Pediatric Obesity

Prevention, Intervention, and Treatment Strategies for Primary Care

2nd Edition

Sandra G. Hassink, MD, FAAP

American Academy of Pediatrics
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To my husband, Bill, and my children,
Matthew, Stephen, and Alexa,
for your unfailing love and support.
Also to all my friends and colleagues along the way
who have dedicated themselves to improving
the lives of children.

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Foreword

Up to one-third of the childhood population has a body mass index (BMI) greater than 85% for age[1] and more than half of these children have a BMI greater than 95%.[2] These children are at risk for and are often already suffering from obesity-related comorbidities such as type 2 diabetes, non-alcoholic steatohepatitis, polycystic ovarian syndrome, sleep apnea, and Blount disease. They are also at risk for a lifetime of obesity with all its attendant medical and psychosocial consequences.

The evolution of obesity in childhood and adolescence is a complex interplay of gene-environment interactions, child temperament, parenting style, family dynamics, and home, school, and community environments. Children are influenced by parental role modeling, television advertising, and commercial food and entertainment offerings. Obesity and obesity-related comorbidities begin in childhood. Children in many ways are at the epicenter of the epidemic.

What does this mean for us as providers of pediatric health care? Not only can we not ignore the effects of obesity on the children we care for, we need to put ourselves in a position to take positive action in developing prevention, intervention, and treatment strategies for obesity.

This manual will help guide these efforts by providing information, strategies, and suggestions for approaches to prevention, intervention, and treatment at the primary care level. Starting with chapters on assessment and evaluation, each subsequent chapter focuses on a specific developmental stage with strategies for prevention of obesity in the normal weight population, intervention for children at risk for obesity, and treatment approaches for those children and adolescents whose BMIs are already greater than 95%. Families play a central role in modeling behavior, buffering the effects of an obesity-promoting environment and changing nutrition and activity habits to achieve a healthy energy balance for their children. Included in each chapter are questions for parents, self-assessment exercises, and points to touch on to enhance parenting information and skill in making family-based change. Additional information on practice-based changes and school interventions for primary care professionals can be used as a springboard for change in their own communities.

Patient handouts, American Academy of Pediatrics policy statements, and coding and reimbursement information are all collected in the appendices to aid the practitioner in implementing practice change aimed at obesity prevention, intervention, and treatment.

In this new second edition, a new chapter titled "Before Birth: Maternal Health" has been added to spotlight the understanding that maternal obesity increases the risk of pregnancy-related complications for both mother and infant. Data from numerous new studies have been incorporated into all chapters, and references have been updated. The nutrition and activity questions and approaches in each of the Health Supervision Visit chapters have been reviewed and enhanced. In the appendices, the growth charts have been updated to include the World Health Organization (WHO) charts, several new AAP policy statements have been added, coding information has been updated, and a brand new section on AAP obesity resources has been added.

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CHAPTER

1 Childhood Obesity: An Overview

1.1 The Rise in Obesity

Childhood obesity has risen to epidemic proportions. Every day pediatricians are faced with children who are overweight or obese and children who are suffering from the comorbidities of obesity. An overview of the problem will help us begin to create a strategy to respond to the needs of these patients and their families.

What happened to the population in the past 3 decades that has given rise to an increase of childhood and adult obesity of epidemic proportions?

There has been an inexorable shift in energy balance over the past 30 years. A combination of decreased activity, increased inactivity, and consumption of excess calories has contributed to the steadily increasing prevalence rates of childhood and adolescent obesity. From 2007 to 2008, 31.7% of children 2 to 19 years of age were above 85% for body mass index (BMI; classified as overweight), 16.9% were above 95% (classified as obese), and 11.9% were above 97%. This increase in obesity is occurring across the board and is alarmingly affecting our youngest patients; 9.5% of infants and toddlers were at or above 95% for weight/length.[1]

Minority populations are being hit even harder. The proportion of Hispanic and African American children aged 2 to 19 years with BMI values greater than 85% were 38.2% and 35.9%, respectively, as compared with 29.3% for non-Hispanic white children (2007–2009). These disparities are magnified by the number of children 2 to 19 years of age with BMI values greater than 95%: Mexican American (20.9%) and African American (20%), compared with non-Hispanic white children (15.3%).[1]

Genetic and Environmental Factors

What genetic and environmental interactions are involved?

