View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by IntechOpen

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 7

Childhood Obesity

Laura Mihaela Trandafir, Ileana Ioniuc and Ingrith Miron

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/65914

Abstract

Currently, the prevalence of obesity among children and adolescents and related complications is considered one of the most important nutritional problems globally. The prevalence of childhood obesity in Europe is 10 times higher now than it was in the 1970s. Initial assessments of these patients should include taking a careful history (investigating comorbidities, family history and potentially modifiable behaviors) and physical examination. The degree of investigation is dependent on the patient's age and severity of obesity, the findings on history and physical examination and associated familial risk factors. Childhood obesity treatment is based on sustained lifestyle changes with family involvement. Management intervention strategies include nutrition, physical activity, behavior and lifestyle changes, medication and surgical considerations.

Keywords: obesity, childhood, adolescents

1. Introduction

Obesity is a chronic disorder of the state of nutrition characterized by an increase in body weight due to excessive adipose tissue, which occurs when the calorie intake exceeds the caloric needs of a body with low energy expenditure. Obesity is currently a significant public health problem, as we have witnessed a dramatic increase in the number of obese and overweight children worldwide in the last years. Children's risk of obesity varies by age and sex groups, ethnic/racial groups, socioeconomic status, geographic and rural/urban regions. The obesity etiology is complex, involving genetic, environmental, psycho-socio-cultural, neuroendocrine and metabolic factors. Complications of pediatric obesity occur during childhood and adolescence and increase the risk for morbidity and mortality in adulthood.

INTECH
open science | open minds

© 2017 The Author(s). Licensee InTech. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. CC BY

2. Nosologic framework of obesity in children and adolescent

2.1. Definition

Obesity is an important pediatric public health problem associated with risk of complications in childhood and increased morbidity and mortality throughout life. Overweight and obesity predispose people to noncommunicable diseases such as heart disease, diabetes mellitus, musculoskeletal and psychological disorders and certain types of cancer [1].

Without intervention, obese infants and young children will likely continue to be obese during childhood, adolescence and adulthood [1–3].

The methodological problem of inconsistency between criteria of childhood obesity classification is a major obstacle in studying global trends for younger age group. The body mass index [BMI: weight (kg)/height $(m)^2$] is the parameter used for the screening of overweight and obesity in childhood because it is easy to determine, it tends to correlate well with body fat, and it has been widely used in adults to define obesity. It decreases until the period called "adiposity rebound" when body fat is at the lowest level (between 3 and 7 years) and after then BMI increases again until the adulthood [4, 5].

The child's BMI must be plotted on nationally recommended BMI—for age charts. The classification of overweight and obesity varies among guidelines, such as those from Centre for Disease Control (CDC), International Obesity Task Force (IOTF) and World Health Organization (WHO).

For example, in UK (IOTF BMI values) the cut off points for and are the BMI >91st and >98th percentile, respectively.

The definition of overweight and obesity using BMI percentiles in the USA: children aged 2 years and older with a BMI between the 85th and 95th percentile is overweight, and those with a BMI greater than the 95th percentile for a specific age and sex subgroup are obese [4–6].

According to WHO, for children aged between 5 and 19 years, overweight is >2 standard deviations and obesity is defined as a BMI-for-age >1 standard deviation, above the WHO growth reference median [1].

The IOTF BMI values represent standard international references that allow the screening of adiposity in children and adolescents worldwide under the same criterion, without variations depending on geographic, social and secular trends [5].

2.2. Epidemiological data

Currently, the prevalence of obesity among children and adolescents and related complications is considered one of the most important nutritional problem globally. The obesity epidemic among children is the result of excess energy intake and inadequate energy expenditure [2]. The prevalence of childhood overweight and obesity has increased worldwide in recent decades, and the numbers of those affected continue to rise at an alarming rate [6].

http://dx.doi.org/10.5772/65914

In recent years, the epidemiology of overweight and obesity is well described in many European countries and the data showed the increasing trends in the prevalence of childhood obesity. The current prevalence of childhood obesity is ten times higher than it was in the 1970s [5].

The incidence of overweight and obesity increased progressively from infancy through adolescence [5, 6]. High prevalence of obesity in 0- to 6-year-old children is warning signs and risk for increased rates of obesity in adolescence and adulthood reported.

North America and some countries in Europe have shown consistent year-on-year increases in prevalence of overweight (20–30%) and obesity (5–15%), although recent surveys indicate that the rising trends are easing, with a plateau in prevalence levels shown since around 2005 [6].

Children's risk of obesity varies by age and sex groups, ethnic/racial groups, socioeconomic status, geographic and rural/urban regions. The key reason for the variations is due to the considerable socioeconomic and lifestyle differences and the differences in differing criteria for obesity and overweight definition [4, 7].

Several countries of Southern Europe appear to be showing high prevalence of childhood obesity (20–35%) in the Italy, Greece, Malta, Portugal and Spain. The highest levels of overweight and obesity were in Southern Italy (30.1 and 33.1% in preschool boys and, respectively, girls) and in various regions of Spain (29.4% in both sexes, increasing to 32.6% among children aged 7- to 10-year old), followed by Greece (19.1 and 23.6% in boys and, respectively, girls) [7–9]. Children and adolescents residing in countries surrounding the Mediterranean Sea show the highest rates ranging from 20 to 40%, too [7]. Studies concerning obesity with regard to its prevalence in Portugal reported a rate of overweight in children under the age of 6 years, 13.6% in boys and 20.4% in girls, and, obesity varied between 6.5% in boys and 6.9%, respectively, in girls [10]. The very high prevalence of childhood obesity in Mediterranean countries could be secondary to lifestyle changes (switching from a healthy Mediterranean diet to a fast food type of diet and lower physical activity levels) [7].

