



Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity

Sarah E. Hampl, MD, FAAP,^a Sandra G. Hassink, MD, FAAP,^b Asheley C. Skinner, PhD,^c Sarah C. Armstrong, MD, FAAP,^d Sarah E. Barlow, MD, MPH, FAAP,^e Christopher F. Bolling, MD, FAAP,^f Kimberly C. Avila Edwards, MD, FAAP,^g Ihuoma Eneli, MD, MS, FAAP,^h Robin Hamre, MPH,ⁱ Madeline M. Joseph, MD, FAAP,^j Doug Lunsford, MD,^k Eneida Mendonca, MD, PhD, FAAP,^l Marc P. Michalsky, MD, MBA, FAAP,^m Nazrat Mirza, MD, ScD, FAAP,ⁿ Eduardo R. Ochoa, Jr, MD, FAAP,^o Mona Sharifi, MD, MPH, FAAP,^p Amanda E. Staiano, PhD, MPP,^q Ashley E. Weedn, MD, MPH, FAAP,^r Susan K. Flinn, MA,^s Jeanne Lindros, MPH,^t Kymika Okechukwu, MPA^u

Greetings

You have in your hands, or at your fingertips, the first edition of the American Academy of Pediatrics clinical practice guideline for evaluation and management of children and adolescents with overweight and obesity. Putting together this guideline was no small task, and the Academy is grateful to the efforts of all the professionals who contributed to the production of this document. This work is a true testament to their passion and dedication to combatting childhood and adolescent overweight and obesity.

The Subcommittee responsible for developing this guideline comprises a diverse group of professionals from a variety of disciplines representing both governmental entities and private institutions. Experts all, they are united by a common desire to provide the finest, most effective care and treatment to children and adolescents with overweight and obesity. Over the course of several months, the members of the Subcommittee reviewed the technical reports produced from the study review, then worked in concert to develop the Key Action Statements and Expert Consensus Recommendations contained within this guideline. These were crafted with meticulous care by the Subcommittee members, to align with current literature and to place appropriate emphasis on each statement.

While representing such a broad spectrum of perspectives, the members of this committee are all keenly aware of the multitude of barriers to treatment that patients and their families face. These barriers impact not only their access to treatment, but their ability to follow prescribed treatment plans. Whereas some patients are able to adopt the lifestyle changes and habitualize elements of their prescribed treatment plans, so many others struggle to do so for a wide variety of reasons. The members of the Subcommittee understand all of this. To assist with optimizing health equity and overcoming these barriers, guidance on a number of multilevel factors related to barriers to treatment have been included in this guideline. During the course of their work, members of the Subcommittee acknowledged that, although so much has been learned to advance the treatment of children and adolescents with overweight and obesity, there is still so much we have yet

^aChildren's Mercy Kansas City Center for Children's Healthy Lifestyles & Nutrition, University of Missouri-Kansas City School of Medicine, Kansas City, Missouri; ^bMedical Director, American Academy of Pediatrics, Institute for Healthy Childhood Weight, Wilmington, Delaware; ^cDepartment of Population Health Sciences, Duke University School of Medicine, Durham, North Carolina; ^dDepartments of Pediatrics and Population Health Sciences, Duke Clinical Research Institute, Duke University, Durham, North Carolina; ^eDepartment of Pediatrics, University of Texas Southwestern Medical Center; Children's Medical Center of Dallas, Dallas, Texas; ^fDepartment of Pediatrics, University of Cincinnati College of Medicine, Cincinnati, Ohio; ^gChildren's Health Policy & Advocacy, Ascension; Department of Pediatrics, Dell Medical School at The University of Texas at Austin, Austin, Texas; ^hDepartment of Pediatrics, The Ohio State University, Center for Healthy Weight and Nutrition, Nationwide Children's Hospital, Columbus, Ohio; ⁱCenters for Disease Control and Prevention; Atlanta, Georgia; ^jDivision of Pediatric Emergency Medicine, Department of Emergency Medicine, University of Florida College of Medicine-Jacksonville, University of Florida Health Sciences Center-Jacksonville, Jacksonville, Florida; ^kFamily Representative; ^lDepartments of Pediatrics and Biostatistics & Health Data Science, Indiana University School of Medicine, Indianapolis, Indiana; ^mDepartment of Pediatric Surgery, The Ohio State University, College of Medicine, Nationwide Children's Hospital, Columbus, Ohio; ⁿChildren's National Hospital, George Washington University, Washington, DC; ^oDepartment of Pediatrics, University of Arkansas for Medical Sciences, Arkansas Children's Hospital, Little Rock, Arkansas; ^pDepartment of Pediatrics, Yale School of Medicine, New Haven, Connecticut; ^qLouisiana State University Pennington Biomedical Research Center, Baton Rouge, Louisiana; ^rDepartment of Pediatrics, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma; ^sMedical Writer/Consultant, Washington, DC; ^tAmerican Academy of Pediatrics, Itasca, Illinois; and ^uAmerican Academy of Pediatrics, Itasca, Illinois

This document is copyrighted and is property of the American Academy of Pediatrics and its Board of Directors. All authors have

To cite: Hampl SE, Hassink SG, Skinner AC, et al. Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents With Obesity. *Pediatrics*. 2023;151(2):e2022060640

to discover. The Subcommittee recognizes the importance that future studies will play in advancing our knowledge and understanding of this chronic disease, knowledge and understanding that will lead to the development of new and more effective treatments. Specific discussion about the needs for future research are included in the guideline.

It is the fervent hope of every member of the Subcommittee that this guideline and the resources that accompany it will provide you with a more complete understanding of the issues, factors, and needs of patients combating overweight and obesity, as well as successful treatment options to assist them in their battle. This guideline and the resources that accompany it are not only for you, they are because of you, and all that you do to care for each and every patient as if they were the most important one. Because, as we all know, they are.

Be well,

Doug Lunsford, Family Representative

I. INTRODUCTION

The current and long-term health of 14.4 million children and adolescents is affected by obesity,^{1,2} making it one of the most common pediatric chronic diseases.³⁻⁵ Long stigmatized as a reversible consequence of personal choices, obesity has complex genetic, physiologic, socioeconomic, and environmental contributors. As the environment has become increasingly obesogenic, access to evidence-based treatment has become even more crucial.

A significant milestone in the fight to counter misperceptions about obesity and its causes occurred in 1998, when the National Institutes of Health (NIH) designated obesity as a chronic disease. The NIH made a further commitment to necessary research in the “Strategic Plan for NIH Obesity Research,” released by the NIH Obesity Task Force in 2011.⁶ In 2013, on the basis of accumulating evidence, the American Medical Association recognized obesity as a complex, chronic disease that requires medical attention.⁷

The scientific and medical community’s understanding of obesity is constantly evolving. Increased understanding of the impact of social determinants of

health (SDoHs, see Definition of Terms section) on the chronic disease of obesity—along with heightened appreciation of the impact of the chronicity and severity of obesity comorbidities—has enabled broader and deeper understanding of the complexity of both obesity risk and treatment.^{8,9} Multiple randomized controlled trials and comparative effectiveness studies have yielded effective treatment strategies, demonstrating that, despite the complex nature of this disease, obesity treatment can be successful.^{10,11}

The knowledge and skills to treat childhood obesity have become necessities for clinical teams in pediatric primary and subspecialty care. For more than 2 decades, the American Academy of Pediatrics (AAP) and its members have had the opportunity to collaborate with multiple scientific and professional organizations to improve the clinical care of children with overweight and obesity. Notable milestones include the 1998 “Expert Committee Recommendations,”¹² the 2007 “Expert Committee Recommendations,”¹³⁻¹⁵ the creation of the AAP Section on Obesity and founding of the Institute for Healthy Childhood Weight, both in 2013; and the Institute’s

“Algorithm for the Assessment and Management of Childhood Obesity” in 2016.¹⁶

This is the AAP’s first clinical practice guideline (CPG) outlining evidence-based evaluation and treatment of children and adolescents with overweight and obesity.

This guideline does not cover the prevention of obesity, which will be addressed in a forthcoming AAP policy statement.

The CPG also does not include guidance for overweight and obesity evaluation and treatment of children younger than 2 years. Children under the age of 2 were not part of the inclusion criteria for the evidence review, because it is difficult to practically define and measure excess adiposity in this age group. The CPG also does not discuss primary obesity prevention, as no studies reporting results of obesity prevention interventions met the inclusion criteria for the evidence review.

Nonetheless, the topics of obesity prevention and evaluation and treatment of children younger than 2 years are very important to reduce this threat to children’s

current and future health. Future CPGs may include these topics; in the meantime, information that may assist pediatricians and other pediatric health care providers (PHCPs) is included on the AAP Institution for Healthy Childhood Weight's Web site (aap.org/obesitycpg). Further information on the CPG's methodology and the writing committee's approach is covered in subsequent sections.

The CPG contains Key Action Statements (KASs), recommendations based on evidence from randomized controlled and comparative effectiveness trials as well as high-quality longitudinal and epidemiologic studies. The CPG writing Subcommittee uses the term "pediatricians and other pediatric health care providers" to include both pediatric primary and specialty care physicians and other medical providers as well as allied health care professionals, since all will encounter and can intervene with children with overweight, obesity, and obesity-related comorbidities. An algorithm with these KASs is provided in Appendix 1.

The KASs are supplemented by Consensus Recommendations that are based on expert opinion and address issues that were not part of the supporting technical reports (TRs). These consensus recommendations are supported by AAP-endorsed guidelines, clinical guidelines, and/or position statements from professional societies in the field and an extensive literature review.

This CPG stands on the shoulders of the pediatricians, other PHCPs, clinical researchers, and other stakeholders who collaborated to create the previous Expert Recommendations, which have been valued sources of guidance for health care professionals, clinical systems, parents, and other key

stakeholders. It is our hope that this CPG will further advance the equitable care of children and adolescents with this chronic disease.

II. APPROACH

Childhood obesity results from a multifactorial set of socioecological, environmental, and genetic influences that act on children and families. Individuals exposed to adversity can have alterations in immunologic, metabolic, and epigenetic processes that increase risk for obesity by altering energy regulation.¹⁷⁻¹⁹ These influences tend to be more prevalent among children who have experienced negative environmental and SDoHs, such as racism.²⁰ Overweight and obesity are more common in children who live in poverty,^{21,22} children who live in underresourced communities,²³ in families that have immigrated,²⁴ or in children who experience discrimination or stigma.²⁵⁻³² As such, obesity does not affect all population groups equally.³³ This fact highlights the importance of understanding the role of SDoHs³⁴ as well as the social context of children and their families in the etiology and treatment of overweight and obesity.

Children with overweight and obesity benefit from health behavior and lifestyle treatment, which is a child-focused, family-centered, coordinated approach to care, coordinated by a patient-centered medical home, and may involve pediatricians, other pediatric health care providers (such as registered dietitian nutritionists [RDNs], psychologists, nurses, exercise specialists, and social workers), families, schools, communities, and health policy.³⁵ Obesity is long-lasting and has persistent and negative health effects, attributable morbidity and mortality, and social

and economic consequences that can impact a child's quality of life.³⁶⁻³⁹ Because obesity is a chronic disease with escalating effects over time, a life course approach to identification and treatment should begin as early as possible and continue longitudinally through childhood, adolescence, and young adulthood, with transition into adult care.^{36,39-41}

A. Health Equity Considerations

It is not uncommon for the differences in disease prevalence and outcomes among population groups to be described in terms of ethnicity, race, gender, and/or age and for these differences to be referred to as "disparities."⁴² Disparity, however, only defines differences *between* groups without referring to inequities that cause these differences among populations (ie, "economic, civil-political, cultural, or environmental conditions that are required to generate parity and equality"⁴²). Precisely because of the intertwining of inequities throughout the life course, health disparities can be found from maternal pregnancy outcomes through adolescence and, as such, can have an inevitable impact on childhood obesity.

This distinction between health disparities and inequities is particularly important when considering chronic disease, because: (1) obesity risk factors are embedded in the socioecological and environmental fabric of children's lives; and (2) there is a danger of stigmatizing children with obesity and their families on the basis of race and ethnicity, age, and gender based on the disparities of outcome—with failure to recognize the systemic challenges that cause and maintain inequities.^{43,44}

Inequities are often associated with each other⁴⁵ and result in disparities in obesity risk and outcomes across the socioecological

spectrum. Importantly, they represent neighborhood-, community-, and population-level factors that can be changed.⁴⁶ Inequities that promote obesity in childhood can have a longitudinal effect leading to disparities in adult health and contributing to adult obesity and chronic disease.⁴⁷

The AAP is dedicated to reducing health disparities and increasing health equity for all children and adolescents.²⁸ Attainment of these goals requires addressing inequities in available resources and systemic barriers to quality health care services for children with obesity.⁴⁸ To that end, “practice standards must evolve to support an equity-based practice paradigm” and payment strategies must promote this approach to care.²⁸

It is our hope that individual clinical encounters with patients and families will provide opportunities to “screen and address the social, economic, educational, environmental, and personal-capital needs of the children with obesity and their families.”⁴⁹ In addition, understanding the wider determinants of the chronic disease of obesity will enable pediatricians and other PHCPs to “raise awareness of the relevance of these social and environmental determinants of childhood obesity in their communities.”⁴⁹

B. Racism

Racism as an SDoH has been defined as a “system of structuring opportunity and assigning value based on the social interpretation of how one looks (race) that unfairly disadvantages some individuals and communities (and) unfairly advantages other individuals and communities ...”^{50,51} that “impacts the health status of children, adolescents, emerging adults and their families.”⁵²

Inequalities in poverty, unemployment, and homeownership

attributable to structural racism have been linked to increased obesity rates.⁵³ Racism experienced in everyday life has also been associated with increased obesity prevalence.⁵⁴ Youth with overweight and obesity have been found to be at increased risk not only for weight-based harassment but also for sexual harassment and harassment based on race and ethnicity, socioeconomic status (SES), and gender.⁵⁵ In adults, studies have found positive associations between self-reported discrimination and waist circumference,^{56,57} visceral adiposity,⁵⁸ and BMI⁵⁷ in both non-Latino and Latino populations.⁵⁹

C. Weight Bias and Stigma Considerations

Individuals with overweight and obesity experience weight stigma, victimization, teasing, and bullying, which contribute to binge eating, social isolation, avoidance of health care services, and decreased physical activity.^{28,43} Importantly, internalized weight bias has been associated with a negative impact on mental health.⁶⁰ Collectively, these factors may adversely affect quality of care, prevent patients with overweight and obesity from seeking medical care, and contribute to worsened morbidity and mortality, independent of excess adiposity.^{28,43,44}

Pediatricians and other PHCPs have been—and remain—a source of weight bias. They first need to uncover and address their own attitudes regarding children with obesity. Understanding weight stigma and bias, and learning how to reduce it in the clinical setting, sets the stage for productive discussions and improved relationships between families and pediatricians or other PHCPs. Acknowledging the multitude of genetic and environmental factors that contribute to the complexity of

obesity is an important mitigator in reducing weight stigma.⁶¹ Additional actions that reduce weight stigma include having appropriately sized office furniture, using appropriate capacity medical equipment, ensuring that aesthetic and/or instructional images posted in the office are inclusive, and avoiding stigmatizing language.²⁸ Accordingly, the CPG utilizes person-first language (ie, using the term “child with obesity, rather than “obese child”) to avoid labeling the child.²⁸ This practice is consistent with recommendations from the AAP and other national organizations, including the Academy of Nutrition and Dietetics, the Obesity Society, and the Obesity Action Coalition.⁶²

D. Adverse Childhood Experiences

Adverse childhood experiences (ACEs) are negative experiences caused by situations or events in the lives of children and adolescents that can pose threats to their current and future physical and mental health.^{63,64} These experiences range from family turmoil and violence to financial hardship, loss of a parent, divorce, abuse, and parental mental illness—to name a few.⁶⁵ ACEs have been associated with obesity, both in adulthood and in childhood.^{66–68} Children and adolescents who live in poverty have a higher likelihood of experiencing ACEs, but risk for ACEs occurs at every income level.^{65,69} The greater the number of ACEs a child or adolescent experiences, the greater the risk for obesity.⁷⁰ The most commonly cited mechanisms linking ACEs to obesity are social disruption, negative health behaviors, and chronic stress response.⁷¹

Approach Summary

The recommendations in the CPG are child-centric and not specific to a particular health care setting and are written to inform pediatricians and other PHCPs about the standard

of care for evaluating and treating children with overweight and obesity and related comorbidities. To reflect the pediatrician's and PHCP's individual relationship with the child and family, the Subcommittee refers to "evaluation" (eg, for comorbidities) rather than "screening." It is anticipated that a pediatrician's or other PHCP's setting, training, and expertise may moderate how elements of the CPG are implemented. Helpful resources can be found in accompanying implementation materials.

Understanding the underlying genetic, biological, environmental, and social determinants that pose risk for obesity is the bedrock of all evaluation and intervention. Allowing the family to have a safe space to understand and process the complexity of obesity and its chronicity requires tact, empathy, and humility. Achieving this goal enables the patient and family to gain the knowledge and understanding needed to recognize risk factors in their environment and behaviors, to honor cultural preferences, and to institute changes independently as well as under the guidance of a trusted and well-trained advocate—such as pediatricians and other PHCPs.

Finally, to emphasize important goals of treatment—both improved weight status and reduction or elimination of comorbidities—the Subcommittee uses the term intensive health behavior and lifestyle treatment (IHBLT) rather than "intensive lifestyle or behavioral modification" or "weight management." Additional definitions are listed in the next section.

III. DEFINITION OF TERMS

BMI: BMI is a measure used to screen for excess body adiposity; it is calculated by dividing a person's weight in kilograms by the square of

height in meters. For children and teens, BMI interpretation is age- and sex-specific. A child's BMI category (eg, healthy weight, overweight) is determined using an age- and sex-specific percentile for BMI rather than the BMI cut-points used for adult categories.⁷²

Capacity-building: "Building individual competencies and technical expertise, strengthening organizational capacities, and enabling supportive structural environments" to maintain or improve health services delivery.⁷³

Children with special health care needs: Children with special health care needs are those who have, or who are at increased risk for, a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally.⁷⁴

Chronic care model: The chronic care model identifies essential elements of a health care system that encourage high-quality chronic disease care: the community; the health system; self-management support; delivery system design; decision support, and clinical information systems.⁷⁵

Chronic disease: The Centers for Disease Control and Prevention (CDC) defines chronic diseases broadly as "conditions that last 1 year or more and require ongoing medical attention or limit activities of daily living or both."⁷⁶ Obesity is a chronic disease that results in altered anatomy, physiology, and metabolism—all of which adversely affect the physical and mental health trajectory of children and adolescents.⁷⁷ The Obesity Medicine Association defines obesity as a "chronic, relapsing, multifactorial, neurobehavioral disease, wherein an

increase in body fat promotes adipose tissue dysfunction and abnormal fat mass physical forces, resulting in adverse metabolic, biomechanical, and psychosocial health consequences."⁷⁸

Comprehensive obesity treatment: Comprehensive obesity treatment (COT) (Fig 1) includes^{79,80}:

- Providing intensive, longitudinal treatment in the medical home
- Evaluating and monitoring child or adolescent for obesity-related medical and psychological comorbidities
- Identifying and addressing social drivers of health
- Using nonstigmatizing approaches to clinical treatment that honor unique individual qualities of each child and family
- Using motivational interviewing that addresses nutrition, physical activity, and health behavior change using evidence-based targets for weight reduction and health promotion
- Setting collaborative treatment goals not limited to BMI stabilization or reduction; including goals which reflect improvement or resolution of comorbidities, quality of life, self-image, and other goals related to holistic care
- Integrating weight management components and strategies across appropriate disciplines, which can include intensive health behavior and lifestyle treatment, with pharmacotherapy and metabolic and bariatric surgery if indicated
- Tailoring treatment to the ongoing and changing needs of the individual child or adolescent, and the family and community context

Comprehensive patient history: A comprehensive patient history includes a review of systems; family history; history of present illness;

and appropriate nutritional, physical activity, and psychosocial history.

Family-based treatment: Family-based treatment centers on the role of family at each stage of child development, includes consideration of the family's critical role in supporting child health, and understands the unique contextual elements that affect the patient and family and influence treatment.

Intensive health behavior and lifestyle treatment: IHBLT educates and supports families in nutrition and physical activity changes that improve weight status and comorbidities and promote long-term health. IHBLT is most often effective when it occurs face-to-face, engages the whole family, and delivers at least 26 hours of nutrition, physical activity, and behavior change lessons over 3 to 12 months. IHBLT is foundational to COT and should continue longitudinally. It should be provided in conjunction with pharmacotherapy and metabolic and bariatric surgery if these treatments are indicated. IHBLT may be available in the form of a defined program and may be offered in pediatrician and other PHCP offices, medical centers or health systems, or in partnership with community organizations.

Longitudinal care: Care provided by a group of health care professionals who monitor a patient's weight and other health indicators over a length of time sufficient to be associated with health improvements. Longitudinal care is continuous and coordinated and should include a plan for transition into adulthood.

Overweight and obesity: Overweight is defined as a BMI at or above the 85th percentile and below the 95th percentile for children and teens of the same age and sex. Obesity is defined as a BMI at or above the

95th percentile for children and teens of the same age and sex.

Pediatricians and other pediatric health care providers: For the purpose of this CPG, pediatricians and other pediatric health care providers refers to a qualified primary or tertiary care medical provider operating within their scope of practice and providing clinical care to children and adolescents. Examples include physicians, nurse practitioners, and physician assistants. (This document also refers to dietitians, licensed psychologists, exercise specialists, and other health care professionals who are not practicing medicine in the same manner.)

Pediatric medical home: The "pediatric medical home" delivers accessible, continuous, comprehensive, patient- and family-centered, coordinated, compassionate, and culturally effective health care. In this venue, well-trained pediatric physicians known to the child and family deliver or direct primary medical care.⁸¹

Pediatric obesity specialist or clinician with expertise: Pediatric obesity specialists and clinicians with expertise are health care professionals with additional training in pediatric obesity medicine. Training may take the form of certification programs specific to obesity, fellowship, or a focus during specialty training, such as within endocrinology or gastroenterology specialty training. It may also take the form of an informal apprenticeship combined with professional workshops. For the purposes of this document, such training occurs within the context of recognized health care professional organizations.

Person-first language: According to the CDC, person-first language emphasizes the individual, not their disabilities.⁸² Hence, this CPG describes

"children with obesity" or "adolescents with overweight," not "obese children" and/or "overweight adolescents."

Severe obesity: The expanded definition of "severe obesity" includes Class 2 and Class 3 obesity.⁸³

- Class 2 obesity ($\geq 120\%$ to $< 140\%$ of the 95th percentile) or a BMI $\geq 35 \text{ kg/m}^2$ to $< 39 \text{ kg/m}^2$, whichever is lower based on age and sex
- Class 3 obesity ($\geq 140\%$ of the 95th percentile) or BMI $\geq 40 \text{ kg/m}^2$, whichever is lower based on age and sex

Social determinants of health (SDoHs): SDoHs are the conditions in the environments where people are born, live, learn, work, play, worship, and age that affect a wide range of health, functioning, and quality-of-life outcomes and risks. SDoHs can be grouped into 5 domains: economic stability, education access and quality, neighborhood and built environment, and social and community context.^{8,84,85}

Treatment intensification: Treatment intensification occurs through increased frequency of contact, increased length of treatment, or other means of increasing the dose of treatment. Treatment intensification could include additional health care professionals and/or additional methods, such as physical therapy, psychotherapy, medical nutrition therapy, pharmacotherapy, or metabolic and bariatric surgery.

IV. METHODOLOGY

A. Subcommittee Process and Support

In 2017, the CDC supported the AAP's Institute for Healthy Child

Weight (the Institute) to conduct an evidence review of obesity treatment and obesity-related comorbidities. The Institute identified a methodologist and convened an evidence review committee consisting of pediatricians and researchers with expertise in pediatric obesity etiologies, diagnosis, and management. This committee, which met regularly in 2018 to 2019, followed established methods (elaborated on below) to create 2 TRs, which capture the evidence review committee's findings and detail the search criteria, systematic review process, and research history. One TR is on overweight and obesity treatment (<https://doi.org/10.1542/peds.2022-060643>) and the second is on overweight and obesity comorbidities (<https://doi.org/10.1542/peds.2022-060643>).