There are hundreds of genes or gene markers associated in some way with energy balance. Predisposition to energy imbalance, increased nutrient partitioning to adipose tissue, and susceptibility to intrauterine programming are among many proposed mechanisms involving gene-environment interaction. Families with 1 or 2 obese parents are at increased risk for having a child or adolescent with obesity.[2] Predisposition for obesity-related comorbidities may also be inherited. For example, it is common to see type 2 diabetes, hypertension, dyslipidemia, and insulin resistance clustered in families. In most cases of obesity, genetic susceptibility to the environment influences outcome.

The interaction between genetics and environment is complex. Nutritional components may influence gene regulation, the intrauterine environment may affect later susceptibility to an energy-abundant environment, and the environment may have a greater effect during periods of rapid growth.

Societal Effects of Obesity

How does this explosion of obesity and associated comorbidities involve the child, family, community, and society at large?

The societal costs of obesity are increasing. There will be a burden of illnesses in the young adult and adult populations never seen before. More young parents will be chronically ill, and this will affect their children. There will be increasing stress on the health care system in terms of economics, time, and personnel. Children with obesity have almost twice the rate of physician visits as children with a BMI less than 95%, as well as increased prescription drug use and hospital admission rates.[3] The length of stay for discharges associated with obesity is longer than that for overall discharges. Costs associated with childhood obesity are estimated at $14 billion annually in direct and indirect health expenses. Annual obesity-related hospital costs for children and adolescents were $238 million in 2005, nearly doubling between the years 2003 and 2005.[3] The overall health effects on the population and the cost to the larger economy are already threatening to become our nation's biggest health expenditure.

1.2 The Medical Effects of Childhood Obesity

What are the medical effects of having an increasing population of children and adolescents with obesity?

Childhood obesity can be thought of as an accelerator of

adult diseases. Children and adolescents are now experiencing comorbidities, including type 2 diabetes, hypertension, dyslipidemia, obstructive sleep apnea, and nonalcoholic steatohepatitis (NASH), which was previously seen predominantly in adults. In addition, childhood obesity gives rise to serious orthopedic problems, such as slipped capital femoral epiphysis (SCFE) and Blount disease, and increases the incidence of less common but serious obesity-related conditions such as pseudotumor cerebri. These are diseases and complications few of us thought we would see in childhood or even adolescence; they give urgency to our need to institute prevention and early intervention, as well as diagnosis and treatment.

Comorbidities

There are a group of obesity-related comorbidities that require immediate attention: pseudotumor cerebri, SCFE, Blount disease, obstructive sleep apnea syndrome, NASH, cholelithiasis, metabolic syndrome, acanthosis nigricans, polycystic ovarian syndrome (PCOS), and type 2 diabetes.

Obesity occurs in 30% to 80% of children with *pseudotumor cerebri.*[4] Pseudotumor cerebri is defined as increased intracranial pressure with papilledema and normal cerebrospinal fluid in the absence of ventricular enlargement. Papilledema is part of the pathology of pseudotumor cerebri but may not occur initially. Presentation may range from an incidental finding on funduscopic examination to headaches, vomiting, blurred vision, or diplopia. Loss of peripheral visual fields and reduction in visual acuity may be present at diagnosis.[5] Neck, shoulder, and back pain have also been reported.[5] Treatment of pseudotumor cerebri includes acetazolamide, lumboperitoneal shunt in severe cases, and weight loss.[6] Pseudotumor cerebri is a diagnosis of exclusion after other causes of increased intracranial pressure are eliminated. It is important to remember that the neurologic deficits are symmetric in pseudotumor. Patients with asymmetric presentations must have other neurologic conditions.

Slipped capital femoral epiphysis is a slipping of the femoral epiphysis through the zone of hypertrophic cartilage cells, which are under the influence of gonadal and growth hormones.[7] From 50% to 70% of patients with SCFE have obesity.[8] Patients can present with limp or complaints of groin, thigh, or knee pain. The characteristic gait is with the leg abducted and externally rotated. Hips should be examined, and radiographs of both hips should be obtained because bilateral slips occur in 20% of cases. Medial and posterior displacement of the femoral epiphysis is seen through the growth plate relative to the femoral neck.[9] Treatment is surgical pinning of the hip.