In children and adolescents residing the Scandinavian countries and Central Western European countries, the prevalence of overweight and obesity is far lower (10–20%). Scandinavian countries have the lowest prevalence of obesity in all age groups, except Finland which reported the prevalence of overweight and obesity in school children of 23.6 and 19.1% for boys and, respectively, girls [7, 11]. It is important to note that among preschool children, the highest prevalence rates of overweight and obesity were in Ireland (26 and 29% in boys and, respectively, girls), United Kingdom (24.1 and 21.4% in boys and, respectively, girls). In school children, the prevalence of overweight and obesity has increased to 32.7% and, respectively, 29.2% in 2007 the United Kingdom [7]. In developed countries, an increasing number of studies suggest that children of lower-income families are vulnerable to becoming obese, possibly due to poor dietary habits and limited opportunities for physical activity [7, 12]. In the UK, the prevalence of overweight and obesity in 11- to 12-year-old children was 25%, with higher rates in girls (29%) and students from lower socioeconomic backgrounds (31%), and the highest rates was observed in black girls (38%) [13].

Data from Eastern European countries indicate the prevalence rate is smaller (15%), but rising. In Lithuania, the Russian Federation, Slovakia and Poland, the overweight and obesity prevalence ranges from 8.46 to 15.8% in children aged 6–12 years. It is likely that the huge economic burden and the associated poverty following the political transition in the 1990s may have contributed to the relatively low obesity prevalence in Eastern Europe [7].

3. The etiology of obesity and the risk factors

Obesity is a multifactorial disease with a complex etiology being involved genetic, environmental, psycho-socio-cultural, neuroendocrine and metabolic factors, intestinal microbiota. The factors involved in its etiology included the 'obesogenic' environment, and the unhealthy dietary behaviors and patterns of physical activity [14].

There is growing interest in the role of experience in early life in the risk of becoming overweight or obese. In children, prenatal life may be a critical period when the long-term regulation of energy balance is permanently 'programmed'. The perinatal parameters and factors implicated in the etiology of obesity include maternal obesity, excessive weight gain in pregnancy, gestational diabetes, and maternal smoking, duration of breastfeeding, rapid infancy weight gain and other cultural or familial factors associated with childhood eating patterns and activity levels [14, 15].

Maternal obesity is a strong predictor of overweight and obesity in children. Obesity in pregnant women was found to be associated to high newborn weight and also causing obesity and metabolic syndrome risk in later life of the individual. Intrauterine growth restriction is associated with the development of central adiposity and adult-onset cardiovascular risk [4, 14].

Child's exposure to passive smoking, since the product conception stage, predisposes to the development of obesity and obesity related diseases as a result [14, 16, 17].

Breastfeeding could help infants to better recognize satiety signals and hence to better self-regulate energy intake. The prevalence and duration of breastfeeding is higher in countries with relatively lower prevalence of childhood obesity such as Sweden, Finland and Austria, in comparison to countries such as Italy, Greece and the UK, where is less [7, 14, 15].

Ethnicity is associated with differences in eating behaviors, preferences, and cultural influences may contribute to obesity among children and youth in minority populations.

The environmental factors are represented by: over-nutrition (high fat, high sugar diets), sedentary lifestyle, short sleep duration, abuse of drugs (antibiotics, corticosteroids, anti-epileptics drugs), smoking and alcohol. Almost all obesity in children is strongly influenced by environmental factors, caused by a sedentary lifestyle or a caloric intake that is greater than needs. However, this explains only a part of obesity risk, but is important targets for treatment such they can be modifiable.

Sugar-sweetened beverages—the literature evidence suggests that consumption of sugar-sweetened beverages is an important factor in the development of obesity in some individuals.

In the United States, sugar-sweetened beverages supplied an average of 270 kcal/day, representing 10–15% of total caloric intake. In a separate randomized trial on children aged 5–12 (primarily normal weight), consuming one serving of a sugar-free beverage daily was associated with less weight gain and fat accumulation than consuming one serving of a sugar-sweetened beverage. Other studies have found that dietary salt intake is associated with increased intake of sugar-sweetened beverages, perhaps because of increased thirst [18, 19].

Television viewing is an environmental influence on the development of obesity in children. Contact of a child with television begins in the newborn stage and tends to increase continuously. In the first year of life, children react to the screen characters with mimics and voice. Toddlers spend approximately 1 h a day watching television, and from the 4th year of life the exposure to other type of media expands and rises significantly to reach 7 out of 24 h per day. The presence of a television in a child's bedroom and any time spent in watching television are directly related to the prevalence of obesity in children and adolescents. A significant association between advert exposure and childhood obesity has been demonstrated in a cross-cultural study which included data from the USA, Australia and eight European countries [18, 20–22].

Video games—the use of PC or console games has been associated with obesity in children. Half of American children have either a DVD, video or game console in their bedroom and, third, a computer with access to Internet [22, 23].

Sleep—there is a reported association between shortened sleep duration and obesity. A causal association arises from a short-term experimental study in which sleep deprivation for 1 week was associated with increased food intake, weight gain and higher leptin levels as compared to the child's usual sleep. Moreover, sleep may have an association with insulin resistance, independent of its association with obesity. The mechanism between sleep duration and obesity has not been well-known, but may comprise dysfunction in serum leptin and/or ghrelin levels, both are involved in the regulation of appetite [4, 18]. A meta-analysis found that sleep is positively associated with fat mass in toddlers. A positive association between nighttime sleep and BMI *z*-scores were observed in the study reported by Kuzik and Carson [24].