Staff from the Institute and the AAP's Council on Quality Improvement and Patient Safety formed a CPG writing Subcommittee, comprising the methodologist and several evidence review committee members; a range of pediatric primary and tertiary care providers; behavioral health, nutrition, and public health researchers; a pediatric surgeon; medical epidemiologists from the CDC Division of Nutrition, Physical Activity and Obesity; an implementation scientist; a parent representative; and a representative from the AAP Partnership for Policy Implementation. Most Subcommittee members also have other national organization affiliations relevant to pediatric overweight and obesity.

The Subcommittee members were identified by the AAP and met regularly in 2019 and 2020 and virtually thereafter. Members were assigned sections and met virtually to complete their sections. Sections were reviewed by the chair or

cochair and outstanding issues were resolved by group consensus. The parent member was an at-large member of all the writing groups and reviewed each section. Members' potential conflicts of interest were identified and considered; no conflicts prevented Subcommittee members from participating in the CPG development process.

B. Scope of the Review

This review was designed to answer 2 overarching key questions (KQs). KQ1 was: "What are effective clinically based treatments for pediatric obesity?" KQ2 was: "What is the risk of comorbidities among children with obesity?"

The Subcommittee developed this focus based on the needs of pediatricians and other PHCPs and the evidence required to inform the future development of clinical practice guidelines. The review did not attempt to address treatment strategies for comorbidities (eg, hypertension [HTN], sleep apnea, type 2 diabetes mellitus [T2DM]), as other guidelines and reviews are available to guide such treatment.⁸⁶⁻⁹⁰

B.1. Rationale for KQ1: Intervention Studies

Pediatricians and other PHCPs are a trusted source of health information for parents, including on issues related to nutrition and physical activity, which are key components of obesity prevention and treatment. To meet this need, pediatricians and other PHCPs need to know the strategies that have high-quality evidence for effectiveness in preventing and treating obesity. Additionally, pediatricians and other PHCPs need guidance on which treatments are effective for their population and how to leverage available resources for obesity treatment efforts.

B.2. Rationale for KQ2: Comorbidity Studies

Previous recommendations have included assessments of comorbidities, including HTN, dyslipidemia, glucose, fatty liver disease, and others. It is not clear whether these assessments lead to improved treatment strategies or outcomes. Additionally, it is not clear whether conducting these assessments would result in an adverse outcome. We examined specific conditions that were previously recommended or that would reasonably require screening: dyslipidemia, HTN, diabetes, fatty liver disease, depression, sleep apnea, and asthma. This is not intended to be a comprehensive list of all conditions comorbid with obesity but represents those most common and for which screening is potentially helpful.

C. Search Strategy

The Evidence Review Subcommittee searched PubMed and CENTRAL (for trials). The initial search was on April 6, 2018, and an additional search was conducted to update the review, covering the time period April 7, 2018 through February 15, 2020. Both searches followed the same procedures, which are described below.

The Subcommittee combined the searches for both KQ1 and KQ2 because of their significant overlap to more efficiently review studies. Because the focus was on interventions that are relevant to primary care, the Subcommittee did not search other discipline-specific databases, such as ERIC or PsycInfo.

The Subcommittee searched for studies of children or adolescents, with a focus on overweight, obesity, or weight status; involving pediatricians, other pediatric health care providers, health care, or other treatment or screening (KQ1); and

examining common comorbidities (KQ2). For both questions, the Subcommittee limited only using key words, not filters, to ensure the inclusion of the newest studies that had not yet been fully indexed. No date limits were placed on searches. In practice, this meant the Subcommittee reviewed studies from 1950 to 2020, although fewer than 2% of the studies were from before 1980.

The complete search strategies are included in Appendix 2 of the accompanying TRs (<https://doi.org/10.1542/peds.2022-060642> and <https://doi.org/10.1542/peds.2022-060643>).

D. Inclusion Criteria

D.1. Inclusion Criteria Common to All Studies

All studies were required to include children ages 2 to 18 years. Studies could also include young adults up to age 25, if this population was stratified from older adult participants, as long as children younger than 18 years were also included in the study. Children could have other conditions (eg, asthma), as long as these conditions were not known to cause obesity, or be taking medications (eg, steroids) other than those known to be significantly obesogenic. Conditions known to cause obesity, such as Prader-Willi syndrome, obesogenic medication (eg, antipsychotics), or known genetic mutations associated with obesity (eg, melanocortin 4 receptor [MC4R]) were excluded.

All studies had to originate from Organization for Economic Cooperation and Development member countries and be available in English.

The race distribution of the samples is reported in the accompanying technical report evidence overview (Appendix 5 in TRs [<https://doi.org/10.1542/peds.2022-060642> and

<https://doi.org/10.1542/peds.2022-060643>]) to assist in interpretation of evidence within a health equity framework. The technical report authors notated in the “special populations” section of Appendix 5 when each study specifically focused on a lower-resourced population, as well as race and ethnicity distributions for all studies.

D.2. Inclusion Criteria for KQ1 (Intervention Studies)

The primary aim of the intervention studies had to be examination of an obesity prevention (intended for children of any weight status) or treatment (intended for children with overweight or obesity) intervention. The primary intended outcome had to be obesity, broadly defined, and not an obesity comorbidity. Studies of obesity interventions that reported other outcomes were included.

Interventions could involve any approach, including screening, counseling, medically managed weight loss, pharmaceutical treatment, or surgery. Regardless of the intervention components, there had to be some level of outpatient clinical involvement in the treatment (ie, not just referral to an outside program), such as screening or a clinic follow-up appointment. Interventions that occurred completely outside the scope of health care were excluded. For example, school-based obesity prevention programs or community-based activity programs with no pediatrician or other pediatric health care provider involvement were excluded.

The Subcommittee did not limit the search by study design but did report experimental and nonexperimental studies separately. Although nonexperimental designs were included, all studies had to have a relevant comparison group to

be included in the TR on interventions (<https://doi.org/10.1542/peds.2022-060642>).

D.3. Inclusion Criteria for KQ2 (Comorbidity Studies)

The Subcommittee included studies that had a primary aim of comparing comorbidities among those with and without obesity or by severity of obesity. Obesity and the comorbidity had to be measured contemporaneously to reflect the practice of clinical screening. Obesity had to be categorized using a BMI-based measure into accepted categories (ie, healthy weight, overweight, class 1 obesity, class 2 obesity, class 3 obesity). These categories could be based on percentiles or z-scores and could use the distributions relevant to the studied population (eg, World Health Organization [WHO] or the CDC).^{91,92}

All studies had to include 1 or more of the following comorbidities: lipids, blood pressure (BP), HTN, liver function, glucose metabolism, obstructive sleep apnea (OSA), asthma, or depression. These were chosen based on known associations with weight and potential for screening in the primary care setting.

The complete inclusion criteria are included in Appendix 3 of the accompanying TR on comorbidities (<https://doi.org/10.1542/peds.2022-060643>).

E. Review Process

The Subcommittee used Covidence to manage the review process (<https://www.covidence.org/>). Covidence is a program for online collaboration and management of systematic reviews. All abstracts were reviewed by 2 independent reviewers on the Subcommittee, who assessed the study's inclusion in the full-text

Treatment Experience of Obesity as a Chronic Disease

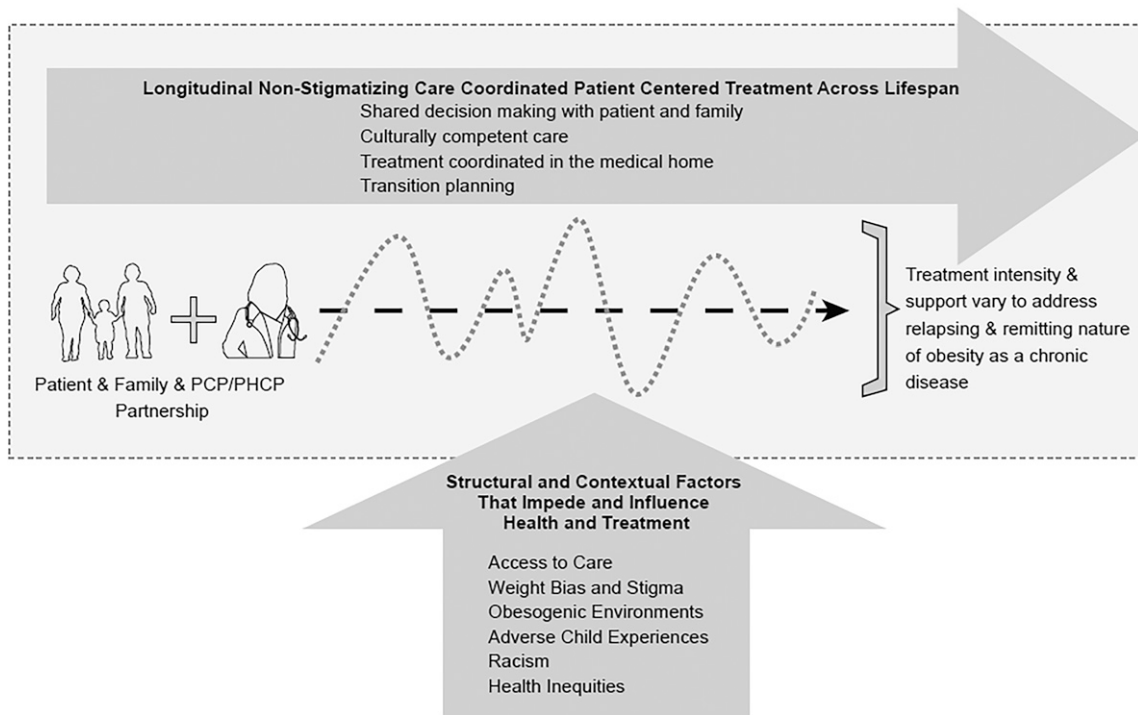


FIGURE 1

Treatment experience of obesity as a chronic disease (this figure illustrates how longitudinal care is important to help address this chronicity and to address and buffer the social and contextual factors that influence a person's health).

review process. All conflicts were discussed and resolved. Articles excluded at this stage were assigned an exclusion reason, with a hierarchy, which is shown in Appendix 4 of the accompanying TRs (<https://doi.org/10.1542/peds.2022-060642> and <https://doi.org/10.1542/peds.2022-060643>).

F. Data Extraction and Quality Assessment

All articles deemed to meet criteria for full text inclusion were categorized into different data extraction strategies. Randomized trials were given a quality assessment using the Cochrane Risk of Bias tool. The Subcommittee decided not to limit studies based on the study quality, because many of them did not reach “high-quality” status (ie, at low risk of bias for most or all domains in the Cochrane Risk of Bias Assessment) using any of the

tools. This occurred largely because studies consisted primarily of behavioral interventions without the possibility of blinding.

All studies, regardless of group, were fully extracted by 2 reviewers, and conflicts were discussed and resolved. Intervention studies were categorized into 5 groups for data extraction.

F.1. Group 1 Extraction

Group 1 articles included randomized trials of diet or “lifestyle” interventions. Extraction of these articles included: sponsorship or funder, design, population information, provider type, detailed intervention strategies and intensity, and BMI-based outcomes. The Subcommittee also identified outcomes other than BMI, including lipids, glucose metabolism, BP, other laboratory values, other obesity measures,

psychosocial outcomes, mental health, behaviors, and other outcomes (primarily parent BMI and child cardiovascular fitness).

The Subcommittee categorized the intensity of interventions in a manner consistent with the US Preventive Services Task Force (USPSTF) to allow for comparisons with its findings, into interventions with a dose (number of hours) of <5 hours; 5 to 25 hours; 26 to 51 hours; and 52 or more hours. All interventions occurred over less than 1 year. The Subcommittee conducted quality assessment for group 1 articles.

F.2. Group 2 Extraction

Group 2 articles included randomized controlled trials of pharmaceutical treatments. Similar information as above was extracted, using a brief description of the

treatment and no categorization of intensity. These articles also received a quality assessment.

F.3. Groups 3 Through 5 Extraction

i) Groups 3 Through 5 Articles Group 3 articles included nonrandomized comparative studies of diet and “lifestyle” treatments, group 4 articles included nonrandomized comparative studies of pharmaceutical treatment, and group 5 articles included any surgical studies.

Because of small numbers, the Subcommittee combined randomized and nonrandomized surgical studies. Brief treatment descriptions and BMI-related outcome data were extracted from these, but the Cochrane Risk of Bias tool was not used because these were observational designs.

F.4. KQ2 Extraction (Comorbidity Studies)

All studies were extracted by 2 reviewers who reported prevalence of comorbidities or mean values of laboratory parameters by weight classification. The Subcommittee included healthy weight, overweight, class 1 obesity, class 2 obesity, and class 3 obesity.

Because all classes of obesity severity were not always reported in the studies, these classes may include higher groups. For example, reporting of ≥95th percentile would only be considered class 1 obesity, although children at higher levels may be included. (See the TR for a detailed description of the KQ1 extraction procedures.)

G. Data Synthesis and Analysis

The Subcommittee’s primary method of data synthesis was narrative. To allow broad inclusion, the Subcommittee did not limit to specific designs or measures that would facilitate meta-analysis. The Subcommittee has reported on studies in each group based on their

Aggregate Evidence Quality	Benefit or Harm Predominates	Benefit and Harm Balanced
Level A Intervention: well designed and conducted trials, meta-analyses on applicable populations Diagnosis: independent gold standard studies of applicable populations	Strong recommendation	Weak recommendation (based on balance of benefit and harm)
Level B Trials or diagnostic studies within minor limitations; consistent findings in from multiple observational studies	Moderate recommendation	
Level C Single or few observational studies or multiple studies with inconsistent findings or major limitations	Weak recommendation (based on low quality evidence)	No recommendation may be made
Level D Expert opinion, case reports, reasoning from first principles		
Level X Exceptional situations in which validating studies cannot be performed, and there is a clear preponderance of benefit or harm	Strong recommendation Moderate recommendation	

FIGURE 2
Grading matrix.

type and design and has reported findings for outcomes other than BMI.

The AAP policy statement, “Classifying Recommendations for Clinical Practice Guidelines,” was followed in designating aggregate evidence quality levels for the available evidence (Fig 2).⁹³ The AAP policy statement is consistent with the grading recommendations advanced by the University of Oxford Centre for Evidence-Based Medicine.

Evidence grades were determined based on the grading matrix in Fig 2. Although we included both trials and observational studies in the technical reports, they are reviewed separately. Study design was considered in the aggregate evidence quality grades, as indicated by the matrix. We did not explicitly use risk of bias scores, but this information was available and used in the Subcommittee’s final assessment.

The Subcommittee reached consensus on the evidence, which was then used to develop the clinical practice guideline’s KASs. When the scientific evidence was at least “good” in quality and demonstrated a preponderance of benefits over harms, the KAS provides a “strong recommendation” or “recommendation.” Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present; clinicians are prudent to follow a recommendation but are advised to remain alert to new information and be sensitive to patient preferences (Fig 2).

Integrating evidence quality appraisal with an assessment of the anticipated balance between benefits and harms leads to a designation of a strong recommendation, recommendation, option, or no recommendation. Once the evidence level was determined, an evidence grade was assigned. AAP policy stipulates that the evidence

supporting each KAS be prospectively identified, appraised, and summarized, and an explicit link between quality levels and the grade of recommendation must be defined.

Possible grades of recommendations range from “A” to “D,” with “A” being the highest:

- Grade A: consistent level A studies;
- Grade B: consistent level B or extrapolations from level A studies;
- Grade C: level C studies or extrapolations from level B or level C studies;
- Grade D: level D evidence or troublingly inconsistent or inconclusive studies of any level; and
- Level X: not an explicit level of evidence as outlined by the Centre for Evidence-Based Medicine. This level is reserved for interventions that are unethical or impossible to test in a controlled or scientific fashion and for which the preponderance of benefit or harm is overwhelming, precluding rigorous investigation.

When it was not possible to identify sufficient evidence, recommendations are based on the consensus opinion of the Subcommittee members.

VI. EPIDEMIOLOGY OF CHILDHOOD AND ADOLESCENT OBESITY

A. Prevalence of Childhood Obesity

Obesity is a common, complex, and often persistent chronic disease associated with serious health and social consequences.⁹⁴ Childhood obesity is typically defined as having a BMI of ≥ 95 th percentile for age and sex.⁹⁵ Severe obesity is defined as BMI $\geq 120\%$ of the 95th percentile for age and sex. The percentage of US children and adolescents affected by obesity has more than tripled from 5% in 1963 to 19% in 2017 to 2018.² In 2017 to 2018, the rise in obesity prevalence slowed in children younger than 6 years of age, but

increases continued among certain populations, including adolescents and non-Hispanic Black and Mexican American youth.⁴ A predictive epidemiologic model estimates that if 2017 obesity trends hold, 57% of children aged 2 to 19 years will have obesity by the time they are 35 years of age, in 2050.³⁶

Obesity prevalence increases with increasing age.³ For example, in 2015 to 2016, the prevalence of obesity in children aged 2 to 5 years, 6 to 11 years, and 12 to 19 years was 13.9%, 18.4%, and 20.6%, respectively.³ Among children younger than 6 years, there were no significant trends in obesity from 1999 to 2018 for those 2 through 5 years of age.⁴ For children 6 through 11 years of age, significant trends in obesity show an increased prevalence from 15.8% in 1999 to 2002 to 19.3% in 2015 to 2018.⁴ Similarly, among adolescents 12 through 19 years, trends show increased obesity in the same time period from 16.0% to 20.9%.⁴ The proportion of children and youth 2 to 19 years of age with severe obesity increased from 4.9% in 1999 to 2000 to 7.9% in 2015 to 2016.^{4,96} The prevalence of severe obesity in youth 12 to 19 years of age in 2015 to 2018 was 7.6%.⁴

The COVID-19 pandemic has significantly affected the lives and routines of children and adolescents. In 1 analysis, the pandemic period was associated with a doubling in the rate of BMI increase compared with the prepandemic period.⁹⁷ Obesity prevention and management efforts should routinely include health care provider screening for BMI, food security, and social determinants of health and increased access to evidence-based pediatric weight management programs and food assistance resources to mitigate such effects in the future.⁹⁷

Disparities exist among children and youth with obesity, including, but not limited to, lower level of parental education, lower income, less access to healthier food options and safe and affordable physical activity opportunities, and higher incidence of ACEs.^{70,98,99} For example, among 5345 children 6 to 9 years of age, those whose parents had lower levels of education had a greater odds of having obesity compared with children whose parents had higher levels of education (odds ratio: 1.78; 95% confidence interval [CI]: 1.36 to 2.32).¹⁰⁰ A cross-sectional analysis of 111 799 children in Massachusetts at the school district level showed that for every 1 percentage point increase in the proportion of children with low SES, there was a 1.17 percentage point increase in the prevalence of obesity.¹⁰¹ Furthermore, children with disabilities, including those with intellectual disabilities, are at higher risk for developing obesity than their peers without disabilities.¹⁰²

Finally, among 43 864 children and adolescents aged 10 to 17 years old, the presence of 2 or more early ACEs was associated with an increased odds of obesity later in childhood and adolescence (odds ratio: 1.21; 95% CI: 1.02 to 1.44).¹⁰³ Together, these disparities highlight the burden of obesity in children from families of lower SES and the need to provide strategies to minimize these inequities.

Disparities also exist in obesity prevalence across ethnic and racial groups. In 2015 to 2018, non-Hispanic Black children and Mexican American youth 6 to 11 years of age had a higher prevalence of obesity compared with non-Hispanic white children (22.7% and 28.2% vs 15.5%, respectively).⁴ An analysis of the Indian Health Services National Data Warehouse showed that in 2015, the prevalence of overweight

and obesity in American Indian and Alaska Native (AI/AN) children and adolescents was 18.5% and 29.7%, respectively.¹⁰⁴

Among children 2 to 5 years of age from lower-income families enrolled in the Special Supplemental Nutrition Program for Women, Infants, and Children program, recent analyses indicate a modest but significant decline in obesity prevalence from 2010 (15.9%) to 2018 (14.4%).¹⁰⁶ Among these children, obesity prevalence ranged across states from 8.5% to 20.2%; disparities persisted by race and ethnicity despite changes in prevalence over time.¹⁰⁵

In addition, children and youth with special health care needs (CYSHCN) have a higher prevalence of obesity and lower levels of physical activity compared with children having typical growth and development.^{106–108} Among CYSHCN, a meta-analysis of studies of adolescents with intellectual disabilities found a pooled odds ratio of obesity of 1.80 compared with adolescents with typical development.¹⁰²

B. Impact of Obesity in Childhood

Children with obesity commonly become adolescents and adults with

obesity; severe obesity during adolescence increases the risk for severe obesity during young adulthood.^{109,110} BMI levels strongly track throughout childhood and adolescence and are predictive of high adult BMI.¹¹⁰

Obesity puts children and adolescents at risk for serious short- and long-term adverse health outcomes later in life, including cardiovascular disease, including HTN; dyslipidemia; insulin resistance; T2DM; and nonalcoholic fatty liver disease (NAFLD).^{38,96,111–113} Additionally, prediabetes in youth with obesity, compared with youth with normal weight, has been associated with elevated systolic blood pressure and low-density lipoprotein, and lower insulin sensitivity.¹¹⁴

In addition to physical and metabolic consequences, obesity in childhood and adolescence is associated with poor psychological and emotional health, increased stress, depressive symptoms, and low self-esteem.¹¹⁵ Several studies have determined that children of some racial and ethnic groups have a greater prevalence of comorbidities associated with childhood obesity, including HTN, T2DM, hypercholesterolemia, and depression, compared with non-Hispanic white children.^{116–120}

Obesity in childhood and adolescence is associated with health care utilization and costs. For example, the most common primary conditions that cooccur with a secondary diagnosis of obesity and may increase costs and utilization include pregnancy, mood disorders, asthma, and diabetes.¹²¹ A modeling study has estimated that the total lifetime medical costs for 10-year-olds with lifelong obesity to be in the range of \$9.4 to \$14 billion for that cohort alone.¹²²

Tracking obesity across the lifespan underscores the importance of primary and secondary prevention and treatment efforts early in life. These efforts include evaluating for obesity using BMI; identifying children at high risk and adolescents; providing or referring to evidence-based obesity treatments for children, youth, and their families; and addressing SDOHs.

VII. DIAGNOSIS AND MEASUREMENT

Although KAS 1 was not explicitly studied and referenced by the TR, most of the TR studies implicitly included measurement of height and weight and calculation and plotting of BMI as part of the study procedures. Thus, the concept of appropriate measurement, calculation, charting, and tracking is

KAS 1. Pediatricians and other PHCPs should measure height and wt, calculate BMI, and assess BMI percentile using age- and sex-specific CDC growth charts or growth charts for children with severe obesity at least annually for all children 2 to 18 y of age to screen for overweight (BMI ≥ 85th percentile to <95th percentile), obesity (BMI ≥ 95th percentile), and severe obesity (BMI ≥ 120% of the 95th percentile for age and sex).

Aggregate Evidence Quality	Grade B
Benefits	Easy to use; reproducible; improved identification of severe obesity; and improved ability to monitor improvements in weight status of youth with severe obesity.
Risks, harms, costs	Screening tool with both false negatives and false positives; may misclassify some populations; development, implementation, and use of separate growth charts for severe obesity requires identification of severe obesity (electronic health record decision prompts can support this); use of severe obesity growth charts may confer stigma associated with obesity; interpretation and explanation for families may be challenging.
Benefit-harm assessment	Benefit outweighs harm.
Intentional vagueness	Growth charts based on reference populations; need to screen or assess adiposity for children among specific populations (eg, CYSHCN); provider education on use and interpretation of charts; training requirements.
Role of patient preference	Patient and family inclusion and discussion is critical to shared decision-making.
Exclusions	>21 y
Strength	Moderate
Key references	79, 123–130

implicit in research-based evidence included in the TR (eg, references^{123,124,126,130}).