The diagnosis of *Blount disease* involves identifying bowing of the tibia and femur. This can affect one or both knees. This condition is thought to result from excessive pressure damage to the medial proximal tibial epiphysis. Obesity is reported in two-thirds of patients with Blount disease.[10] Treatment requires surgical correction and weight loss.

Obstructive sleep apnea syndrome is a common diagnosis associated with obesity. This syndrome is defined as a breathing disorder characterized by prolonged partial upper airway obstruction or intermittent complete obstruction that disrupts normal ventilation during sleep and normal sleep patterns.[11] Symptoms can include nighttime awakening, restless sleep, difficulty awakening in the morning, daytime sleepiness, napping, enuresis, decreased concentration and memory, and poor school performance.[12] Nighttime polysomnography is the diagnostic procedure of choice. If left untreated, obstructive sleep apnea can result in pulmonary hypertension, systemic hypertension, and right-sided heart failure.[11] Weight gain, hypertrophy of the tonsils and adenoids, and intercurrent upper respiratory infections can provoke symptoms.

Nonalcoholic steatohepatitis is suspected when elevated liver enzymes are found in the context of fatty liver discovered by ultrasound in the absence of other causes of liver disease. From 20% to 25% of children with obesity have evidence of steatohepatitis.[13] The definitive diagnosis is made on the basis of the liver biopsy, in which evidence of inflammatory infiltrates and fibrosis can be seen. Nonalcoholic steatohepatitis can progress to cirrhosis and end-stage liver disease.[14] Weight loss reduces fatty infiltration and may decrease fibrosis.

Cholelithiasis symptoms in children include right upper quadrant abdominal pain and tenderness; diagnosis is made on the basis of ultrasound and appropriate laboratory studies. Of cases in adolescents, 50% are associated with obesity.[15]

Metabolic syndrome is a cluster of conditions characterized by insulin resistance. The components in childhood are central adiposity, elevated blood pressure, elevated triglyceride levels, decreased high-density lipoprotein (HDL) cholesterol, increased low-density lipoprotein cholesterol, and impaired glucose tolerance or hyperinsulinemia.

Acanthosis nigricans is often associated with metabolic syndrome, insulin resistance, and type 2 diabetes. This condition is characterized by hyperpigmentation and a velvety thickening that occurs in the neck, axillae, and groin.

Polycystic ovarian syndrome can occur in adolescence and is characterized by insulin resistance in the presence of elevated androgens. Clinical signs and symptoms include oligomen-

orrhea or amenorrhea, hirsutism, acne, polycystic ovaries, and obesity. There is some evidence that girls with premature adrenarche are at risk for PCOS.[16]

Type 2 diabetes occurs when the diagnosis of hyperglycemia is made in the presence of insulin resistance and an elevated insulin level. Type 2 diabetes can present with hyperglycemic hyperosmolar state (HHS), diabetic ketoacidosis, or symptoms of polyuria, polydipsia, and weight loss. Diagnosis can also be made in a patient with obesity on the basis of symptoms of hyperglycemia, such as abdominal pain, vomiting, dizziness, and weakness.

Obesity Emergencies

Complications of obesity can be life-threatening and even life-ending. Some severe obesity-related emergencies include HHS, diabetic ketoacidosis, pulmonary embolism, and cardiomyopathy of obesity. All of these have been seen in children and adolescents with obesity.

Hyperglycemic hyperosmolar state can rarely be the first manifestation of type 2 diabetes. Patients may initially present with symptoms of vomiting, abdominal pain, dizziness, weakness, polyuria and polydipsia, weight loss, and diarrhea.[17] If unrecognized, patients may develop hyperosmolar nonketotic coma and death.[18] Diagnostic criteria for HHS include a plasma glucose level greater than 600 mg/dL, serum carbon dioxide level greater than 15 mmol/L, small ketonuria, absent to low ketonemia, an effective serum osmolality greater than 320 mOsm/kg, and stupor or coma.[17,18]

Diabetic ketoacidosis can be an initial manifestation of type 2 diabetes even though most pediatricians associate it primarily with type 1. Insulin resistance often accompanies obesity and results in low baseline insulin sensitivity and relative insulin deficiency, which leads to increased lipolysis, increased free fatty acids in circulation, ketonemia, and ketonuria. Diabetic ketoacidosis should be treated as such, with the diagnosis of the diabetes type made when the patient is stabilized.

