 2024; Młynarska et al., 2025; Trang and Grant, 2023; Wójcik and Zachurzok, 2024).

Parental obesity not only determines children's health via genetic and environmental factors but also through epigenetic mechanisms. Such changes involve DNA methylation at CpG sites, histone modifications, and control by non-coding RNAs. Methylation of CpG islands, which are generally found in the promoter areas of genes, usually results in their suppression, while methylation inside a gene tends to increase its expression. In people who are obese, reduced methylation levels have been found in the promoter of the leptin gene, whereas the methylation of genes related to insulin signaling is increased (Młynarska et al., 2025; Sivakumar et al., 2024; Wójcik and Zachurzok, 2024).

Modifications of histones, for example methylation and acetylation, can lead to changes in chromatin structure and thus influence the activity of genes that regulate adipogenesis and appetite. It is also possible for changes in short and long non-coding RNAs to affect gene expression and play a role in the development of obesity (Minabe et al., 2025; Wójcik and Zachurzok, 2024).

Prenatal and Perinatal Factors

Numerous pre- and perinatal factors additionally influence the occurrence of metabolic diseases in children. The parents' diet, body weight, the use of assisted reproduction techniques, and environmental exposures all affect fetal development. Prenatal and early life exposure to chemicals, including pesticides and air pollution, is also substantial, as these factors influence the mesenchymal differentiation of stem cells into adipocytes and osteoblasts. Such changes may lead to an increase in the number of adipocytes, a change that can be passed on to subsequent generations. Children of mothers who were obese before pregnancy are at an increased risk of obesity as well (Drozd et al., 2021; Romanelli et al., 2020; Szczyrka, 2023; Umamo et al., 2025). Apart from that, smoking during pregnancy, gestational diabetes, and excessive weight gain during pregnancy add to the development of obesity among the pediatric population (Drozd et al., 2021; Yuan et al., 2024).

Parental Body Weight

Many studies have shown that when parents have a high body weight, the children are at a greater risk of becoming obese. Children who have one obese parent are more likely to become obese than those whose parents have a healthy body weight (Tragomalou et al., 2020). The risk is even greater if both parents are obese (Chen et al., 2025). It has also been found that the mother's BMI (Body Mass Index) has a greater effect on the child's body weight (Szczyrka, 2023).

It has been suggested that maternal obesity during both pregnancy and lactation could affect the way genes and the neuroendocrine pathways responsible for appetite control, energy balance, and metabolic function in the developing fetus are expressed, possibly leading to children having altered eating habits and metabolic problems later in life. Moreover, maternal excess body weight might be linked to abnormal distribution of adipose tissue in children, and it is believed to encourage the development of white adipose tissue while at the same time impairing that of brown adipose tissue (Umamo et al., 2025).

Maternal malnutrition during pregnancy can also affect the metabolic programming of the fetus and increase the likelihood of obesity developing in the offspring (Sarni et al., 2022).

Mode of Delivery

There is a growing body of evidence showing that children who are born by cesarean section seem to be at a higher risk of becoming obese when compared to those who are delivered vaginally (Szczyrska, 2023; Umano et al., 2025). It is believed that this link is in part due to differences in the microbial exposure experienced at birth. Babies born vaginally tend to have a more diverse gut microbiome at the start of life. Those delivered by cesarean section usually have lower amounts of both *Bifidobacteria* and *Bacteroides*, organisms which are considered to be protective against excessive weight gain (Akagbosu et al., 2022; Zhang et al., 2022).

The composition of the early gut microbiota might affect how efficiently energy is extracted from the diet and could thus contribute to later adiposity (Bała and Staszko, 2026; Romanelli et al., 2020). Research also indicates that cesarean section under elective circumstances is linked to lower cortisol levels in newborns, which possibly contributes to metabolic dysregulation and the later development of obesity (Zhang et al., 2022).

Birth Weight and Early Weight Gain

Evidence shows that a higher birth weight is associated with an increased risk of developing obesity later in life (Szczyrska, 2023). On the other hand, a low birth weight has been connected with a greater tendency for central fat distribution (Drozd et al., 2021). In children who are particularly susceptible to obesity, the pattern of their growth is mainly determined by their body weight in early life, and the values of their body mass index during childhood generally carry over into adolescence and later adulthood. Babies and young children who gain weight rapidly after birth—this being shown by exceeding the appropriate weight-to-length percentiles in the first 3 to 6 months or by showing rapid increases in their BMI between the ages of 2 and 6—are at a higher risk of being obese when they are 12 to 14 years old (Deal et al., 2020).