A. Use of BMI as a Screening and Diagnosis Tool

The gold standard measurement of body composition—dual-energy x-ray absorptiometry—to identify, locate, and quantify body fat, and can be expensive and difficult to implement. In clinical practice, BMI is frequently used as both a screening and diagnostic tool for detecting excess body fat because it is easy to use and inexpensive. BMI is a validated proxy measure of underlying adiposity that is replicable and can track weight status in children and adolescents.^{38,131,132} Because of its ease of use, BMI is also frequently used to follow a child or adolescent's weight trajectory over time. The CDC BMI growth curves are frequently used to visualize BMI trajectory over time. Furthermore, BMI is often used to evaluate the success or impact of interventions to improve weight status.

For most individuals, BMI is generally well-correlated with direct measures of body fat, including skinfold thickness measurements, bioelectrical impedance, densitometry, and dual-energy x-ray absorptiometry.^{131,133–139}

BMI has limitations, however, including high specificity and low sensitivity for detecting excess adiposity.¹³² BMI does not directly measure body composition and fat content and may under- or overdetect excess adiposity in certain racial and ethnic groups.^{140,141} Finally, children and adolescents who have high fat-free mass may have a high BMI and, as a result, be incorrectly classified as having overweight or obesity.¹⁴²

The CDC's 2000 Growth Charts are based on NHANES data from the 1960s through the early 1990s and include age- and sex-specific BMI-

for-age charts.¹⁴³ The CDC Growth Charts provide a historical comparison of children's weight status relative to a time before the current obesity epidemic during that healthier growth patterns predominated; thus, percentiles on the Growth Charts do not equate to the current population distribution of BMI. The CDC Growth Charts are recommended for clinically tracking BMI patterns among US children and adolescents aged 2 to 18 years; although the CDC Growth Charts can be used for adolescents aged 19 to 21 years, in practice, most pediatricians and other PHCPs transition to adult BMI calculation and categorization for patients older than 18 years.¹⁴³

"Overweight" is defined as a BMI at or above the 85th percentile and below the 95th percentile for age and sex; "obesity" is defined as a BMI at or above the 95th percentile for age and sex. "Severe obesity" is defined as a BMI equal to or more than 120% above the 95th percentile, which approximates the 99th percentile. The CDC Growth Charts were not intended to track growth of children with extremely high BMI values. Because of limited data on children and adolescents above the 97th percentile in the reference population, higher percentile curves could not be generated. Caution was recommended in extrapolation of percentiles beyond the 97th percentile, as this may generate unusual or unexpected results.¹⁴⁴ In older adolescents, the adult cut-off of a BMI equal to or greater than 30 kg/m² can be used to define obesity if this value is less than the 95th percentile BMI for age and sex.

Conversion of BMI percentiles to z-scores (a statistical measure that describes a value's relationship to a population mean) derived from the CDC Growth Charts have historically been used for assessing

longitudinal change in adiposity over time among children and adolescents with obesity.¹²⁷ The change in z-score, however, may not accurately detect meaningful changes in weight status or comorbidity risk over time, particularly for children and adolescents with severe obesity caused by compression of z-scores corresponding to extremely high BMI values into a very narrow range.¹⁴⁵ Consequently, investigators have proposed and described various alternative options, including using the degree to which, expressed in percentage, a particular BMI percentile was above the 95th percentile, or the median, for age and sex (referred to as percentage above the 95th percentile, or percentage above the median, respectively).

The "extended" method for calculating BMI z-scores and percentiles at extremely high BMI values was developed to address these limitations. This method incorporates data on children and adolescents with obesity from more recent NHANES surveys to better characterize the BMI distribution above the 95th percentile while retaining the 2000 CDC Growth Chart BMI distribution below the 95th percentile.

The CDC and the AAP recommend that weight status in children up to 2 years of age be tracked using the WHO's weight-for-length, age-, and sex-specific charts.^{146,147} Specialized growth charts for children and adolescents with certain conditions, such as trisomy 21, can provide useful growth reference information for special populations. These charts may, however, be limited, for example, by the small sample sizes used in developing them, which may not be representative of all children and youth with trisomy 21.¹⁴⁸

B. The Clinical Utility of BMI

BMI is a useful evaluation measure to clinically identify children with overweight and obesity for appropriate

treatment—such as family-based behavioral therapy—which can lead to improvements in BMI and related comorbidities.^{123,124,127–130,149–152}

Following comprehensive systematic reviews, the USPSTF issued a Grade B recommendation that pediatricians and other PHCPs screen children and adolescents aged 6 years or older annually for obesity—defined by BMI percentile—and offer, or refer children and adolescents to, a comprehensive, intensive, family-based behavioral treatment to improve weight status.⁷⁹ (A “comprehensive, intensive behavioral treatment” was defined as a treatment of 26 hours or more over a period of 2 to 12 months.) (See Evaluation and Treatment sections.)

Furthermore, the AAP’s *Bright Futures* recommendations, which are based on systematic reviews and expert panels, offer prevention guidelines including annual assessment of BMI alongside dietary nutrition and physical activity counseling for children and adolescents starting at 2 years old^{127,153} (Appendix 2).

Appendix 2 describes the USPSTF recommendations, *Bright Futures* recommendations, and the recommendations reflected in this CPG’s KAS 1. All 3 sources recommend annual screening for excess weight using BMI, with the USPSTF beginning at 6 years old and both *Bright Futures* and this CPG beginning at age 2 years. For children or adolescents with a BMI \geq 95th percentile for age and sex, the USPSTF provides recommendations for primary care providers to offer, or refer them to, a comprehensive, family-based weight management intervention. *Bright Futures* recommends that primary care providers screen for excess weight and provide dietary nutrition and physical activity counseling for all children and adolescents with

either overweight or obesity (BMI \geq 85th and $<$ 95th percentile for age and sex). *Bright Futures* also provides implementation tips and guidance for pediatricians and other PHCPs including, for example, providing counseling using motivational interviewing. *Bright Futures* offers guidance to states by offering a framework for meeting national performance standards under Title V. Finally, *Bright Futures* suggests how communities and families can support healthier lifestyles and prevention. This CPG recommends referral to evidence-based weight management interventions for all children 2 years and older who have a BMI \geq 95th percentile for age and sex (see KAS 1, above).

The practice of annual BMI measurement at well-child visits is recommended and central to the management and tracking of overweight and obesity in children.^{127,153,154} Limitations to this approach include missed opportunities to track and manage weight changes that occur in less than a 12-month period.^{127,153} However, other visit opportunities can be used to assess BMI outside the well-child visit.¹⁵³ This CPG’s KAS on evaluation, based on the evidence described above, and in concordance with USPSTF and the *Bright Futures* recommendations, continues to highlight the critical importance of annual evaluation for excess weight and the provision of, or referral to, evidence-based interventions, as indicated, to promote the health and well-being of all children and adolescents.

C. Communication of BMI and Weight Status to Children and Parents

Despite its limitations, BMI is currently the most appropriate

clinical tool to screen for excess adiposity and make the clinical diagnosis of overweight or obesity. Thus, the BMI must be communicated to the patient and family, as it guides next steps for comprehensive evaluation and treatment of obesity and related comorbidities. Weight-related discussions can be uncomfortable for clinicians who want to avoid stigmatizing children because of their shape or size. Avoiding this discussion may, however, cause delays or barriers to patients receiving evidence-based care. In addition, obesity stigma can result in patient avoidance of health care and disruption of clinician-patient relationships. There is evidence that having conversations about obesity can facilitate effective treatment.^{155–157}

Three key factors can facilitate a nonstigmatizing conversation about weight with patients and families:

1. Ask permission to discuss the patient’s BMI and/or weight.
2. Avoid labeling by using person-first language (“Child with obesity”; not “obese child” or “my patient is affected by obesity; not “my patient is obese”).¹⁵⁸
3. Use words that are perceived as neutral by parents, adolescents, and children. In several studies inclusive of diverse racial, ethnic, and rural and urban populations, preferred words include: “unhealthy weight, gaining too much weight for age, height, or health, *demasiado peso para su salud* (too much weight for his or her health).” Words perceived as most offensive include: “obese, morbidly obese, large, fat, overweight, chubby, or *sobrepeso* (overweight).”¹⁵⁶

Recognize that discussing BMI with children, adolescents, and families, even when using nonstigmatizing language and preferred terms, can

elicit strong emotional responses including sadness or anger. Acknowledging and validating those responses, while keeping the focus on the child's health, can help to strengthen the relationship between the pediatrician or other PHCP and patient and family to support ongoing care.¹⁵⁹

VIII. RISK FACTORS FOR CHILD AND ADOLESCENT OVERWEIGHT AND OBESITY

Obesity is a chronic disease that has a multifactorial etiology. Risk factors for overweight and obesity—many of which are SDoHs—include broader policies and systems factors; institutional or organizational (ie, school); neighborhood and community; and family, socioeconomic, environmental, ecological, genetic, and biological factors (Table 1).^{21,160} These individual, social, and contextual risk factors often overlap and/or influence one another and can operate longitudinally throughout childhood and adolescence, initiating weight gain and escalating existing obesity. Children and their families interact with their environment at all of these levels and have a unique and “insider’s” point of view that needs to be understood in delivering culturally sensitive care.¹⁶¹

Pediatricians and other PHCPs need to be aware of the risk factors for pediatric obesity to provide early anticipatory guidance for obesity prevention, monitor their patients closely, and intervene early when weight trajectory increases.

Consensus Recommendation

The CPG authors recommend pediatricians and other pediatric health care providers:

- perform initial and longitudinal assessment of individual, structural, and contextual risk factors to provide individualized and tailored

treatment of the child or adolescent with overweight or obesity.

A. Policy Factors

The larger macroenvironment—including societal attitudes and beliefs, government policies, food industry practices, and the educational and health care systems—can influence obesity risk.¹⁶² It is difficult to make or sustain healthy behavior changes in an obesogenic environment that promotes high-energy intake, unhealthy dietary choices, and sedentary behavior.

A.1. Marketing of Unhealthy Foods

Marketing of unhealthy food and beverages directed at children tends to negatively impact their dietary choices and behaviors.^{163–166} Foods and beverages embedded in entertainment media have been shown to influence eating behavior choices of children and also increase consumption of foods during or after exposure to the embedded foods.¹⁶⁷

A systematic review and meta-analysis showed that even short exposure to unhealthy food and beverage marketing targeted to children resulted in increased dietary intake and behavior during and after the exposure.¹⁶⁸ Both younger children and male children (sex assigned at birth) tend to be more susceptible to the food and beverage marketing,¹⁶⁸ and because of their stage of cognitive development, younger children are more likely to be susceptible to advertising and interpret it as factual.^{169,170}

Currently, marketing to children targets highly palatable relatively inexpensive energy-dense foods and beverages.¹⁶⁶ This marketing occurs via television, websites, online games, at supermarkets, and outside schools.¹⁶⁶ Children are, unfortunately, frequently exposed

to foods of low nutritional values from advertisements; therefore, it is not surprising that they have preferential increase in consumption of foods of low nutritional value.¹⁶⁷

A.2. Underresourced Communities

Underresourced communities are settings in which obesity risk factors can predominate over health-promoting factors. Children and families in these settings may be unable to access fresh fruits and vegetables and safe physical activity spaces and may suffer from food insecurity.^{170–172} Limitations in transportation, cost, affordability, and availability may reduce access to health care and obesity treatment. Families may be struggling with poverty, access to healthy foods, lack of social supports, racism, and/or immigration status. Understanding these contextual factors that impact each child and family is crucial in being able to provide compassionate and effective obesity treatment.

A.2.a. Socioeconomic Status

Obesity has been shown to disproportionately affect children and adolescents who have low SES.^{173–175} Even though the prevalence of obesity has been stabilizing among US children overall, the rates continue to increase among children with low SES.^{173,176} According to the Children’s Defense Fund, the poverty rate among US children is alarmingly high.¹⁷⁷

A longitudinal analysis of predominantly non-Hispanic white children in the United States found that low SES before 2 years of age was associated with higher obesity risk by adolescence in both boys and girls; this analysis also indicated that the effect of early poverty endures later in life.¹⁷⁸ Similarly, another study found that low SES in

early childhood had a long-term impact on overweight and obesity.¹⁷⁹ This study found that the risk of experiencing overweight or obesity in adulthood was not altered by either upward or downward mobility of poverty after early childhood,¹⁷⁹ indicating the long-term effect of poverty-related stress in early childhood.

Low SES may also be associated with higher risk for obesity by increasing the child's experience with toxic stress. In addition, poverty may limit access to healthy foods and opportunities for physical activity.¹⁸⁰⁻¹⁸² Another study of a large dataset of children followed longitudinally from 9 months of age to kindergarten entry showed that SES played a major role in BMI z-score gaps in Hispanic children, whereas rapidity of weight gain in the first 9 months "accounted for much of the disparity between white children and children" of other races and ethnicities (other than Hispanic children).¹⁸⁰

A.2.b. Children in Families That Have Immigrated

For decades, researchers have believed that despite poverty and other negative SES factors, recently arrived immigrants are healthier than their US-born counterparts. Recent studies, however, have examined large datasets in novel ways and now call this idea into question when it comes to children in families that have immigrated.¹⁸³

As families who have immigrated try to adjust to a new culture, they may adopt Americanized foodways, which are high in fat, sugar, and salt. This tendency could be heightened by children's exposure to media advertising these foods and high-energy snacks and by reduced physical activity.^{184,185}

Patterns of childhood overweight and obesity among families that have immigrated vary substantially by both ethnicity and generational status. Immigrants to the United States generally originate from countries that have a lower prevalence of obesity, but as families acculturate to US eating and activity patterns, rates of obesity may increase. One study found that second-generation Hispanic immigrants were 55% more likely to have obesity than nonimmigrant white children, whereas first-generation Asian immigrants had a 63% lower risk of having obesity.¹⁸⁰

Several studies have indicated different patterns of developing obesity in Mexican-origin populations among adults and children. Obesity among adults of Mexican origin in the United States has been associated with longer stays in the United States and with being born in the United States versus Mexico, which are 2 proxies for acculturation. This pattern differs in children, in whom "significantly higher obesity prevalence has been observed for first-generation young adult males (ages 18-24) and adolescent females (ages 12-17)."¹⁸⁶

In addition, in some cultures, larger body sizes may be preferred as an indication of health and wealth.¹⁸⁷ This cultural factor may make it more difficult for parents to understand the gravity of their children's overweight or obesity. For this and many other reasons, it is vital to ensure that children and families who have immigrated and who are native-born have access to culturally competent health care.¹⁸⁸

A.3. Food Insecurity

The literature positing an association between food insecurity and overweight and

obesity in children has been inconsistent when looking at general child populations. Children living in households with food insecurity have been found, however, to have higher BMI z-scores and waist circumference measurement and a greater likelihood of having overweight or obesity.¹⁸⁹ The correlation between food insecurity and obesity has been found to be higher in adolescents, who may have had more exposure to food insecurity over their life-course.¹⁹⁰ Female children appear to more at risk for obesity in food-insecure environments, compared with male children.¹⁹¹

Food insecurity is highly associated with poverty, and the cost of fruits and vegetables¹⁹² and fast food have been found to influence consumption in low-income families^{193,194} and to be positively related to overweight in children.¹⁹⁵ Associations between consuming more sugar from sugar-sweetened beverages, and less frequency of eating breakfast and eating dinner with family have also been noted in families with food insecurity.^{190,196} Family dynamics around feeding may change in situations of food insecurity and include pressure to eat as well as monitoring and restrictive eating practices.^{191,197} Experiences of food insecurity are stressful for children and families and may add to the burden of chronic stress, which can result in altered eating patterns in the direction of either restricting intake or increasing consumption of energy-dense foods.^{190,196}

The AAP and Food Research and Action Center's toolkit, *Screen and Intervene: A Toolkit for Pediatricians to Address Food Insecurity*, is designed to help pediatricians identify and address childhood food insecurity (available at <https://prac>.

TABLE 1 Selected Examples of Multilevel Influencers and Contributors to Obesity

Example	Description
A. Policy factors	<ul style="list-style-type: none"> ● Marketing of unhealthy foods ● Underresourced communities ● Food insecurity
B. Neighborhood and community factors	<ol style="list-style-type: none"> 1. School environment 2. Lack of fresh food access 3. Fast food proximity 4. Access to safe physical activity 5. Environmental health
C. Family and home environment factors	<ol style="list-style-type: none"> 1. Parenting feeding style 2. Sugar-sweetened beverages 3. Portion sizes 4. Snacking behavior 5. Dining out and family meals 6. Screen time 7. Sedentary behavior 8. Sleep duration 9. Environmental smoke exposure 10. Psychosocial stress 11. Adverse childhood experiences
D. Individual factors	<ol style="list-style-type: none"> D.1. Genetic factors <ol style="list-style-type: none"> a. Monogenetic syndromes and polygenetic effects b. Epigenetic effects D.2. Prenatal risk <ol style="list-style-type: none"> a. Parental obesity b. Maternal weight gain c. Gestational diabetes d. Maternal smoking D.3. Postnatal risk <ol style="list-style-type: none"> a. Birth weight b. Early breastfeeding cessation and formula feeding c. Rapid weight gain during infancy and early childhood d. Early use of antibiotics D.4. Childhood risk <ol style="list-style-type: none"> a. Endocrine disorders b. Children and youth with special health care needs <ol style="list-style-type: none"> 1. Children with autism spectrum disorder 2. Children with developmental and physical disabilities 3. Children with myelomeningocele c. Attention-deficit/hyperactivity disorder d. Weight-promoting appetitive traits e. Medication use (weight-promoting medications) f. Depression

org/aaptoolkit). The Toolkit assists pediatricians and other PHCPs to: (1) better identify children living in households struggling with food insecurity; (2) sensitively address the topic; (3) connect patients and their families to federal nutrition programs and community resources; and (4) advocate for greater

food security and improved overall health of children and their families. The toolkit also includes the “Hunger Vital Sign,” a simple, validated 2-question tool that can be used in the clinical setting to evaluate for food insecurity (see link to toolkit above).

B. Neighborhood and Community Environment Influences or Contributors to Obesity

Environmental factors play an important role in obesity prevalence. Families’ dietary and physical activity opportunities and practices (mentioned above) are influenced by their neighborhoods (Table 1).

B.1. School Environment

Children spend most of their time in school. Therefore, schools play an important role in influencing children’s food choices and physical activity level and, ultimately, their body weight. For example, the presence of fast foods, vending machines, and/or sweetened beverages in schools may negatively influence children’s food choices.¹⁹⁸

Systematic reviews have shown an association between fast food outlets and convenience stores located near schools and obesity in children.^{199,200} When analyzed by subgroups, a positive association has been seen between fast-food outlets and proximity to schools among Hispanic, Black, and white children. Although the association was seen for all grade levels, the effect was larger in younger grades.¹⁹⁹ This review also reported a stronger association between fast-food outlets and grocery stores located near schools and obesity in socioeconomically underresourced neighborhoods.

B.2 Lack of Fresh Food Access

A neighborhood’s food environment has been shown to have mixed association with children’s BMI. Although some studies have shown that a 1.6-km distance or shorter from a home to a supermarket is associated with a lower BMI,²⁰¹ other studies have found that the greater the number of supermarkets located near a child’s home, the higher the child’s BMI.²⁰² Similarly,

a systematic review reported mixed association, with some studies showing a negative association between supermarket accessibility and childhood and adolescent obesity, and other studies either showing a positive effect or no association.²⁰³

Some of the differences were attributed to variations in assessment measures and lack of adjustment for confounding variables. Hence, it is not only the presence of supermarkets that is important, but also other factors that may impact dietary choices—such as the type of foods stocked, pricing, etc. Some, but not all, studies have reported a positive association between neighborhood poverty and childhood and adolescent obesity.²⁰⁴

It has been suggested that lack of access to fresh fruits and vegetables may be a risk factor for childhood and adolescent obesity, as it may lead to an increased reliance on, and consumption of, unhealthy foods. The data for this association have been inconsistent, however. A recent systematic review showed that, although there was a negative association between access to fresh fruits and vegetables and healthy eating behavior, the association between access to fresh fruits and vegetables and overweight and obesity was inconclusive.²⁰⁵

B.3. Presence of Fast-food Restaurants

Fast-food restaurants generally serve relatively low-priced and calorie-rich fast foods with high levels of saturated fat, simple carbohydrates, sugar, and sodium. Because of their easy availability, taste, and marketing strategies, fast foods tend to be popular with children and adolescents.²⁰⁶

Fast-food consumption has been associated with weight gain.²⁰⁷ Some, but not all, studies have shown an

association between access to fast-food restaurants and pediatric obesity.²⁰⁸ A meta-analysis and recent systematic review showed a mixed association between access to fast-food restaurants and weight-related behaviors and weight status in children and adolescents.²⁰⁹ This association was shown to be stronger in populations with lower SES.²⁰⁹

B.4. Access to Safe Physical Activity

A child's environment may influence the amount of physical activity they get. For example, living in an urban environment that lacks safe walkable and/or green spaces in which children can play may result in decreased physical activity levels. Greater exposure to green space has been shown to be associated with higher levels of physical activity and lower risk of obesity.²¹⁰

A recent systematic review of the literature on the influence of the built environment and childhood obesity found significant association between childhood obesity and traffic air pollution and indicators of walkability (which included intersection density and presence and amount of park area in the neighborhood).²¹¹

In addition to green spaces, other aspects of the environment—including safety—are important in these spaces' use. A study of low-income preschool children in New York City reported an association of lower obesity risk in neighborhoods with trees alongside the streets and a positive association between obesity and higher homicide rates in the neighborhood.²¹²

B.5. Environmental Health

Exposure to environmental hazards during the prenatal period, infancy, and childhood can have impacts on the health and well-being of children. Endocrine-disrupting chemicals (EDC) can cross the placental barrier and affect the fetus.²¹³ There are some data that show an association

between prenatal exposure to bisphenol A and polyfluoroalkyl and childhood obesity.^{214,215}

In the postnatal and infancy period exposure may occur through breastfeeding, inhalation, ingestion, or absorption through the skin. Children get exposed to chemicals that are used in household products including cleaning agents, food packaging, pesticides, fabrics, upholstery, etc. Leaching of chemical products (eg, bisphenols, phthalates, parabens, and other EDCs) has been reported in baby feeding bottles, clothing, diaper creams, etc. Exposure to EDCs during early childhood can affect programming of several systems, including endocrine and metabolic systems, which may affect BMI, cardiovascular, and metabolic outcomes later in life.²¹³

C. Family and Home Environment Factors

The family's dietary preferences and lifestyle habits have a crucial role in influencing the child's weight.²¹⁶ Parenting feeding practices and modeling of eating behavior and the type and quantities of foods and beverages in the home have been reported to be important influences in children's appetitive behaviors and food preferences.²¹⁷

C.1. Parenting Feeding Styles

Parenting styles differ and may have an impact on a child's risk for obesity. Four types of parent feeding styles have been described: *authoritative* (responsive and warm with high expectations); *authoritarian* (not responsive but with high expectations); *permissive or indulgent* (responsive and warm but lenient with few rules); and *negligent* (not responsive with few rules). The 4 parenting styles discussed were initially defined by Baumrind (1966) and later expanded by Maccoby and Martin (1983).^{218,219} (Restrictive feeding

was not included as 1 of the parenting styles.)

Authoritative feeding, where parents respond to the child's cues of hunger and satiety, is considered to be protective against excessive weight gain. Children from authoritative parenting homes have been shown to eat more healthy foods, be more physically active, and have healthier BMI, compared with children raised in homes with authoritarian, permissive or indulgent, or negligent parenting styles.^{220,221}

One possible mechanism of parenting style's influence on a child's weight status is thought to be from interference in the child's ability to self-regulate their dietary intake. An authoritarian parent, for example, may not respond to a child's cues for energy intake, resulting in poor ability on the part of the child to self-regulate their own energy intake, and a higher likelihood of overindulging when presented with an opportunity to eat.^{222,223}

A large cross-sectional study showed that, among preschool- and school-aged children, authoritarian or negligent parenting is associated with a higher risk of obesity,²²⁴ whereas authoritative parenting was associated with healthy BMI.²²¹ Among preschoolers, the effect of the parenting feeding style was found to be modulated by poverty, with the effect only being seen among children who were not living in poverty.²²⁴

C.2. Family Home Environment Organization

A systematic review of associations between the organization of the family home environment and childhood obesity found that greater organization of the home environment, which included practices such as having family

routines and setting limits, was inversely associated with obesity.²²⁵ This relationship was present for younger and older children. Most but not all of the 32 studies included in the review controlled for sociodemographic factors.