Medications that may cause weight gain in children include cortisol and other glucocorticoids, sulfonylureas, tricyclic antidepressants (TCAs), monoamine oxidase inhibitors, such as phenelzine, oral contraceptives, insulin (in excessive doses), thiazolidinediones, risperidone, clozapine [25].

Other environmental factors that have been proposed as possible contributors to obesity include the influences of gut microbiota, toxins and viruses. Due to the influence of gut microbiota, it has been suggested that there exists a relationship between the resident intestinal bacteria and the potential for weight gain. Effect of microbiota may be partially responsible for increased rate of obesity in children born via cesarean section. Intestinal bacteria seem to influence several factors leading to development of obesity complications such us non-alcoholic steatohepatitis, cardiovascular disease and insulin resistance in humans [26]. It has been suggested that obesity can be triggered or worsened by exposure to a virus. Adenovirus 36 increases body fat in several animal models [18].

Other epidemiologic studies highlight the possibility that obesity could be triggered or exacerbated by exposure to environmental endocrine disrupting chemicals (dichlorodiphenyltrichloroethane—DDT and bisphenol A—BPA). Some studies in adults and children establish an association between urinary BPA levels and obesity or obesity related diseases, as well as diabetes and cardiovascular disease [18].

Endocrine disruptors can disturb every level of the endocrine system. They can interrupt the action of enzymes involved in steroidogenesis. The endocrine disruptors inducing obesity are called obesogens and have been revealed to target transcription regulators that function to control intracellular lipid homeostasis as well as proliferation and differentiation of adipocytes. The main group of regulators that are targeted represent a group of nuclear hormone receptors recognized as peroxisome proliferator-activated receptors (PPARα, δ and γ). PPARγ is considered to be the master regulator of adipogenesis and plays key roles in nearly all aspects of adipocyte biology. Other endocrine disruptors are known to promote adipogenesis, but probably do not act through PPARγ, these include BPA, organophosphate pesticides and monosodium glutamate [27].

Hormonal disorders associated with childhood obesity include growth hormone deficiency, growth hormone resistance, hypothyroidism, leptin deficiency or resistance to leptin action, glucocorticoid excess (Cushing syndrome), precocious puberty, polycystic ovary syndrome (PCOS), prolactin-secreting tumors. Furthermore, in obese individuals, dysfunction in the gut-brain hypothalamic axis and ghrelin/leptin hormonal pathway has been proposed to have a role in excess energy intake and abnormal appetite control [28].

3.1. Genetic factors

Specific syndromes and single gene defects that are linked to obesity in children have been identified. These are very rarely causes of obesity, Generally, monogenic forms of childhood obesity are very rare, accounting for <1% in children. Mutations in only a few genes are known to cause the development of severe obesity in early childhood. Single gene disorders that can cause obesity include deficiency in leptin or its receptor, mutation in leptin gene, deficiency of proopiomelanocortin (POMC), haploinsufficiency receptor 4 and accessory protein receptor 2 of melanocortin, also disorders of protein convertase 1 [4, 18]. The leptin/leptin receptor system regulate food intake through reduce feeding and increased energy expenditure. Some forms of monogenic obesity like congenital leptin deficiency benefits from leptin substitution therapy that leads to significant decrease in weight [29].

Moreover, children with genetic syndromes associated with obesity typically have early onset obesity and characteristic signs on physical examination, including dysmorphic features, developmental delay, short stature or intellectual disability, retinal changes or deafness. The Prader Willi syndrome is the most common among obesity syndromes and is characterized by hypotonia and feeding difficulties during infancy, hyperphagia and obesity developing during early childhood and developmental delay. Other syndromes associated with childhood obesity are Pseudohypoparathyroidism, Laurence Moon Biedl (Bardet Biedl) syndrome, Cohen syndrome, Down syndrome and Turner syndrome [30].

http://dx.doi.org/10.5772/65914

4. Pathophysiology of comorbidities of pediatric obesity

Complications of pediatric obesity occur during childhood and adolescence and increased the risk for morbidity and mortality into adulthood.

Obesity, particularly abdominal, has been shown to be an important risk factor for a number of chronic diseases in adults. Associated with obesity in childhood is a wide range of health serious complications and increased risk of premature onset year of illnesses. The most important organic complications are dyslipidemia, arterial hypertension, type 2 diabetes mellitus, nonalcoholic fatty liver disease, polycystic ovaries syndrome, orthopedic and respiratory complications. The metabolic syndrome (central obesity, hypertension, glucose intolerance and hyperlipidemia) increases risk for cardiovascular morbidity and mortality. The most frequent psychological complications are disorders concerning body image, eating habits and depression [4, 19].

Insulin resistance is defined as a decreased response of tissue to the action of insulin, and due to lowering of the capacity of insulin to stimulate glucose utilization by muscle cells and fat cells and to suppress hepatic glucose production, and insulin resistance in the protein and lipid metabolism. The association of obesity with insulin resistance is well-known: the factors and the mechanism by which the insulin resistance compensation is produced by beta islet cells and those that lead to the "failure" of the pancreatic beta cells in obese patients. It seems that microvascular changes associated with diabetes begin early stages still hyperinsulinemia with normal glycaemia or impaired oral glucose tolerance test. A central role in regulating central nervous system appears to have fat in the body's glucose metabolism by integrating information neural hormonal and nutritional. Insulin via the insulin receptor in the central nervous system regulates food intake and energy homeostasis. Adipose tissue seems to play a role in insulin resistance by metabolites, hormones and adipocytokines influencing different stages of insulin action. Fat distribution is an important determinant of insulin resistance, abdominal fat tissue lipolysis is easier and is less sensitive to insulin anti-lipolysis than subcutaneous adipose tissue. Total fat in children correlates well with the visceral and the relationship of visceral adipose tissue and the cardiovascular risk factors demonstrated in adults appears to differ [4, 31].

