Obesity in children and adolescents: epidemiology, causes, assessment, and management

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This Review describes current knowledge on the epidemiology and causes of child and adolescent obesity, considerations for assessment, and current management approaches. Before the COVID-19 pandemic, obesity prevalence in children and adolescents had plateaued in many high-income countries despite levels of severe obesity having increased. However, in low-income and middle-income countries, obesity prevalence had risen. During the pandemic, weight gain among children and adolescents has increased in several jurisdictions. Obesity is associated with cardiometabolic and psychosocial comorbidity as well as premature adult mortality. The development and perpetuation of obesity is largely explained by a bio-socioecological framework, whereby biological predisposition, socioeconomic, and environmental factors interact together to promote deposition and proliferation of adipose tissue. First-line treatment approaches include family-based behavioural obesity interventions addressing diet, physical activity, sedentary behaviours, and sleep quality, underpinned by behaviour change strategies. Evidence for intensive dietary approaches, pharmacotherapy, and metabolic and bariatric surgery as supplemental therapies are emerging; however, access to these therapies is scarce in most jurisdictions. Research is still needed to inform the personalisation of treatment approaches of obesity in children and adolescents and their translation to clinical practice.

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For the **WHO definition** see https://www.who.int/health-topics/obesity

Introduction

Obesity in children and adolescents is a global health issue with increasing prevalence in low-income and middle-income countries (LMICs) as well as a high prevalence in many high-income countries.[1] Obesity during childhood is likely to continue into adulthood and is associated with cardiometabolic and psychosocial comorbidity as well as premature mortality.[2–4] The provision of effective and compassionate care, tailored to the child and family, is vital. In this Review, we describe current knowledge on the epidemiology and causes of child and adolescent obesity, considerations for assessment, and current management approaches.

Epidemiology

Definitions of overweight and obesity in children and adolescents

WHO defines overweight and obesity as an abnormal or excessive fat accumulation that presents a risk to health. For epidemiological purposes and routine clinical practice, simple anthropometric measures are generally used as screening tools. BMI (weight/height^2; kg/m^2) is used as an indirect measure of body fatness in children and adolescents[5] and should be compared with population growth references adjusted for sex and age. The WHO 2006 Growth Standard is recommended in many countries for children aged 0–5 years, and for children aged 0–2 years in the USA.[6] For older children and adolescents, other growth references are used, including the WHO 2007 Growth Reference, recommended for those aged 5–19 years (overweight defined as BMI ≥1SD and obesity as BMI ≥2SD of the median for age and sex), and the United States Centers for Disease Control and Prevention (CDC) Growth Reference for those aged 2 to 20 years (overweight is >85th to <95th percentile and obesity is ≥95th percentile based on CDC growth charts).[6,7] The International Obesity Task Force tables for children aged 2 to 18 years are used for epidemiological studies.[8]

Abdominal or central obesity is associated with increased cardiometabolic risk in children and adolescents.[9] For waist circumference there are regional and international growth references allowing adjustment for age and sex.[10–12] A waist-to-height ratio of more than 0·5 is increasingly used as an indicator of abdominal adiposity in clinical and research studies, with no need for a comparison reference.[13]

Various definitions have been suggested to identify more extreme values of BMI in children and adolescents. The International Obesity Task Force defined morbid obesity as equivalent to age-adjusted and sex-adjusted BMI of 35kg/m^2 or more at age 18 years, a definition specifically for epidemiological use.[14] The American Heart Association characterises severe obesity as a BMI of 120% or more of the 95th percentile of BMI for age and sex (based on CDC2000 growth charts), a definition that can be used in both clinical practice and research.[15] There are marked limitations in transforming very high BMI values to z-scores, particularly when using CDC2000 growth charts because reductions in BMI can be underestimated.[15]

Prevalence

The prevalence of paediatric obesity[16] has increased worldwide over the past five decades. From 1975 to 2016, the global age-standardised prevalence of obesity in children and adolescents aged 5–19 years increased from 0·7% (95% credible interval [CrI] 0·4–1·2) to 5·6% (4·8–6·5) for girls and from 0·9% (0·5–1·3) to 7·8% (6·7–9·1) for boys.[17] Since 2000, the mean BMI has plateaued, usually at high levels, in many high-income countries but has continued to rise in LMICs. In 2016, obesity prevalence in this age group was highest (>30%) in many Pacific Island nations and was high (>20%) in several countries in the Middle East, north Africa,

Micronesia (region of the western Pacific), Polynesia (subregion of Oceania), the Caribbean, as well as in the USA.[17]

In 2019, the World Obesity Federation estimated there would be 206 million children and adolescents aged 5–19 years living with obesity in 2025, and 254 million in 2030.[1] Of the 42 countries each estimated to have more than 1 million children with obesity in 2030, the top ranked are China, followed by India, the USA, Indonesia, and Brazil, with only seven of the top 42 countries being high-income countries.

The prevalence of severe obesity in the paediatric population has grown in many high-income countries, even though overall prevalence of obesity has been stable.[18–21] In a survey of European countries, approximately a quarter of children with obesity were classified with severe obesity, a finding that has implications for delivery of obesity clinical services, because such children will need more specialised and intensive therapy.[19]

There are socioeconomic disparities in paediatric obesity prevalence within countries. In lower-income to middle-income countries, children of higher socioeconomic status are at greater risk of being affected by overweight or obesity than children of a lower socioeconomic status, whereas in high-income countries, it is children living in socioeconomic disadvantage who are at higher risk.[22–24]

Reports from China, Europe, and the USA have documented increased weight gain among children and adolescents during the COVID-19 pandemic compared with the rate before the pandemic,[25–29] an apparent consequence of decreases in physical activity, increased screen time, changes in dietary intake, food insecurity, and increased family and individual stress.[30]