C.3. Sugar-Sweetened Beverages

A systematic review of 20 prospective cohort studies and randomized controlled trials from 2013 to 2015 found that sugar-sweetened beverages (SSBs) were positively associated with obesity in children in all but 1 study (96%).²²⁶ Based on this review and others demonstrating a link between SSB and multiple other medical and dental diseases, the AAP published a policy statement on SSBs in 2019, calling for broad implementation of policies restricting SSB consumption in children and adolescents.²²⁷

C.4. Portion Sizes

Much of the research on the influence of portion size on children's intake has been performed in laboratory settings providing a single meal to preschool-aged children. A comprehensive review of this research reported that children who serve or are served larger portions of commonly liked energy-dense foods typically consume larger amounts but cautioned that long-term studies of the effects of larger portions over time on a number of variables, including body weight, are lacking.²²⁸

C.5. Snacking Behavior

A recent systematic review of body fat and consumption of ultra-processed foods (defined as snacks, fast foods, junk foods, and convenience foods) in children and adolescents found a positive association but noted that longer-term studies examining the association of these foods and obesity are needed.²²⁹

C.6. Dining Out and Family Meals

Eating outside of the home has been shown to be associated with higher energy intake in both children and adults.^{230,231} In the United States, food eaten outside the home is characterized by higher fat content, larger portions, and greater energy intake.²³⁰

In a systematic review of pediatric and adult studies, eating at fast-food establishments was associated with much higher weight gain, compared with eating at other types of restaurants.²³¹

Take-away food has also been associated with high BMI.²³¹ Hence, eating outside of the home—irrespective of the type of restaurant establishment visited—is associated with higher risk of weight or BMI gain. Conversely, 2 meta-analyses found that increased frequency of eating family meals was associated with lower risk of childhood obesity.^{232,233}

C.7. Screen Time

Some, but not all, studies report an association between screen time duration, childhood adiposity,^{234–236} and adult BMI.²³⁷ Some studies have shown a dose-response effect of screen time and childhood adiposity,²³⁵ with screen time greater than 2 hours per day being positively associated with higher risk of overweight or obesity.²³⁶ A recent meta-analysis reported 42% greater risk of overweight or obesity with more than 2 hours per day of television (TV) compared with 2 or fewer hours.²³⁸

There is evidence to support the association between screen time and consumption of unhealthy diet and high energy intake.²³⁵ The appearance or depiction of food items while engaging in screen time may affect a child's dietary behavior. A systematic review examining food

choice and intake showed that food included in entertainment media affects eating behaviors of children.¹⁶⁷ Children and adolescents are more exposed to food and beverage advertisements when watching TV.²³⁹ Additionally, increase in screen time may displace physical activity and interfere with sleep.^{235,240}

Although “screen time” includes TV, computer, video or videogames, mobile phones, and other digital devices, the majority of the studies published examined the effect of TV viewing.²³⁵ Male children and adolescents tend to spend more time on media screen devices and other Internet technology than female children and adolescents do.²³⁶

C.8. Sedentary Behavior

The association between sedentary behavior and adiposity has been shown to range from small to inconsistent.²⁴¹ Studies examining the effect of sedentary behavior alone on weight using accelerometer measures have shown no association between sedentary behavior and obesity. Teasing out the effects of sedentary behavior alone in treatment studies may be challenging, as this is often confounded with other behaviors such as physical activity, screen time, or increased intake of unhealthy foods.²⁴¹

C.9. Sleep Duration

Short sleep duration is associated with higher risk of obesity in children.^{242–244} A meta-analysis of prospective cohort studies demonstrated a dose-response inverse association between sleep duration and risk of childhood overweight and obesity.²⁴⁴ Children 13 years and younger with short sleep duration (~10 hours) had a 76% increased risk of overweight or obesity compared with their

counterparts with longer sleep duration (12.2 hours).

Sleep restriction may be associated with increased calorie consumption.^{245,246} Additionally, fatigue and decreased physical activity has also been associated with short sleep duration. It is unclear whether the inverse association between sleep and adiposity is causal or a consequence of hormonal or metabolic disturbance.²⁴⁷ Although the exact mechanism for this association is unknown, some of the consequences of short sleep duration include hormonal and metabolic alterations—such as increased ghrelin and decreased leptin—which may lead to increased hunger.²⁴²

C.10. Environmental Smoke Exposure

Children exposed to environmental tobacco smoke (ETS) have been found to have higher BMI compared with their nonexposed counterparts, according to a systematic review of ETS exposure and growth outcomes in children up to 8 years of age.²⁴⁸

C.11. Psychosocial Stress

Psychosocial stress in the prenatal period may have an effect on endocrine function (hypothalamic-pituitary-adrenal axis and glucose–insulin metabolism) in the child’s life course. A meta-analysis showed that prenatal psychological stress was associated with higher risk of childhood and adolescent obesity.²⁴⁹

Psychosocial and emotional issues may lead to weight gain through maladaptive coping mechanisms, including eating in the absence of hunger to suppress negative emotions, appetite up-regulation, low-grade inflammation, decrease in physical activity, increase in sedentary behavior, and sleep disturbance.^{154,250,251} Depression

has been shown to be a risk factor in both pediatric and adult obesity.²⁵¹ The association between depression and obesity could be reciprocal, as obesity may increase depression risk.

C.12. Adverse Childhood Experiences

A number of studies have documented an association between ACEs and the development of overweight and obesity. ACEs impact occurs via toxic stress, which occurs “when a child experiences strong, frequent, and/or prolonged adversity—such as physical or emotional abuse, chronic neglect, caregiver substance abuse or mental illness, exposure to violence, and/or the accumulated burdens of family economic hardship—without adequate adult support.”^{252,253}

ACEs include a history of physical, emotional, or sexual abuse; exposure to domestic violence; household dysfunction from parental divorce or substance abuse; economic insecurity; mental illness; and/or loss of a parent because of death or incarceration.^{69,254–257}

A US study found that cumulative ACEs doubled the risk of children having overweight or obesity, compared with their counterparts with no history of ACEs.²⁵⁸ Unresolved stress and emotional issues may result in maladaptive coping strategies—such as binge eating, eating in the absence of hunger, impulsive eating, and poor sleep hygiene—which may result in further weight gain.

Poverty and associated toxic stresses in utero and early childhood have been suggested to initiate neuroendocrine and/or metabolic adaptations that produce biological phenotypes and obesogenic behaviors that lead to obesity.^{259,260} These effects may persist throughout the lifetime.²⁶⁰

D. Individual-Level Influences or Contributors to Obesity

D.1. Genetic Factors

Heritability studies suggest that there is a 40% to 70% genetic contribution to an individual's obesity risk.^{261–263} Genome-wide association studies have identified 32 loci of significance to obesity predisposition.²⁶⁴ Genetic causes of obesity include both common and rare genetic variants that involve impairment of gene expression or function.²⁶⁴

D.1.a and D.1.b. Monogenic Syndromes and Polygenetic Effects
Polygenetic causes of obesity are by far the most common, and single gene defects are rarer causes of obesity. For example, MC4R, a heterozygous mutation, is the most common form of monogenic obesity and accounts for only 2% to 5% of severe obesity in children.^{265,266} Polygenic inheritance refers to a single inherited phenotypic trait that is controlled by 2 or more different genes. Polygenic variants, on their own, have little effect on an

individual's phenotype. The phenotypic effect manifests only in the presence of, or in combination with, other predisposing factors.

Children with genetic causes of obesity may present with characteristic clinical features that have historically included findings such as short stature, dysmorphic features, developmental delay, skeletal defects, deafness, retinal changes, or intellectual disability. It is important to note that more recently discovered genetic

TABLE 2 Genetic Syndromes Associated With Obesity

Genetic Syndrome	
Monogenic disorders	
MC4R deficiency	Increased lean body mass, accelerated linear growth. Hyperinsulinemia. May have lower blood pressure.
Leptin deficiency	Normal linear growth with reduced adult height. Rapid-onset obesity with hypothalamic dysfunction (hypogonadotropic hypogonadism, hypothyroidism). Alterations in immune function. Responsive to leptin treatment.
Leptin receptor deficiency	Normal linear growth with reduced adult height. Rapid-onset obesity with Hypothalamic dysfunction (hypogonadotropic hypogonadism, hypothyroidism). Alterations in immune function. Not responsive to leptin therapy.
POMC deficiency	Accelerated childhood growth. Adrenocorticotrophic hormone deficiency, mild hypothyroidism. Red hair, light skin (in non-Hispanic white individuals).
Proprotein subtilisin or kexin type 1 deficiency	Failure to thrive in early infancy. Hypoglycemia, adrenocorticotrophic hormone deficiency. Intestinal malabsorption, diarrhea.
SRC1 deficiency	Impaired leptin-induced POMC expression.
Syndromic forms of obesity	
Prader-Willi syndrome	In neonatal period poor feeding, failure to thrive, and hypotonia. By 4–8 y, hyperphagia with food impulsiveness. Short stature. Growth hormone deficiency, hypogonadism. Dysmorphia, intellectual disability, behavioral difficulties.
Alstrom syndrome	Short stature. Insulin resistance, T2DM, hypogonadism, hyperandrogenism in females, hypothyroidism. Visual impairment, hearing loss, cardiomyopathy, hepatic dysfunction, renal failure.
Bardet-Biedl syndrome	Normal stature. Hypogonadism, polydactyly, retinal dystrophy, renal malformation, cognitive disabilities, polyuria, and polydipsia.
Smith-Magenis syndrome	Short stature. Disrupted melatonin signaling. Craniofacial anomalies, intellectual disability, self-injurious behaviors, sleep disturbance.
SH2B1 deficiency	Hyperinsulinemia, delayed speech and language development, aggressive behavior.
Sim1 deficiency	Short stature. Hypopituitarism. Neonatal hypotonia, facial dysmorphism, developmental delay.
16p11.2 microdeletion syndrome	Developmental delay, intellectual disability, autism spectrum disorder, impaired communication, and socialization skills.
Brain derived neurotrophic factor deficiency	Hyperphagia, impaired short-term memory, hyperactivity, learning disability. Patients with Wilms tumor-aniridia (WAGR syndrome) have subset of deletions on chromosome 11p.12 including brain derived neurotrophic factor locus.
Albright's hereditary osteodystrophy	Short stature, round face, brachydactyly, subcutaneous ossifications. Some patients may have mild developmental delay. If inherited from the mother, may be associated with pseudohypoparathyroidism type 1a.
Cohen syndrome	Hypotonia, intellectual disability, distinctive facial features with prominent upper central teeth, broad nasal tip, smooth or shortened philtrum, thick hair and eyebrows, long eyelashes, retinal dystrophy, acquired microcephaly, joint hyperextensibility.
Beckwith-Wiedemann syndrome	Macrosomia, macroglossia, hemihyperplasia, anterior abdominal wall defects, visceromegaly, neonatal hypoglycemia, embryonal tumors, renal anomalies. Genetic alteration in chromosome 11p15.5.

Adapted from Pediatric Obesity-Assessment, Treatment, and Prevention: An Endocrine Society Clinical Practice Guideline.²⁶⁸

disorders associated with obesity are not necessarily characterized by these findings in childhood; for instance, short stature is not a hallmark of leptin deficiency in children. Table 2 lists selected monogenetic causes and syndromes associated with obesity.

Early onset of severe obesity and the presence of hyperphagia are the 2 clinical characteristics that distinguish genetic disorders of obesity. “Early onset” refers to the presence of obesity before age 5. As noted previously, “severe obesity” is defined as BMI \geq 120% of the 95th percentile for age and sex. “Hyperphagia” is the presence of insatiable hunger in which the individual’s time to satiation is long, the individual’s duration of satiation is shorter, the individual’s feelings of hunger are prolonged, and the individual has a severe preoccupation with food and experiences distress if denied food.²⁶⁷

D.1.c. Epigenetic Factors

Epigenetic factors can result in alterations in gene expression without alteration in genetic code. These epigenetic factors may modify the interaction of environmental and individual factors in promoting weight gain.²⁶⁹ One of the critical periods in the establishment of the epigenome is considered to be during embryonic development.²⁷⁰ Prepregnancy maternal or paternal obesity, for example, may influence epigenetic changes during subsequent pregnancy, increasing the risk of overweight or obesity in the offspring.²⁶⁹ Other risk factors during pregnancy—such as gestational diabetes or maternal excessive weight gain—may result in epigenetic changes and increase the risk of obesity in the offspring.

D.2. Prenatal Risk Factors

The perinatal environment plays an important role in a child’s later development of overweight or obesity. The mechanisms by which the fetal environment predisposes to the development of obesity are complex and poorly understood. They probably include gene-environment interactions or epigenetic changes attributable to several environmental factors, including maternal diet, physical activity, and/or other environmental contaminants.^{271,272}

Preterm infants have a greater likelihood of developing childhood obesity.^{273–275} Although the exact mechanisms for this association are uncertain, several risk factors have been postulated, including feeding patterns leading to accelerated weight gain in preterm infants.²⁷³

D.2.a. Parental Obesity

Parental weight is a strong predictor of pediatric obesity. Children are at greatest risk of developing obesity as an adult if at least 1 of their parents has obesity.²⁷⁶ A meta-analysis reported an increased risk of adolescent excess adiposity if either parent had overweight or obesity; the risk increased if *both* parents had obesity.²⁷⁷ Contributors to this association include genetic, environmental, and behavioral factors or the interaction of these factors, resulting in intergenerational transmission of adiposity.

Maternal BMI is a stronger predictor of childhood and adolescent obesity, compared with paternal obesity.²⁴⁷ Maternal obesity more than doubles the risk of adult obesity (see below). Paternal obesity has been associated with childhood and adolescent obesity and has an additive effect to maternal obesity.²⁷⁸

D.2.b. Maternal Weight Gain

Prepregnancy adiposity and weight gain during pregnancy are associated with neonatal, infancy, and childhood adiposity.²⁴⁷ The known effect of maternal weight on the offspring led the Institute of Medicine (IOM) to recommend different ranges for weight gain during pregnancy, varying from 12.5 to 18 kg for underweight women to 5 to 9 kg for women with obesity (BMI > 30 kg/m²).²⁷⁹ Yet, between 1997 and 2007, almost half of pregnant US individuals gained more than the weight recommended by the IOM.²⁸⁰

Excess maternal adiposity has been suggested to affect fetal metabolic programming and make the offspring more vulnerable to the obesogenic environment and increase the risk of obesity. This effect was illustrated in metabolic and bariatric surgery studies, in which children born to mothers with obesity after gastric bypass surgery had lower prevalence of macrosomia and severe obesity at adolescence, compared with their siblings born before the mothers’ surgery.²⁴⁷ Fetal or infant macrosomia and gestational diabetes are some of the complications associated with maternal obesity and serve as risk factors for later onset of obesity and T2DM in the offspring.²⁸¹

The exact mechanism by which maternal obesity predisposes to adverse outcomes in the offspring is unclear. It has been suggested that the pathways that are affected control the central regulation of appetite and insulin sensitivity and cardiovascular regulation.²⁸¹ Alteration of the fetal hypothalamic-pituitary-adrenal axis function has been implicated in programming the metabolic syndrome of the offspring of mothers with obesity.²⁸²

D.2.c. Gestational Diabetes

Infants and children of mothers with gestational diabetes mellitus (GDM) have higher fat mass and BMI than their counterparts whose mothers did not have GDM.²⁸³

Adjusting for maternal BMI and other potential confounders, GDM was shown to be associated with childhood obesity with odds ratio of 1.6 to 2.8.^{284,285} The odds of developing higher waist circumference (≥ 95 th and percentile) in children of mothers with GDM was also found to be higher after controlling for potential confounders (OR, 1.55; 95% CI, 1.03–2.35).²⁸⁵ Sibling studies controlling for shared genetics and environment have shown higher BMIs in offspring exposed to diabetes in utero compared with their unexposed siblings.²⁸⁶

Although the exact mechanisms of the effect of GDM are not fully understood, it has been postulated that the effect may be mediated through insulin. Pregnant women with GDM have higher insulin resistance compared with pregnant women without GDM.^{287,288} It has been suggested that maternal insulin resistance and hyperglycemia causes fetal hyperinsulinemia, resulting in excessive fetal growth with associated macrosomia and increased adiposity.²⁸⁷ Maternal hypertriglyceridemia from insulin resistance has also been thought to lead to increased adiposity and birth size even when glucose levels are well-controlled.²⁸⁹ Additionally, maternal diabetes is associated with increased leptin synthesis in the offspring.²⁹⁰ Epigenetic changes in infants of mothers with GDM is another suggested mechanism, affecting gene expression regulation body fat accumulation or other related metabolic pathways.²⁹¹

D.2.d. Maternal Smoking

Exposure to ETS has been shown to increase the prevalence of childhood and adolescent obesity.^{292–294} A systematic review and meta-analysis reported an association between prenatal ETS and childhood and adolescent obesity; children exposed to ETS in utero had about 1.9 times greater risk of developing obesity, compared with their nonexposed counterparts.²⁹⁵ Prenatal exposure to the risk from tobacco smoke can occur both directly from smoking mothers and indirectly through ETS, although maternal smoking was found to more strongly predict obesity.

Children exposed to smoking in utero have a dose-dependent increased risk of developing overweight and obesity.²⁹⁶

D.3. Postnatal Risk Factors

As with the prenatal environment, the postnatal environment is important to the later development of overweight and obesity. In addition to epigenetic mechanisms, behavioral habits begin to get set at an early age. Acceptance of foods, availability of high calorie foods, establishment of the microbiome, and early eating habits are only a few of the proposed mechanisms for postnatal factors to influence later weight status.²⁹⁷

D.3.a. Birth Weight

Several studies have shown a U-shaped or J-shaped distribution between birth weight and adult BMI.²⁴⁷ Infants with both low (<2500 g) and high (>4000 g) birth weight have been shown to have higher risk of obesity, compared with infants with birth weight between 2500 and 4000 g.²⁹⁸ A high BMI and central adiposity are more prevalent among low-birth weight infants.²⁹⁹ Maternal prepregnancy weight and nutritional status are strong predictors of neonatal outcomes, with underweight

prepregnancy increasing the risk of preterm birth and small-for-gestational-age neonates.³⁰⁰ Maternal pregnancy overweight and obesity are significantly associated with large-for-gestational-age babies.³⁰⁰

D.3.b. Early Breastfeeding Cessation and Formula Feeding

Some, but not all, studies have reported decreased risk of childhood and adolescent obesity in breastfed infants.²⁴⁷ The majority of evidence is derived from observational studies and may include confounding effects.²⁴⁷ Some studies have reported that, compared with bottle-fed infants, breastfed infants are better able to regulate their energy intake and have lower risk of childhood excess weight gain.³⁰¹ Other studies have also shown that body weight gain is slower in breastfed infants.²⁴⁷

Breastfeeding has been found to be inversely associated with overweight risk in the first year of life, independent of maternal BMI and SES. Breastfeeding cessation before 6 months was associated with an increased risk of rapid weight gain and overweight by 12 months of age, compared with exclusive breastfeeding.³⁰²

A systematic review of feeding practices associated with rapid infant weight gain found that certain practices (such as overfeeding, inappropriately concentrating formula, placing infants in bed with a bottle, or adding cereal to a bottle) may lead to rapid infant weight gain.³⁰³ In addition, infants fed high-protein formulas are at greater risk of elevated BMI later in childhood.^{247,304}

D.3.c. Rapid Weight Gain During Infancy and Early Childhood

In resource-abundant countries, rapid weight gain in infancy and

during the first 2 years of life is associated with higher risk of obesity both in later childhood and in adulthood.^{247,305} A systematic review and meta-analysis found that children who experienced rapid weight gain from birth to age 2 were up to 3.6 times more likely to have overweight or obesity in childhood or adulthood, with the relationship being stronger between rapid infant weight gain and childhood overweight or obesity.³⁰⁶

Therefore, rapid weight gain in infancy and early childhood can be viewed both as a risk factor for later excess weight gain and also as a signal, as mentioned previously, for pediatricians and other PHCPs to look for other underlying risk factors and causes for excess weight gain. For instance, early introduction (at younger than 4 months of age) of complementary foods has been found to increase the risk of childhood obesity in several systematic reviews.^{307,308}

D.3.d. Early Use of Antibiotics

Literature on antibiotic exposure in early life (<2 years) is mixed, with some suggestion that it may slightly increase the risk of childhood and adolescent obesity.^{309–312} The association is stronger with repeated antibiotic exposure,^{313,314} exposure within the first 6 months of infancy,³¹⁴ and broad-spectrum antibiotic use.³¹⁰ With similar antibiotic exposure, boys appear to be more susceptible to weight gain than girls.²⁹⁵ Gut microbiota is usually established during the first years of life; it is hypothesized that the effect of antibiotics is mediated through the alteration of the gut microbiome, which plays a role in energy balance.

D.4. Childhood Risk Factors

Various medical conditions that present in childhood and adolescence are associated with the

development and progression of overweight and obesity. Similarly, certain behaviors established in childhood and adolescence can increase the risk of later development of overweight and obesity.

D.4.a. Endocrine Disorders

Endocrine disorders account for less than 1% of all the causes of pediatric obesity. These disorders can be associated with endogenous or exogenous glucocorticoid excess (eg, Cushing syndrome, use of corticosteroid medications). Short stature or growth failure and abnormally high BMI may result from pseudohypoparathyroidism type 1a, growth hormone deficiency, or hypothyroidism.^{268,315}

D.4.b. Children and Youth With Special Health Care Needs Impacting Nutrition and Physical Activity

D.4.b.1 Children With Developmental and Physical Disabilities

A survey of data from NHANES, the National Health Interview Survey, and the National Survey of Children's Health found that children with disabilities were from 27% to 59% more at risk for obesity than children without disabilities.³¹⁶

In addition to factors experienced by children without disabilities, factors that affect children with disabilities that have been implicated in their greater obesity risk are: more difficulty breastfeeding,³¹⁷ disrupted appetite regulation,³¹⁸ weight-gain promoting medications,^{319,320} food selectivity and sensitivity issues,³²¹ behavioral disorders,³²² physical activity limitations,³²³ and use of food rewards.³¹⁶ A lack of adaptive physical education or sports,³²⁴ and specialized supervision and instruction³²⁴ also play a role in increasing obesity risk.

Furthermore, it is important to consider that children with disabilities are at a disadvantage when it comes to obesity treatment strategies that are tailored to their needs. For example, most community or school weight management, nutrition or physical activity interventions are not readily adapted for children with disabilities. Therefore, many children with disabilities do not have the support or strategies that they need to address excess weight. Finally, children may face bullying or stigmatization and bias in school. They may also receive unhealthy incentives as rewards from caregivers increasing their risk for obesity. These systemic trends and biases make providing adequate care for children with disabilities extremely difficult.

D.4.b.2 Children With Autism Spectrum Disorder

Children and youth with autism spectrum disorder (ASD) have a higher risk of developing overweight or obesity. In the United States, children and adolescents 2 to 18 years of age with ASD have a 43.7% greater risk of obesity compared with their counterparts without ASD.³²⁵ Although the exact mechanisms through which ASD increases the risk for excess weight gain is unknown, a recent meta-analysis of international studies showed that positive moderators to this association include children of certain races and ethnicities, female biological sex, increased age, and living in the United States.³²⁵ This meta-analysis did not control for other risk factors for obesity, however, such as use of antipsychotic medications, food intake challenges, or limited physical activity. Hence, the variable of race could be reflective of a negative SDoH.