Feeding Practices in Early Life

It has been suggested that breastfeeding in the first six months of life could provide some protection against the development of obesity later in life. This might be due to hormones present in breast milk, for example, leptin, adiponectin, and ghrelin, which assist in regulating the baby's appetite. Infants who are breastfed usually gain weight more slowly in early life; on the other hand, quick weight gain in infants is known to be a risk factor for obesity in childhood (Umano et al., 2025).

Moreover, certain studies show that babies who are breastfed have a more favorable composition of gut microbiota than those who are fed on formula, which possibly further reduces the risk of developing obesity (Romanelli et al., 2020).

It is difficult to evaluate the effect of feeding infants formula milk in regard to the development of obesity and to compare such cases with those who have been breastfed during the first six months of life because of methodological problems. A meta-analysis from 2013 shows that longer breastfeeding is linked to a 13% lower risk of obesity in childhood. It is recommended that babies should be breastfed exclusively for six months. In the United States, the Healthy People 2020 program has set a target of 60%, although at present only about 25% of infants are breastfed. Breastfeeding is less common in families with an income below 200% of the poverty level, among mothers who have a lower level of education, younger mothers, and in African American and Hispanic communities (Deal et al., 2020).

Sedentary Lifestyle and Screen Time

A sedentary lifestyle is a well-established risk factor for childhood obesity (Chen et al., 2025; Pietrzak et al., 2025; Wilhite et al., 2023). Children who take part in less physical activity burn fewer calories. If this is coupled with an excessive intake of energy, it results in the buildup of extra fat tissue. Children with only minimal daily physical activity are considerably more likely to become obese than their more active counterparts (Verduci et al., 2021; Wilhite et al., 2023).

The risk is made worse by an overuse of screens, for example, by watching television, playing video games, and using other screen-based media (Khadilkar et al., 2023). In children, the longer they spend watching television, the more likely they are to consume energy-dense snacks. While taking part in a program, children may keep on eating even when they have reached a state of satiety (Ali et al., 2024). Furthermore, long periods in front of screens reduce children's total energy expenditure and disrupt their sleep patterns, thereby worsening the effect on their energy balance and metabolic regulation (Ali et al., 2024; Minabe et al., 2025).

Sleep Length and Quality

Several studies have shown that poor sleep quality and short sleep duration are linked to an increased risk of obesity in children (Ali et al., 2024; Littlejohn, 2023; Wilhite et al., 2023). Sleeping less than 8–9 hours each night is strongly associated with a higher prevalence of obesity. Furthermore, the time at which children go to bed is important. Those who go to bed late and wake up early tend to eat more high-calorie foods and show less healthy eating habits (Ali et al., 2024). It has also been found that late-sleeping children are more likely to skip breakfast and make up for it by eating highly processed foods or 'snacks' (Ali et al., 2024; Yuan et al., 2024). The fact that one gets insufficient sleep is linked to hormonal imbalances, which are involved in the development of obesity. In children who suffer from inadequate sleep, the level of ghrelin—a hormone that stimulates appetite—is higher while that of leptin—which indicates a feeling of fullness—is lower. These children also exhibit disturbances in cortisol secretion and increased insulin resistance (Minabe et al., 2025).

Dietary Factors and Processed Foods

Poor nutrition is one of the main risk factors for childhood obesity (Romanelli et al., 2020; Yuan et al., 2024). The food that caregivers consume has a strong effect on the way in which infants are fed (Khadilkar et al., 2023). Overfeeding, the consumption of sweetened drinks, and eating a large amount of processed foods such as fast food can result in an excess of calories and lead to obesity (Khadilkar et al., 2023; Yuan et al., 2024).

Even among children breastfed up to 6 months of age, early introduction of beverages containing added simple sugars and fruit juices is conducive to developing obesity later in life (Khadilkar et al., 2023). The process is also influenced by low vegetable consumption, frequent dining out, snacking, and high meat intake (Chen et al., 2025; Khadilkar et al., 2023; Magiera et al., 2024). The promotion of highly processed foods, rich in sugar, salt, and fat, in television and online advertisements contributes to excessive weight gain in children (Khadilkar et al., 2023; Yuan et al., 2024).