Causes

Development and perpetuation of obesity: a bio-socioecological framework

The development and perpetuation of obesity in modern society can largely be explained by a bio-socioecological framework that has created the conditions for a scenario in which biological predisposition, socioeconomic forces, and environmental factors together promote deposition and proliferation of adipose tissue and resistance to efforts of obesity management. A high degree of biological heterogeneity exists in bodyweight regulation and energy dynamics such that some individuals can maintain healthy levels of adipose tissue with little effort while others face a lifelong struggle with regulating levels. Further, adipose tissue is heterogeneous such that white, brown, and beige forms exist with a variety of physiological functions.[31] The anatomical sites where adipose tissue is stored can translate into varying health risks (eg, central accumulation of adipose tissue is associated with cardiometabolic disease compared to peripheral stores).[32] At a fundamental level, the relative function of the energy regulatory system (the complex interplay of central and peripheral pathways driving appetite, satiety, pleasure-seeking behaviours, and metabolic efficiency) strongly influences body composition. More specifically, the bodyweight set point theory posits the existence of a tightly regulated and complex biological control system, which drives a dynamic feedback loop aimed at defending a predetermined relative or absolute amount of adiposity.[33] Support for this theory comes from evidence in adults demonstrating immediate and sustained alterations in levels of hormones driving appetite and satiety, perceptions of food palatability, and resting energy expenditure following attempts at weight loss.[34,35] Other biobehavioural factors such as poor sleep quality, adversity, stress, and medications (causing iatrogenic weight gain) can also serve to exacerbate dysfunction of the energy regulatory system favouring weight gain.

Environmental and behavioural associations of obesity

Over the past few decades the rise in obesity prevalence has been profoundly influenced by changes in the broader obesogenic environment.[36] These changes operate at the level of the family (eg, family modelling of physical activity, food habits, sleep, screen use), local community (eg, child care and schools, parks, green space, public transport and food outlets), or the broader sociopolitical environment (eg, government policies, food industry, food marketing, transport systems, agricultural policies and subsidies). Such influences have been described as having the ability to exploit people's biological, psychological, social, and economic vulnerabilities.[37] Figure 1 depicts a socioecological model incorporating some of the personal and environmental factors influencing paediatric obesity.[38]

Dietary factors contributing to obesity risk in children and adolescents include excessive consumption of energy-dense, micronutrient-poor foods; a high intake of sugar-sweetened beverages; and the ubiquitous marketing of these and fast foods.[39,40] The relative effect of other factors such as specific eating patterns (eg, frequent snacking, skipping breakfast, not eating together as a family, the window of time from first to last daily meal), portion sizes, the speed of eating, macronutrient intake, and glycaemic load on obesity development remain unclear, although all might be important.[41,42]

The link between screen time and obesity in childhood and adolescence was initially documented through cross-sectional and longitudinal studies of television viewing.[43,44] The past two decades have seen the increase of mobile and gaming devices. Screen exposure influences risk of obesity in children and adolescents via increased exposure to food marketing, increased mindless eating while watching screens, displacement of time spent in more physical activities, reinforcement of sedentary behaviours, and reduced sleep time.[44,45]

Children's physical activity levels decline around the age of 6 years and again at age 13 years, with girls usually exhibiting more marked declines than boys. Overall, children with obesity tend to engage in lower levels of moderate-vigorous activity than leaner peers.[46–48] Sedentary

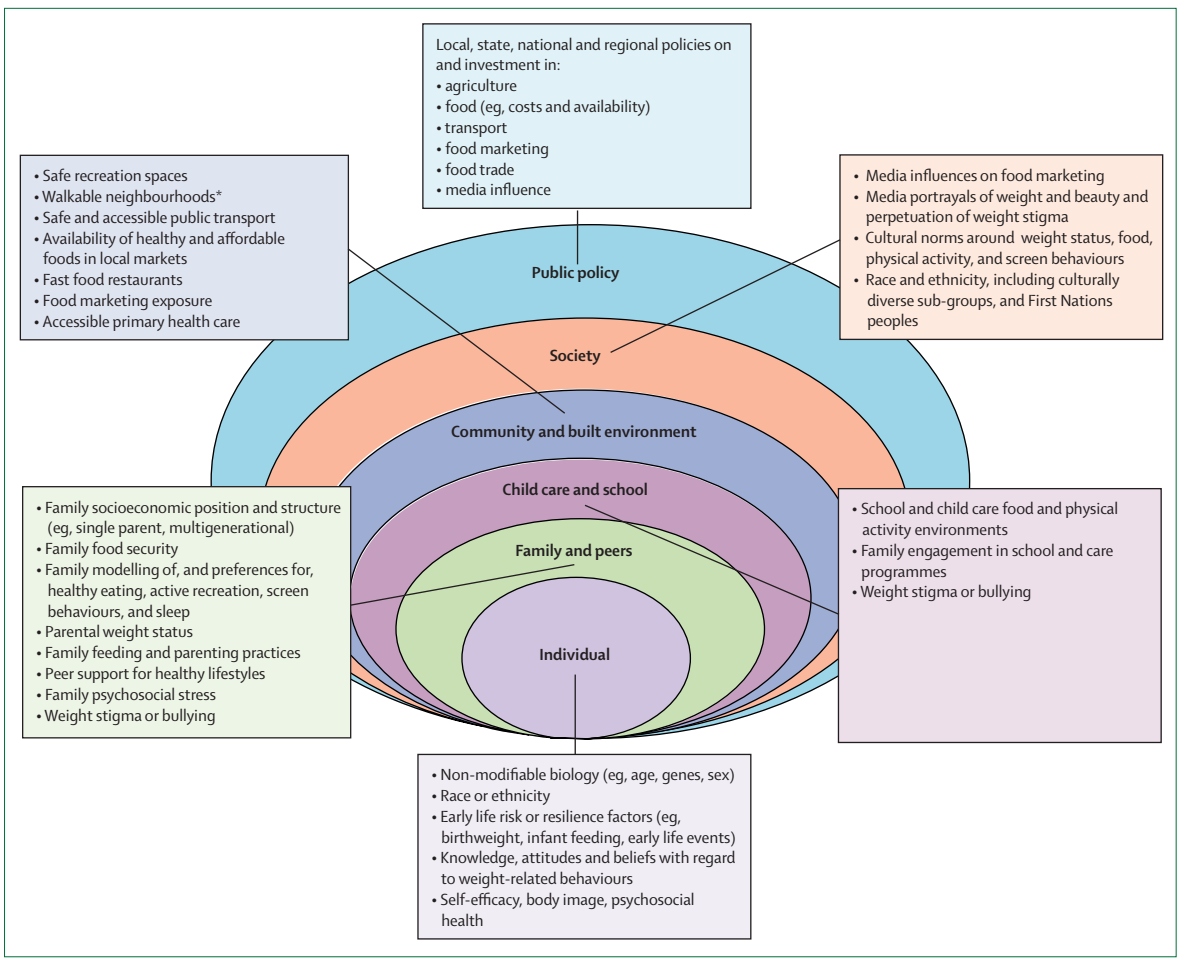

Figure 1: **A socioecological model for understanding the dynamic interrelationships between various personal and environmental factors influencing child and adolescent obesity.**
Adapted from the Centers for Disease Control and Prevention social-ecological model framework for prevention.[38] *Defined as being traversable on foot, compact, physically enticing, and safe.