Several etiological factors have been postulated to contribute to the

association between ASD and obesity, including: genetic variants (eg, 16p11.2 deletion and microdeletion 11p14.1),^{326,327} prenatal exposure to certain infections or medications,^{328,329} pre and postnatal exposure to toxins,^{330,331} maternal diabetes,³³² maternal obesity,³³³ intrauterine growth restriction and preterm birth,^{334,335} food selectivity,^{336,337} and physical limitations.^{338,339}

D.4.b.3 Children With Myelomeningocele

Several studies report increased rates of obesity of children with myelomeningocele,^{340–342} with children having more severe disease tending to have higher BMIs.³⁴⁰ Children and adolescents with myelomeningocele have increased total body fat³⁴³ and lower energy expenditure,³⁴² compared with children without myelomeningocele. Risk factors for obesity in this population include limited ambulation, sedentary lifestyle, decreased lean body mass, and reduced resting energy expenditure.³⁴⁴ In addition, children with myelomeningocele may be less likely to have routine weight and height, and primary care providers' discussions may be lacking with respect to addressing healthy lifestyles.

D.4.c. Attention-Deficit/Hyperactivity Disorder

A systematic review and meta-analysis showed significant association between attention-deficit/hyperactivity disorder (ADHD) and obesity among unmedicated individuals with ADHD—but not among medicated individuals.³⁴⁵ The prevalence of obesity was found to be 40% higher among children and adolescents with ADHD, compared with those without ADHD. This association is not affected by gender or by study setting, country, or quality. Causality between ADHD and obesity could not be inferred from this meta-

analysis, because the studies were cross-sectional; however, some prospective studies have shown that ADHD precedes the diagnosis of obesity.^{346,347}

Some of the known symptoms of ADHD may contribute to weight gain. For example, binge eating, which is a manifestation of impulsivity in individuals with ADHD, may result in increased energy intake. Inattentiveness, another symptom of ADHD, may lead to lack of planning, or of following through on a plan, resulting in missed meals or the consumption of unhealthy meals and snacks.³⁴⁸ Other psychiatric comorbidities that are often associated with ADHD—such as depression, anxiety, and circadian rhythm disturbances—may also be risk factors for obesity.³⁴⁸

Dopamine plays an important role in some of the addictive behaviors of ADHD and obesity. Functional MRI studies have identified shared neuropsychiatric circuits that are associated with reward, response inhibition, and emotional regulation in obesity, ADHD, and abnormal eating behavior.³⁴⁸

D.4.d. Weight-Promoting Appetitive Traits

Differences in children's appetitive traits manifest as early as infancy (for example, suckling behavior) and may become more pronounced when children get exposed to an obesogenic food environment.²¹⁷ Although the exact reasons why some children have better control of their energy intake is unknown, interaction between genetic predisposition and children's early environment may explain some of the individual differences in appetitive traits. Parent feeding style, as discussed, has been shown to be of importance.²¹⁷

Systematic review and meta-analysis of adult data showed a positive association between eating quickly and higher BMI,³⁴⁹ and in longitudinal studies, faster eating rate was associated with excess weight gain.³⁴⁹ Similarly, 2 cross-sectional pediatric studies have reported a positive association between eating fast and childhood and adolescent obesity.^{350,351} Eating quickly has been suggested to result in greater energy intake.

A recent American Heart Association policy statement on caregiver influences on young children's eating behaviors synthesized appetitive traits consistently associated with child adiposity. In addition to more rapid eating pace, these traits include eating in the absence of hunger, high enjoyment of food, low responsiveness to satiety, and low level of restrained eating.³⁵²

D.4.e. Medication Use

Medications within many categories have been associated with weight gain. The magnitude of risk associated with medication use is not fully known; therefore, there is an urgent need for more research in this area as well as mediating strategies. Medications implicated include glucocorticoids, sulfonyleureas, insulin, thiazolidinediones, antipsychotics, tricyclic antidepressants, and antiepileptic drugs.^{353–356} In particular, second-generation antipsychotics (ie, risperidone, clozapine, quetiapine, and aripiprazole) can lead to rapid weight gain and comorbidities such as prediabetes, diabetes, and dyslipidemia.^{357,358}

A recent review discusses the more commonly prescribed medications in children and adolescents with obesity and comorbidities, and offers suggestions on alternative therapeutic agents (Table 3).³⁵⁹

TABLE 3 Selected Examples of Commonly Prescribed Medications and Weight Gain in Pediatric Practice³⁵⁹

Medication	Obesogenic Medications	Nonobesogenic Medications
Allergies and asthma management	<ul style="list-style-type: none"> • antihistamines • steroids (systemic) 	<ul style="list-style-type: none"> • inhaled nasal steroids • montelukast
Antidepressants	<ul style="list-style-type: none"> • amitriptyline • nortriptyline • paroxetine • sertraline 	<ul style="list-style-type: none"> • bupropion • imipramine HCL • buspirone • trimipramine maleate • citalopram • protriptyline HCL • desipramine HCL • trazadone • venlafaxine • doxepin • escitalopram • fluoxetine • fluvoxamine
Antiepileptics	<ul style="list-style-type: none"> • carbamazepine • gabapentin • pregabalin • valproate • vigabatrin 	<ul style="list-style-type: none"> • felbamate • lamotrigine • levetiracetam • phenytoin • topiramate • zonisamide
Antipsychotics	<ul style="list-style-type: none"> • aripiprazole • clozapine • haloperidol • mirtazapine • olanzapine • perphenazine • quetiapine • risperidone • sertindole • thioridazine • ziprasidone 	<ul style="list-style-type: none"> • molindone • pimozide
Anxiolytics	not applicable	<ul style="list-style-type: none"> • alprazolam • lorazepam
Migraine management	<ul style="list-style-type: none"> • amitriptyline • atenolol • divalproex sodium • flunarizine • gabapentin • imipramin • nortriptyline • pizotifen • propranolol 	<ul style="list-style-type: none"> • lamotrigine • levetiracetam • protriptyline • timolol • topiramate • zonisamide
Mood stabilizers and antimania	<ul style="list-style-type: none"> • carbamazepine • gabapentin • lithium • valproate 	<ul style="list-style-type: none"> • lamotrigine • topiramate • zonisamide
Psychostimulants	not applicable	<ul style="list-style-type: none"> • amphetamine • methylphenidate • dextroamphetamine sulfate

This is not an exhaustive list; it is included as an example of medications that may result in weight gain and possible alternatives.

D.4.f. Depression

Children with obesity are more likely to have anxiety and depressive symptoms compared with their peers of healthy weight. It is not clear whether obesity is a risk

factor for these symptoms.^{360,361}

Some earlier research reported bidirectional associations between obesity and depression and anxiety. Limitations of some of the studies included small samples; self-

reported data on anthropometry; assessment of symptoms based on self-administered questionnaires; and not controlling for potential confounders, such as family history, neuropsychiatric disorders, and SES. A more recent study showed that obesity was a risk factor for anxiety and depression among children and adolescent after adjusting for SES, neuropsychiatric disorders, and family history of anxiety or depression.³⁶²

The association between obesity and depression and anxiety may be attributable to interactions and shared pathophysiological mechanisms between these conditions.^{363,364} Some of the shared risk factors include genetic, physiologic, and environmental factors. Obesity is associated with subclinical inflammation and oxidative stress, which have been shown to be important etiological factors for depression, and this has been suggested as possible common link between obesity and depression.³⁶³ Other factors that can potentially impact the association between obesity and anxiety and depression include sleep disturbance, unhealthy diet, physical activity, anti-or bullying of children/or bullying of children.

IX. EVALUATION OF THE PEDIATRIC PATIENT WITH OVERWEIGHT OR OBESITY

A. Evaluation of Patients With Overweight or Obesity

This evaluation is an important part of COT (see COT section in the Treatment section). As with all chronic diseases, a complete history, review of systems (RoS), and physical examination are important for treatment. Specific elements of both history and physical relating to obesity are of special importance. Evaluation of the patient and family's readiness to change behavior is critical to effectively

KAS 2. Pediatricians and other PHCPs should evaluate children 2 to 18 y of age with overweight (BMI \geq 85th percentile to <95th percentile) and obesity (BMI \geq 95th percentile) for obesity-related comorbidities by using a comprehensive patient history, mental and behavioral health screening, SDOH evaluation, physical examination, and diagnostic studies.

Aggregate Evidence Quality	Grade B
Benefits	Early detection and treatment can reduce future serious sequelae, detection of comorbidity may motivate treatment engagement.
Risks, harm, costs	Increased anxiety, overtesting, time needed for counseling, potential false-negative or false-positive tests, costs of tests.
Benefit-harm assessment	Potential to identify and manage comorbidities that have short and long term serious sequelae exceeds potential harm especially for high-risk patients.
Intentional vagueness	Frequency of evaluation, patient level of risk.
Role of patient preferences	Family history, families' concern about the test, ease and accessibility for testing must be considered.
Exclusions	\leq 24 mo old.
Strengths	Strong recommendation.
Key references	80, 365

help with obesity treatment (see algorithm in Appendix 1).

The early and accurate classification of overweight and obesity and identification of obesity-related comorbidities is fundamental to the provision of timely and appropriate treatment (see the Comorbidities section, below). The routine classification of weight status allows for early recognition of abnormal weight gain. This is particularly important because patients—including children and adolescents—often do not perceive overweight and obesity as a health problem.³⁶⁶ Caregivers, families, pediatricians, other pediatric health care providers, and other health care providers³⁶⁷ can also be slow to recognize abnormal weight status, even in the presence of severe obesity.^{368,369}

Patients and caregivers identify pediatricians and other PHCPs as trusted and preferred sources of information about weight status,³⁷⁰ starting with discussions of feeding practice in infancy and continuing with evaluation of healthy nutrition and activity into adulthood. Pediatricians and other PHCPs are also uniquely qualified to evaluate patients for overweight, obesity, and related comorbidities.

Routine well-child checks (WCCs) in the medical home are an opportune time for the evaluation of a child or

adolescent with overweight and obesity, but this can occur during problem-focused visits as well. When the discussion of weight status is normalized and nonstigmatizing, the family and provider can exit a WCC or other visit with a clear and practical plan to improve health and quality of life. Successfully and sensitively treating overweight and obesity can be highly rewarding for both the family and the pediatrician (or other pediatric health care provider), because families suffering from overweight and obesity often have experienced previous shaming or negative experiences with treatment.^{28,371}

Shaming of children with regard to their weight may happen at school and even at home in misguided efforts to “motivate” the child to adopt healthier behavior. Overt or subtle and unintended bias in health care leads to adverse health, behavioral, and psychological outcomes.⁶¹ In addition, when feeding practices are identified as unhealthy, parents may feel blame. It is important, although challenging, for pediatricians and other PHCPs to communicate support and alliance with children, adolescents, and parents as they diagnose and guide obesity treatment.⁶²

In the AAP statement on obesity bias, steps to provide supportive

and nonbiased behavior include recognition of the complex genetic and environmental influences on obesity. Recommendations include use of neutral words like “BMI” or “excess weight” rather than “fat” or “chubby,” use of people-first language (ie, “a child with high weight” or “a child with obesity” rather than “an overweight child” or “an obese child”), an office set-up that accommodates different body sizes, and a private weighing station.²⁸ Ongoing successful communication of support and empathy during obesity treatment is essential to reduce the effect of weight bias, because families will not continue to seek help if they experience stigma.^{372,373}

B. Medical History

Both a complete medical history and physical examination are necessary to evaluate *any* patient with a chronic disease. Obesity is no exception and, like other chronic diseases, requires comprehensive evaluation in certain areas of both the history and physical examination, which may require additional time to that which is allocated in a routine visit. The medical history includes the chief complaint, history of the present illness, and family history.

- The *chief complaint* is notable for determining whether overweight and obesity is a concern for the

patient and family. An open-ended question such as “What concerns, if any, do you have about your child’s growth and health?” can provide a wealth of insight on this issue.

- The *history of the present illness* provides a more comprehensive picture of the trajectory of overweight and obesity. Starting with an inquiry about maternal weight gain during pregnancy and prenatal factors that predispose to obesity, and then moving on to childhood and later adolescent factors that predispose to obesity, the pediatrician or other pediatric health care provider can glean valuable information on causes and therefore management for a particular patient’s

obesity. These prenatal and postnatal causes are described in detail in the Risk Factors section. Information about the onset of excess weight gain and consistency of weight status over time (including a review of the growth curve and previous weight control attempts) can provide an understanding of what weight status represents for the patient. It can also offer clues as to root causes, necessary diagnostic evaluation, and potential therapeutic targets.

- The *family history* focuses on obesity-related comorbidities and potential genetic causes of obesity in addition to other family health problems. A family history of obesity and obesity-

related comorbidities may influence both evaluation and treatment. Although shared environment, SDoHs, and stress can contribute to obesity within the same family, a family history of obesity can also provide a clue to genetic susceptibility to obesity—especially if the family history includes severe obesity resulting in metabolic and bariatric surgery or severe obesity present in multiple family members and generations.

- The *medication history* should be complete and should include medications associated with weight gain, such as antipsychotics, especially atypical antipsychotics; antidepressants including selective serotonin reuptake inhibitors;

TABLE 4 Special Considerations in the Review of Systems for the Patient With Overweight or Obesity

System	Symptom	Possible Obesity-Related Causes
General	Poor or slowed linear growth velocity	Endocrinologic contributor (eg, hypothyroidism, Cushing syndrome)
	Hyperphagia from early childhood, developmental delay, obesity onset under age 5 y, or syndromic features	Various genetic etiologies (see Table 2, genetic syndromes associated with obesity)
Respiratory	Shortness of breath	Obesity-related asthma phenotype, deconditioning
Gastrointestinal	Snoring, apnea, disordered sleep	Obstructive sleep apnea (OSA)
	Asymptomatic vague abdominal pain	NAFLD, NASH
	Heartburn, dysphagia, chest pain, regurgitation	Gastroesophageal reflux disease
	Abdominal pain, enuresis, encopresis, anorexia	Constipation
	Right upper quadrant pain	Gall bladder disease
Endocrine	Hyperphagia	Prader-Willi, other genetic causes
	Polyuria, polydipsia	Diabetes mellitus (DM) type 1 or 2
GYN	Oligomenorrhea, dysfunctional uterine bleeding	Polycystic ovarian syndrome
Orthopedic	Hip, thigh, or groin pain, painful or uneven gait	Slipped capital femoral epiphysis (SCFE)
	Knee pain	SCFE, Blount disease
	Foot pain	Increased weight bearing
	Back pain	Increased weight
	Proximal muscle wasting	Cushing syndrome
Mental health	Sadness, depression, anhedonia, body dissatisfaction, school avoidance, poor self-image	Depression or anxiety, bullying, sexual, physical, or emotional abuse
	Impulsive eating, distractibility, hyperactivity	ADHD
	Purging, restricting intake, binge-eating, night eating	Disordered eating or eating disorders
	Flat affect	Depression or anxiety
Urinary	Nocturia, enuresis	DM, OSA
Dermatologic	Rash	Intertrigo
	Darkened skin on flexural surfaces	Acanthosis nigricans
	Pustules, abscesses	Hidradenitis suppurativa
	Hirsutism in females	PCOS
	Flesh-colored striae	Rapid weight gain
	Purplish striae	Cushing syndrome
	Skin fold irritation	Candida
	Neurologic	Morning headaches
Daytime sleepiness	OSA	
Persistent headache	Idiopathic intracranial hypertension (IIH)	

Adapted from Krebs et al.¹⁴

steroids; anticonvulsants; antihypertensives; birth control agents, including injected forms; and medications used in diabetes mellitus.

Table 4 summarizes the RoS and provides a valuable framework for investigating a variety of obesity-related conditions.

B.1. Social History

A thorough social history is helpful in the evaluation of the child or adolescent with overweight and obesity. An understanding of family living arrangement will identify resources and barriers that are unique to the patient and their family. Factors such as eating routines and schedules; eating at multiple households; and eating environments, such as family meals, eating at a table, eating with or without screens, are all important elements in assessing contributors to and potential treatment targets for excess weight gain. Determining a family's relationship with food is also important (eg, Is food a common reward? How is food used in celebrations? Is there pressure for the child to eat?).

Because overweight and obesity tends to cluster in social groups as well as families,³⁷⁴ discussions of neighborhood, school, and friend groups can guide pediatricians, other PHCPs, and families to productive areas for treatment. Social history can heighten an awareness of, and provide insight

into, patients who are most exposed to negative SDoHs. Given that inequities exist in obesity risk factors, an SDoH evaluation is important to increase awareness and provide insight in identifying patients who are more vulnerable to obesity. Assessment of SDoHs is also important to contextualize the patient's and family's treatment challenges. Standardized tools for use in primary care exist and include the Safe Environment for Every Kid model³⁷⁵ and the Accountable Health Communities Health-Related Social Needs Screening Tool.³⁷⁶

Being alert to and recognizing SDoHs are the initial steps in trauma-informed care (TIC). ACEs can have a profound impact on health over a lifetime and, as noted, include stressors as diverse as harsh parenting, food insecurity, and parental incarceration. These factors can trigger physiologic abnormalities that increase a patient's risk for obesity, cancer, and numerous other diseases. TIC is characterized by screening and recognition of these ACEs, responding to them, and working to prevent reexposure to trauma. Initial recognition of the importance of ACEs on health occurred in the field of adult obesity treatment. The importance of TIC and addressing ACEs in pediatric obesity management is ongoing.^{68,377,378}

B.2. Nutrition and Physical Activity History

Gathering a *nutrition history* and *physical activity history* often takes the form of a patient and/or caregiver completing a healthy habits survey before seeing the pediatrician or other pediatric health care provider (Table 5). Electronic health records, waiting room kiosks, and emailed previsit surveys can all be used to help gather this information.

There are many additional tools to assess nutrition and physical activity. These include: 24-hour recalls, electronic and written food diaries, telephone- and text-prompted diaries, and various smartphone applications that track food intake. Pedometers and other wearable activity monitors can assist with physical activity assessment. Pediatricians and other PHCPs may find some of these applications and tools at their disposal.

Cultural dietary habits, limited English proficiency, and limited literacy levels may influence the accuracy of the tool used. In comparison with adults, physical activity assessment is challenging, because children and adolescents are less reliable in performing recall of performed activity.³⁷⁹ And, because of the greater burden of overweight and obesity on people of certain race and ethnicities, these differences should be acknowledged and any limitations should be

TABLE 5 Assessment Components

Dietary Intake Can Be Addressed by Assessing the Following:	Physical Activity Can Be Addressed by Assessing Physical Literacy ³⁷⁹ :
Eating outside the home	Physical literacy: The motivation, confidence, physical competence, knowledge, and understanding to engage in age-appropriate physical activity for a lifetime. Routine ambient activity is built into daily living.
Consumption of sweet drinks	Sedentary time, especially recreational screen time.
Portion size	Moderate activity levels, characterized by a mild increase in pulse and respiratory rate but still able to talk.
Meal habits, including skipping meals	Vigorous activity levels, characterized by increased breathing, elevated heart rate, or sweating.
Snack habits	
Fruit and vegetable consumption	

mitigated. An example of a healthy habit survey can be found at <https://mainehealth.org/-/media/lets-go/files/childrens-program/pediatric-family-practices/full-healthcare-toolkit.pdf>. Sensitivity to cultural, economic, and literacy barriers is necessary with the *nutrition history* and *physical activity history*, as with other assessments. Furthermore, the presence of eating disorders, obsessive-compulsive disorder, and other mental health conditions may preclude the use of certain tools that require intensive tracking.

B.3. Assessments for Behavioral Health and Disordered Eating Concerns

Because rates of behavioral health illnesses are greater in patients with obesity than other patients, it is important for pediatricians and other PHCPs to evaluate the emotional health of children with overweight and obesity.³⁸⁰ A common comorbidity of obesity in children is weight-based bullying and teasing.^{28,43} If a patient responds affirmatively when asked if they have ever been teased or bullied about their weight, pediatricians and other PHCPs can consider provision of resources (such as those found at stopbullying.gov) to the child and parent as well as local counseling referral.

Various in-office tools can be used to address behavioral health disorders seen in greater prevalence in patients with obesity. Overall behavioral functioning can be assessed through the Pediatric Symptom Checklist's parent or teen versions.³⁸¹ Evaluation for depression can be conducted through the teen version of the Patient Health Questionnaire 2- or 9- question version.³⁸² Assessment of anxiety by tests such as the General Anxiety Disorder assessment or the Screen for Child

Anxiety Related Disorders assessment.^{383,384} In addition, ADHD can be assessed by the Vanderbilt ADHD Rating Scales.^{385,386}

As discussed in the AAP clinical reports, "Preventing Obesity and Eating Disorders in Adolescents"³⁸⁷ and "Identification and Management of Eating Disorders in Children and Adolescents,"³⁸⁸ adolescents with obesity may engage in unhealthy practices to lose weight. These practices include skipping meals, using diet pills or laxatives, and inducing vomiting. Therefore, it is important for pediatricians and other PHCPs to evaluate the adolescent with overweight or obesity for these and other related behaviors, and to examine the growth chart for evidence of more rapid than expected decline in BMI.