4.1. Dyslipidemia

Research on elucidating the relationship between obesity and atherogenic dyslipidemia appears to show a close relationship with insulin resistance. Three major events are based on atherogenic dyslipidemia of obesity with insulin resistance: excessive production of very low density lipoprotein-cholesterol, lipoprotein catabolism and defective catabolism of high density lipoprotein-cholesterol. Visceral fat is associated with impaired insulin-glucose homeostasis, the plasma lipoprotein, in particular increased triglycerides and decreased high density lipoprotein-cholesterol [4].

Arterial hypertension is recognized as an important component of metabolic syndrome in adults, but in children, its role is not very clear. While some studies hypertension is considered

the direct effect of obesity, insulin resistance in others, it is considered a predictor of hypertension, independent of BMI. Arterial hypertension in the pathogenesis of obesity and insulin resistance may play a role in which leptin resistance physiological actions of insulin that leptin central nervous system vessels and kidneys should be changed. Studies suggest the involvement of oxidative stress in the pathogenesis and hypertension by stimulating reactive oxygen species by the renin–angiotensin–aldosterone system [32].

5. Clinical assessment of obesity

Obesity is more complicated to diagnose in children than in adults because children increase in height, weight and body fat naturally as they grow. The criteria for defining obesity in children are the fat mass assessment, the distribution of the body fat measure by age and sex and a centile cut off to define the point in the body fat measure distribution corresponding to obesity [33].

Initial assessments of these patients should include taking a careful history (investigating comorbidities, family history and potentially modifiable behaviors) and physical examination with BMI plotted on a BMI-for-age chart.

The careful history includes as follows: elements of perinatal life (gestational diabetes, maternal obesity, birth weight, infant feeding, medications—glucocorticoids, some antiepileptics, antipsychotics), weight history (onset of parental and child obesity, current eating behaviors, management interventions), complications (psychological, sleeping disorders, gastrointestinal and orthopedic complications, menstrual disturbances in girls), family history (ethnicity, history of obesity, type 2 diabetes, cardiovascular disease, dyslipidemia, obstructive sleep apnea, polycystic ovary syndrome, bariatric, surgery, eating disorders) and lifestyle history (detailed exploration of family eating, nutritional, and activity patterns, sleep) [34].

Physical examination should include the following: anthropometric data (weight, height, BMI, abdominal circumference), adiposity distribution (central versus generalized), assess blood pressure, markers of comorbidities and physical stigmata of a genetic syndrome, endocrine disorders, congenital or acquired hypothalamic alterations (fewer than 5% of cases) [4, 34].

The child's BMI must be plotted on nationally recommended BMI—for age charts. Children and adolescents with a BMI ≥99th percentile are even more likely to have comorbidities [4].

Abdominal circumference (AC) is also used for assessing excess fatty tissue is an indirect method for assessing abdominal fat tissue. Given the strong association between body fat distribution and risk of metabolic complications, it is helpful to calculate in all children with excess weight from the age of 5 years and upwards the relationship between waist circumference and height.

Other methods of measuring fat, such as bioelectrical impedance, and total body water measurement are used in research, but not in clinical evaluation [4].

Careful screening for hypertension using an appropriately sized blood pressure cuff is important (e.g., hypertension is diagnosed if systolic or diastolic blood pressure falls over 95th percentile for age, gender and height in at least three occasions) [35, 36].

Endocrine problems must be considered carefully on signs suggesting hypothyroidism (goiter), insulin resistance (acanthosis nigricans), polycystic ovary syndrome (hirsutism, excessive acne) and Cushing syndrome (violaceous striae, moon face) [36].

Symptoms of polyuria, nocturia or polydipsia may be the result of type 2 diabetes mellitus. Depending on their durations, overweight and obesity are important potential risk factors for respiratory complications (asthma, sleep apnea), abdominal pain or hepatomegaly (gastroesophagial reflux, nonalcoholic fatty liver), musculoskeletal problems (hip or knee pain, genu valgum, slipped capital femoral epiphysis, Blount disease) and psychological disorders (depression, body dissatisfaction, bulimia nervosa impaired social relationships and decreased health-related quality of life depression) [4, 37–39].

Reproductive system and Tanner stage disturbance can reveal premature puberty, apparent micropenis (but normal penis may be hidden in fat), undescended testis/micropenis (Prader Willi syndrome) and must be evaluated [4].

The degree of investigation is dependent on the patient's age and severity of obesity, the findings on history and physical examination, and associated familial risk factors.

First-line investigations recommended in cases of childhood obesity include fasting plasma glucose, triglycerides, low-density lipoprotein and high-density lipoprotein cholesterol, liver function tests and, possibly, insulinemia [4, 34, 35].

The investigations for overweight children include the fasting lipid screening test. If this children present risk factors represented by hypertension, dyslipidemia and family history of diabetes, it is necessary to evaluated the serum levels of fasting glucose, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) to every 2 years (increased value of ALT and AST is associated with possible non-alcoholic fatty liver disease) [36].

In obese, children is necessary to evaluated serum levels of fasting lipids, glucose, ALT and AST every 2 years, and insulinemia [4, 34, 35].

Second-line investigations may include liver ultrasound, an oral glucose tolerance test, more detailed endocrine assessment and polysomnography [4, 34].