time increases from the age of 6 years in general, although accelerometery studies report no differences between children with obesity compared with leaner peers.[48] Lower levels of physical activity and increasing sedentary behaviours throughout childhood in all children contribute to obesity development.[49] In most countries, children and adolescents are not sufficiently active due to the loss of public recreation space, the increase in motorised transport and decrease in active transport (eg, cycling, walking, public transport), perceptions of lack of safety in local neighbourhoods leading to less active behaviour, as well as an increase in passive entertainment.[39,49]

There is growing evidence that short sleep duration, poor sleep quality and a late bedtime are associated with a higher obesity risk, sedentary behaviours, poor dietary patterns, and insulin resistance. In addition, there is a possible link with increased screen time, decreased physical activity, and changes in ghrelin and leptin levels.[50] Many of these obesity-conducive behaviours co-occur. For example, increased screen time is associated with delayed sleep onset and shortened sleep duration, and insufficient sleep is associated with increased food intake and lower levels of physical activity.[50]

Early life factors

Several factors in early life put children at increased risk of developing obesity. These factors include maternal obesity before pregnancy, excessive gestational weight gain, and gestational diabetes, all associated with increased birth weight.[51,52] Infant and young child feeding practices have variable influences on childhood obesity. Meta-analyses from systematic reviews suggest that breastfeeding has a modest but protective effect against later child obesity.[53,54] There is some evidence suggesting that the very early introduction of complementary foods and beverages, before the age of 4 months, especially in formula-fed babies, is associated with higher odds of overweight and obesity.[55] Parental approaches to feeding, especially in the

preschool age group (aged 1–4 years), might influence obesity risk, with a systematic review showing a small but significant association between controlling child feed practices (eg, restriction of specific foods or the overall amount of food) and higher child weight.[56] Studies of the role of responsive feeding, whereby the caregiver attends to the baby's cues of hunger and satiety, show that non-responsive feeding is associated with increased child BMI or overweight or obesity.[57,58] By contrast, a responsive feeding style that recognises the child's cues of hunger and satiety appears to support healthy weight gain trajectories.[58,59] However, in all such studies of infant and young child feeding, the effect of residual confounding on child weight status cannot be discounted.

Other environmental exposures in early life that influence child obesity risk include maternal smoking during pregnancy,[60] second-hand exposure to smoke, and air pollution.[61] Antibiotic exposure in infancy is associated with a slight increase in childhood overweight and obesity, especially if there are repeated treatments, an association that might be mediated by alterations in the gut microbiome.[62] Importantly, there is increased recognition that adverse childhood experiences, such as abuse, family dysfunction and neglect are associated with the development of childhood obesity. This association appears to be especially the case for sexual abuse and for co-occurrence of multiple adverse experiences.[63]

Medical conditions associated with obesity

Obesity might occur secondary to a range of medical conditions including several endocrine disorders (eg, hypothyroidism, hypercortisolism, growth hormone deficiency), central nervous system damage (ie, hypothalamic-pituitary damage because of surgery or trauma) and post-malignancy (eg, acute leukaemia). Several pharmacological agents are associated with excess weight gain, including glucocorticoids, some anti-epileptics (eg, sodium valproate), insulin, and several atypical antipsychotics (eg, risperidone, olanzapine, clozapine).[64] The rapid and large weight gain associated with the latter class of drugs suggests that anticipatory weight management strategies should be formally used when commencing such therapy, although evidence is largely from adult studies.[65]

Weight stigma

Weight stigma refers to the societal devaluation of a person because they have overweight or obesity, and includes negative stereotypes that individuals are lazy and lack motivation and willpower to improve health.[66,67] Higher body mass is associated with a greater degree of weight stigma, although longitudinal studies have shown associations between weight stigma and BMI to be bidirectional.[68] Stereotypes manifest in different ways, leading to discrimination and social rejection, often expressed as teasing, bullying and weight-based victimisation in children and adolescents.[66,67] Bodyweight is consistently reported to be the most frequent reason for teasing and bullying in children and adolescents, with a quarter to half of youth reporting being bullied based on their bodyweight.[69] Parents and health-care providers can also be sources of weight stigma.[69,70] Weight stigma is associated with poor mental health, impaired social development and education, and engagement in disordered eating behaviours including binge eating.[69] Of concern, youth who have experienced weight related teasing or bullying have higher rates of self-harm behaviours and suicidality compared with peers of the same weight who have not felt stigmatised.[67]

Experience of weight stigma is a barrier to accessing health care.[67] Health professionals have a responsibility to help reduce weight stigma experienced by children, adolescents, and families through the use of supportive, compassionate, and non-stigmatising language while providing care.[69] In 2020, an international consensus statement was endorsed by more than 100 organisations pledging to reduce weight stigma.[66] Additionally, the American Academy of Paediatrics recommends paediatricians help mitigate weight stigma within clinical practice by role-modelling professional behaviours, using non-stigmatising language, using patient-centred behaviour change counselling, creating a safe and welcoming clinical environment accommodating of all body sizes, and conducting behavioural health screening for signs of weight-based bullying including emotional comorbidities.[67]