As noted in the clinical reports above, pediatricians "should be knowledgeable about the variety of risk factors and early signs and symptoms of eating disorders in both male and female children and adolescents. Pediatricians should evaluate patients for disordered eating and unhealthy weight-control behaviors at annual health supervision visits. Pediatricians should evaluate weight, height, and BMI by using age- and sex-appropriate charts, assess menstrual status in girls, and recognize the changes in vital signs that may signal the presence of an eating disorder."³⁸⁸ For more information on this evaluation, please see the AAP clinical report.³⁸⁸

B.3.a. Physical Evaluation

A complete physical examination is necessary in the patient with overweight and obesity because of the disease's complex and multisystem effects. The 2015 article "Physical Examination Findings Among Children and

Adolescents with Obesity: An Evidence-Based Review," by Armstrong et al provides a thorough explanation of special considerations for patients with or at-risk for weight-related illness.³⁶⁵ Pediatricians and other PHCPs are encouraged to reference this AAP-published review. The physical exam also requires focused attention to certain obesity-related findings related to physical evaluation (Table 6). These include:

- *Vital signs* such as heart rate, pulse, and blood pressure should be taken; blood pressure should be measured accurately with an appropriately sized cuff.⁸⁷
- *Other important signs:* short stature may be a sign of a genetic or endocrinologic cause for overweight and obesity. Flat affect may indicate depression, and anxious mood may indicate anxiety. Attention-seeking may be a signal for underlying distress over overweight and obesity. Syndromic features may also offer indications of the presence of an underlying genetic cause for obesity.
- *Skin examination* should be performed to look for intertrigo and hidradenitis suppurativa associated with excess skin folds as well as acanthosis nigricans associated with insulin resistance. Flesh-colored striae may be seen on the abdominal wall and/or thighs as an indication of rapid weight gain. The combination of purplish abdominal striae, slowed linear growth, cervicodorsal fat accumulation, proximal muscle wasting, full facies, and hypertension should prompt evaluation for Cushing syndrome.
- Examination of the *head, ears, eyes, nose, and throat* should occur to look for papilledema associated with pseudotumor cerebri, tonsillar hypertrophy associated with sleep

TABLE 6 Physical Examination Findings in Children and Adolescents With Obesity

	Physical Examination Finding	Definition	Other Causes and Differential
Vital signs	Hypertension	SBP or DBP \geq 95th percentile on at least 3 readings	Numerous, including essential, stress-induced, renal parenchymal or vascular disease, cardiovascular disorders, obstructive sleep apnea syndrome, substance abuse or medication side effect, pheochromocytoma, anemia, hyperthyroidism, Cushing syndrome, Williams syndrome, Turner syndrome
	Increased HR	Heart rate above upper limit of normal for age	Numerous, including fever, anemia, drugs, anxiety, pain, arrhythmia, myocarditis, substrate deficiency, hypovolemic shock, sepsis, anaphylaxis, toxic exposure, hyperthyroidism, Kawasaki disease, acute rheumatic fever, pheochromocytoma
Anthropometric	Changes in height velocity	Early height velocity increase	True pattern characteristic of obesity, but early height increases can also be: familial tall stature, precocious puberty, gigantism, pituitary gland tumor
	Changes in weight gain	Early weight gain before age 5 y Earlier onset of peak height velocity	Genetic causes, overfeeding Slowing of height can be attributable to medications, inflammatory bowel disease, hypothyroidism, hypercortisolism, dysplastic or genetic syndrome, constitutional delay, growth hormone deficiency
HEENT	Papilledema	Slowing of height age 8–18 y Edema of the optic disc secondary to increased intracranial pressure (Frisen scale)	Intracranial mass lesion, hydrocephalus, cerebral venous thrombosis, medications, autoimmune disorders, anemia, and cranial venous outflow abnormalities
	Dental caries	White, brown, or black spots (noncavitory) or eroded areas of enamel or dentin (cavitory)	Developmental disease of the tooth and gum, trauma, infection
	Tonsillar hypertrophy	Tonsils occupy at least 50% of the oropharynx (Brodsky classification 3+ and 4+).	Infectious causes
Chest	Gynecomastia	>2 cm of breast tissue in biological males	Hyperaromatase syndrome; hypoandrogenism, hyperprolactinemia, chronic liver disease, and medications, particularly H2 antagonists
	Cervicodorsal hump	Fibrous fatty tissue over the upper back and lower neck	Endogenous (Cushing syndrome) or exogenous corticosteroid exposure, adrenal carcinoma, adrenal adenoma; HIV with secondary hyperinsulinemia
Gastrointestinal	Liver enlargement (hepatomegaly)	Liver span >5 cm in 5-y-olds and 15 cm in adults or liver edge palpable below the right costal margin by >3.5 cm in adults or >2 cm in children	Multiple, including hepatitis, storage disorders, infiltrative, impaired outflow, and biliary tract disorders
Genitourinary	Buried penis	Suprapubic fat accumulation leading to the appearance of a shortened penile shaft	Trapped penis, webbed penis, and micropenis

TABLE 6 Continued

	Physical Examination Finding	Definition	Other Causes and Differential
Musculoskeletal	Gait	Collapse into hip (“waddle”), Trendelenburg or antalgic gait (external rotation or out-toeing on affected side)	Arthritis, SCFE
	Lordosis	Trunk sway associated with postural adaptations	Spondylolisthesis, achondroplasia, muscular dystrophy, other genetic conditions
	Hip pain and/or limp	Knee or hip pain, subacute onset, pain with external rotation of hip	Multiple problems present with chronic hip, knee, or thigh pain including slipped capital femoral epiphysis (SCFE), growing pains, femoral neck fracture, groin injury, Perthes disease, osteonecrosis associated with systemic disease, juvenile idiopathic arthritis, reactive arthritis, overuse injuries, chondrolysis, tumors, osteitis pubis
	Genu varum or valgum	Genu varum (bow legs)	Tibia vara (Blount disease), rickets, skeletal dysplasia, celiac sprue, collagen disorder and hypermobility syndromes (eg, Marfan syndrome), Loays-Dietz, classic Ehler Danlos syndrome) ¹⁵
		Genu valgum (knock-kneed)	Physiologic in children under 6 y; in older children and adolescents, consider postaxial limb deficiency, neoplasms, genetic and metabolic disorders, neurofibromatosis, and vitamin D-resistant rickets
	Pes planus	Rigid versus flexible, sometimes with pain	Posterior tibial tendon insufficiency, tarsal coalition, congenital vertical talus, rheumatoid arthritis, trauma, neuropathy
Skin	Acanthosis	AN is thickened and darker skin, occasionally pruritic at the nape of the neck (99%), axillae (73%) and, less commonly, groin, eyelids, dorsal hands, and other areas exposed to friction	Medication side effect, and uncommonly, visceral malignancy.
	Hirsutism or acne		Hirsutism: familial, Cushing syndrome, thyroid disorders Acne: physiologic, folliculitis, rosacea
	Striae	Linear, usually symmetrical smooth bands of atrophic skin that initially appear erythematous, progressing to purple then white; perpendicular to the direction of greatest tension in areas with adipose tissue	Pregnancy, Cushing syndrome, and topical corticosteroid use
	Intertrigo	Macerated, erythematous plaques in skin folds	Inflammatory diseases, metabolic disorders, malignancies (rare in pediatrics), and various infections by site
	Pannus	Excess skin and subcutaneous fat below the umbilicus	Pregnancy, malignancy

Adapted from Table 4 and used with permission by Armstrong et al. et al.³⁶⁵ HEENT, head, eye, ear, nose, and throat examination.

apnea and goiter associated with thyroid disease.

- *A cardiopulmonary examination* should be performed to look for a

spectrum of impairment that can be associated with overweight and obesity. Simple deconditioning may present with tachypnea, dyspnea,

or tachycardia. Wheezing may be suggestive of intrinsic or exercise-induced asthma. Tonsillar hypertrophy may be a sign that increases

the likelihood of sleep apnea. In more severe obesity, congestive heart failure may present with basilar rales or other signs of more significant cardiac disease.

- *Liver size* should be assessed by palpation and auscultation. If present, right upper quadrant tenderness should be noted.
- *Genito-urinary examination* should be performed to assess pubertal status and genital appearance looking for signs of endocrine or genetic abnormality. Hypogonadism may be present in certain syndromes associated with obesity or be a result of obesity.^{389,390} More commonly, biological males with abdominal obesity may have a suprapubic fat pad obscuring the penis, a so-called “vanishing penis,” and need instruction on proper voiding and genital hygiene to avoid development of skin breakdown.
- *Neurologic evaluation* may reveal papilledema, as described above, as well as paresthesia.
- *Orthopedic findings* associated with obesity include abnormal gait, knee tenderness, pes planus, genu valgum (“knock knees”), genu varum (leg bowing), foot pain, back tenderness, and hip pain. Obesity may also make detection of scoliosis more difficult.
- *Neuromuscular evaluation* of obesity, as with the orthopedic evaluation of obesity, includes assessment of bone structure, gait and pain, but also includes assessment for balance, coordination, lower limb muscle strength, flexibility and motor skill proficiency. Patients with obesity frequently experience impairment in these areas. Such limitations can result in further reduction of ability to engage in physical activity.³⁹¹

C. Assessment of Patient Readiness to Change

“Readiness to change” refers to a patient’s interest in changing a

behavior (Importance) and their belief that they can bring about this change (Confidence). This evaluation is important when discussing healthy nutrition and activity with patients who have BMI in the healthy range; it assumes even greater importance with a patient and family who are struggling with overweight, obesity, or severe obesity where health concerns are elevated. This evaluation of readiness to change is central to deciding how and when to embark on obesity treatment. Motivational interviewing (MI), discussed in the Treatment section, provides a useful framework for evaluating and discussing a patient’s readiness to change.³⁹²

Readiness to change, perceptions of weight status, health challenges, nutrition habits, and access to physical activity are influenced by familial, cultural, and socioeconomic factors. For this reason, understanding these factors is beneficial in forging a productive relationship with children and their families. It is also important to remember that patients and families care about their health and their child’s health regardless of race, ethnicity, and/or SES. Caregivers should be reminded that the presence of overweight or obesity is NOT an indication of poor parenting.

D. Laboratory Evaluation

Based on BMI classification—and augmented by findings in the history, physical examination, and patient readiness to change assessments—laboratory evaluation of the patient represents the next important step in evaluation. This laboratory evaluation and its connection to the delineation of more common comorbid illnesses is described in the Comorbidities section. Other laboratory evaluations can be performed as clinically indicated.

X. COMORBIDITIES OF PEDIATRIC OVERWEIGHT AND OBESITY

Introduction to Comorbidities

Children and adolescents with obesity have increased prevalence of comorbidities, and a greater risk for obesity in adulthood, morbidity, and premature death.^{36,393–395} The risk for obesity-related comorbidities increases with age and severity of obesity and prevalence varies by ethnicity and race.³⁹⁶ For example, there is a higher prevalence of NAFLD in Hispanic children and a lower prevalence in Black children.^{397,398} AI/AN, Black, and Hispanic youth have higher prevalence of prediabetes and diabetes, compared with white youth.^{399,400} Pediatricians and other PHCPs need to recognize that the association between ethnicity and race and obesity and related comorbidities in both children and adults likely reflects the impact of epigenetic, social, and environmental factors, such as SDoHs, low SES, exposure to structural racism, neighborhood deprivation, and inadequate built environment in these subpopulations.^{399,401–408}

Obesity and related comorbidities should be evaluated concurrently with an obesity-specific history and review of systems, family and social history, physical examination, and laboratory testing. This evaluation provides pediatricians and other PHCPs with an opportunity to assess for both the etiology and complications of obesity (see the Evaluation section). Pediatricians and other PHCPs need to take into consideration patient-specific factors that may increase the risk for comorbidities. For example, prediabetes and diabetes occur more frequently among children who are 10 years and older, are in early pubertal stages, or have a family history of T2DM.^{399,400,409}

There is compelling evidence that obesity increases the risk for comorbidities and that weight loss

interventions can improve comorbidities.^{80,396,410} Thus, the recommendations for comorbidity evaluation uses input from the technical report on comorbidities for the prevalence, age, and weight category³⁹⁶ associated with comorbidities, and the technical report on treatment of obesity intervention outcomes on dyslipidemia, prediabetes and diabetes, HTN, and NAFLD.⁸⁰ Studies on optimal age, frequency, benefits, and harms of evaluating for comorbidities for children with obesity remain limited. To address when to begin evaluation, what tests to obtain, and frequency of testing, input from other clinical practice guidelines was also considered.^{87,88,90,411,412}

The KASs in this section are limited to comorbidities addressed in the technical reports and/or guidelines from professional organizations or societies. Consensus recommendations are included to cover the breadth of relevant comorbidities associated with pediatric overweight and obesity and to provide context for implementation. Each KAS or consensus recommendation is drawn from the technical reports, an extensive review of the literature, and clinical guidelines or position statements from premier organizations or professional

TABLE 7 Inclusion Criteria for Guidelines or Position Statements Reviewed for Comorbidities

Inclusion Criteria
<ul style="list-style-type: none"> • Clinical guideline or position statement was published in the last 15 y. • The organization or professional society is recognized as the leading scientific expert in the field. • The clinical guideline or position statement uses an established grading matrix to assess the evidence.

societies in the field. The inclusion criteria for the guidelines and position statements are in Table 7. When there was more than 1 guideline from the same organization or professional society, the most recent guideline was given precedence. Other considerations for inclusion were guidelines supported by a technical report or endorsed by the AAP.

The following section is divided into 3 sections:

- Overall KASs for Laboratory Evaluation of Obesity-Related Comorbidities for children with overweight and obesity (KASs 3–3.1);
- Concurrent Treatment of Obesity and Obesity-Related Comorbidities (KAS 4); and
- Specific Recommendations for Evaluation for Common Comorbidities (KASs 5–8) and Guidelines for Other Comorbidities.

Recommendations for reevaluation and initial management of

common comorbidities are in Appendix 3.

A. Laboratory Evaluation of Obesity-Related Comorbidities for Children With Overweight and Obesity

The 2007 AAP Expert Committee on Child Obesity recommended laboratory evaluation for children with obesity for dyslipidemia, prediabetes, and NAFLD starting at 10 years by obtaining a fasting lipid panel, fasting glucose, alanine transaminase, and aspartate transaminase levels every 2 years.⁹² For children with overweight, the recommendation was only for a fasting lipid panel unless additional risk factors were present (such as family history of obesity-related diseases, elevated BP, elevated lipid levels, or tobacco use).¹⁴ KASs 3 and 3.1 build on the 2007 recommendations—taking into account recent studies, guidelines, and pediatrician and other PHCP behaviors—while balancing the harm versus benefit of evaluation at the individual and population levels.

KAS 3. In children 10 y and older, pediatricians and other PHCPs should evaluate for lipid abnormalities, abnormal glucose metabolism, and abnormal liver function in children and adolescents with obesity (BMI ≥ 95th percentile) and for lipid abnormalities in children and adolescents with overweight (BMI ≥ 85th percentile to <95th percentile).

Aggregate Evidence Quality	Grade B.
Benefits	Allows for early detection and management to reduce risk factors for future cardiometabolic disease. Result will guide treatment. May motivate treatment engagement.
Risks, harm, costs	Cost, access, patient anxiety, labeling with chronic medical condition, stress, and time of undergoing treatment.
Benefit-harm assessment	Identification and management of cardiometabolic comorbidities in childhood and adolescence exceeds potential harm, especially for high-risk patients.
Intentional vagueness	Age.
Role of patient preferences	Parent and patient knowledge, family history, families' concern about the test, ease and accessibility of testing should be considered.
Exclusions	<24 mo old.
Strengths	Strong.
Key references	80, 86, 88, 90, 396, 397, 413–416

Children ≥ 10 Years

To encourage a pragmatic and efficient evaluation strategy, KAS 3 and 3.1 recommend that, for children with obesity, evaluation for lipid abnormalities, abnormal glucose metabolism, and liver dysfunction be obtained at the same time and begin at age 10 years. The expectation is that pediatricians and other PHCPs will find it easier to adhere to recommendations when all tests are obtained at the same time. They may order fasting laboratory tests for the evaluation, because a fasting lipid panel is still the recommended test to evaluate for dyslipidemia for children and adolescents with overweight and obesity (see the dyslipidemia section, below, for additional information).

Children 2–9 Years

For children 2 to 9 years of age with obesity, evaluation for lipid abnormalities may be considered (KAS 3.1). This recommendation aligns with the 2011 National Heart Lung Blood Institute (NHLBI) Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents.⁸⁶ In population-based studies, lipid abnormalities occur in children younger than 10 years, with higher rates among children with obesity.^{80,395,417} High

triglycerides (TG) and low high-density lipoprotein (HDL) levels (the typical pattern of dyslipidemia that occurs with obesity) have been reported in children with obesity as young as 3 years.³⁹⁵

As the risk profile for NAFLD and diabetes mellitus in children younger than 10 years is lower (especially in the absence of severe obesity), obtaining tests for abnormal glucose metabolism or liver function is not universally recommended for this population.^{88,90,415,418}

Detailed and specific recommendations are provided in the following sections on dyslipidemia, prediabetes and diabetes mellitus, and NAFLD.

Children With Overweight

For children 10 years and older with overweight, evaluating for lipid abnormalities is recommended in the absence of additional risk factors (KAS 3).^{86,412} For evaluation of type 2 diabetes mellitus (T2DM), additional risk factors need to be considered, which include: family history, history of gestational diabetes, signs of insulin resistance (such as acanthosis nigricans), and use of obesogenic psychotropic medication.^{90,358,419} For NAFLD, additional risk factors include family

history of NAFLD, central adiposity, signs of insulin resistance, prediabetes or diabetes mellitus, dyslipidemia, and sleep apnea.⁸⁸

Considerations for Testing

Among children with obesity, there is clustering of comorbidities, a higher risk profile for more severe disease and/or progression than may be commonly or previously recognized.^{396,421–423} For example, regardless of the definition used for metabolic syndrome, the prevalence is 0% to 4.7% among children with healthy weight and increases to 14.5% to 35% among children and adolescents with obesity.³⁹⁶ In a cohort of 675 children with NAFLD from 12 clinical centers across the United States, one-third had T2DM or prediabetes,⁴²² 2 conditions that have significant morbidity in childhood. Adolescents with severe obesity—who have comparable BMI and metabolic profiles as adults—are more likely to present with advanced liver damage and severe systemic inflammation, suggesting that pediatric NAFLD may be more aggressive.⁴²⁴ Similarly, in T2DM, children have a more rapid rate of progression of islet β cell failure and dysglycemia compared with adults.^{425–427}

Concerns about overtesting and cost are warranted but are balanced by the significant impact of obesity and

KAS 3.1 In children 10 y and older with overweight (BMI ≥ 85th percentile to <95th percentile), pediatricians and other PHCPs may evaluate for abnormal glucose metabolism and liver function in the presence of risk factors for T2DM or NAFLD. In children 2 to 9 y of age with obesity (BMI ≥ 95th percentile), pediatricians and other PHCPs may evaluate for lipid abnormalities.

Aggregate Evidence Quality	Grade C.
Benefits	Limit evaluation to populations with increased risk. Allows for early detection and management to reduce risk factors for future cardiometabolic disease. Result will guide treatment.
Risks, harm, costs	Cost, access, patient anxiety, labeling with chronic medical condition, stress, and time of undergoing treatment.
Benefit-harm assessment	Identification and management of cardiometabolic comorbidities in childhood and adolescence may exceed potential harm, especially children at increased risk.
Intentional vagueness	Age. Need for testing may vary by condition and individual presentation.
Role of patient preferences	Parent and patient knowledge, family history, families' concern about the test, ease and accessibility of testing should be considered.
Exclusions	<24 mo old.
Strengths	Moderate.
Key references	80, 86, 88, 90, 358,396, 397, 413–415, 420

comorbidities on morbidity and mortality. Almost half (43%) of children and adolescents with obesity have at least 1 abnormal lipid level,⁴¹⁷ and 1 in 5 US adolescents have prediabetes,⁴²³ which are both precursors for future cardiometabolic disease. Although the prevalence of T2DM in children is low, at approximately 1%, the incidence has increased from 9 in 100 000 in 2002 to 13.8 in 100 000 in 2015, a worrisome annual percentage change of 4.8%.³⁹⁹ NAFLD is considered one of the most common chronic liver diseases in children^{88,397,398,428} and occurs more frequently in male children, older children, and Hispanic children.^{397,429}

Finally, although obesity prevalence rates continue to rise, the rate of evaluating for obesity or comorbidities in practice is low—suggesting that any concerns about overtesting are likely to be more theoretical than real.^{3,394,430–432}

See Appendix 3 for information on frequency of testing for comorbidities.

B. Concurrent Treatment of Obesity and Obesity-Related Comorbidities

There is substantial evidence to support concurrent treatment of obesity and comorbidities to achieve weight loss, avoid further weight gain, and improve obesity-related comorbidities. The majority of studies

reviewed in the technical report on comorbidities³⁹⁶ demonstrate an association between overweight and/or obesity, severity of obesity, and higher prevalence of comorbidities. Studies also report improvement in comorbidities with intensive lifestyle treatment, weight loss medication, and/or bariatric surgery.^{80,126,438} Specifically, cardiometabolic markers improved significantly in children with obesity who underwent intensive pediatric obesity treatment of 3 to 6 months, which provides an opportunity for clinicians to emphasize health outcomes of lifestyle management.^{80,410,439} Interventions that meet the intensity or “dose” threshold of 26 hours or more over 2 to 12 months can lead to clinically significant improvements in BMI,⁷⁹ and decreases in BMI can lead to clinically meaningful improvements in comorbidities.^{440–444}

Guidelines for dyslipidemia, T2DM, NAFLD, and HTN all recommend lifestyle treatment of the primary management of the comorbidity.^{86–88,90,414,415,419,420} Although the specific dietary recommendation may differ slightly (eg, CHILD-1 and 2 for dyslipidemia, low-glycemic diet for prediabetes, limiting sugary beverages for NAFLD, and a Dietary Approaches to Stop Hypertension [DASH] Diet for elevated BP), there is overlap between the dietary recommendations and all comorbidities improve with weight stabilization and reduction.^{80,410,436}

Children are often seen at least once a year for WCCs, at which the pediatrician or other pediatric health care provider reviews the growth chart, provides anticipatory guidance on growth, feeding, nutrition, sedentary screen time, and participation in physical activity. At a minimum, the WCC can include evaluation for comorbidities for children with overweight and obesity, and anticipatory guidance on risk for comorbidities with increasing BMI or obesity. It may be helpful for pediatricians and other PHCPs to include the diagnosis of obesity to the problem list to heighten awareness and remind providers to address weight concerns at subsequent clinic encounters.^{445,446} In a large adult study, documentation of an obesity diagnosis on a problem list was independently predictive of at least 5% weight loss.⁴⁴⁵ To avoid any harmful effects related to potential weight bias and stigma, however, pediatricians and other PHCPs need to be mindful of how this diagnosis is conveyed to the child and/or caregiver.²⁸

There may also be a potential benefit for improved weight outcomes with comorbidity evaluation. In adult studies, identifying obesity-related comorbidities has been shown to be a motivating factor to address weight concerns.^{447–449} The evidence in pediatrics is, however,

KAS 4. Pediatricians and other PHCPs should treat children and adolescents for overweight (BMI ≥ 85th percentile to <95th percentile) or obesity (BMI ≥ 95th percentile) and comorbidities concurrently.

Aggregate Evidence Quality	Grade A
Benefits	Awareness of relationship between comorbidities and weight status, treating weight improves comorbidities. Improved continuity of care and outcomes. Provides an opportunity to discuss health.
Risks, harms, costs	Anxiety about weight or comorbidities, time needed for education counseling, potential for disordered eating. Health care costs.
Benefit-harm assessment	Benefits exceed potential harm.
Intentional vagueness	Pediatricians and other PHCPs may not have access to appropriate subspecialists.
Role of patient preference	Increased visit time, patient or parent experience with prior weight-related counseling.
Exclusions	<24 mo.
Strength	Strong.
Key references	80, 86–88, 90, 396, 410, 414, 415, 419, 420, 433–437

sparse and inconsistent.^{434,450} Adolescents identify a desire for improved health as a primary motivation for change.⁴⁵¹ Another study analyzed clinic records of 4000 youth aged 10 to 18 years with overweight or obesity in an academic primary care network and found that youth who were evaluated ($n = 2815$) with a glycosylated hemoglobin (HbA1c) had a decrease in BMI-z slope per year after the HbA1c test compared with similar peers ($n = 2087$) who had not been evaluated. Among those who had an HbA1c test, the decline in BMI-z slope per year was greater for youth with HbA1c in the prediabetes-range.⁴³⁴ An earlier study with a similar pediatric clinic population but a smaller sample size ($n = 128$) did not find a positive effect on BMI change following cholesterol evaluation.⁴⁵⁰ There is a need for more studies before definitive conclusions can be reached about whether evaluating families for comorbidities increases engagement, adoption of healthy choices, and weight loss or has unintended negative effects.

C. Specific Guidelines for Initial Evaluation for Comorbidities

The following sections provide specific recommendations on initial comorbidity evaluation. Guidance on repeat evaluation and initial

comorbidity management may be found in Appendix 3.

G1. Dyslipidemia

Children and adolescents with overweight and obesity have increased prevalence of abnormal lipid levels.³⁹⁶ The combination of hypertriglyceridemia and low high-density lipoprotein (HDL) levels, driven largely by underlying insulin resistance, is the most common type of dyslipidemia seen with overweight and obesity. Children and adolescents with overweight and obesity can also have elevated total cholesterol and low-density lipoprotein (LDL) levels.⁸⁶ NHANES data from 2011 to 2014 showed that prevalence of abnormal lipid level was 3 times higher among children and adolescents with obesity, compared with those with a healthy BMI (43% vs 14%).⁴¹⁷

Studies indicate that cardiovascular risk factors track from childhood into adult life and that lifestyle treatments can improve outcomes with respect to these risk factors.^{393,413,452} Being aware of the association of these “silent” cardiovascular comorbidities with overweight and obesity—as well as their persistence into adulthood with potential serious health consequences—obliges pediatricians and other PHCPs to perform

laboratory testing, educate patients and families about the long-term risks of cardiovascular disease and provide nutrition and activity counseling.

This KAS is supported by both the 2011 NHLBI Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents and 2018 American Heart Association and American College of Cardiology Guidelines, which recommend evaluation for early risk of atherosclerotic cardiovascular disease and counseling on risk-reduction behaviors in children and adolescents.^{86,411} Evaluation for dyslipidemia with obesity is recommended for younger children, as well as for children 10 years and older. Although data are limited in young children, 1 population-based study showed that 10% of children with obesity aged 3 to 5 years have elevated TG and low HDL levels.³⁹⁵

In addition to obesity, other risk factors for dyslipidemia include cigarette use, HTN, diabetes, and a family history of cardiovascular disease in a first- or second-degree relative (≤ 55 years for males and ≤ 65 years for females) with a history of myocardial infarction, sudden death, or HTN.^{86,437} All of these conditions

KAS 5. Pediatricians and other PHCPs should evaluate for dyslipidemia by obtaining a fasting lipid panel in children 10 y and older with overweight (BMI \geq 85th percentile to <95 th percentile) and obesity (BMI \geq 95th percentile) and may evaluate for dyslipidemia in children 2 through 9 y of age with obesity.