Pulmonary embolism has been reported as a complication of gastric bypass in adolescence.[19] The risk factors for pulmonary embolism include obesity, obesity-hypoventilation syndrome, obstructive sleep apnea syndrome, and coagulation disorder; symptoms include dyspnea, chest pain, decreased oxygen concentration, and hemoptysis.

Congestive heart failure resulting from obesity has been seen in adolescents with morbid obesity. The effect of obesity on the heart is known as *cardiomyopathy of obesity* and is thought to result from high metabolic activity of excessive fat, which increases total blood volume and cardiac output and leads to left ventricular dysfunction. Dilation, increased left ventricular wall stress, and compensatory left ventricular hypertrophy then occur. Pulmonary hypertension caused by upper airway obstruction can also occur. Signs and symptoms of cardiac failure should point to this diagnosis.[20]

Bariatric Surgery

As more adolescents are undergoing bariatric surgery, pediatricians may see either immediate or late-onset surgical complications (Table 1.1).

1.3 The Pediatrician's Role in the Obesity Epidemic

Today's Children at Risk

How does the obesity epidemic involve individual patients and our interaction with them and their families?

As you end this chapter, ask yourself, "What, as pediatricians, do we want for our patients?" Provide a healthy start in life? A chance to participate in the normal activities of childhood? A childhood free of disease? The vignettes that close this chapter show us how close this problem is to our everyday practice. This manual will help you begin to address the prevention and treatment of obesity in your daily encounters with patients and families.

You are seeing newborns in the nursery and examine Marta, a 1-day-old little girl born of Hispanic parents. Her chances of developing type 2 diabetes over her lifetime is 50%.

The lifetime risk of developing diabetes for an average person born in the United States in 2000 until his or her death is

- Male—1 in 3 chance
- Female—2 in 5 chance
- Hispanic female—1 in 2 chance (high risk)

Progression to diabetes among those with prediabetes is not inevitable. Studies suggest that weight loss and increased physical activity among people with prediabetes prevent or delay diabetes and may return blood glucose levels to normal.[21]

You are seeing Tom, a 14-year-old who wants to play football, for a sports physical. His BMI is 35, which is greater than 97%.

Adolescents who are obese have approximately an 80% likelihood of being obese adults.[22]

Table 1.1. Possible Complications of Bariatric Surgery

Complication Type	Complications
Early onset	• Bleeding • Bowel perforation • Deep vein thrombosis • Pulmonary embolism • Dehydration • Dysphagia • Nausea/vomiting • Dumping syndrome • Small bowel obstruction • Anastomotic leak • Peritonitis • Anastomotic stricture • Abdominal adhesions
Late onset	• Cholecystitis • Dysphagia • Gastroesophageal reflux • Incisional hernia • Malnutrition • Pancreatitis • Ulcers • Renal calculi • Internal hernia • Small bowel obstruction
Deficiencies	• Protein deficiency – Hair loss – Edema – Hypoalbuminemia – Anemia – Fatigue • Vitamin/mineral deficiencies – B_{12} – Folic acid – Iron – Fat-soluble vitamin
Gastric banding	• Intraoperative conversion to open gastronomy • Hemorrhage • Port infection • Stomal obstruction • Perforation • Late mechanical dysfunction • Hiatal hernia • Erosion • Band or port slippage

Sources: Zitsman JL, Fennoy I, Witt MA, et al. Laparoscopic adjustable gastric banding in adolescents: short-term results. *J Pediatr Surg.* 2011;46(1):157–162; Decker GA, Swain JM, Crowell MD, et al. Gastrointestinal and nutritional complications after bariatric surgery. *Am J Gastroenterol.* 2007;102(11):2571–2580

You are asked to speak at a preschool parents' meeting on nutrition and obesity.

The number of children with obesity has tripled in the past 3 decades.[23] One in 3 children is currently overweight or obese (BMI at 85% or higher).[1]

You meet Mr. and Mrs. Brown for a prenatal visit. Both the Browns are significantly obese.