The health effects of poor nutrition and high BMI contributed to approximately 11 million adult deaths worldwide in 2017. Rapid and profound changes in the availability of food and beverages have led Americans to derive about 80% of their calories from packaged products, more than 70% of which are highly processed. The harmful effects of added sugars on cardiovascular and metabolic health, including leading to obesity and the development of type 2 diabetes, are well established. Between 2013 and 2018, added sugars accounted for about 14% of the daily energy intake of American children over 2, while guidelines advise limiting them to 5-10% of total calories. Consumption of sugar-sweetened beverages, which account for more than half of all added sugars in the US diet, begins in the first year of life. In 2009, 43% of infants and 72% of young children consumed at least one sweetened beverage or sweetened dessert per day. Consumption of sugar-containing beverages is associated with increased BMI in children (Deal et al., 2020; Tester et al., 2020).

Socioeconomic and Environmental Factors

Poverty, food insecurity, stress, living in rural areas, and lower levels of parental education are all among the main factors that contribute to obesity in early childhood (Ali et al., 2024; Au et al., 2019; Deal et al., 2020).

A high number of children experiencing poverty face limited access to nutritious foods such as fresh vegetables and fruits, as they are often more expensive than easily accessible ultra-processed ones. Thus, the latter are more frequently chosen by families with limited financial resources (Tester et al., 2020).

Food insecurity, which is the inability of people to obtain a steady and dependable supply of nutritious food, is experienced by more than 12% of households in the United States and involves nearly 17 million children who do not know when or whether they will get their next meal. When food is short in supply from time to time, people tend to eat more calorie-rich but nutrient-deficient foods whenever food is available. In these circumstances, the average daily intake of added sugars goes above 70 g, whereas the recommended amount should be 25 g or less (Deal et al., 2020).

Food insecurity is also linked to a higher prevalence of stress and anxiety in children, and chronic stress can cause the adrenal cortex to secrete more cortisol, thereby also contributing to the development of obesity (Tester et al., 2020). The fact that people live in areas with high levels of air pollution may cause fewer children to engage in physical activity and spend less time outdoors. Moreover, exposure to noise and artificial light at night causes sleep disturbances (Yuan et al., 2024).

Environmental pollution by endocrine disruptors also contributes to the development of obesity, since these external compounds are referred to as obesogenic substances. They can have a direct effect on adipocytes by causing their hyperplasia and hypertrophy, and their presence is also linked to disturbances in the regulation of hunger and satiety, changes in the gut microbiota, metabolic disorders, and hormonal disorders (Sarni et al., 2022).

4. CONCLUSION

Childhood obesity is a complicated condition that results from both factors that can be modified and those that cannot. A child's risk of becoming obese is greatly affected by genetic predisposition and influences during early life, mainly because these affect metabolism and the regulation of appetite. At the same time, a number of factors connected with lifestyle have a direct effect on the development of obesity. They are unhealthy eating habits, a low level of physical activity, too much time spent in front of screens, and insufficient sleep. When diet is poor and physical activity is inadequate, a positive energy balance results, and this in turn leads to an increase in body fat. Efforts to prevent obesity should focus on the child's everyday environment. Caregivers play a key role in shaping health-related behaviors, making their education particularly important. Preventive strategies should focus on supporting healthier eating habits, increased physical activity, better sleep patterns, and reduced screen time, and should be introduced as early as possible.

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Informed consent

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Conflict of interest

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Data and materials availability

No new data were created or analyzed in this study. Data sharing is not applicable to this article.

REFERENCES

1. Akagbosu CO, Nadler EP, Levy S, Hourigan SK. The role of the gut microbiome in pediatric obesity and bariatric surgery. *Int J Mol Sci.* 2022;23(23):15421. doi: <https://doi.org/10.3390/ijms232315421>.

