Chapter

Obesity in Children: Recent Insights and Therapeutic Options

Mirjam Močnik and Nataša Marčun Varda

Abstract

Obesity in children, including adolescents, is nowadays, in the light of the COVID-19 pandemic, an even more pressing problem than before it, leading to increased prevalence of obesity and its comorbidities at young age. A simple and correct approach to diagnosis is essential, and some new insights in epidemiology, pathophysiology, and diagnosis are currently under investigation. Obesity in preschool children and metabolically healthy obesity are new entities that are recently being defined and written about. Additionally, several new factors that might influence obesity development are being researched, such as pollutants, sleep duration, and gut microbiota. In this chapter, we briefly present them as possible therapeutic targets in the future along with current therapeutic options in the pediatric population, namely lifestyle change, pharmaceutical options, and surgery. A child is always significantly affected by his/her family lifestyle, home, and social environment, which has to be considered in childhood obesity management.

Keywords: obesity, children, adolescents, treatment, recent discoveries

1. Introduction

Obesity, defined by the World Health Organization, as an excess in fat mass great enough to increase the risk of morbidity, altered physical, psychological or social well-being, and/or mortality [1], has reached pandemic proportions in recent decades [2]. Simultaneously, several "adult diseases," such as type 2 diabetes mellitus, hypertension, and nonalcoholic fatty liver disease, are emerging also in the pediatric population, therefore increasing their cardiovascular risk at a young age. Rarely, children can have an underlying endocrine or monogenic cause of their weight gain. Therefore, obesity in childhood is mostly the result of energy intake and expenditure imbalance [2].

In this chapter, we will review some new insights into childhood obesity with possible therapeutic options.

2. Epidemiology

The increasing prevalence of childhood obesity with advancing age is a well-known fact in recent decades and varies by racial, ethnic, and socioeconomic factors [2].

Children of obese parents have an increased risk for obesity development, not only because of hereditary but also due to environmental factors [3]. Additionally, childhood, and especially adolescent obesity, are associated with obesity in adulthood [4, 5], creating a vicious cycle.

After a dramatic increase in obesity prevalence approximately 40 years ago, some studies after the year 2000 showed a steady state; however, these studies were not replicated and a true trend of stabilizing obesity incidence was not proven [2]. Some isolated studies still brought some hope in decreasing obesity in children, but then the COVID-19 pandemic happened and had a significant impact on childhood obesity.

2.1 COVID-19 and obesity in children

The pandemic of childhood obesity has collided with the pandemic of COVID-19 in the last few years [6]. Many countries from all over the world are reporting an increased prevalence of obesity in childhood [7]. Billions of people were subjected to home confinement leading them to change their lifestyles and eating behaviors, including buying and consuming large quantities of preserved and processed food. Simultaneously, sedentary behavior and increased screen time led to decreased physical activity. Isolation, restricted activity, and unhealthy food choices, associated also with financial reasons, are the most commonly identified culprits for increased obesity, along with other psychosocial, behavioral, and environmental factors [6, 7]. Additionally, preexisting disparities in obesity in terms of race, ethnicity, and socioeconomic status increased [8].

3. Etiology and pathophysiology

Obesity in children is usually the consequence of an imbalance between nutrient consumption and energy expenditure, rarely, other causes can be identified. Still, genetic etiology or at least predisposition might play an important role in the pathogenesis of obesity. Genetic predisposition to obesity has been widely accepted; however, there are only few articles associating specific gene pathways to obesity in children, namely Toll-like receptor 4 signaling pathway [9], leptin-melanocortin signaling pathway [10], c-Jun N-terminal kinase signaling pathway [11], and adenosine monophosphate-activated protein kinase signaling pathway [12]. Several genes, however, they were associated with overexpression in visceral fat and subcutaneous adipose tissues, including the phospholipid transfer gene, ras, adipsin, and calcyclin [13, 14] with gene variants associated with severe obesity in both childhood and adulthood [15, 16].

In addition, differentially expressed genes in children with obesity were identified recently in pathways associated with the immune system. Also, matrix metalloproteinase 9 and acetyl-CoA carboxylase ß were identified as hub genes in the protein-protein interaction network and might be marker genes for childhood obesity [17].

Another important association, found recently, is between obesity and sleep deprivation, highlighted by growing evidence, described below. In these studies, autonomic dysfunction is presented as a new possible pathophysiological pathway to obesity development, which was supported by sympathovagal imbalance, particularly during the night in obese children compared to healthy controls [18]; however, the evidence is not conclusive.