Patients with fasting blood glucose >100 mg/dL or overweight children (BMI 85th to 95th percentile) who have a family history of diabetes mellitus or signs of insulin resistance (acanthosis nigricans), polycystic ovary syndrome, or metabolic syndrome should also be evaluated with an oral glucose tolerance test [4, 34, 35]. If the result of oral glucose tolerance test is more than 126 mg/dL, counseling and repeating test is necessary because pediatric obesity can lead to impaired glucose tolerance. The value of HbA1c of 40 mmol/mol (5.8%) is an appropriate screening tool for diagnosing impaired glucose tolerance [40, 41].

Liver ultrasound is recommended for all obese children and adolescents. In children with confirmed ALT >40 IU/L or palpable liver, more thorough diagnostic tests are advisable with gamma-GT and differential diagnosis of hepatitis [41].

Other laboratory tests such as thyroid function tests (if there is a faster increase in weight than height), pelvic ultrasound and hormonal doses in cases of suspected polycystic ovary syndrome have been recommended [4, 41].

Psychological and psychiatric evaluations are essential to identify psychological disturbances including depression, loss-of-control eating, unhealthy and extreme weight control behaviors, and decreased health-related quality of life which are warning signs of bulimia nervosa and binge-eating disorder [39].

In patients with hypertension more diagnostic tests should be done: cardiac exam: ECG and echocardiogram, standard urinalysis, microalbuminuria, creatinine and potassium levels [4, 41].

We should realize screening for diagnosis of metabolic syndrome in the presence of at least three of the following situations: BMI indicate obesity or waist circumference/height ratio >0.5, systolic and/or diastolic blood pressure >95th percentile, fasting blood glucose >100 mg/dL, serum level of triglycerides >95th percentile, serum level of HDL cholesterol [4, 41].

According to International Diabetes Federation (IDF), the consensus definition of metabolic syndrome in children (older than 6 years) and adolescents are as follows:

- for children aged 6–10 years:

- obesity mean a waist circumference >90th percentile

- in case of metabolic syndrome that cannot be diagnosed, the measurements should be made if there is a family history of metabolic syndrome, type 2 diabetes mellitus, dyslipidemia, cardiovascular disease, hypertension or obesity.

- IDF suggests for weight reduction to use an appropriate message in patients with abdominal obesity.

- for children aged 10–16 years:

- obesity mean a waist circumference >90th percentile (or adult cut-off if lower).

- serum triglycerides level >1.7 mmol/l

- serum HDL cholesterol level <1.03 mmol/l

- systolic blood pressure >130 mmHg or diastolic blood pressure >85 mmHg

- blood glucose >5.6 mmol/l (oral glucose tolerance test recommended) or medical history of type 2 diabetes mellitus

- for children >16 years:

- should use the IDF criteria for adults. According to the recent IDF definition, a person with metabolic syndrome must have central obesity (defined as waist circumference using ethnicity-specific values) and any two of the following four factors:
- serum triglyceride levels >150 mg/dl (1.7 mmol/l) or specific treatment recommended for this abnormality
- reduced serum HDL cholesterol level <40 mg/dl (1.03 mmol/l) in males and <50 mg/dl (1.29 mmol/l) in females, or specific treatment for this lipid abnormality
- raised blood pressure: systolic blood pressure >130 mmHg or diastolic blood pressure of 85 mmHg or treatment for previously diagnosed hypertension
- raised fasting plasma glucose >100 mg/dl (5.6 mmol/l) or previously diagnosed type 2 diabetes (if above 5.6 mmol/l or 100 mg/dl, the oral glucose tolerance test is strongly recommended but is not necessary to define the presence of this syndrome) [32, 42].

Vitamin D deficiency is common in obese children and is associated with risk factors for type 2 diabetes in obese children, but they are not still recommended by national clinical practice guidelines as routine measures [43].

6. Management of child obesity

Prevention is the best cost/benefit approach for the management of obesity in children and, in the future, of adulthood. Childhood obesity is a multifaceted problem embedded in physiological, behavioral, genetic, socioeconomic, environmental and political contexts, and the actions to prevent childhood obesity must therefore be taken in multiple settings. Public awareness campaigns, social marketing and behavior-change communication related to nutrition and physical activity implemented in countries together are very important strategies regarding childhood obesity prevention. The critical periods of pediatric obesity characterized by important changes in adiposity growth velocity or obesity related behavior are represented by the first year of life, the preschool ("adiposity rebound") and adolescence years. The transition period from childhood to adolescence is characterized by important behavioral changes and decreased physical activity [44]. The obesity primary prevention begins in pregnancy period (healthy food diet), continuing with promoting the breastfeeding in the first 2 years of life, and then with support for healthy eating habits (low sugar consumption, eat breakfast every day, eat at home with family, avoid fast—food meals, avoid television in the first years of life and limit television to less than 2 h per day after then, etc. [4, 41, 45, 46].

Management of obesity should be based on risk factors, including age, severity of overweight and obesity and comorbidities, as well as family history and support. Management intervention strategies are available and include nutrition, physical activity, behavior and lifestyle changes, medication and surgical considerations. Treatment largely focuses on sustained lifestyle changes with family involvement. There are several broad principles of conventional management: management of comorbidities, family involvement, taking a developmentally

appropriate approach, the use of a range of behavior change techniques, long-term dietary change, increased physical activity and decreased sedentary behaviors. The primary goal for all children with uncomplicated obesity is the long-term improvement of physical health through healthy lifestyles. In obese children with a secondary complication, specific treatment of the complication is an important goal. Effective weight reduction is one of the key elements in the treatment of comorbidities. In morbid obesity, bariatric surgery and laparoscopic sleeve gastrectomy have been used in adolescence [4, 34, 41].