There is a 75% chance that children will be overweight if both parents are obese—there is a 25% to 50% chance if just 1 parent with obesity.[3]

Parents of a high school sophomore with obesity ask you if there is anything they can do to help him lose weight.

More than 90% of high schools have vending machines, stores, or snack bars, but only 21% sell low-fat yogurt, fruits, or vegetables.[23] Only 50% of schools offer intramural activity or clubs for students.[3] Only 6% to 8% of schoolchildren have daily physical education.[23]

You are interviewed by your local newspaper and asked about the cost of the epidemic of childhood obesity.

Costs associated with childhood obesity are estimated at $14 billion annually in direct and indirect health expenses. Annual obesity-related hospital costs for children and adolescents were $238 million in 2005.[3]

Parents of an 8-year-old who is overweight ask you about her health risks during a well examination.

Children with a BMI greater than 85% are more likely to have elevated cholesterol and triglyceride levels and a lower HDL-C level and higher blood pressure than children of normal weight.[24]

A mother of a 4-year-old boy who is overweight and drinking 5 to 6 cups of juice a day believes that she is giving him a healthy drink.

Children increase their chances of becoming obese 1.6 times for each additional can or glass of sugared beverage they consume each day.[25]

Obesity prevention, intervention, and treatment will be integral to the practice of pediatrics in primary care practices, subspecialty pediatrics, and hospital-based care. Pediatricians are in a primary position to help children and families increase their knowledge and skills to combat obesity and obesity-related comorbidities.

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CHAPTER

2 Assessment of Obesity

2.1 Introduction

Pediatricians have always focused on helping children maintain normal growth. One of the founding principles of pediatrics is that good nutrition is essential for optimal health and growth. In 1897, L. Emmett Holt, Sr, MD, in *The Diseases of Infancy and Childhood,* wrote, "Nutrition in its broadest sense is the most important branch of pediatrics." In the late 19th and early 20th centuries, cycles of gastroenteritis and resultant malnutrition and recurrent infections caused the death of countless infants. Efforts were made to promote breastfeeding, ensure a safe milk supply, and create formula tailored to infant needs. Vitamin deficiencies were identified and treated, and the government created programs, such as the food stamp program (now known as the SNAP program); the Special Supplemental Nutrition Program for Women, Infants, and Children (commonly referred to as WIC); and school lunch and breakfast programs, to ensure that children's nutritional needs were met.

Pediatricians vigorously identified and treated children with failure to thrive and weight or height deceleration with the knowledge that growth disturbances were often the early signs of significant illness. Growth charts became tools to assess overall health, and there was always a reassurance when children "stayed on the growth chart." Attention to the optimal growth of premature infants, children with chronic disease, and disadvantaged children has all become integral parts of pediatric practice.

With a focus on optimal growth as a cornerstone to preventive care, pediatricians:

- Understand the importance of height, weight, and body mass index (BMI) as measures of overall health.
- Recognize the importance of breastfeeding in supporting healthy growth.
- Give attention to environmental, social, and psychosocial factors as parameters that are crucial to healthy growth.
- Recognize the importance of social supports in maintaining optimal nutrition and growth.
- Recognize the effect of family life events on the growth of children.
- Recognize the link between deviations in growth and disease.

The obesity epidemic challenges us to use these same skills to examine our strategies for prevention, intervention, and treatment to help children stay on the growth curve. We recognize that children with obesity are at risk for and suffer from diseases that were previously seen only in adults. Pediatricians have the opportunity to be involved with children and families from birth throughout adolescence. We have experience in taking a broad approach to growth and can use insights from our experiences to help our patients and families.

2.2 Assessing Obesity

With the onset of the obesity epidemic, assessing the growth status of all children is more important than ever. Preventive efforts that focus on maintaining normal healthy weight gain require that no opportunity be missed to measure height and weight and calculate BMI. Frequent assessment of BMI also makes it possible to identify children who are overweight or obese and allows for early intervention to reinforce and reestablish good nutrition and activity habits. Children who have BMI values greater than 95% require screening for obesity-related comorbidities and intervention to manage obesity.