Health complications

All body systems can be affected by obesity in the short, medium, or longer term, depending upon age and obesity severity. Figure 2 depicts the possible complications of obesity that can occur anywhere from childhood and adolescence to adulthood. It is important that complications are assessed in childhood and treated alongside obesity to prevent progression of both. Recent reviews provide additional detail regarding complications.[2–4,48,72–74,76–79,84–88,91,92,94,95]

Clinical assessment

A detailed clinical examination screens for underlying causes of obesity, and assesses for possible obesity-related complications, risk of future disease, and whether potentially modifiable behavioural factors exist. Adapted from various national or regional level clinical practice guidelines,[96–103] summaries of the main aspects to be explored in history taking and physical examination are included in the panel and table. Laboratory tests can complement clinical assessment, looking for cardiometabolic complications and some underlying causes of obesity. These tests are appropriate in most adolescents with obesity, and in all patients with severe obesity, with clinical signs or history suggestive of complications (eg, acanthosis nigricans), or with a family history of cardiometabolic disease. Investigations would generally include liver function tests, lipid profile, fasting glucose, and

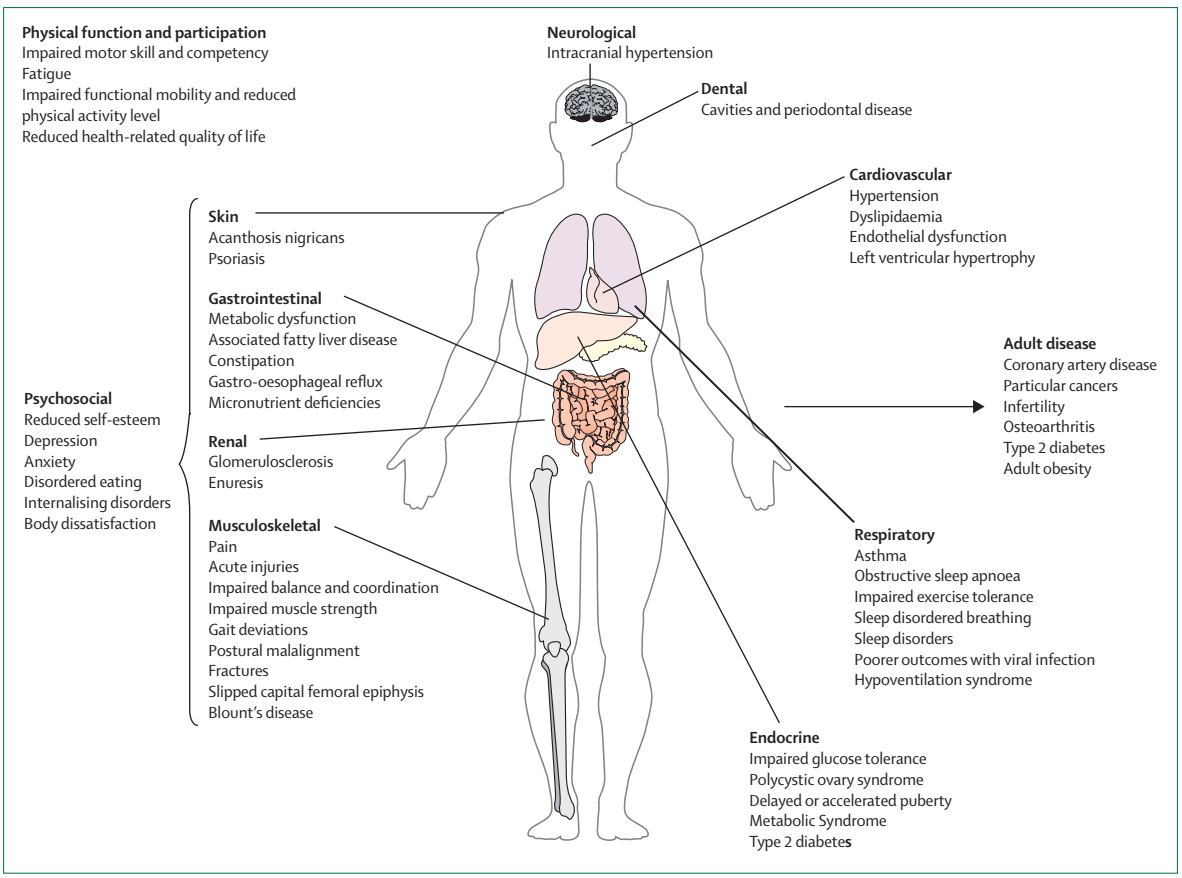

Figure 2: **Short-term and long-term health complications and comorbidities associated with child and adolescent obesity**
Health complications and comorbidities include neurological,[71] dental,[72] cardiovascular,[2,73–75] psychosocial,[2,4,76–78] respiratory,[79–83] endocrine,[73,84,85] musculoskeletal,[80,86–88] renal,[89,90] gastrointestinal,[90,91] skin,[92] function, and participation.[48,93]

glycated haemoglobin, and might include an oral glucose tolerance test and additional endocrine or genetic studies.[96–102]

Management

Overview

Obesity treatment in children and adolescents aims to reduce adiposity, improve related physical and psychosocial complications, and prevent the development of chronic diseases. The degree of BMI reduction needed to improve obesity related complications is currently unknown. However, some evidence suggests that BMI z-score reductions greater than 0·25 and 0·5 might represent clinically important thresholds.[104] Several high-quality clinical practice guidelines are in use internationally.[96–102] Treatment type and intensity depends upon obesity severity, the age and developmental stage of the child, needs and preferences of the patient and family, clinical competency and training of the clinician(s), and the health-care system in which treatment is offered.[105] Treatment integrates multiple components including nutrition, exercise and psychological therapy, pharmacotherapy, and surgical procedures. It should be delivered by suitably qualified paediatric health professionals who incorporate behavioural support and non-stigmatising child-focused and youth-focused communication into their practice.[69]

Multicomponent behavioural interventions

Behavioural support strategies in obesity management include a combination of addressing dietary intake, physical activity, sedentary behaviours, sleep hygiene, and behavioural components within the context of a family-based and developmentally appropriate approach aiming for long-term behaviour modification.[106–108] Tailoring of interventions to various subgroups based on age, gender, and culture might be needed. For example, with young children the therapy might be largely parent-focussed[109] and for adolescents a greater degree of autonomy might be required.[107]