Aggregate Evidence Quality	Grade B: Children ≥ 10 y of Age With Obesity. Grade C: Children 2 Through 9 y of Age
Benefits	Allows for identification and management of specific cardiovascular risk factors, fasting status limits number of blood draws. May motivate treatment engagement.
Risks, harms, costs	Convenience, cost, access, patient anxiety, labeling with chronic medical condition, stress and time of undergoing treatment.
Benefit-harm assessment	Identification and management of specific cardiometabolic comorbidities in childhood and adolescence exceeds potential harm of no evaluation, especially for high-risk patients. When obtaining a fasting lipid panel is not possible, the pediatrician or other PHCP may assess the benefit of evaluating for dyslipidemia with a nonfasting lipid panel in certain circumstances.
Intentional vagueness	None.
Role of patient preference	Ease and accessibility of testing; families concern about the test.
Exclusions	≤ 24 mo old.
Strength	Strong (10 y and older). Moderate (2–9 y).
Key references	80, 86, 395, 396, 410, 411, 417, 453, 454

TABLE 8 NHLBI Criteria for Lipid Testing Results

Lipid Category	Low (mg/dL)	Acceptable (mg/dL)	Borderline High (mg/dL)	High (mg/dL)
Total cholesterol	—	<170	170–199	≥200
LDL cholesterol	—	<110	110–129	≥130
HDL cholesterol	<40	>45		—
Triglycerides				
0–9 y	—	<75	75–99	≥100
10–19 y	—	<90	90–129	≥130
Non-HDL cholesterol	—	<120	120–144	≥145

Adapted from the Expert Panel on Integrated Guidelines for Cardiovascular Health.⁸⁶ —, not applicable.

warrant laboratory evaluation and may help guide clinical decisions for assessment of dyslipidemia in younger children. Additionally, awareness of an association of social factors, specifically ACEs, with cardiovascular risk factors is important.⁴⁵⁵

C.1.a. Laboratory Tests for Diagnosis of Dyslipidemia

The NHLBI expert panel recommends a fasting lipid panel for evaluation of dyslipidemia for children with overweight and obesity.⁸⁶ Because dietary fats and carbohydrates (particularly simple sugars) increase serum TG concentrations, 8 to 12 hours of fasting before testing is recommended.⁴⁵⁶ Given that a combination of high TG and low HDL cholesterol is the most common pattern of dyslipidemia observed in children with overweight and obesity, the recommendation to obtain a fasting lipid panel is important, because nonfasting TG levels will not be accurate.⁸⁶

For practical purposes, a nonfasting lipid panel using the non-HDL level may be easier to obtain for routine evaluation in the primary care setting. The non-HDL level is the total cholesterol minus the HDL cholesterol level. If the non-HDL cholesterol level

is abnormal (non-HDL ≥145 mg/dL) and/or the HDL level is <40 mg/dL, a fasting lipid panel needs to be obtained for diagnosis.⁴¹² The nonfasting lipid panel is recommended for all children 9 to 11 years of age to evaluate for familial hypercholesterolemia.⁸⁶ Estimates are that approximately 25% of children would be referred for a fasting lipid panel because of elevated non-HDL lipid evaluation.⁴⁵⁷ Because of the elevated risk of lipid abnormality among youth with overweight and obesity, a fasting lipid panel is recommended. See the implementation guide for additional information.

The cut-off criteria for lipids in the 2011 NHLBI guidelines are the same across different age groups, except for triglycerides, as indicated in Table 8.

See Appendix 3 for information on frequency of laboratory testing and information about initial management of dyslipidemia.

C.2. Prediabetes and Type 2 Diabetes Mellitus

T2DM is now increasingly diagnosed in the pediatric population. Between

2002 and 2015, the incidence of T2DM among 10- to 19-year-olds in the United States increased from 9.0 to 13.8 per 100 000.^{290,399,458} Based on the 2005 to 2016 NHANES, 1 in 5 adolescents (12–18 years) have prediabetes.⁴²³ Although uncommon, T2DM has been diagnosed in children younger than 10 years, some as young as 4 years of age.^{459,460} For this reason, pediatricians and other PHCPs should consider risk factors and symptoms of altered glucose metabolism in all ages (eg, polydipsia, polyphagia, polyuria, blurred vision, unexplained weight loss).

Because obesity is a strong predictor for developing prediabetes and T2DM,^{423,461,462} pediatricians and other PHCPs need to have an increased index of suspicion when caring for children with obesity, especially in the presence of other risk factors (Table 9).^{90,408,414,415,463} Both genetics and SDoHs account for some of the racial and ethnic disparities observed in the incidence of T2DM.^{401,408}

The pathogenesis of prediabetes and T2DM is a peripheral and hepatic resistance to insulin accompanied by progressive loss of islet cell function.

TABLE 9 Other Risk Factors for Prediabetes and T2DM^{90,358,415,419}

Risk Factors
<ul style="list-style-type: none"> • Maternal history of diabetes or gestational diabetes • Family history of diabetes in first- or second-degree relative • Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidemia, polycystic ovary syndrome, or small-for-gestational-age birth weight) • Use of obesogenic psychotropic medications

KAS 6. Pediatricians and other PHCPs should evaluate for prediabetes and/or diabetes mellitus with fasting plasma glucose, 2-h plasma glucose after 75-g oral glucose tolerance test (OGTT), or glycosylated hemoglobin (HbA1c).^a

Aggregate Evidence Quality	Grade B.
Benefits	Tests sensitive for glucose intolerance have established diagnostic cut-offs.
Risks, harms, costs	Cost, access, time, patient anxiety with testing, varying levels of sensitivity and specificity, concordance between tests is imperfect, HbA1c levels can vary by age, ethnicity, anemia or hemoglobinopathies.
Benefit-harm assessment	Benefit outweighs harm.
Intentional vagueness	Consideration for patient access and timing of tests.
Role of patient preference	Families' concern about developing T2DM, concerns about the test, ease and accessibility for testing, length of time for OGTT.
Exclusions	Fasting insulin test is not reliable measure of insulin resistance.
Strength	Moderate.
Key references	90, 414–416, 464

^a Per KAS 3 and 3.1: pediatricians and other PHCPs should evaluate children 10 y and older with obesity (BMI ≥ 95th percentile) for abnormal glucose metabolism and may evaluate children 10 y and older with overweight (BMI ≥ 85th percentile to <95th percentile) with risk factors for T2DM or NAFLD for abnormal glucose metabolism. (Refer to evidence tables for KAS 3 and 3.1.)

Insulin resistance, when assessed by the homeostatic model assessment of insulin resistance test, varies across weight categories, with highest levels observed among children with severe obesity.³⁹⁶ Some children with T2DM have rapidly progressive disease, which underscores the need for early identification and intensive treatment in collaboration with a pediatric endocrinologist.⁴²⁵

C.2.a. Laboratory Tests for the Diagnosis of Prediabetes and T2DM
Testing for T2DM should always be performed if there is suspicion of hyperglycemia in a patient with symptoms and signs of hyperglycemia, such as new onset thirst (polydipsia), frequent urination (polyuria) or new onset bedwetting, excessive hunger and eating (polyphagia), blurred vision, unexplained or unexpected weight loss, or fatigue.

Diagnostic tests for prediabetes and T2DM are fasting plasma glucose (FPG), 2-hour plasma glucose after oral glucose tolerance test (OGTT), and HbA1c.⁹⁰ There are several clinical guidelines that do not recommend one test over the other for evaluation.^{90,268,415,416,419,420,465} Pediatricians and other PHCPs need to be aware of the strengths and shortfalls of each test and take patient preferences and test accessibility into consideration. In addition, the concordance between all 3 tests is imperfect.⁴¹⁶ For instance, the FPG is highly reproducible; the OGTT, which does not fare as well on reproducibility, is effective in identifying dysglycemia. This is a good reason to use the OGTT as a confirmation test if the initial test result is equivocal.^{466–470} The OGTT, however, may not be readily available at some medical settings, requires fasting before the test, lasts at least 2 hours, and includes an

unpalatable glucose drink—all of which are factors that can limit its use in pediatric outpatient settings as an evaluation test.

The HbA1c test is easy to obtain as fasting is not required. It provides a measure of chronic hyperglycemia, and use of the test has been shown to increase evaluation for T2DM in primary care settings.⁴⁷¹ It is also the recommended test for monitoring prediabetes.^{90,414–416,418,464,472} The sensitivity of HbA1c for diagnosing diabetes is lower in children⁴⁷³ when compared with adults.^{473,474} Pediatricians and other PHCPs also need to be aware that HbA1c levels can be 0.1% to 0.2% higher in individuals with iron deficiency anemia.^{475,476}

Fasting insulin is not recommended for diagnosis of prediabetes or T2DM because the levels are highly variable

TABLE 10 Criteria for Diagnosing Prediabetes and T2DM⁹⁰

	Prediabetes or Impaired Glucose Tolerance	Diabetes Mellitus ^a
Fasting plasma glucose (FBG) ^b	100–125 mg/dL	≥126 mg/dL
2-h plasma glucose (OGTT) ^c	140–199 mg/dL	≥200 mg/dL
Random plasma glucose (RBG) ^d	Not applicable	≥200 mg/dL
HbA1c ^e	5.7% to 6.4%	≥6.5%

^a In the absence of unequivocal hyperglycemia, diagnosis is confirmed if 2 different tests are above threshold or a single test is above threshold on 2 separate occasions.

^b Fasting for at least 8 h with no calorie intake.

^c Oral glucose tolerance test (OGTT) using a load 1.75 g/kg of body weight of glucose with a maximum of 75 g.

^d In patients with hyperglycemic crises or classic symptoms of hyperglycemia (eg, polyuria, polydipsia).

^e Glycosylated hemoglobin (HbA1c) is the preferred test for monitoring prediabetes.⁴⁷⁸

TABLE 11 Risk Factors for Diagnosis and Progression of NAFLD^{88,397,433}

NAFLD	Risk Factors ^a
Diagnosis	Male sex, ≥10 y, obesity, sibling with NAFLD, prediabetes or diabetes mellitus, obstructive sleep apnea, dyslipidemia
Progression	Adolescent ≥14 y; higher or increasing alanine transaminase; elevated baseline aspartate transaminase, γ glutamyl transferase (GGT), and LDL cholesterol; prediabetes or diabetes mellitus; obstructive sleep apnea; increasing wt or waist circumference

^a Consideration should be given to groups of certain races/ethnicities with higher rates of NAFLD (eg, Hispanic, Asian), for which higher prevalence can be attributed to genetic, socioeconomic, and environmental factors.⁴⁸⁰

and do not reliably correlate with the level of insulin resistance.^{268,477}

The cut-off values are similar for pediatric and adult populations, as illustrated in Table 10, above. If the results are unequivocally high and indicative of T2DM, obtaining a second or repeat confirmatory test is not recommended; instead, treatment should be initiated.^{90,415} Guideline recommendations for tracking glycemic control over time use the HbA1c test; however, the FPG can be substituted using the cut-off criteria in Table 10. See the implementation guide for further discussion on use of OGTT or FBG tests.^{90,414,415,419,464}

See Appendix 3 for more information on frequency of evaluation and on initial management of prediabetes and T2DM.

C.3. Nonalcoholic Fatty Liver Disease

NAFLD is a chronic liver disease marked by steatosis (fat accumulation), inflammation, and fibrosis. The underlying pathogenesis is insulin resistance, which alters the process of fat oxidation in the liver, increasing oxidative stress and inflammation—with resultant liver damage. Among children with obesity, rates as high as 34% have been reported.⁴²⁹

Three diagnostic terms are used to describe the histology of the disease progression: NAFLD, nonalcoholic fatty liver (NAFL), and nonalcoholic steatohepatitis (NASH). NAFLD refers to the whole spectrum of the disorder, from mild steatosis to cirrhosis of the liver. NAFLD is

divided into steatosis (NAFL) and steatohepatitis (NASH). In NAFL, the milder form of the condition, there is fatty infiltration in ≥5% of the liver, with or without fibrosis. In NASH, there is inflammation, steatosis, and fibrosis with ballooning injury to the hepatocytes.⁴³³

The risk profile and natural history of the disorder in the pediatric population are still evolving, given that there are limited long-term studies in children. Pediatric NAFLD may reflect the early onset of a chronic disease with a more aggressive course, particularly once NASH has occurred.^{424,479} Preadolescent children with NAFLD have higher rates of mortality over 20 years, compared with their peers without NAFLD.⁴⁷⁹ Children with increasing weight gain; higher levels of alanine transaminase (ALT), γ glutamyl transferase (GGT), and cholesterol at baseline; worsening levels of HbA1c; and an incident diagnosis of T2DM are more likely to have severe disease or progression (Table 11).^{433,479} However, in a recent study of children 8 to 17 years of age with

biopsy-confirmed NAFLD who received standardized nutrition and exercise counseling consistent with the 2007 AAP Expert Recommendations at 12-week intervals over 1 to 2 years, about half demonstrated any improvement in resolution of NASH or regression of fibrosis. Among children with borderline or definite NASH, resolution occurred in about one-third. Adolescents were more likely to develop worsening steatosis and less likely to experience any resolution of NASH or regression in fibrosis than younger children.⁴³³

C.3.a. Laboratory Tests for Diagnosis of NAFLD

The 2017 North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN) clinical practice guidelines recommend ALT as the preferred test for NAFLD.⁸⁸ ALT is more specific for liver disease than aspartate transaminase (AST), easily accessible at laboratory centers, minimally invasive relative to other testing modalities for NAFLD, and has been used most often in pediatric NAFLD studies. Higher levels of ALT correlate

KAS 7. Pediatricians and other PHCPs should evaluate for NAFLD by obtaining an alanine transaminase (ALT) test.^a

Aggregate Evidence Quality	Grade A.
Benefits	Minimally invasive, fair correlation to histology.
Risks, harms, costs	Test may not correlate with disease severity.
Benefit-harm assessment	Benefit outweighs harm.
Intentional vagueness	Future research needed.
Role of patient preference	None.
Exclusions	Not applicable.
Strength	Strong.
Key references	88, 396, 481

^a Per KAS 3 and 3.1: Pediatricians and other PHCPs should evaluate children 10 y and older with obesity (BMI ≥ 95th percentile) for abnormal liver function and may evaluate children 10 y and older with overweight (BMI ≥ 85th percentile to <95th percentile) with risk factors for TD2M or NAFLD for abnormal liver function. (Refer to evidence tables for KAS 3 and 3.1.)

with more advanced liver disease with steatosis and fibrosis; however, a normal ALT does not definitively exclude NAFLD.⁴²⁹ In a population of children older than 10 years with overweight and obesity who were referred from a primary care clinic, an ALT level ≥ 80 IU/L had a sensitivity of 57% and a specificity of 71% for NASH.⁴⁸¹ Elevations in AST and GGT, especially at baseline, can be indicative of severe disease or rapid progression.^{433,481}

NAFLD is less common in children younger than 10 years. In an autopsy study of 742 children, 3.3% of 5- to 9-year-old children had fatty liver, compared with 11.3% in 10- to 14-year-olds and 17.3% in 15- to 17-year-olds.³⁹⁷ There is a higher risk for NAFLD in young children 2 to 9 years of age who have severe obesity, however.⁸⁸ Thus, pediatricians and other PHCPs may consider evaluating NAFLD by obtaining an ALT level every 2 years in these children.

See Appendix 3 for more information on the frequency of evaluation for NAFLD and on managing NAFLD.

C.4. Hypertension

The prevalence of HTN among children and adolescents with overweight and obesity ranges from 5% to 30%, with higher prevalence with increasing BMI percentile.³⁹⁶ Children with excess weight also

have abnormal diurnal variation in BP. One-third of children with obesity have a decreased nocturnal BP dip, increasing the potential risk for end-organ damage.⁴⁸² Among children with obesity, additional cardiometabolic risk factors—such as insulin resistance or dyslipidemia—may affect BP, independent of obesity.

Studies indicate that HTN during childhood and adolescence increases the risk for adult HTN and cardiovascular disease.^{483–485} More concerning, studies have shown that, among children with obesity, HTN is associated with vascular changes, increased left ventricular mass, and carotid intima media thickness during childhood.^{486,487} These findings support the importance of evaluating for HTN early and consistently throughout childhood and adolescence among individuals with overweight and obesity.^{413,488}

C.4.a. Evaluation for HTN

Obesity is the strongest risk factor for HTN in childhood.^{87,488} Elevated BP is observed in early childhood and prevalence increases with age and BMI category.³⁹⁶ A large study conducted in primary care settings found that 8% of children 3 to 5 years of age with obesity had elevated BP levels; the percentage increased to 20% among children 11 to 15 years of age.⁴⁸⁹ HTN prevalence varies by race and

ethnicity, with highest prevalence occurring among non-Hispanic Black and Hispanic youth.⁴⁹⁰ SES is also a risk factor for HTN,⁴⁹¹ as are adverse childhood experiences, both prenatally and during childhood.⁸⁷ These factors may contribute to the higher prevalence of hypertension observed among non-Hispanic Black and Hispanic youth.⁴⁹⁰

Pediatricians and other PHCPs should obtain a history of salt intake (eg, addition of salt while cooking and/or at meals) and sources of sodium from processed, frozen, and fast foods, because high sodium intake is associated with childhood HTN. Obtaining a history of physical activity and inactivity levels is also recommended, because decreased activity levels are associated with childhood HTN.⁸⁷ Finally, evaluation of sleep duration and disordered breathing are recommended because of the association between abnormal sleep duration and OSA and elevated BP.^{492,493}

This KAS aligns with the 2017 AAP's "Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents," which recommends evaluation of elevated BP and HTN for children with obesity at every clinic visit beginning at 3 years of age.⁸⁷ Frequent monitoring of BP among children with overweight and

KAS 8. Pediatricians and other PHCPs should evaluate for hypertension by measuring blood pressure at every visit starting at 3 y of age in children and adolescents with overweight (BMI ≥ 85 to < 95 th percentile) and obesity (BMI ≥ 95 th percentile).

Aggregate Evidence Quality	Grade C
Benefits	Early detection of HTN, opportunity to address weight's impact on BP and health, prevention of HTN-related morbidity.
Risks, harms, costs	Improper measurement techniques, misclassification, discomfort, time needed, possible inaccuracy during acute care visits when patient may be in pain.
Benefit-harm assessment	Benefits exceed potential harm.
Intentional vagueness	None.
Role of patient preference	Increased visit time, discomfort with cuff.
Exclusions	< 3 y of age.
Strength	Moderate.
Key references	87, 396, 413, 453, 488

TABLE 12 BP Categories by Age and Number of Visits Needed for Diagnosis

BP Category	Children 1–13 Years of Age	Children ≥13 Years of Age	Number of Visits to Diagnosis
Normal	BP < 90th percentile	BP <120/80 mm Hg	NA
Elevated	BP ≥ 90th percentile to <95th percentile	120/<80 to 129/<80 mm Hg	3
Stage 1	BP ≥ 95th percentile to <95th percentile + 12 mmHg	130/80 to 139/89 mm Hg	3
Stage 2	BP ≥ 95th percentile + 12 mm Hg	≥140/90 mm Hg	2

Used with permission and adapted from the AAP HTN CPG,⁸⁷ Fig 2, and AAP Pediatric Obesity Clinical Decision Support Chart.⁴⁹⁴ NA, not applicable.

obesity fosters earlier detection of elevated BP.

C.4.b. Diagnosis of HTN

In 2017, the AAP published a CPG on HTN that included recommendations for evaluation for elevated BP and updated HTN

definitions of “elevation,” “stage 1 BP,” and “stage 2 BP” (see Table 12). This CPG recommended that an elevated initial BP measurement (≥90th percentile), taken either by oscillometry or auscultation, should be repeated twice with auscultation and averaged, at the same visit, to

determine accurate BP measurement and category. For diagnosis, BP by auscultation should be repeated with confirmed elevated BP measurements on 3 separate clinic visits for elevated BP and stage 1 HTN, and on 2 separate visits for stage 2 HTN.⁸⁷

TABLE 13 Summary of KASs for Evaluation of Comorbidities Among Children and Adolescents With Overweight and Obesity

KAS #	Key Action Statement (KAS)	Evidence Quality, Recommendation Strength
A. laboratory evaluation of obesity-related comorbidities		
3	In children 10 y and older, pediatricians and other PHCPs should evaluate for lipid abnormalities, abnormal glucose metabolism, and abnormal liver function in children and adolescents with obesity (BMI ≥ 95th percentile) and for lipid abnormalities in children and adolescents with overweight (BMI ≥ 85th percentile to < 95th percentile)	B, Strong
3.1	In children 10 y and older with overweight (BMI ≥ 85th percentile to < 95th percentile), pediatricians and other PHCPs may evaluate for abnormal glucose metabolism and liver function in the presence of risk factors for T2DM or NAFLD. In children 2 to 9 y of age with obesity (BMI ≥ 95th percentile), pediatricians and other PHCPs may evaluate for lipid abnormalities.	C, Moderate
B. Concurrent treatment of obesity and obesity-related comorbidities		
4	Pediatricians and other PHCPs should treat children and adolescents for overweight (BMI ≥ 85th percentile to < 95th percentile) or obesity (BMI ≥ 95th percentile) and comorbidities concurrently.	A, Strong
C. Evaluation for diagnosis of dyslipidemia, prediabetes, T2DM, NAFLD, and hypertension		
5	Pediatricians and other PHCPs should evaluate for dyslipidemia by obtaining a fasting lipid panel in children 10 y and older with overweight (BMI ≥ 85th percentile to < 95th percentile) and obesity (BMI ≥ 95th percentile) and may evaluate for dyslipidemia in children 2 through 9 y of age with obesity.	B, Strong (10 y and older); C, moderate (2–9 y of age)
6	KAS 6. Pediatricians and other PHCPs should evaluate for prediabetes and/or diabetes mellitus with fasting plasma glucose, 2-h plasma glucose after 75-g oral glucose tolerance test (OGTT), or glycosylated hemoglobin (HbA1c). ^a	B, Moderate
7	KAS 7. Pediatricians and other PHCPs should evaluate for NAFLD by obtaining an alanine transaminase (ALT) test. ^b	A, Strong
8	Pediatricians and other PHCPs should evaluate for hypertension by measuring blood pressure at every visit starting at 3 y of age in children and adolescents with overweight (BMI ≥ 85 percentile to < 95th percentile) and obesity (BMI ≥ 95th percentile).	C, Moderate

^a Per KAS 3 and 3.1: pediatricians and other PHCPs should evaluate children 10 y and older with obesity (BMI ≥ 95th percentile) for abnormal glucose metabolism and may evaluate children 10 y and older with overweight (BMI ≥ 85th percentile to <95th percentile) with risk factors for T2DM or NAFLD for abnormal glucose metabolism. (Refer to evidence tables for KAS 3 and 3.1.)

^b Per KAS 3 and 3.1: pediatricians and other PHCPs should evaluate children 10 y and older with obesity (BMI ≥ 95th percentile) for abnormal liver function and may evaluate children 10 y and older with overweight (BMI ≥ 85th percentile to <95th percentile) with risk factors for T2DM or NAFLD for abnormal liver function. (Refer to evidence tables for KAS 3 and 3.1.)

BP measurements should be taken with an appropriately sized cuff; the bladder length should be 80% to 100% of the circumference of the arm, and the width should be at least 40% of the arm circumference.⁸⁷ (See https://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf)

For children and adolescents with excess weight, a larger cuff size may be required to obtain accurate measurements. For children and adolescents with severe obesity, a thigh cuff may be needed. Additionally, for children and adolescents with obesity, ambulatory blood pressure monitoring (ABPM) is recommended to assess HTN severity and identify possible abnormal circadian BP patterns, which increases risk for end-organ damage. ABPM also helps to identify masked HTN and/or “white coat” HTN.⁸⁷

Elevated BP in the office setting is unrecognized in approximately 25% of cases.⁴⁹⁵ The AAP’s CPG on HTN provides pediatricians and other PHCPs with practical tools to assist with identification of elevated BP and HTN. Improved identification of children at high risk and youth allows for a thorough evaluation, treatment, and follow-up, with the goal of decreasing long-term cardiovascular morbidity and mortality.

See Appendix 3 for more information on repeat evaluation for HTN and on management of HTN. Table 13 lists the KASs for the comorbidities covered in the TR.