Identifying excess adiposity in childhood is important for determining risk of obesity-associated comorbidities and future risk of obesity and obesity-related disease in adulthood. Direct measures of adiposity, such as hydrodensitometry, dual-energy x-ray absorptiometry, and magnetic resonance imaging, are used in research. Body mass index (weight/height^2) has been shown to correlate with direct measures of adiposity.[1] Because adipose tissue stores change as children grow and differ between boys and girls, BMI charts are specific for age and gender. Growth charts for 2 to 20 years are included in Appendix A.2; boys on page 123 and girls on page 125.

Body mass index should be calculated at least once a year in all children and adolescents[2] and can be calculated as weight

in kilograms divided by height in meters squared or weight in pounds multiplied by 703 and divided by height in inches squared. Once BMI is calculated, BMI classification can be assigned (Table 2.1).

Table 2.1. Current Classification of Weight Status by Body Mass Index (BMI)

BMI	Classification
<5%	Underweight
5%–85%	Normal weight
85%–95%	Overweight
>95%	Obese

Body mass index measurements are an important screening tool, but when applied to an individual patient, they need to be used in the context of that individual. For example, a highly trained, muscular athlete may have an increased BMI but no excess adiposity. History and physical assessment of nutrition and activity and anthropometric measurements, such as skinfold thickness, help put BMI into proper focus for each individual.

For patients younger than 2 years of age, weight-for-length graphs should be used to characterize appropriate growth. Growth charts for birth to 24 months are included in Appendix A.2; boys on page 122 and girls on page 124. If weight is accelerating ahead of height, attention should be paid to identifying any underlying medical or metabolic cause of obesity and ensuring that optimal nutrition and activity habits are being fostered.

Rapid change in BMI can also be used to identify a rate of excessive weight gain relative to linear growth. Sequential BMI measurements should be plotted and compared with BMI charts for age and gender. Often identifying the point at which excessive weight gain begins allows changes in risk factors for obesity to be addressed.

A Case in Point: CS

CS is an 11-year-old girl you are seeing for her yearly checkup. As you plot her height and weight and calculate her BMI, you note that she has gained 20 pounds (9.1 kg) since last year and has a BMI placing her at 90% for her age and gender.

Over the past year she visited your office several times for respiratory illnesses but has no other problems. She is not taking any medications. You ask about school, and she and her mother report that her grades are good but she is experiencing some teasing. When you ask CS about this, she becomes upset and tells you that no one wants to play with her. Mom says she is worried about CS's self-esteem. You acknowledge the difficulty of getting teased and ask CS how she responds. She says she gets upset and usually says something back to the girls who tease her. You briefly discuss some responses to teasing, and CS expresses some interest in trying to ignore their comments and walk away.

You then share the growth and BMI charts with Mom and CS and explain that sometimes eating and activity habits change when someone is being teased and feeling bad. You let them know that staying healthy is important and begin to ask specifically about her eating and activity. Mom notes that CS comes home from school starving and that as soon as she finishes dinner she asks for more food. CS says she is watching television or is on the computer a lot, which Mom translates to about 4 hours a day.

You then begin to explore CS's interest in out-of-school activities to help give her another venue for physical activity and peer interaction. Mom says they have a YMCA membership; you ask CS and Mom to look into 2 or 3 possible activities to join, such as a dance, karate, or swimming, and have CS to choose one of them. You ask Mom if she would prepare an after-school snack for CS instead of having her daughter get her own, to which Mom agrees.

You go over the laboratory studies that you want to order for CS based on her BMI; then you ask CS and Mom to come back in 3 to 4 weeks to review her laboratory studies, to see how the changes in her eating and activity are going, and most important, to see how she is doing with the kids at school.

Second Visit

A month later CS returns to your office. You reviewed the laboratory studies; her triglyceride levels are mildly elevated, but there are no other abnormalities. Her weight decreased by 1 pound (0.5 kg). She joined a karate class at the YMCA and has been going 2 to 3 times a week. Mom prepares an after-school snack, and CS is not asking for extra food. CS seems happier and says she has met some kids at karate that she likes. CS and Mom say they are willing to keep some diet records to address CS's elevated triglyceride levels at the next visit. You schedule the next visit and plan to continue supporting CS and her family with the goal of normalizing her triglyceride levels and returning her to her previous growth trajectory.

Several states now mandate that schools measure or report BMI. This may provide additional opportunities to address risk for obesity and actual obesity in the school-age population if linked with communication with children's pediatricians.