Dietary intervention

Dietary interventions might include dietary education alone or combined with a moderate energy restriction,[110] with structured dietary plans or advice preferred over broad dietary principles, particularly for adolescents.[111] Principles of dietary education focus on adoption of dietary

***Panel*: Elements of history taking**

General history
- Prenatal and birth history, including gestational obesity, gestational diabetes, maternal smoking, gestational age, birth weight, and neonatal concerns
- General medical history, including psychiatric or behavioural diagnoses and previous malignancy
- Developmental history, including any delays in motor, speech, or cognitive developmental, and therapy received
- Infant feeding, including breastfeeding and duration and timing of introduction of complementary foods
- Current medications, including glucocorticoids, anti-epileptics (eg, sodium valproate), and antipsychotics (eg, clozapine, risperidone, and olanzapine)

Growth history
- Height and weight growth trajectories
- Onset of obesity and timing of weight concerns of child or adolescent and family
- Previous obesity management, whether supervised or self-initiated
- Previous and current dieting and exercise behaviours or use of supplements

Complications history
- Psychological impacts of obesity, including bullying, poor self-esteem, anxiety, depression, and disordered eating
- Sleep routines, presence of snoring or possible sleep apnoea (eg, poor refreshment after sleep, daytime somnolence, and witnessed apnoea)
- Exercise tolerance, exercise-induced bronchoconstriction, dyspnoea, hypertension, and fatigue levels
- Specific symptoms including acne and hirsutism (girls), morning headache and visual disturbance, nocturnal enuresis, daytime dribbling, constipation, hip and knee joint pain, and gastrointestinal complaints (vomiting, abdominal pain, constipation, and gastrointestinal reflux)
- Menstrual history (girls)

Family history
- Ethnicity (high risk groups for cardiometabolic complications include First Nations peoples, Latin American, south Asian, east Asian, Mediterranean, and Middle Eastern)
- Family members with a history of obesity; type 2 diabetes and gestational diabetes; hypertension, dyslipidaemia, and cardiovascular disease; obstructive sleep apnoea; polycystic ovary disease; bariatric surgery; eating disorders; and mental health disorders
- Home environment including household members, parental relationship, parental employment, hours, and home supervision

Social history, including welfare, and safety
- Housing or accommodation situation (stable or homeless) and residential care
- Family income (or proxy) and food insecurity
- Previous social services involvement
- School attendance, additional educational assistance, learning difficulties, and behavioural difficulties
- Hobbies and interests
- Friends in school or neighbourhood
- Use of tobacco, alcohol, or recreational drugs
- Parenting style and child–parent interactions

Behavioural risk factors
- Nutrition and eating behaviours: breakfast consumption; eating patterns including snacking, grazing, sneaking or hiding food, fast-food intake, binge-eating; beverage consumption (sodas, juices, other sugary drinks); family routines around food and eating; and active dieting
- Physical activity: transport to and from school; participation in physical education class; participation in organised sport, dance, or martial arts; gym membership; after-school and weekend recreation; and family activities
- Sedentary behaviours: time spent sitting each day; screen-time per day (television, video game, mobile phone, tablet, computer use); number of devices in the household and bedrooms; patterns of screen viewing (eg, during meals, at night); and use of social media
- Sleep behaviours: bedtime routines; sleep and wake times on weekdays and weekends; and daytime napping

intake patterns consistent with local dietary guidelines—eg, increased intake of vegetables and fruit, reductions in energy-dense nutrient-poor foods and sugar sweetened beverages, and improvement in dietary behaviours such as encouraging mealtime routines and family meals.[110,112] One common approach, the traffic light diet, categorises foods by energy density, with green low-energy foods that can be eaten freely, yellow foods eaten moderately, and red foods eaten occasionally due to a higher energy-density.[107] Dietary approaches aim to be nutritionally complete and to address and prevent nutritional deficiencies.[113,114] However, children and adolescents might present for obesity treatment with relatively poor diet quality;[115] therefore, an initial goal of improving the baseline diet might be appropriate. Selection of dietary strategies should be informed by individual preference and circumstances, family environment, and available support.

Physical activity

Physical activity components might include provision of education or a structured exercise programme, or both, in line with local guidelines. The goals of exercise interventions should be to offer a safe, supportive, fun, and non-judgemental environment for children with

	Possible diagnosis or additional comments
General	
Flat affect	Depression, hypothyroidism
Dysmorphic features	Syndromic obesity
Developmental delay	Syndromic obesity, risk factor for obesity
Anthropometry	
Standardised measurement of weight, height or length, and waist	Chart weight, height or length, and BMI for age; calculate waist-to- height ratio
Tall stature	Growth acceleration in common obesity (pre-pubertal)
Short stature	Hypothyroidism, hypercortisolism, monogenic obesity, or syndromal obesity
Blood pressure	
Hypertension	Hypercortisolism (use appropriately sized cuff and compare with age, sex, and height adjusted references)
Skin and subcutaneous tissue	
Violaceous striae	Possible hypercortisolism
Acanthosis nigricans	Insulin resistance
Acrochordons (skin tags)	Insulin resistance
Acne	Possible hyperandrogenism, hypercortisolism
Hirsutism	Possible hyperandrogenism
Folliculitis, intertrigo, thigh chafing	Infection in skin folds, skin rubbing
Coarse and brittle hair	Hypothyroidism
Pseudogynaecomastia	Excess body fat over pectoral muscle in males
Head and neck	
Crowded pharynx or tonsillar enlargement	Obstructive sleep apnoea
Palatal bruise	Bulimia
Teeth erosions	Bulimia
Dental cavities	Poor dietary intake
Goitre	Hypothyroidism
(Table continues in next column)	
(Continued from previous column)	
Neurological	
Papilloedema	Idiopathic intracranial hypertension
Cardiorespiratory	
Exercise intolerance	Poor cardiorespiratory fitness, asthma
Wheeze	Asthma
Heart rate	Cardiorespiratory fitness assessment
Gastrointestinal	
Hepatomegaly	Fatty liver (can be difficult to palpate)
Abdominal tenderness	Gallstones
Faecal masses	Constipation
Endocrine	
Goitre	Hypothyroidism
Extensive violaceous striae, dorsocervical fat pad, hypertension	Hypercortisolism
Pubertal staging	Premature common obesity; delayed monogenic or syndromal obesity
Micro-orchidism	Monogenic or syndromal obesity
Reduced growth velocity	Hypothyroidism, hypercortisolism
Pseudogynaecomastia	..
Psychosocial	
Altered mood	Depression, anxiety, child protection or welfare concerns, bullying or teasing
Musculoskeletal	
Reduced range of hip rotation and waddling gait	Slipped capital femoral epiphysis
Tibial bowing	Blount disease
Musculoskeletal tenderness on palpation	Bony malalignment
Pes planus; poor posture; reduced strength, balance, and coordination	Impaired physical fitness