4. Diagnosis and risk factors

Evaluation of children with obesity is aimed at determining the diagnosis and the cause of weight gain and assessing for comorbidities resulting from excess weight.

4.1 Establishing the diagnosis

Clinical diagnosis is based on anthropometric measurements, usually widely used as body mass index (BMI). BMI cut-off points in adults (25 and 30 kg/m^2 for overweight and obesity, respectively) are used due to the observed increased health risks at these levels and are adjusted in the pediatric population according to age, sex, and height. BMI has some disadvantages; mainly it does not predict body fat percentage and it does not distinguish between lean and fat mass. Additional measurement of waist/hip circumference is a tool to identify central obesity, associated more strongly with complications, such as insulin resistance, dyslipidemia, and nonalcoholic fatty liver disease [19].

Measuring waist/hip circumference can also be challenging and time-consuming in terms of behavioral, cultural, and environmental issues. Additionally, waist circumference may be affected after eating by abdominal distension. Given these limitations, new strategies to find a better anthropometric obesity measure are evolving. One of them is neck circumference measurement, which has been associated with central obesity and abnormal metabolic status. It can be reliably used to screen overweight and obesity in children with identification of those with high BMI [20, 21].

Interestingly, dermatoglyphic differences have also been observed in children and adolescents with obesity. In participants with normal weight, a higher frequency of ulnar loops on the index and middle finger were noted along with presence of radial loops on the middle finger. In children with obesity, a greater frequency of whorls on the index and middle fingers were observed in males along with arches in the middle finger [22].

Body fat mass can further be estimated by several other methods, namely dual-energy X-ray absorptiometry, bioelectrical impedance assay, computed tomography and magnetic resonance imaging of abdomen, measurement of skinfold thickness at multiple sites, air displacement plethysmography, and stable isotope dilution techniques [19].

4.2 Metabolically healthy obesity in children

Metabolically healthy obesity in children is a new entity mainly regarded as obesity without cardiometabolic risk factors; however, definitions are variable. An international panel of 46 experts agreed that criteria for metabolically healthy obesity in children include high-density lipoprotein-cholesterol >1.03 mmol/l (or > 40 mg/dl), triglycerides ≤1.7 mmol/l (or ≤ 150 mg/dl), systolic and diastolic blood pressure ≤ 90th percentile, and a measure of glycemia [23]. Due to the heterogeneous definition, the prevalence of metabolically healthy obesity varies from 3 to 80% [23]. Specific genetic predispositions and environmental factors or absence (family history, specific gene variants, and decreased physical activity) of them contribute significantly to metabolic health [24]; however, they were not widely researched and accepted yet. It is still unknown if these children have lower cardiovascular risk and can be managed less aggressively.

4.3 Challenges of obesity in children under six years of age

With increasing obesity, preschool years present a point of opportunity for children to be active, develop healthy habits, and maintain a healthy lifestyle, however, the prevalence of obesity in this age group is inconsistent in European countries and varies up to one-third of 5-year-old children. Measuring overweight and obesity in this group may be challenging [25]. The prevalence was lower among children born to parents with high education, and higher among children born to foreign parents and overweight mothers [26].

In younger children with obesity, there is also an increased risk for monogenic or underlying endocrine disease and should be excluded, especially when severe obesity is present. However, in this age group, establishing the diagnosis should be done with caution when obesity is not as severe. It was shown that young children with a high BMI percentile have lower fat mass than older children with the same BMI percentile and therefore obesity in this age group can be over diagnosed [27].

In order to plan appropriate management of obesity in younger children, we need a better understanding of potential modifiable factors. Specific risk factors for obesity in children under six years of age include maternal diabetes, maternal smoking, gestational weight gain, and rapid infant growth [28]. In preschool children, parents can have a significant influence on modifiable risk factors associated with severe obesity already in this age group and also in further growth. These risk factors include inappropriate nutrition (sugar-sweetened beverages and fast food and skipping breakfast), inactivity (low frequency of outdoor play and excessive screen time), behaviors (lower satiety responsiveness, sleeping with a bottle, lack of bedtime rules, and short sleep duration) and socio-environmental risk factors (informal childcare setting, maternal smoking, and maternal obesity) [27, 29].