In order to plan a developmentally appropriate approach, it is essential to consider the developmental age of the patient and the resultant level of parental engagement that will be required. Most successful interventions have been family based and take into account the child's developmental age. In preadolescent children, a parent-based program, without direct engagement of the child, might be more appropriate than a child centered approach.

Depending on the age of the child, the present of parents must be or not compulsory. For example, if we talk about adolescents, the present of parents in not recommended. However, the parents must participate at counseling session that are designed for them.

Because obesity is multifactorial, not all children and adolescents will respond to the same approach. Behavior therapy, healthy diet and increasing physical activity are the great sections of obesity treatment. Referral to multidisciplinary, comprehensive pediatric weight—management programs is ideal for obese children whenever possible [4, 34].

Behavior modification strategy has a large effect on weight reduction. The set of techniques employed to change thought processes and actions associated with eating, physical activity and sedentary are components of behavior strategies. For the obese adolescent, there are several ways to help him acquiring a healthy lifestyle: to log daily his physical effort and food intake; to participate to motivational interview; to receive permanent psychological support for positive lifestyle changes [34, 41, 46].

Dietary interventions are usually part of a broader lifestyle change program can be effective in achieving relative weight loss in children and adolescents. Dietary interventions should follow national nutrition guidelines which have an emphasis on:

- at least five meals over the day (three meals + two snacks)

- restrict/replace specific high calorie foods with others less rich in calories

- the protein content: it is suggested the 14 meals per week: meat, three to four times a week; fish, three to four times a week; legumes, three to four times a week; cheese and eggs, once a week.

- carbohydrates should account for at least 50% of total calories, preferring low glycemic index foods: cereals such as pasta, barley and whole wheat products—twice a day; legumes; fruit and vegetables (not canned or pureed) five servings a day) and by limiting foods that combine a high glycemic index to a high glycemic load (bread, rice, potatoes, sweets, sugar, fruit juices, sweet drinks).

- the total fat in the diet should account for no more than 30% of total calories.

- the adequate intake of fiber in grams/day: five servings a day of fruits and vegetables in season, not canned or pureed, and legumes four times a week are recommended.

- decreased portion sizes.

- drinking water as the main beverage and reduction in sugary drink intake.

- involvement of the entire family in making sustainable dietary changes [34, 41].

Increasing physical activity can decrease risk for cardiovascular disease, improve well-being and contribute to weight loss:

- walking or cycling for transport,

- undertaking household chores and playing,

- organized exercise programs,

- limiting television and other small screen recreation to less than 2 h per day is particularly strategic, but may be challenging,

- is recommended at least 60 min of moderate exercise.

Parental involvement is vital and may include monitoring and limiting television use, role modeling of healthy behaviors and providing access to recreation areas or recreational equipment.

Existing recommendations on management of pediatric obesity suggest that drug therapy can be used in the treatment of severely obese adolescents. Orlistat can be useful as an adjunct to lifestyle changes in severely obese adolescents and metformin can be used in older children and adolescents with clinical insulin resistance [4, 34, 41].

Bariatric surgery should be considered in adolescents with complete or near-complete skeletal maturity, who are severely obese with a body mass index of more than 40 kg/m^2 or weight exceeding 100% of ideal body, and a medical complication resulting from obesity, after they have failed 6 months of a multidisciplinary weight management program. Preoperative care and counselling is very important if we want to have good long-term results for bariatric surgery patients. This care must be provided by specialist in various medical fields: endocrinology, gastroenterology, cardiovascular, pneumology, etc. All this effort must by sustained with nutritional and psychological support [4, 34, 41,47, 48].

Childhood obesity treatment is based on sustained lifestyle changes with family involvement. Behavior therapy, healthy diet and increasing physical activity are the great sections of obesity treatment.

7. Conclusions

Childhood and adolescent obesity is a major health problem. The prevalence of childhood obesity in Europe is ten times higher than it was in the 1970s. The increasing occurrence in

children of disorders, such as type 2 diabetes, is a consequence of this obesity epidemic. Initial assessments of these patients should include taking a careful history (investigating comorbidities, family history and potentially modifiable behaviors) and physical examination. The degree of investigation is dependent on the patient's age and severity of obesity, the findings on history and physical examination, and associated familial risk factors. The increased prevalence of obesity in childhood and adolescence highlights the need for effective treatment approaches. There are several broad principles of conventional management: management of comorbidities, family involvement, the use of a range of behavior change techniques, long-term dietary change, increased physical activity and decreased sedentary behaviors. Pediatric patients and their families should be counselled on nutritional interventions including limiting sugar-sweetened beverages, eating nutrient-dense breakfasts, limiting eating out at fast food restaurants, families eating together, increased exercise and decreased time in front of computer and TV screens. For adolescents with severe obesity, lifestyle changes are mandatory. This change must be supported with medical therapy (the only drugs approved by the health organizations are Orlistat and Metformin; these treatments do not exclude metabolic surgery). Finally, given the high prevalence and chronic nature of obesity, coordinated models of care for health service delivery for the management of pediatric obesity are needed.