Table: **Clinical findings on examination by organ system**

obesity to engage in active play. It can also enable socialisation with peers and facilitate motor competence, confidence, and optimisation of fundamental motor skills. The aims of exercise itself are to increase physical fitness, reduce or attenuate obesity-related complications, improve quality of life, and support the child to reach age-appropriate physical activity levels.[116,117] Studies have found that the most effective exercise interventions consist of sessions lasting 60 min or more on at least 3 days per week for at least 12 weeks duration.[118] Training programmes should be tailored to the child's physical abilities and fitness level evaluated at baseline using standardised and age-appropriate outcome measures. Intervention should be fun, leverage the preferences of the child while following frequency, intensity, duration, type, volume, and progression principles.[119]

Children with obesity often experience personal barriers to movement and exercise. Therefore tailoring and adapting paediatric exercise interventions will often be necessary, particularly for those that report musculoskeletal pain, high rates of fatigue, urinary incontinence, skin chafing, or have impaired motor skills. Additionally, the presence of intellectual or physical disabilities should be considered. As such, the type of exercise intervention offered will vary according to the child's clinical presentation and the desired outcome (eg, improvements in aerobic fitness, increased enjoyment, or reduction of fat-mass). The health professional might need to consider whether the intervention incorporates weight-bearing or non-weight bearing games, aerobic, proprioceptive and resistance exercises, individual or group-based work, or whether specific physiotherapy approaches might also need to be integrated to address underlying impairments. Appropriate monitoring and evaluation of the exercise

intervention is recommended and should include the perspective of the child in addition to psychometrically robust outcome measurement. Additional guidance is available elsewhere.[120–122]

Screen time and sedentary behaviours

Sedentary behaviours, including screen time, are distinct from physical activity and need to be addressed as part of a comprehensive behavioural change programme. Interventions that are successful in decreasing screen time in the short term include strong parental engagement, structural changes in the home environment (eg, removing or replacing home or bedroom electronic games access), and e-monitoring of time on digital devices.[123] These interventions tend to be more effective in young children.

Sleep

There are few trials targeting sleep in the treatment of paediatric obesity, especially in older children and adolescents. Sleep interventions in preschool-aged children are associated with reduced weight gain.[124] Improvements in sleep hygiene, such as a consistent bedtime routine, regular sleep-wake times, and reduced screen times in the evening, are likely to have many co-benefits and positive effects on other weight-related behaviours.

Behavioural support strategies

Changes in dietary intake, physical activity, sedentary behaviours, and sleep are underpinned by strategies supporting behaviour change with the vast majority of interventions using a form of behavioural therapy. Common behaviour change techniques include goal setting, stimulus control (modifying the environment), and self-monitoring.[107,125] Motivational interviewing techniques such as reflective listening and shared decision making might also be used by healthcare workers to improve motivation for behaviour change.[126,127]

The effectiveness of behaviour change interventions are well described, with modest reductions in weight-related outcomes[128,129] and improvements in cardiometabolic health.[130] The 2017 Cochrane reviews[128,129] found that behaviour changing interventions were more successful than no treatment or usual care comparators in reducing BMI (–0·53 kg/m² [95% CI –0·82 to –0·24], low-quality evidence in children; –1·18 kg/m² [–1·67 to –0·69], low-quality evidence in adolescents), and BMI z score (–0·06 units [–0·10 to –0·02], low-quality evidence in children; –0·13 [–0·21 to –0·05], low quality evidence in adolescents).[128,129] Effects were maintained at 18 to 24 months' follow-up for both BMI and BMI z-score in adolescents.[128] In children and adolescents aged 5–18 years, behavioural interventions are also associated with reductions in total cholesterol, triglycerides, fasting insulin, and HOMA-insulin resistance[130] as well as increased sleep duration and a reduced prevalence of obstructive sleep apnea.[131]

A systematic review of 109 randomised controlled trials (RCTs) found that dietary interventions achieve a modest reduction in energy intake, reduced intake of sugar sweetened beverages, and increased intake of fruit and vegetables in children and adolescents aged 2–20 years.[132] The beneficial effects of supervised exercise in children and adolescents with obesity on measures of anthropometry and adiposity include reductions in BMI, bodyweight, waist circumference, and percent body fat.[133] Improvements in obesity-related complications are also observed, independent of changes in anthropometry including increased cardiorespiratory fitness,[134] improved muscle performance[80] and fundamental motor skills,[135] reductions in insulin resistance, reductions in fasting glucose and insulin levels,[136] improved lipid profile,[137] and reductions in blood pressure.[138,139] Exercise might also yield additional benefits related to appetite and response to food cues.