2. Ali A, Al-Ani O, Al-Ani F. Children's behaviour and childhood obesity. *Pediatr Endocrinol Diabetes Metab.* 2024;30(3):148-158. doi: <https://doi.org/10.5114/peddm.2024.142586>.
3. Au LE, Zhu SM, Nhan LA, Plank KR, Frongillo EA, Laraia BA, Gurzo K, Ritchie LD. Household food insecurity is associated with higher adiposity among US schoolchildren ages 10-15 years: the Healthy Communities Study. *J Nutr.* 2019;149(9):1642-1650. doi: <https://doi.org/10.1093/jn/nxz108>.
4. Bała K, Staszko N. The role of the gut microbiota in childhood obesity. *Qual Sport.* 2026;49:67143. doi: <https://doi.org/10.12775/QS.2025.49.67143>.
5. Brambilla I, Bellanca E, Pistone C, De Filippo M, Votto M, Tondina E, Licari A, Guarracino C, Marseglia GL. Pediatric obesity: a mini-review for pediatrician. *Acta Biomed.* 2022;93(S3). doi: <https://doi.org/10.23750/abm.v93iS3.13078>.
6. Chen K, Zheng F, Zhang X, Wang Q, Zhang Z, Niu W. Factors associated with underweight, overweight, and obesity in Chinese children aged 3-14 years using ensemble learning algorithms. *J Glob Health.* 2025;15:04013. doi: <https://doi.org/10.7189/jogh.15.04013>.
7. Chung ST, Krenek A, Magge SN. Childhood obesity and cardiovascular disease risk. *Curr Atheroscler Rep.* 2023;25(7):405-415. doi: <https://doi.org/10.1007/s11883-023-01111-4>.
8. Daley SF, Balasundaram P. Obesity in pediatric patients. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2025. <https://www.ncbi.nlm.nih.gov/books/NBK570626/>.
9. Deal BJ, Huffman MD, Binns H, Stone NJ. Perspective: childhood obesity requires new strategies for prevention. *Adv Nutr.* 2020;11(5):1071-1078. doi: <https://doi.org/10.1093/advances/nmaa040>.
10. Drozd D, Alvarez-Pitti J, Wójcik M, Borghi C, Gabbianelli R, Mazur A, Herceg-Čavrak V, Lopez-Valcarcel BG, Brzeziński M, Lurbe E, Wühl E. Obesity and cardiometabolic risk factors: from childhood to adulthood. *Nutrients.* 2021;13(11):4176. doi: <https://doi.org/10.3390/nu13114176>.
11. Górczyńska-Kosiorz S, Kosiorz M, Dzięgielewska-Gęsiak S. Exploring the interplay of genetics and nutrition in the rising epidemic of obesity and metabolic diseases. *Nutrients.* 2024;16(20):3562. doi: <https://doi.org/10.3390/nu16203562>.
12. Khadilkar V, Shah N, Harish R, Ayyavoo A, Bang A, Basu S, Chatterjee S, Chhatwal J, Elizabeth KE, Gbate S, Gupta A, Kinjawadekar U, Kumar R, Mishra S, Sakamuri K, Saxena V, Singh H, Singh P, Sud A, Tiwari S. Indian Academy of Pediatrics revised guidelines on evaluation, prevention and management of childhood obesity. *Indian Pediatr.* 2023;60(12):1013-1031. doi: <https://doi.org/10.1007/s13312-023-3066-z>.
13. Littlejohn E. Pediatric obesity-a looming problem for the pediatric population. *Pediatr Ann.* 2023;52(2). doi: <https://doi.org/10.3928/19382359-20230102-01>.
14. Magiera B, Rybak J, Magiera K, Bator P, Razik M, Rozwadowska P, Ramian J, Razik W. Childhood obesity - risk factors and prevention strategies. *J Educ Health Sport.* 2024;60:175-187. doi: <https://doi.org/10.12775/JEHS.2024.60.012>.
15. Mahmoud R, Kimonis V, Butler MG. Genetics of obesity in humans: a clinical review. *Int J Mol Sci.* 2022;23(19):11005. doi: <https://doi.org/10.3390/ijms231911005>.
16. Minabe S, Sutoh Y, Otsuka-Yamasaki Y, Komaki S, Nakao M, Ohmomo H, Hasegawa Y, Ishigaki Y, Tanno K, Sasaki M, Shimizu A. Risk factors and prediction for pediatric obesity: current status and future perspectives. *Endocr J.* 2025;72(7):765-779. doi: <https://doi.org/10.1507/endocrj.EJ24-0724>.
17. Młynarska E, Bojdo K, Bulicz A, Frankenstein H, Gąsior M, Kustosik N, Rysz J, Franczyk B. Obesity as a multifactorial chronic disease: molecular mechanisms, systemic impact, and emerging digital interventions. *Curr Issues Mol Biol.* 2025;47(10):787. doi: <https://doi.org/10.3390/cimb47100787>.
18. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in underweight and obesity from 1990 to 2022: a pooled analysis of 3663 population-representative studies with 222 million children, adolescents, and adults. *Lancet.* 2024;403(10431):1027-1050. doi: [https://doi.org/10.1016/S0140-6736\(23\)02750-2](https://doi.org/10.1016/S0140-6736(23)02750-2).
19. Pietrzak K, Wasilewska M, Polok S, Demel K. Youth obesity: sport as a solution strategy. *Qual Sport.* 2025;40:59778. doi: <https://doi.org/10.12775/QS.2025.40.59778>.
20. Romanelli R, Cecchi N, Carbone MG, Dinardo M, Gaudino G, Miraglia Del Giudice E, Umamo GR. Pediatric obesity: prevention is better than care. *Ital J Pediatr.* 2020;46(1):103. doi: <https://doi.org/10.1186/s13052-020-00868-7>.
21. Sarni ROS, Kochi C, Suano-Souza FI. Childhood obesity: an ecological perspective. *J Pediatr (Rio J).* 2022;98(Suppl 1). doi: <https://doi.org/10.1016/j.jped.2021.10.002>.
22. Sivakumar S, Lama D, Rabhi N. Childhood obesity from the genes to the epigenome. *Front Endocrinol (Lausanne).* 2024;15:1393250. doi: <https://doi.org/10.3389/fendo.2024.1393250>.
23. Szczyrńska J. Pediatric obesity - time to act as early as possible. *Pediatr Endocrinol Diabetes Metab.* 2023;29(4):267-273. doi: <https://doi.org/10.5114/peddm.2023.133122>.