Author details

Laura Mihaela Trandafir*, Ileana Ioniuc and Ingrith Miron

*Address all correspondence to: trandafirlaura@yahoo.com

Pediatric Department, University of Medicine and Pharmacy "Grigore T. Popa", Iasi, Romania

References

[1] World Health Organization. Obesity and Overweight [Internet]. 2015 [Updated: 2016]. Available from: http://www.who.int/mediacentre/factsheets/fs311/en/

[2] Hill Jo: Understanding and addressing the epidemic of obesity: an energy balance perspective. Endocrine Reviews. 2006;27:750–761. doi:10.1210/er.2006-0032

[3] World Health Organization. Global Status Report on Non-communicable Diseases [Internet]. 2014. Available from: http://www.who.int/nmh/publications/ncd-status-report-2014/en/ [Accessed: 2016]

[4] Gahagan S. Overweight and Obesity. In: Kliegman RM, Stanton BF, St. Geme JWIII, Schor NF, editors. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Elsevier Saunders; 2016. p. 307–316. doi:978-1-4557-7566-8, 2823-2831

[5] Rodríguez G, Pietrobelli A, Wang Y, Moreno LA. Methodological Aspects for Childhood and Adolescence Obesity Epidemiology. In: Moreno AL, Pigeot I, Ahrens W, editors.

Epidemiology of Obesity in Children and Adolescents—Prevalence and Etiology. New York: Springer; 2011. p. 21–40. doi:10.1007/978-1-4419-6039-9

[6] Lobstein T. Prevalence and Trends Across the World. In: M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. ebook.ecog-obesity.eu:2015.

[7] Manios Y, Costarelli V. Childhood Obesity in the WHO European Region. In: Moreno AL, Pigeot I, Ahrens W, editors. Epidemiology of Obesity in Children and Adolescents—Prevalence and Etiology. New York: Springer; 2011. p. 43–68. doi:10.1007/978-1-4419-6039-9

[8] Larranaga, N., Amiano, P., Arrizabalaga, J., Bidaurrazaga, J.J., Gorostiza E. Prevalence of obesity in 4-18-year-old population in the Basque Country, Spain. Obesity Reviews. 2007;**8**(4):281–287. doi:10.1111/j.1467-789X.2006.00306

[9] Kontogianni, M.D., Vidra, N., Farmaki, A.E., Koinaki, S., Belogianni, K., Sofrona, S., Magkanari, F., Yannakouli M. Adherence rates to the mediterranean diet are low in a representative sample of Greek children and adolescents. Journal of Nutrition. 2008; **138**:1951–1956.

[10] Moreira P. Overweight and obesity in Portuguese children and adolescents. Journal of Public Health. 2007;**15**(3):155–161. doi:10.1007/s10389-007-0109-1

[11] Hakanen, M., Lagstrom, H., Kaitosaari, T., Niinikoski, H., Nanto-Salonen K., Jokinen, E., Sillanmaki, L., Viikari, J., Ronnemaa, T., Simell, O. Development of overweight in an atherosclerosis prevention trial starting in early childhood. The STRIP study. International Journal of Obesity.2006;**30**:618–626.

[12] Kumanyika S.K. Environmental influences on childhood obesity: ethnic and cultural influences in context. Physiology & Behaviour. 2008;**94**(1):61–70. doi:10.1016/j.physbeh.2007.11.019

[13] Wardle J., Brodersen N.H., Cole T.J., Jarvis M.J., Boniface D.R. Development of adiposity in adolescence: five-year longitudinal study of an ethnically and socioeconomically diverse sample of young people in Britain. British Medical Journal. 2006;**332**:1130–1135. doi:10.1136/bmj.38807.594792.AE

[14] Hollis J, Robinson S. Prenatal Experience and Childhood Obesity. In: M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. ebook.ecog-obesity.eu 2015.

[15] Socha P. Metabolic Programming, Breastfeeding and Later Risk of Obesity. In: In M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. Retrieved from ebook.ecog-obesity.eu. 2015.

[16] Oken E., Levitan E., Giillman M. Maternal smoking during pregnancy and child overweight systematic review and meta-analysis. International Journal of Obesity. 2008;**32**(2):201–210. doi:10.1038/sj.ijo.0803760

[17] Riedel C., Schönberger K., Yang S., Koshy G., Chen Y.C., Gopinath B., Ziebarth S., von Kries R. Parental smoking and childhood obesity: higher effect estimates for maternal smoking in pregnancy compared with paternal smoking—a meta-analysis. International Journal of Epidemiology. 2014;**43**(5):1593–1606. doi:10.1093/ije/dyu150

[18] Klish W.J. Definition; epidemiology; and etiology of obesity in children and adolescents, http://www.uptodate.com/contents/definition-epidemiology-and-etiology-of-obesity-in-children-and-adolescents [Internet].

[19] Klish W.J. Comorbidities and Complications of Obesity in Children and Adolescents. http://www.uptodate.com/contents/comorbidities-and-complications-of-obesityin-children-and-adolescents [Internet].

[20] Tu A.W., Mâsse L.C., Lear S.A., Gotay C.C., Richardson C.G. Exploring the mediating roles of physical activity and television time on the relationship between the neighbourhood environment and childhood obesity. Canadian Journal of Public Health. 2016;**107**(2):168–175. doi:10.17269/cjph.107. 5268

[21] Lobstein T., Dibb S. Evidence of a possible link between obesogenic food advertising and child overweight. Obesity Reviews. 2005;**6**(3):203–208. doi:10.1111/j.1467-789X.2005. 00191.x

[22] Mazur A., Radziewicz-Winnicki I. Obesity and Media. In: In M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. Retrieved from ebook.ecog-obesity.eu. 2015. The ECOG's eBook

[23] Bhadoria A.S., Kapil U., Kaur S. Association of duration of time spent on television, computer and video games with obesity amongst children in National Capital Territory of Delhi. Journal of Preventive Medicine. 2015;**6**(1):80. doi:10.4103/2008-7802.164090

[24] Kuzik N., Carson V. The association between physical activity, sedentary behavior, sleep, and body mass index z-scores in different settings among toddlers and preschoolers. ***BMC Pediatrics. 2016;**16**. doi:10.1186/s12887-016-0642-6

[25] emedicine. http://emedicine.medscape.com/article/985333-overview#a5 [Internet]

[26] Telega G. Intestinal Microbiome. In Obesity. In: In M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. Retrieved from ebook.ecog-obesity.eu. 2015.