Behavioural obesity treatment is also associated with improved psychosocial health, including improved quality of life,[128,140] and body image[141] compared with no treatment or usual care comparators post-intervention and improvements in self-esteem at latest follow-up in those aged 4–19 years at baseline.[141] In assessing mental health, no difference between intervention and no-treatment comparator groups have been seen for the changes in symptoms of depression,[142] anxiety,[142] and eating disorders,[143] during the intervention period. However, symptoms of depression, anxiety, and eating disorders are reduced post-intervention or at follow-up in intervention arms, with no worsening of symptoms within groups.[142–145] Adverse effects of lifestyle interventions are poorly reported.[128,129] Where reported, no significant differences in adverse events between intervention and control groups are seen.[146]

Psychological interventions

Psychological interventions, incorporated alongside traditional behavioural obesity treatment strategies, or as stand-alone interventions, target psychological factors that might contribute to eating behaviours and obesity, including distorted body image, negative mood, and stimulus control.[125,147] A core objective of psychological interventions is to reduce barriers for behaviour change.[147] Cognitive behavioural therapy (CBT) is the most frequently used approach, and addresses the relationship between cognitions, feelings, and behaviours using behavioural therapy techniques to modify behaviours and cognitive techniques to modify dysfunctional cognitions.[125] CBT-based interventions have been shown to result in healthier food habits, improved psychosocial health, quality of life, self-esteem, and anthropometric variables including BMI and waist circumference in children and adolescents.[125] Acceptance and commitment therapy (ACT), which encourage acceptance rather than avoidance of internal experiences (eg, food cravings), have shown to be effective in the management of obesity

in adults and are an emerging area of research in adolescence. Pilot studies have found ACT-based interventions to be feasible and acceptable in adolescents with obesity,[148,149] with further research underway. Weight-neutral interventions, aiming to promote healthy behaviours and improve physical and psychosocial health without promoting weight loss, are an emerging area of practice in adults. There is currently insufficient evidence to guide the use of weight-neutral approaches in paediatrics.

Mode of intervention delivery

Evidence for behavioural change programmes encompass a variety of modes of delivery including group programmes, one-on-one therapist sessions, and various forms of e-health support.[128,129,150,151] No one form is necessarily superior to another, although a combination of such approaches might be used in a comprehensive integrated programme. Availability of resources; time constraints for health professionals, patients, and families; and appropriate health professional training will influence treatment provided alongside the child's developmental stage and patient or parent preferences. The COVID-19 pandemic has highlighted the need for effective interventions that can be delivered remotely without exacerbating existing social and technological disparities.[152] A 2021 review describes the considerations for successful implementation for such telemedicine approaches.[153]

Eating disorders risk management

Children and adolescents with obesity are vulnerable to the development of eating disorders because obesity and eating disorders have several shared risk factors.[76,154] Disordered eating attitudes and behaviours, as precursors to eating disorders, are also elevated in children and adolescents with obesity.[155] Although obesity treatment helps improve eating disorder symptoms, including binge eating and loss of control in most youth with obesity,[143,144] a small number undergoing obesity treatment might develop an eating disorder during or after an intervention.[143] Although whether this low risk of developing eating disorders differs in youth who do not present for clinical treatment remains unclear, it is an important consideration for clinicians providing obesity care. For over a decade, it has been recommended that there be screening of disordered eating attitudes and behaviours before obesity treatment,[76,154] particularly with the use of dietary interventions,[112] to identify undiagnosed eating disorders. However, guidance on how this should occur in practice is scarce. Screening tools that assess for binge eating disorder specifically in children[156] and adolescents[157] with obesity are available but a self-report screening tool to assess for the broad spectrum of eating disorders for those with obesity and with adequate diagnostic accuracy is lacking. Eating disorder symptoms should not prevent the provision of obesity care;[158] rather, a combination of self-report questionnaires and clinical assessment might be needed to assess and monitor eating disorder risk in practice.[76]

Intensive dietary interventions

Use of intensive dietary interventions is an emerging area of research and practice, particularly in post-pubertal adolescents with obesity related comorbidity or severe obesity.[110,159] Prescriptive dietary approaches may be delivered within the context of a multicomponent behavioural intervention, by experienced paediatric dietitians with medical supervision.[159] Very Low Energy Diets (VLEDs), consisting of an energy prescription of approximately 800 kcal/day or less than 50% of estimated energy requirements, often involving the use of nutritionally complete meal replacement products, are one such option. A meta-analysis of 20 studies found VLEDs to be effective at inducing rapid short term weight loss in children and adolescents with obesity (–10·1kg [95% CI 8·7 to 11·4] over 3 to 20 weeks), though follow-up beyond 12-months is scarce.[160] Data on VLEDs in the treatment of youth with early onset type 2 diabetes are limited to a small pilot study[161] and a medical chart review,[162] however, they have shown early short-term success and the possibility of reducing the requirement for medication, including insulin, and inducing remission of diabetes.[163] However, there is need for further research.[163] Variations in macronutrient distribution have been widely studied due to hypothesised effects on satiety, particularly higher protein (20–30% of energy intake from protein) approaches and very low carbohydrate diets (<50g per day or <10% energy from carbohydrate) aiming to induce ketosis. Although lower carbohydrate approaches show a significantly greater reduction in weight-related outcomes in the short-term (<6 months), dietary patterns with varied macronutrient distribution do not show superior effects in the longer term in children and adolescents.[164] Pilot studies on the use of various regimens of intermittent energy restriction in adolescents with obesity have shown these to be feasible and acceptable.[165,166] Larger trials are underway.

Anti-obesity medications

Anti-obesity medications are an important part of comprehensive obesity treatment. Pharmacotherapy, when combined with behavioural change interventions, can be particularly useful in patients for whom behavioural approaches alone have proven suboptimal or unsuccessful in reducing BMI and improving obesity-related complications. Although regulatory approval and availability varies by country and region, there is one anti-obesity medication that is approved by most regulatory agencies (including the United States Food and Drug Administration and the European Medicines Agency) for chronic obesity treatment in adolescents aged 12–18 years, which is liraglutide at 3 mg daily. Liraglutide, delivered

via subcutaneous injection, belongs to the glucagon-like peptide-1 receptor agonist class, which acts on its receptors in the hypothalamus to reduce appetite, slow gastric motility, and act centrally on the hind brain to enhance satiety.[167] In the largest RCT of liraglutide 3 mg among adolescents (12 to <18 years old) with obesity, whereby all participants also received lifestyle therapy, the mean placebo-subtracted BMI reduction was approximately 5% with one year of treatment.[168] More participants in the liraglutide versus placebo group had a decline in BMI by 5% (43·3% liraglutide *vs* 18·7% placebo) and 10% (26·1% liraglutide *vs* 8·1% placebo). Importantly, no new safety signals were observed in the adolescent trial in relation to previous adult trials. The most reported adverse events were gastrointestinal and were more frequent in the liraglutide group (64·8% liraglutide *vs* 36·5% placebo). No statistically significant improvements in cardiometabolic risks factors or quality of life were observed between groups.