4.4 Risk factors for obesity development

The development of obesity is very complex and includes many risk factors. Although we mostly define the etiology as an imbalance between calorie intake and consumption, the reality is not as simple. Genetic, biological, and socio-environmental factors, including family, school, community, and national policies, can play a crucial role. The complexity of risk factors among the pediatric population leads to difficulty in treatment and many interventional trials have been proven ineffective [30]. Therefore, early identification of possibly modifiable factors at an individual, local, and global level should be done to allow appropriate management and policies for obesity prevention and treatment.

Traditional risk factors in school children and adolescents include genetic predisposition, diet (fast food and sugar-sweetened food), eating patterns, lack of physical activity and increased sedentary time (viewing television, playing video games, and using computer), unresolved stress, environmental settings (home, school, and community), sociocultural factors [30].

Some new risk factors for obesity development are under investigation and could present a possible modifiable target in obesity management. Additionally, some new insights into already-known risk factors are presented.

4.4.1 Screen time

The relationship between screen time and increased risk of obesity is well documented. Screen media exposure leads to obesity in children and adolescents through

increased eating while viewing, increased sedentary time and reduced sleep duration. Randomized controlled trials of reducing screen time in community settings have reduced weight gain in children, demonstrating a cause-and-effect relationship. However, some evidence also suggests a promise for using interactive media to improve eating and physical activity behaviors to prevent or reduce obesity [31].

Studies, reporting associations between obesity and playing video games are almost half positive, and the rest reported no association. There was preliminary evidence on the effectiveness of exergame (physically active) play for weight reduction and to attenuate weight gain; however, there was little indication that interventions effectively reduced video game play or general screen time [32].

4.4.2 Obesity and sleep duration

The present studies indicate that short sleep duration increases the risk of childhood obesity, making appropriate sleep duration another priority in a child's life [33, 34]. Few studies examined other dimensions of sleep, namely quality, efficiency, and bed/wake time in association with weight status. Even when present, there were variations in defining and measuring these dimensions, making their comparison and potential discrepancies difficult to assess [35].

There are several mechanisms proposed to link sleep loss and the risk of weight gain, based on hormonal and neuroendocrine changes associated with sleep restriction. One of the potential may lie within the hypothalamus or within hypothalamic communication with the peripheral system, and through hormonal systems via leptin and ghrelin (with peripheral metabolic indicators, such as glucose and cholecystokinin). Two distinct neuropeptides, orexin A in B, are synthesized mainly by the neurons in some parts of the hypothalamus and are believed to play a key role in the interaction between sleep and feeding. Orexins induce and support arousal and promote feeding, and are involved in the regulation of many functions, such as sleep-wakefulness, locomotor activity, feeding, thermoregulation, and neuroendocrine and cardiovascular control. Orexin neurons may be disinhibited by low levels of leptin and glucose and excited by ghrelin. Orexin activity is associated with an increased sympathetic tone, which in sleep deprivation may further inhibit leptin release and stimulate ghrelin release, consistent with the effects of short sleep on the peripheral levels of both hormones already observed in adult population [36]. Increased catecholamine levels in sleep deprivation also inhibit insulin secretion and promote glycogen breakdown, increasing the risk of hyperglycemia and insulin resistance seen in obesity [36]. Additionally, it has been suggested that C-reactive protein (CRP) is a leptin-binding protein, and increased CRP levels, seen in obesity, could present a possible mechanism for leptin resistance [36]. There may also be a role for the reward system in modulating food intake and energy storage following a state of sleep loss. Voluntary sleep restriction has been shown to increase snacking, the number of meals eaten per day, and the preference for energy-dense foods [37]. Decreased sleep may also have an adverse effect on energy expenditure. It is also possible that behavioral changes and poor parent-child dynamics may influence sleep deprivation in children, contributing further to weight gain, present already due to other socioeconomic factors [36].

4.4.3 Obesity and gut microbiota

Antibiotics are one of the most commonly prescribed drugs in childhood and their use can cause unwanted problems. Among these, antibiotic-induced gut

microbiota dysbiosis has been associated with obesity. This problem is even more relevant to children that are frequently treated with antibiotics. The microbiota composition and its disturbance in neonatal and in early childhood have a profound impact further in life. It can contribute to a decrease in the number and composition of microbiomes affecting glucose and lipid metabolism and immune system development [38]. Evidence suggests that short-chain fatty acids made by the gut microbiota directly or indirectly modulate physiological and pathological processes in relation to obesity. At first glance, excessive short-chain fatty acids represent an additional energy source and could cause an imbalance in energy regulation. Simultaneously, however, short-chain fatty acids participate in glucose-stimulated insulin secretion and release of peptide hormones, which control appetite [39]. However, the causal effect has not been defined and no definitive therapeutic approach has been elucidated. Probiotics and prebiotics could play a role in treating microbial dysbiosis. The addition of specific bacterial strains has been associated with normal weight gain [38]. Dietary modulation has been proven effective also in children with genetic obesity [40].