2.3 Identifying Children and Families at Risk for Obesity

Risk factors for obesity can be identified in children and include

- Genetic and medical disorders associated with obesity
- Parental obesity
- Nutrition and activity patterns associated with obesity
- Living in an at-risk environment

Obesity associated with genetic syndromes is a rare but important cause of childhood obesity.[3] Syndromes associated with obesity are frequently characterized by developmental delay, short stature, dysmorphic features, and involvement of specific organ systems.

Prader-Willi syndrome, which most commonly results from a deletion of 15q11q13, is the most common single-gene obesity-associated syndrome, occurring in 1 out of every 10,000 to 15,000 births. Primary features include infantile hypotonia, a poor sucking reflex, developmental delay, mental retardation, short stature, and skin picking. Obesity becomes apparent as early as 2 years of age, along with hyperphagia, hypogonadism, and behavioral difficulties.[4] With early diagnosis and counseling, excessive weight gain can often be controlled. Additional obesity-associated genetic syndromes are listed in Table 2.2.

Medical conditions that may result in obesity include injury to the hypothalamus as a result of trauma or malignancy, surgery, or radiation treatment (Table 2.3). Endocrinologic causes of obesity include Cushing syndrome, hypothyroidism, and growth hormone deficiency. Psychosocial conditions may also be associated with obesity.

Drug therapy can present a risk for weight gain. Table 2.4 shows commonly prescribed drugs that may be associated with obesity.

Children from pregnancies complicated by diabetes and/or cigarette smoking and children born small for gestational age with accelerated catch-up growth have a greater incidence of obesity (see Chapter 3).

Table 2.2. Genetic Syndromes Associated With Obesity

Syndrome	Associated Issues
Prader-Willi syndrome	Infantile hypotonia; poor feeding followed by hyperphagia and weight gain, developmental delay, mental retardation, short stature, skin picking, behavioral and psychosocial problems
Bardet-Biedl syndrome	Polydactyly, cognitive delay, short stature, retinitis pigmentosa, renal disease, hypogonadism
Alström syndrome	Nerve deafness, diabetes, pigmentary retinal degeneration, cataracts
Albright hereditary osteodystrophy	Short stature; may have pseudohypoparathyroidism, ectopic calcifications, hypocalcaemia
Hereditary Cushing syndrome	Carney complex, an autosomal dominant syndrome of multiple neoplasia, spotty skin pigmentation, multiple endocrine neoplasia, testicular neoplasia, ovarian cysts[5]
Isolated growth hormone deficiency	Short stature, central obesity[5]
X-linked syndromic mental retardation	X-linked mental retardation with a high prevalence of obesity from mutations in the *MECP2* gene[5]

Table 2.3. Medical Conditions Associated With Obesity

Condition	Associated Issues
Hypothalamic obesity	• Head injury; central nervous system malignancy, radiation, or surgery
	• Associated neurologic and endocrinologic deficits
Cushing syndrome	• Hypertension, centripetal obesity, striae
	• Elevated cortisol
Hypothyroidism	• Constipation, linear growth delay, myxedema, lethargy
Growth hormone deficiency	• Short stature
Depression or anxiety	• Change in eating and activity behavior or pharmacotherapy
Abuse	• Signs and symptoms consistent with physical, psychological, and/or sexual abuse[3]

Table 2.4. Drugs Associated With Obesity

• Glucocorticoids	• Carbamazepine
• Phenothiazines	• Beta-adrenergic blockers[6]
• Tricyclic antidepressants	• Insulin
• Valproic acid	• Selective serotonin reuptake inhibitors[6]

Parental obesity is a strong predictor of childhood and adolescent obesity. A child of 1 obese parent has a 30% likelihood of becoming and obese adult. A child of 2 obese parents has a 70% chance. The strong predictive value of parental obesity can allow early identification of children at risk for obesity and early intervention in helping families develop good nutrition and activity patterns. This is particularly important because activity and nutritional patterns are formed at a very early age. For example, studies show that preschool-age children from obese or overweight families share food preferences with their parents and prefer sedentary activity.[7]

Nutritional patterns that may increase the risk of obesity include eating meals at restaurants, eating take-out meals, consuming increased portion sizes, snacking, and skipping meals,[8] as well as drinking soda, sugar-sweetened beverages, and juice.[9,10] Other major sources of risk for obesity are increased television watching, decreased physical activity,[2] and decreased sleep.[11] It is important to identify nutritional and activity patterns that increase the risk of obesity because many are directly influenced by parental and family behavior patterns and could be amenable to lifestyle change.