Other medications have been evaluated for the treatment of paediatric obesity yet are not approved in the EU and many other countries. These medications include orlistat (mean placebo-subtracted BMI reduction <3%), phentermine (no randomised, controlled trials conducted in children or adolescents), topiramate (mean placebo-subtracted BMI reduction <3%), and metformin (mean placebo-subtracted BMI reduction of around 3%).[169–172] Two anti-obesity medications are currently under evaluation for the treatment of adolescent obesity: semaglutide 2·4 mg weekly (NCT04102189) and the combination of phentermine and topiramate (NCT03922945). Results from these clinical trials are expected in 2022 with regulatory review and perhaps approval in 2022–23. Additional therapies are available for monogenic forms of obesity,[173] details of which are beyond the scope of the current Review.

Metabolic and bariatric surgery

Metabolic and bariatric surgery is the most effective and durable treatment for inducing weight loss in adolescents with obesity, with average BMI reductions in various longitudinal studies of the Roux-en-Y gastric bypass and vertical sleeve gastrectomy ranging from approximately 25–40% at 1–9 years post-surgery.[174–178] Beyond weight loss, metabolic and bariatric surgery leads to clinically meaningful improvements in obesity-related complications, cardiometabolic risk factors, musculoskeletal pain, and functional mobility.[174,178–180] Importantly, although the relative degree of weight loss with metabolic and bariatric surgery is similar between adults and adolescents, emerging data suggest that serious complications like type 2 diabetes and hypertension might be more likely to remit in adolescents than in adults.[177] Improved quality of life and reduced symptoms of depression are also seen following metabolic and bariatric surgery in the short term.[181,182] However, incidence and remission of mental health problems are highly variable following metabolic and bariatric surgery; a proportion of youth will continue to experience mental health concerns post-surgery that can persist long-term, with a subset experiencing suicidal ideations and behaviours.[183–186] Pre-surgical and post-surgical psychological support is recommended[187] and is associated with improved psychosocial health and weight loss maintenance.[188] Mortality rates 5 years following metabolic and bariatric surgery in adolescents (1·9%) appear to be similar to that of adults (1·8%) but adolescents tend to require abdominal reoperations more frequently than adults and have a higher incidence of low levels of ferritin,[177] requiring monitoring of nutritional status.[189] Until approximately 5 years ago, clinical practice guidelines for metabolic and bariatric surgery restricted eligibility to older adolescents having reached skeletal maturity. However, contemporary guidelines have now suggested that there need not be any lower age limit for consideration of metabolic and bariatric surgery as long as other medical eligibility requirements are met (detailed rationale and other considerations such as pubertal progression, linear growth, and pregnancy).[190,191] The uptake of metabolic and bariatric surgery has been extremely limited as surgery is typically reserved for the most severe forms of obesity and for patients with significant obesity-associated complications.[192] Other factors probably contributing to the low use of metabolic and bariatric surgery among adolescents include the perceived invasiveness and irreversibility of the procedures, lingering concerns regarding long-term safety, lack of access to surgical centres, poor insurance coverage, and referral bias.

Treatment selection

Treatment outcomes for paediatric obesity are highly variable[193–195] and a thorough baseline assessment guides the health professional on the appropriate treatment at a given time for a given patient. Baseline factors known to negatively predict weight-related outcomes with treatment include high levels of picky eating in preschool age children,[196] poor family functioning, or low self-concept[193] in the child and maternal psychological distress.[197] Positive predictors of treatment effect on weight include younger age, lower baseline BMI, higher global self-esteem, and adherence[198] to follow up. Predictors of treatment response represents a critically important area of future research as the ultimate goal is to match the appropriate patient to the treatment most likely to provide benefit and minimise potential risks from a non-suitable treatment plan.

Barriers to obesity treatment

Despite its growing evidence base, there remain many barriers to delivery of effective obesity treatment. For example, most RCTs have not been undertaken in culturally diverse populations, in people with complex health needs or disabilities, nor in those living with social

Search strategy and selection criteria

References for this Review were identified through searches of Medline (PubMed) and the Cochrane Database of Systematic Reviews for articles published up to Nov 1, 2021, using combinations of terms such as "child", "adolescent", "obesity", "epidemiology", "aetiology", "complications", "co-morbidity", "treatment", "behaviour change", "prevention", "bariatric surgery", "metabolic surgery", "pharmacotherapy", and "BMI". Articles published in English were included. We also reviewed reference lists of published manuscripts, clinical guidelines, and other relevant reviews and meta-analyses.

disadvantage, all of which might make adherence to standard therapies more challenging.[199] Further, there are failures in implementing the known evidence into routine service delivery. An audit of adult and paediatric obesity services in 68 countries revealed poor resourcing and staffing of clinical services; a lack of integration of services across primary, secondary and tertiary level care; inadequate health professional training; widespread health system stigma towards people with obesity; and frequent unaffordability and inaccessibility of services.[200] Future research is needed to both develop the evidence base for obesity treatment in priority populations and in LMICs, as well as bring an implementation science perspective to obesity service delivery.

Conclusions

Structured, supported and life-long care for children and adolescents with obesity and their families is essential. The provision of care will change over time based on growth, development, life stage, and available support. More research is needed on management of obesity in disadvantaged communities and in those from LMICs, and on the real-world implementation of management approaches. Importantly, clinical care needs to be underscored by modifications to the social, commercial, and built environments which currently promote, rather than protect against, obesity, together with associated policy changes.

Contributors

All authors contributed to the literature search, writing, reviewing, and editing of the manuscript.

Declaration of interests

ASK serves as an unpaid consultant for Novo Nordisk, Vivus, Eli Lilly, and Boehringer Ingelheim; and receives donated drug and placebo from Vivus for a National Institutes of Health funded clinical trial. LAB serves on the Advisory Committee of the ACTION Teens study, sponsored by Novo Nordisk, for which an honorarium is paid to her hospital research cost centre. All other authors declare no competing interests.

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