[27] Yang O., Kim H.L., Weon J.I., Seo Y.R. Endocrine-disrupting Chemicals: Review of Toxicological Mechanisms Using Molecular Pathway Analysis. Journal of Cancer Prevention. 2015;**20**(1):12–24. doi:10.15430/JCP.2015.20.1.12

[28] http://emedicine.medscape.com/article/985333-overview#a5 [Internet].

[29] Funcke J.B., Schnurbein J., Lennerz B., Lahr G., Debatin K.M., Fischer-Posovszky P., Wabitsch M. Monogenic Forms of Childhood Obesity due to Mutations in the Leptin Gene [Internet]. 2014 . Available from: http://molcellped.springeropen.com/articles/10.1186/s40348-014-0003-1

[30] http://emedicine.medscape.com/article/985333-overview [Internet].

[31] Cinti S. The Adipose Organ: Implications for Prevention and Treatment of Obesity. In: In M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. Retrieved from ebook.ecogobesity. 2015.

[32] Wabitsch M, Körner A. Definitions. In: Kiess W, Wabitsch M, Maffeis C, Sharma AM, editors. Metabolic Syndrome and Obesity in Childhood and Adolescence. Pediatr Adolesc Med. Basel, Karger; 2015. p. 1–12. doi:10.1159/000368101

[33] Tim J Cole. How to Measure Obesity in Children [Internet]. 2014. Available from: https://thewinnower.com/discussions/14-how-to-measure-obesity-in-children

[34] Baur LA. Management of Child and Adolescent Obesity. In: Berthold Koletzko, editors. Pediatric Nutrition in Practice. 2nd ed. Basel: Karger; 2015. p. 161–167. (1660–2242)

[35] Balow SE. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;**120**(Suppl 4):164–192.

[36] Zahra Alizadeh, Shahrzad Khosravi,Sima Borna/Iraninan Journal of Pediatrics. Obese and Overweight Children and Adolescents: An Algorithmic Clinical Approach [Internet]. 2013. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4025117/

[37] Ruiz Extremera A., Carazo A., Salmerón A., León J., Casado J., Goicoechea A., Fernandez J.M., Garofano M., Ocete E., Martín A.B., Pavón E., Salmerón J. Factors associated with hepatic steatosis in obese children and adolescents. Journal of Pediatric Gastroenterology and Nutrition. 2011;**53**(2):1962–1901. doi:10.1097/MPG. 0b013e3182185ac4

[38] Thielemann F., Guenther K.P., Stiehler M. Orthopaedic Aspects of Obesity in Children and Adults. In: Kiess W, Wabitsch M, Maffeis C, Sharma AM, editors. Metabolic Syndrome and Obesity in Childhood and Adolescence. Pediatr Adolesc Med. Basel, Karger; 2015. p. 84–98. doi:10.1159/000368101

[39] Vander Wal J.S., Mitchell E.R. Psychological complications of pediatric obesity. Pediatric Clinics of North America.2011;**58**(6):1393–1401. doi:10.1016/j.pcl.2011.09.008

[40] Lee HS, Park HK, Hwang JS. HbA1c and glucose intolerance in obese children and adolescents. Diabetic Medicine.2012;**29**(7):102–105.

[41] Maffeis C., Licenziati M.R., Vania A., Garofalo P., Di Mauro G., Caroli M., Morino G., Siani P., Chiamenti G. Childhood Obesity. In: P. Sbraccia, editor. Clinical Management of Overweight and Obesity: Recommendations of the Italian Society of Obesity (SIO). Switzerland: Springer International Publishing; 2016. p. 131–147. doi:10.1007/978-3-319-24532-4_10

[42] International Diabetes Federation. Metabolic Syndrome in Children and Adolescents [Internet]. 2007. Available from: http://www.idf.org/webdata/docs/Mets_definition_children.pdf

[43] Olson M.L., Maalouf N.M., Oden J.D., White P.C., Hutchison M.R. Vitamin D deficiency in obese children and its relationship to glucose homeostasis. Journal of Clinical Endocrinology & Metabolism. 2012;**97**(1):279–285. doi:10.1210/jc.2011-1507

[44] Joan C. Han, Debbie A. Lawlor, Sue Y.S. Kimm. Childhood Obesity –2010: Progress and Challenges [Internet]. 2011. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3073855/

[45] Kugelberg S., Jewell J., Breda J. Prevention Across Europe. In: M.L. Frelut, editor. The ECOG's eBook on Child and Adolescent Obesity. Retrieved from ebook.ecog-obesity.eu. 2015.

[46] Han J.C., Lawlor D.A, Kimm S.Y.S. Childhood obesity–2010: Progress and challenges. Lancet. 2010;**15**:1737–1748. doi:10.1016/S0140-6736(10)60171-7

[47] Baur L.A., Hazelton B., Shrewsbury V.A. Assessment and management of obesity in childhood and adolescence. Nature Reviews Gastroenterology and Hepatology. 2011;**8**:635–645doi:10.1038/nrgastro.2011.165

[48] Barlow S.E. Expert committee: Expert committee recommendations regarding the prevention, assessment, and treatment and obesity: Summary report. Pediatrics. 2007;**120** (suppl 4):S164–S192.

IntechOpen