The exact microbiota status in obesity has not been elucidated yet; however, some studies have tackled this challenge. One study showed associations between *Firmicutes spp*. to obesity and *Bifidobacterium spp*. with a healthy weight in children [41]. Additionally, the addition of *Lactobacillus casei* strain was associated with weight loss while also improving lipid metabolism in obese children via significant increase in the fecal *Bifidobacterium* numbers [42].

4.4.4 Obesity and pollutants

Due to the increase in pollutants in our environment, their concentration has been associated with childhood obesity several times. Recent meta-analyses showed that air pollution is correlated with a substantially increased risk of childhood obesity [43, 44]. The biological and physiological mechanisms regarding the cause-and-effect between air pollution and obesity are still unclear [43]. Animal studies showed that ambient air pollution exaggerated adipose inflammation and insulin resistance [45]. This may further affect the basal metabolic rate and appetite control of exposed individuals [43]. Another animal experiment indicated that exposure to air pollution results in Toll-like receptor 2/4-dependent inflammatory activation in lipid oxidation could lead to metabolic dysfunction and weight gain [46]. Air pollution might also prevent people from going out, causing excess sedentary time, especially in some heavily polluted parts of the world [43].

Along with pollution, other chemicals may affect obesity development. They are called obesogens and are defined as exogenous chemicals belonging to the group of endocrine-disrupting chemicals and are believed to interfere with obesity development. The major mechanism through which obesogens can contribute to obesity is believed to be the activation of nuclear receptors involved in adipogenesis, lipid metabolism, inflammation, and maintenance of metabolic homeostasis. Several chemicals are under investigation as potential causal factors in obesity development; however, these associations remain controversial and it is difficult to find evidence for direct causality between environmental exposure and disease [47]. These chemicals include bisphenol A [47], phthalates [48], perfluoroalkyl substances [49], polycyclic aromatic hydrocarbons [50, 51], etc.

5. Obesity prevention

Disease prevention is even more important in obesity since there are numerous complications, presented in **Table 1**, associated with obesity and even more pronounced when obesity is present early in life [30].

Obesity prevention is therefore a major challenge that has yet to be tackled appropriately. So far, the success of our intervention showed that our understanding of effective prevention is still not achieved [52].

Preventive activity is divided into primordial, primary, secondary, and tertiary. It is primarily achieved by implementing measures to ensure a normal weight and health, and prevent the development of obesity. Public health and national policy strategies play an important role in primordial prevention. The approaches are cost-effective and reduce the overall burden of obesity. With primary prevention, we want to eliminate or reduce exposure to all factors that cause obesity. Effective secondary prevention of obesity is based on early detection and population screening. Tertiary prevention strategies are focused on reducing or delaying the long-term complications that can be caused by obesity (**Table 1**) [53, 54].

Our understanding of childhood obesity, energy balance-related behaviors, determinants of behavior, and effective components of prevention programs is still deficient [52]. Mostly, interventions consist of diet combined with physical activity at individual or community level. Combined interventions had moderate success in children younger than 5 years; however, weaker evidence is present with only dietary or only physical activity interventions [55]. In contrast, in older children, interventions focused only on physical activity can reduce the risk of obesity, but there is no evidence that interventions focused only on diet are effective. Importantly, combined diet and exercise might prove more effective; however, behavioral prevention programs were associated with small improvements in weight outcomes [55, 56].

Cardiovascular	dyslipidaemia, hypertension, left ventricular hypertrophy, coagulopathy, chronic inflammation, endothelial dysfunction
Pulmonary	obstructive sleep apnea, asthma, exercise intolerance
Gastrointestinal	gastroesophageal reflux, non-alcoholic fatty liver disease, steatohepatitis, gallstones, constipation
Endocrine	insulin resistance, type 2 diabetes, precocious puberty, polycystic ovary syndrome (girls), hypogonadism (boys)
Renal	glomerulosclerosis
Neurological	idiopathic intracranial hypertension (pseudotumour cerebri)
Dermatologic	acanthosis nigricans, intertrigo, hidradenitis suppurativa, furunculosis, stretch marks
Musculoskeletal	slipped capital femoral epiphysis, Blount's disease, forearm fracture, back pain, flat feet
Psychosocial	poor self-esteem, anxiety, depression, eating disorders, social isolation, lower educational attainment
Long-term risks	carotid artery atherosclerosis, colorectal carcinoma, ischemic heart disease, stroke, short life span, premature death

Table 1.
Complications of childhood and adolescent obesity [30].