24. Tester JM, Rosas LG, Leung CW. Food insecurity and pediatric obesity: a double whammy in the era of COVID-19. *Curr Obes Rep.* 2020;9(4):442-450. doi: <https://doi.org/10.1007/s13679-020-00413-x>.
25. Tragomalou A, Moschonis G, Kassari P, Papageorgiou I, Genitsaridi SM, Karampatsou S, Manios Y, Charmandari E. A national e-health program for the prevention and management of overweight and obesity in childhood and adolescence in Greece. *Nutrients.* 2020;12(9):2858. doi: <https://doi.org/10.3390/nu12092858>.
26. Trang K, Grant SFA. Genetics and epigenetics in the obesity phenotyping scenario. *Rev Endocr Metab Disord.* 2023;24(5):775-793. doi: <https://doi.org/10.1007/s11154-023-09804-6>.
27. Umano GR, Bellone S, Buganza R, Calcaterra V, Corica D, De Sanctis L, Di Sessa A, Faienza MF, Improda N, Licenziati MR, Manco M, Ungaro C, Urbano F, Valerio G, Wasniewska M, Street ME. Early roots of childhood obesity: risk factors, mechanisms, and prevention strategies. *Int J Mol Sci.* 2025;26(15):7388. doi: <https://doi.org/10.3390/ijms26157388>.
28. Verduci E, Bronsky J, Embleton N, Gerasimidis K, Indrio F, Köglmeier J, de Koning B, Lapillonne A, Moltu SJ, Norsa L, Domellöf M; ESPGHAN Committee on Nutrition. Role of dietary factors, food habits, and lifestyle in childhood obesity development: a position paper from the European Society for Paediatric Gastroenterology, Hepatology and Nutrition Committee on Nutrition. *J Pediatr Gastroenterol Nutr.* 2021;72(5):769-783. doi: <https://doi.org/10.1097/MPG.0000000000003075>.
29. Wilhite K, Booker B, Huang BH, Antczak D, Corbett L, Parker P, Noetel M, Rissel C, Lonsdale C, Del Pozo Cruz B, Sanders T. Combinations of physical activity, sedentary behavior, and sleep duration and their associations with physical, psychological, and educational outcomes in children and adolescents: a systematic review. *Am J Epidemiol.* 2023;192(4):665-679. doi: <https://doi.org/10.1093/aje/kwac212>.
30. Wójcik M, Zachurzok A. Obesity in children: inheritance and treatment - state of art 2024. *Pediatr Endocrinol Diabetes Metab.* 2024;30(3):112-115. doi: <https://doi.org/10.5114/peddm.2024.144042>.
31. Yuan C, Dong Y, Chen H, Ma L, Jia L, Luo J, Liu Q, Hu Y, Ma J, Song Y. Determinants of childhood obesity in China. *Lancet Public Health.* 2024;9(12). doi: [https://doi.org/10.1016/S2468-2667\(24\)00246-9](https://doi.org/10.1016/S2468-2667(24)00246-9).
32. Zhang S, Qin X, Li P, Huang K. Effect of elective cesarean section on children's obesity from birth to adolescence: a systematic review and meta-analysis. *Front Pediatr.* 2022;9:793400. doi: <https://doi.org/10.3389/fped.2021.793400>.