The school environment can increase the risk of obesity if sugar-sweetened beverages and snacks are available. Lack of physical education, longer school days, and a decrease in recess may also contribute to an environment that promotes obesity. Less walking and biking to school have also been implicated in rising obesity trends.[8]

Once obesity is identified, screening for obesity-related comorbidities and intervening to normalize weight need to occur. These topics will be addressed at length in the following chapters.

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Health Supervision Visits:

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CHAPTER

3 Before Birth: Maternal Health

3.1 What Do We Know?

Improving maternal health before and during pregnancy is a focal point for improving pregnancy outcomes, and it is important to understand that maternal obesity increases the risk of pregnancy-related complications for both mother and infant (Table 3.1).[1,2]

Maternal morbidity increases with increasing body mass index (BMI); therefore, attention to obesity in women in their childbearing years has important implications for maternal health. More than one-third of women between 20 and 39 years of age have a BMI greater than 30. More than 18% have a BMI greater than 35 and 4.2%, greater than 40. In this age group, obesity rates with a BMI greater than 30 vary by ethnicity: non-Hispanic white women (31.3%), non-Hispanic black women (47.2%), and Hispanic women (37.6%).[10] This means that one-third to almost half of women between 20 and 39 years of age are at risk for obesity-related complications of pregnancy.

Complications of pregnancy increase as BMI increases. Maternal BMI of 50 or greater was positively associated with large-for-gestational-age (LGA) newborns, gestational diabetes (GDM), preeclampsia, cesarean delivery, and a 5-minute Apgar score below 7.[11] The rates of cesarean section and induced preterm delivery also rise with increasing maternal BMI,[12,13] as does the rate of neonatal death (5.3/100,000 in nonobese women to 7.5/100,000 in women with morbid obesity).[11]

Maternal obesity results in altered infant body composition. Newborns of mothers with obesity have increased fat mass, body fat, and ponderal index (weight/height^3) compared with newborns of nonobese mothers.[14] Newborns of mothers with obesity are more insulin-resistant than infants born to nonobese mothers. The degree of insulin resistance in an infant correlates with maternal insulin resistance and infant adiposity.[15]

Children of mothers who are obese in pregnancy are at increased risk of being obese in childhood and adolescence.[16,17] Overweight rates were even higher in offspring of mothers who were both overweight and had GDM.[4] One study reported that adequate treatment of GDM during pregnancy may attenuate this risk.[18] This increased risk may be attributable to the increased rates of prediabetes in this population.[19]

The incidence of GDM has increased in the general population, paralleling the increase in obesity.[20] The GDM rate for women with a BMI between 30 and 35 is 4.8%, and the rate is 11.5% for women with a BMI greater than 35 compared with 0.7% in women of normal weight.[21] Maternal diabetes alters the intrauterine environment. In a study of Pima Indians, siblings born after their mother developed type 2 diabetes were more likely to develop diabetes and obesity than those born before the onset of maternal diabetes.[22]

Mothers with obesity are also at an increased risk for hypertension, preeclampsia, and eclampsia during pregnancy. Risk of hypertension is 1.7% for women with a BMI between 25 and 29.9 and 2.2% for those with a BMI greater than 30 compared with women with a BMI less than 21.[23] The risk of preeclampsia doubles when a woman enters pregnancy overweight and triples when obese.[24]

The Institute of Medicine (IOM) recommends beginning pregnancy at a healthy weight and revised its pregnancy weight gain guidelines to improve pregnancy outcomes.[25]

Table 3.1. Adverse Outcomes of Pregnancy Associated With Maternal Obesity Gestational Diabetes[3]

Hypertension[4]
Preeclampsia[4]
Eclampsia[4]
Infection[1]
Cesarean delivery[5]
Thromboembolic event[1]
Surgical complications[1]
Early miscarriage[6]
Induced preterm delivery[2]
Fetal/neonatal death[7]
Macrosomia[1]
Early neonatal death[1]
Suboptimal ultrasound visualization[8]
Maternal mortality[9]