6. Therapeutic options

Obesity management in childhood is based on lifestyle interventions with an emphasis on dietary and physical activity modifications, possibly involving the whole family. However, only a modest effect of lifestyle interventions is seen in severe obesity [57], requiring a consideration about which therapeutic approach to use according to the severity of obesity and the presence of obesity-related comorbidities. Pharmaceutical and surgical options are limited, as presented below.

6.1 Lifestyle interventions

Lifestyle interventions include behavioral measures to alter dietary habits and increase physical activity and are the preferred methods to treat overweight and obesity in children and adolescents. A variety of multicomponent lifestyle interventions may improve BMI in children and adolescents with varying degrees of overweight and obesity [57]. Commonly, they have produced losses from 5 to 20% of excess weight over 3 to 6 months in children [58]. Over 6 to 12 months, the change has ranged from 25% loss to 10% increase in excess weight [58]. In children up to the age of 6 years, a reduction in BMI Z-scores up to 2-year follow-up showed beneficial effects of diet, physical activity, and behavioral interventions. In older children, the beneficial effect of the same measures was found in at least of 6 months of interventions duration [59]. The durability of weight loss is often limited by physiologic systems that are evolutionarily designed to promote weight gain, making lifestyle interventions even more difficult. Continued treatment preventing relapse with face-to-face interventions with a multidisciplinary approach is recommended [60].

Physical activity of at least 60 minutes a day at a moderate-vigorous level is usually recommended [61]. Dietary approaches to weight loss focus on caloric restriction with limited guidelines recommending a very low-carbohydrate/ketogenic diet. Dietary approaches beyond simply caloric restriction with individual assessment and care are recommended for optimal patient outcomes [62]. Additionally, eating behavior has an important impact on obesity development. It was shown that skipping breakfast in the family had significantly increased the risk of childhood overweight and obesity [63]. The same applies to high-energy intake at dinner or late-night snacking [64].

Children that were breastfed for at least 12 months had a significantly lower risk of being overweight or obese than those breastfed for less than 17 weeks. The age of introduction of solid food was not associated with the risk of excess weight at 2 or 3 years of age [65]. Interestingly, dairy products consumption later in life has also been associated with decreased risk of obesity [66].

Not only the amount but also the composition of food intake is of crucial importance. Despite the increased intake, children with obesity often exhibit vitamin D deficiency, usually associated with decreased outdoor activities [67, 68], making appropriate dietary management even more demanding.

All lifestyle interventions should be supported by psychosocial support and treatment that are carried out simultaneously to achieve maximal success. Well-established psychological treatments include family-based behavioral treatment and parent-only behavioral treatment for children utilizing behavioral strategies. Appetite awareness training and regulation of cues treatments are considered experimental. Additional research is needed to test a stepped care model for treatment and to establish the ideal dosage (number and length of the sessions), duration, and intensity of treatments for long-term sustainability of healthy weight management [69].

Especially in severe obesity, lifestyle changes alone, although of fundamental importance, are frequently insufficient. Drug therapies for children are limited and surgical approaches associated with potential morbidity have less known long-term consequences. However, for children with severe obesity, a multifaceted behavioral, pharmacological and surgical approach may be implemented [70].

6.2 Pharmaceutical options

The current use of pharmacotherapy for the treatment of obesity in the pediatric population is limited. Several anti-obesity medications have been approved by the Food and Drug Administration for use among adult patients; however, they are used off-label in the pediatric population [71]. Most commonly prescribed drugs include metformin, topiramate, sibutramine, orlistat, and combinations with fluoxetine and exenatide [71, 72]. A systematic review showed that pharmacological interventions using metformin, sibutramin, orlistat, or fluoxetine may have small effects on BMI reduction. However, trials conducted were generally of low quality, with many having a short or no post-intervention follow-up period and high dropout rates. Adverse effects have also been reported but not in a standardized manner [72]. Therefore, the current endocrine society practice guidelines recommend that these drugs should be used only in clinical trials [73] and only orlistat for patients older than 12 years of age, and phentermine in those older than 16 years are currently approved by the Food and Drug Administration [71, 74].

6.3 Surgical options

The most commonly known surgical option is bariatric surgery, which has been shown to result in a significant and sustained decrease in BMI and improved comorbidities in adult patients. In the pediatric population, there has been an increase in bariatric surgery use, although it is still infrequently performed. Several types of bariatric surgery are being used, most commonly laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass. The first one carries a lower risk of micronutrient deficiencies (the procedure does not result in malabsorption) and is less complex to do. The Roux-en-Y gastric bypass results in restriction of caloric intake (reduced capacity and neuroendocrine mechanisms) and malabsorption of food along with vitamins and minerals [2]. Adjustable gastric banding is also done in adolescents. Appropriate patient selection is of utmost importance, and the following criteria have been developed [modified according to references 2 and 75]:

1. Body mass index >35 kg/m^2 and a severe comorbidity,

2. Physical maturity (completing 95% of predicted adult stature based on bone age or reaching Tanner stage IV),

3. History of lifestyle changes to lose weight,

4. Ability and motivation of the patient and family to adhere to pre- and postoperative treatments,

5. Understanding of the risks and benefits of surgery,

6. Family support [2, 75].

Contraindications are rare but include a medically correctable cause of obesity, a medical, psychiatric, psychosocial, or cognitive condition that prevents adherence to postoperative dietary and medication regimens, current or planned pregnancy within 12 to 18 months of the procedure, an inability on the part of the patient or parent to comprehend the risks and benefits of the surgical procedure and an ongoing substance abuse problem [75]. Short-term complications include wound infections, leakage at anastomotic sites, pulmonary embolism, small bowel obstruction, gastrojejunal strictures, and gastrogastric fistula. Long-term complications include nutritional deficiencies of iron, vitamin B12, thiamine, and vitamin D. Lifelong vitamin and mineral supplementation is recommended to prevent the development of nutritional deficiencies as a result of decreased intake or malabsorption [2].

According to research, adolescents reached similar weight loss benefits as adults five years after gastric bypass surgery. Improvements in diabetes and hypertension were even greater than in adults [76].

Another possibility is endoscopic procedures, such as an intragastric balloon, for a limited period of time as an adjunct, which showed good efficacy in weight loss and comorbidities improvement with a good safety profile [76].

7. Psychosocial and parental influence

When managing behavioral changes in children, psychosocial and parental factors have to be considered as two elements necessary for effective management.

Childhood is regarded as the most important period of life affecting adulthood. Health problems or illnesses during this period would be brought to maturity or become a risk factor for the onset of diseases in adulthood [77]. The family and environment provide a great impact on social, cognitive, behavioral, and health aspects, including overweight and obesity. Interventions in this field should begin in childhood. Parents play a critical role in shaping child's healthy lifestyle from an early age. They should pay attention to children's weight, eating behavior, and food intake. They are also role models for their children. With their involvement, the long-term effects of lifestyle interventions are enhanced. The most effective way of preventing and controlling overweight and obesity is through family empowerment, including parental knowledge about nutrition, its influence on food choice, eating patterns, sedentary habits, and physical activity. The beliefs and parent's lifestyle and health promotion are also of vital importance due to learning by imitation [77]. Interestingly, parent-only interventions had a similar effect compared with parent-child interventions in children 5 to 11 years [78].

Several socioeconomic factors also influence obesity development in multifaceted ways from an early age on and should be accounted for in childhood obesity management [79, 80].

8. Conclusions

Obesity in children is a significant medical problem, leading to several comorbidities. Several new insights are being researched in diagnosis, management, and treatment options. Additionally, several new factors that might influence obesity development are being identified and researched, such as pollutants, sleep duration, and gut microbiota. In this chapter, we briefly presented them as a possible

therapeutic target in the future along with current therapeutic options in the pediatric population, namely lifestyle change, pharmaceutical, and surgical options. A child is always significantly affected by his/her family lifestyle, home, and social environment, which has to be considered in child obesity management.

Conflict of interest

The authors declare no conflict of interest.

Author details

Mirjam Močnik[1*] and Nataša Marčun Varda[2]

1 Department of Pediatrics, University Medical Center Maribor, Maribor, Slovenia

2 Department of Pediatrics, University Medical Center Maribor and Medical Faculty, University of Maribor, Maribor, Slovenia

*Address all correspondence to: mirjammocnik91@gmail.com

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