D. Other Comorbidities

D.1. Obstructive Sleep Apnea

OSA is a sleep disorder “characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts normal ventilation during sleep.”⁸⁹ The condition is associated with cardiovascular complications, neurocognitive impairment, and decreased quality of life. Children with obesity have a higher prevalence of OSA: 45% among children obesity compared with 9% among children with healthy weight. One study indicated that a 1-unit increase in the BMI SD score increased the odds of having OSA by a factor of 1.9 independent of age, sex, tonsillar hypertrophy, and asthma.⁴⁴¹

Evaluation for OSA is based on history of symptoms and examination. Children with obesity, tonsillar hypertrophy, craniofacial anomalies, trisomy 21, and neuromuscular disorders are at higher risk for OSA. Common symptoms include frequent snoring, gasps or labored breathing during sleep, disturbed sleep, daytime

sleepiness, inattention and/or learning problems, nocturnal enuresis, and headaches. Examination findings may include tonsillar hypertrophy, adenoidal facies, micro- or retrognathia, high-arched palate, and elevated BP. Diagnosis is made by obtaining a polysomnography, the gold standard test, with an apnea-hypopnea index of 1 or more episodes per hour in children.⁴⁹⁶ Because of limited availability of sleep centers with pediatric expertise, referral to a pediatric otolaryngologist for further evaluation, diagnosis, and management may be needed.

Consensus Recommendations

The CPG authors recommend pediatricians and other PHCPs obtain:

- A sleep history, including symptoms of snoring, daytime somnolence, nocturnal enuresis, morning headaches, and inattention, among children and adolescents with obesity to evaluate for OSA.
- A polysomnogram for children and adolescents with obesity and at least 1 symptom of disordered breathing.

See Appendix 3 for more information on the initial management of OSA.

TABLE 14 Definitions and Criteria for PCOS

Definition	Diagnostic Criteria
National Institutes of Health	Requires the presence of: <ol style="list-style-type: none"> 1. Hyperandrogenism (clinical and/or biochemical) 2. Ovarian dysfunction
American Society of Reproductive Medicine (Rotterdam)	Requires the presence of at least 2 criteria: <ol style="list-style-type: none"> 1. Hyperandrogenism (clinical and/or biochemical) 2. Ovulatory dysfunction 3. Polycystic ovarian morphology
American Endocrine Society	Requires the presence of hyperandrogenism (clinical and/or biochemical) and either: <ol style="list-style-type: none"> 1. Ovulatory dysfunction 2. Polycystic ovarian morphology
Androgen Excess and Polycystic Ovary Syndrome Society	Requires the simultaneous presence of: <ol style="list-style-type: none"> 1. Hyperandrogenism (clinical and/or biochemical) 2. Ovarian dysfunction (ovulatory dysfunction and/or polycystic ovarian morphology)

All of the diagnostic criteria for PCOS require the exclusion of other disorders of adrenal excess, such as nonclassic or late-onset congenital adrenal hyperplasia, Cushing syndrome, hyperprolactinemia, hypothyroidism, acromegaly, premature ovarian failure, a virilizing adrenal or ovarian neoplasm, or a drug-related condition.

D3. Polycystic Ovarian Syndrome

Polycystic ovarian syndrome (PCOS) is a heterogeneous disorder characterized by hyperandrogenism and disordered ovulatory function and is often associated with obesity and insulin resistance. The condition increases risk for infertility, T2DM, cardiovascular disease, and cancer.⁴⁹⁷

Four different sets of criteria have been published for diagnosis of PCOS in adults as outlined by differing professional organizations (Table 14).⁴⁹⁸ Establishment of diagnostic criteria for PCOS in adolescence has been difficult, because characteristic features of PCOS can be normal physiologic events during early adolescence.⁴⁹⁹ International pediatric and adolescent specialty societies have made recommendations for diagnosis specific to adolescents, which include the following: (1) evidence of clinical or biochemical hyperandrogenism, and (2) persistent irregular menstrual cycles (<20 days or >45 days) 2 years after menarche.⁵⁰⁰ Limited data are available on prevalence of PCOS in adolescents. Estimates range from 3% to 11%, depending on the criteria for diagnosis.⁵⁰¹

Evaluation for PCOS in an adolescent requires first excluding other medical conditions that may cause menstrual dysfunction (oligomenorrhea or amenorrhea) and/or signs of androgen excess (acne, hirsutism, or alopecia). Additionally, for adolescents, evaluation should occur 2 years after menarche, because irregular menstrual cycles are not uncommon during this timeframe. Laboratory testing may include: 17-hydroxyprogesterone, total testosterone, free testosterone, sex hormone-binding globulin, dehydroepiandrosterone sulfate, androstenedione, luteinizing hormone, follicle-stimulating hormone, estradiol,

prolactin, free thyroxine, thyroid stimulating hormone, and insulin. Interpretation of laboratory results should be made in the context of age-appropriate reference ranges; therefore, referral to a laboratory that can perform ultrasensitive pediatric assays is recommended. Routine ovarian imaging is not indicated for the diagnosis of PCOS in adolescents.^{498,502} An algorithm for evaluation is provided in the implementation materials from previously published consensus recommendations.⁵⁰³

See Appendix 3 for more information on the initial management of PCOS.

Consensus Recommendation

The CPG authors recommend pediatricians and other PHCPs:

- Evaluate for menstrual irregularities and signs of hyperandrogenism (ie, hirsutism, acne) among female adolescents with obesity to assess risk for PCOS.

D4. Depression

The relationship between pediatric obesity and depression is less well understood than the physical comorbidities; however, identification of depression is an important component of the assessment and management of pediatric obesity, given its potential impact on treatment outcomes. A systematic review and meta-analyses conducted in 2019 showed that children 18 years and younger with obesity have a 32% increased odds of having or developing depression compared with children of healthy weight, with the highest odds (44%) among females with obesity.⁵⁰⁴

Studies are limited on the effect of treatment of pediatric obesity on depression. A recent meta-analysis of 36 studies found a small but

significant reduction in depressive symptoms following structured pediatric obesity treatment. Notably, no adverse mental health outcomes were reported.⁵⁰⁵ Additionally, the interventions technical report indicates that obesity treatment may improve psychosocial outcomes for youth with obesity, including quality of life.⁸⁰ Further research in this area is needed; however, pediatricians and other PHCPs should be aware that obesity treatment interventions have not been associated with increased symptoms of depression.⁸⁰

Evaluation for depression includes awareness of symptoms and risk factors. Symptoms include irritability, fatigue, insomnia, excessive sleeping, decline in academic performance, family conflict, and weight changes. Risk factors include personal or family history of depression, substance use, trauma, frequent psychosomatic complaints, psychosocial stressors, and other mental health conditions. The AAP CPG for depression recommends evaluating adolescents 12 years and older for depression annually using a formal self-report tool, such as the Patient Health Questionnaire-9.⁵⁰⁶ Additionally, routine monitoring of psychosocial function and using an evaluation tool when a patient presents with symptoms of depression is recommended.

If initial evaluation for depression is positive, evaluation with a standardized depression tool should be conducted. Assessment for depression should also include direct, separate interviews with the patient and family members to include functional impairment at home, school, and peer settings and safety and/or suicide risk.⁵⁰⁶ The implementation materials include additional information and resources, including tools for pediatricians and other PHCPs, in

addition to an assessment and management algorithm.⁵⁰⁶

See Appendix 3 for more information on the initial management of depression.

Consensus Recommendation

The CPG authors recommend pediatricians and other PHCPs:

- Monitor for symptoms of depression in children and adolescents with obesity and conduct annual evaluation for depression for adolescents 12 years and older with a formal self-report tool.

D5. Orthopedic Comorbidities

D.5.a. Slipped Capital Femoral Epiphysis

Slipped capital femoral epiphysis (SCFE) is the most common hip disorder in the adolescent period. It occurs between 9 and 16 years of age, spanning periods of rapid linear growth. There is a 1.5:1 male-to-female ratio, and SCFE occurs more often in Black, Hispanic, and AI/AN children.^{507,508} SCFE is bilateral in 25% to 80% of cases.⁵⁰⁷ Weakening of the proximal femoral physis (growth plate) causes a slip in the physis, with a corresponding displacement of the epiphysis (femoral head). Risks such as obesity exert mechanical stress on the physis, whereas metabolic conditions (eg, hypothyroidism, hypopituitarism) weaken the physis, creating the ideal setup for a slip.

The common presentation is hip pain, although many children may

present with knee pain alone or in addition to hip pain. The pain can happen only with weight bearing or be constant. On physical examination, there is external rotation with passive hip flexion, limitation of internal rotation and antalgic gait. Pain can also be elicited passively with internal rotation of the hip. SCFE is characterized as stable if the child can bear weight with or without crutches and as unstable when weight-bearing is not possible.⁵⁰⁹ Because SCFE can be bilateral, the pediatrician or other pediatric health care provider needs to remember to obtain a history and exam for the contralateral leg. The history and examination should also exclude differential diagnoses for hip pain (eg, infections, inflammation or autoimmune conditions, neoplasms, and trauma).

As the pathophysiologic process continues, the child is at greater risk for increased morbidity, including avascular necrosis. Thus, the importance of early diagnosis cannot be overemphasized. Once SCFE is suspected, pediatricians and other PHCPs should confirm the diagnosis and place an emergent referral to the orthopedic surgeon. The mainstay for diagnosis is plain radiographs of the hip and pelvis (Table 15). Ultrasonography and computerized tomography are not useful. In cases with equivocal radiography results and a high index of suspicion, MRI, which is more sensitive at assessing the physis, can be obtained.

See Appendix 3 for more information on the initial management of SCFE.

D.5.b. Blount Disease

Blount disease is a growth disorder that primarily affects the proximal medial tibial physis and epiphysis.^{510,511} It often presents as a triad of asymmetric tibia vara, tibial torsion, and procurvatum. As with SCFE, excess weight is a risk factor, because it increases mechanical stress on the physis. Blount disease disproportionately affects non-Hispanic Black or Hispanic children.^{510,511} The reason for this predilection is unclear, but it may reflect epigenetic, social, or cultural factors that affect early ambulation, growth, or obesity. Other risk factors include a family history of Blount disease and ambulation before 12 months of age.^{510,511} Symptoms and signs include leg pain, abnormal gait with bowing of the lower legs, and leg-length discrepancy.

Blount disease is classified into 2 categories: (1) infantile or early-onset, and (2) late-onset or adolescent Blount disease, based on whether the onset occurred before or after age 10 years, respectively. Infantile Blount disease is bilateral but asymmetric, occurs more often in males, and often includes a preceding history of early ambulation. For young children, pediatricians and other PHCPs should exclude physiologic bowing typically seen during toddlerhood, which is bilateral but symmetrical and resolves by age 3 or 4 years. In

TABLE 15 Recommended Imaging for Slipped Capital Femoral Epiphysis and Blount Disease

Condition	First-Line Imaging	Additional Tests
SCFE	Bilateral hip (anteroposterior and lateral), frog-leg radiographs	If SCFE is unstable, cross-table lateral radiograph. MRI for equivocal imaging results, or assess blood supply to femoral head.
Blount	Long leg (anteroposterior and lateral), knee (anteroposterior and lateral) radiographs	MRI of the knee to delineate level and extent of deformity, assess blood supply to physis. ^a

^aAdditional tests are determined by orthopedic surgeon.

the adolescent subtype, the tibia vara deformity is milder, unilateral, and predominantly associated with severe obesity.⁵¹⁰⁻⁵¹³

Plain radiographs are the initial imaging of choice (Table 15). When used, MRI provides a more sensitive investigation of the deformity.

See Appendix 3 for more information on the initial management of Blount disease.

Consensus Recommendations

The CPG authors recommend pediatricians and other PHCPs:

- Perform a musculoskeletal review of systems and physical examination (eg, internal hip rotation in growing child, gait) as part of their evaluation for obesity.
- Recommend immediate and complete activity restriction, nonweight-bearing with use of crutches, and refer to an orthopedic surgeon for emergent evaluation, if SCFE is suspected. PHCPs may consider sending the child to an emergency department if an orthopedic surgeon is not available.

D.5.c. Idiopathic Intracranial Hypertension

Idiopathic intracranial hypertension (IIH) (previously known as pseudotumor cerebri) is a neurologic condition with serious long-term morbidity.^{514,515} It occurs most often in females of child-bearing age, and obesity is a well-established risk factor.⁵¹⁶⁻⁵¹⁸ In a

population-based study in Olmstead County, Minnesota, the incidence of IIH among adult females aged 15 to 44 years with obesity was 3.5-fold higher than that of all females in that age group.⁵¹⁷

The pathogenesis is unclear, hence the name; however, 3 hypothesized mechanisms are increased venous pressure, decreased cerebrospinal fluid (CSF) drainage, and increased CSF production. Other factors associated with for IIH include medications (eg, doxycycline, tetracyclines, retinoic acid, sulfonamides), autoimmune disorders (eg, systemic lupus erythematosus), and hormonal disorders (eg, Cushing disease, Addison disease). A higher prevalence of IIH has also been reported in females with PCOS.⁵¹⁹

Typical symptoms are persistent headaches, pulsatile synchronous tinnitus, and visual changes or loss, although the history can be variable. Physical examination includes a fundoscopy for papilledema and a thorough neurologic evaluation for cranial nerve deficits such as sixth nerve palsy. The presence of altered consciousness or neurologic deficit with localized peripheral findings should prompt pediatricians and other PHCPs to consider another etiology. The serious sequelae for IIH is vision loss. Thus, a review of system should be obtained to evaluate any child with obesity who has significant or progressive

headaches. There should also be a high index of suspicion for IIH with new-onset headaches and significant weight gain (5% to 15% of body weight), particularly when it occurs in the prior 12 to 18 months.^{518,520}

Initial evaluation for IIH involves conducting comprehensive evaluation by the neurologist and ophthalmologist or at an integrated IIH clinic.

See Appendix 3 for more information on the initial management of IIH.

Consensus Recommendation

The CPG authors recommend pediatricians and other PHCPs:

- Maintain a high index of suspicion for IIH with new-onset or progressive headaches in the context of significant weight gain, especially for females.

D.6. Summary

In summary, a thorough history and physical examination is invaluable in guiding pediatricians' and other PHCPs' assessment for comorbidities. This section of the CPG, the algorithm in Appendix 1, Appendix 3, and the accompanying implementation resources provide a framework for evaluation, reevaluation, and initial management. Obesity is a linchpin disorder with attendant comorbidities, some of which are not covered in this section or in the technical report (eg, pes planus). For these comorbidities,

KAS 9. Pediatricians and other PHCPs should treat overweight (BMI ≥ 85th percentile to <95th percentile) and obesity (BMI ≥ 95th percentile) in children and adolescents, following the principles of the medical home and the chronic care model, using a family-centered and nonstigmatizing approach that acknowledges obesity's biologic, social, and structural drivers.

Aggregate Evidence Quality	Grade B
Benefits	Early intervention, reduction of comorbidities, long-term health care cost savings, efficiency with coordinated care.
Risks, harms, costs	Time, resources, and cost to family, provider, and clinical practice. Stigma felt by patient and parent.
Benefit-harm assessment	Benefits outweigh the harms.
Intentional vagueness	Frequency of monitoring and duration of longitudinal care not solidified by scientific literature.
Role of patient preference	Family and patient's willingness to discuss weight, attend weight-related visits, undergo obesity-related clinical and laboratory evaluation.
Exclusions	None.
Strength	Strong.
Key references	521

pediatricians and other PHCPs are encouraged to seek resources available through the AAP and other professional societies.

XI. TREATMENT OF CHILD AND ADOLESCENT OVERWEIGHT AND OBESITY

A. Obesity is a Chronic Disease

Obesity is a chronic disease and should be treated with intensive and long-term care strategies, provision of ongoing medical monitoring and treatment of associated comorbidities and ongoing access to obesity treatment. As noted previously, obesity is associated with increased prevalence of comorbidities, including abnormal lipids, glucose dysregulation and other endocrinopathies, abnormal liver enzymes, and elevated BP. A key component of treating obesity is to concurrently monitor and treat the comorbidities (see Comorbidities section).

The chronic care model requires care to be delivered within the context of individual patient factors, taking into consideration the child's household and familial influences, access to healthy food and activity spaces, and other SDoHs.⁵²² Recommendations for obesity treatment should be integrated within existing community and social systems.⁵²¹ The medical home model is the preferred standard of care for children who have chronic conditions,⁵²² and the child's medical home should serve as a care coordinator in the treatment of children with obesity, coordinating with subspecialists, including obesity treatment specialists, and community resources.

Treatment of obesity varies based on individual-level factors. No specific studies were found that compare different treatments by a patient's underlying condition, special needs, or developmental

status. Nonetheless, it is important to recognize that the following recommendations will require adaptation based on the patient's unique medical, family, developmental, social, and environmental factors. No evidence exists, however, to exclude children with special health care needs, complex disease, or developmental limitations from the treatment options outlined below, except where specifically noted.

The evidence for pediatric obesity treatment that is presented in this CPG shows that several treatments are effective in treating both obesity and related comorbidities. It is important to note, however, that in all of these studies, if the treatment is discontinued, children tend to regain weight and lose the attendant health benefits. There is limited longitudinal evidence about durability of weight change after treatment. The natural course of obesity across the lifespan is characterized by responses to treatment and relapse when treatment ends.⁷⁷ Therefore, continuous coordinated care is required to support ongoing obesity treatment throughout childhood and adolescence into young adulthood.⁵⁰³

B. Evidence-based Pediatric Obesity Treatment Reduces Risks for Disordered Eating

In the field of pediatric nutrition, in the treatment of both obesity and eating disorders, concerns have been raised as to whether diagnosis and treatment of obesity may inadvertently place excess attention on eating habits, body shape, and body size and lead to disordered eating patterns as children grow into adulthood. The literature refutes this relationship, however. Cardel et al refer to multiple studies that have demonstrated that, although obesity and self-guided dieting consistently place children at

high risk for weight fluctuation and disordered eating patterns, participation in structured, supervised weight management programs decreases current and future eating disorder symptoms (including bulimic symptoms, emotional eating, binge eating, and drive for thinness) up to 6 years after treatment.^{378,505,523} The structure and underlying principles of the primary care-based and intensive health behavior and lifestyle programs described here share multiple similarities with eating disorder programs. These include a focus on increasing healthful food consumption, participating in physical activity for enjoyment and self-care reasons, and improving overall self-esteem and self-concept. Structured and professionally run pediatric obesity treatment is associated with reduced eating disorder prevalence, risk, and symptoms.^{505,523}

C. Motivational Interviewing

MI (also discussed in the Evaluation section) is a patient-centered counseling style that identifies and reinforces a patient's own motivation for change—in contrast to the more traditional approach in which a provider prescribes behavior change. MI guides families to identify a behavior to change, based on what the parent(s) or child feels is important and can be accomplished.⁵²⁴

MI does not impose a particular goal but is successful when the family changes the selected behavior—which could be nutritional, such as reducing sugar-sweetened beverages; increasing physical activity; or engaging in other behaviors, such as eating meals together or improving sleep hygiene. The target of MI is the person who is responsible for the behavior change. Pediatricians and other PHCPs focus on parent motivation

when patients are preadolescent or younger, and transition to patient motivation, usually combined with parent motivation, when patients reach adolescence.

MI consists of 4 processes: engaging, focusing, evoking, and planning. These processes, described below, are particularly salient when discussing weight status and devising a change plan.⁵²⁴

The MI process of *engaging* is facilitated by the existing medical pediatrician-patient relationship. Through engaging, MI can help answer the question of whether to attempt behavior change. Attempts at obesity treatment often fail because of a disconnect between pediatricians and other PHCPs who see impending health problems with regard to weight status and a caregiver who sees a thriving, growing child. The presence of other challenges—such as financial constraints and other SDOHs, mental illness, or competing health considerations—may make obesity treatment a low priority.

An evaluation of these factors is necessary before any action. The longitudinal nature of many pediatrician-family relationships can enable ongoing monitoring of weight, health status, and family challenges. More focused obesity treatment efforts can ensue when

families are ready. And regardless of their readiness to engage in more “active” obesity treatment, all patients need follow-up, encouragement, and monitoring of health status.

The MI process of *focusing* furthers respect for the autonomy of the patient and family. Behavior change is the patient’s and family’s decision, not the pediatrician’s or other PHCP’s. Identifying behaviors to change is a collaborative process. Caregivers and pediatricians (or other pediatric health care providers) tend to feel that the locus of control for pediatric behavior change resides with the caregiver longer than it actually does, however. Between 6 and 12 years of age, a steady shift of control occurs from caregiver to patient. By the early teen years, the vast majority of behavioral decisions reside in the patient—not the caregiver. Therefore, the patient should increasingly be the target of the *readiness to change* assessment and *focusing* as they age.

The MI principle of *evocation* advances the autonomy of the patient and family. Pediatricians and other PHCPs can evaluate values that are important to the patient and family. Speaking to an adolescent patient who is more concerned about athletic performance than health,

pediatricians and other PHCPs may have better traction discussing what the patient hopes to accomplish physically than attempting to incite concern about potential future disease. Likewise, encouraging a very self-conscious patient to exercise in a public setting may come across poorly. Taking time to evaluate individuals’ values, goals, and barriers is a critical piece of assessing *readiness to change*.

With regard to the MI process of *planning*, pediatricians and other PHCPs can evaluate a patient’s knowledge of what is necessary for a particular strategy and what resources and support are available to them. As a consultant, pediatricians and other PHCPs play a crucial role providing support and guidance for the patient’s collaboratively chosen course of action. Because obesity treatment is characterized by frequent setbacks and relapses, pediatricians and other PHCPs can also serve as valuable experts who can assess why behaviors may have reverted and what strategies might be appropriate for patients who seek to “get back on track.”

Table 16 summarizes MI processes as a way of evaluating and responding to patient *readiness to change*. Note that the MI tools are suggestions; in practice, each tool

TABLE 16 Possible Use of MI to Evaluate and Respond to Readiness to Change³⁹²

MI Process	Phase of Evaluation	Goal	Possible MI Tool
Engaging	Early, getting to know patient	Establishing collaborative role, understanding patient issues	Open-ended questions, affirmations, nonjudgmental graphics, empathic reflections
Focusing	Early and when desire to change weight status is expressed	Identifying appropriate and productive strategies to change weight status	Readiness ruler, elicit-provide-elicited, healthy habits survey, identifying and responding to change talk and sustain talk
Evoking	When behavior change is desired	Triggering internal motivation, empowering change	Values statement, double-sided and amplified reflections
Planning	When embarking on change	Carrying out effective change plan, dealing with relapse	Readiness ruler, action reflections, summarization, teach back, SMART goals (specific, measurable, achievable, realistic, and timely)

Aggregate Evidence Quality	Grade B
Benefits	Improvement in BMI, including in low-intensity interventions.
Risks, harms, costs	Minimal. Use of MI can add some time to the standard visit.
Benefit-harm assessment	Benefit outweighs risk.
Intentional vagueness	None.
Role of patient preference	Patient preference is central.
Exclusions	Patients who are not responsible for their behavior change, such as children who are young or with developmental or cognitive impairment. In these situations, MI should target the parent or other caregiver.
Strength	Moderate.
Key references	525

may find utility in every phase of evaluation.

C.1. Motivational Interviewing and Weight Status

Prospective studies specifically examining MI have demonstrated that the approach has positive effect on weight status, compared with controls.⁵²⁵⁻⁵²⁸ The outcomes included greater decline in BMI percentile or BMI SD score (also known as z-score), and less of an increase in BMI. These studies were all low-intensity treatments (ie, less than 5 hours) that were delivered in pediatric primary care practices by medical providers and dietitians who successfully learned and used MI.

MI is a tool used with many different strategies aimed at encouraging nutrition and physical activity change, and so the use of MI does not guarantee effect. Tables 2 and 3 in the technical report on interventions (<https://doi.org/10.1542/peds.2022-060642>) provide an overview of all the programs, including impact and use of MI. Of the 2 additional effective

low-intensity studies (not aimed at MI evaluation), 1 included MI in both arms⁵²⁹ and 1 did not use MI.⁵³⁰

Approximately 23 low-intensity studies were ineffective, of which 14 included MI.⁵³¹⁻⁵⁵¹ These programs varied in participant age, sample size, duration, and other components.

Among moderate-intensity interventions (5–25 hours), about one-third of the approximately 20 effective interventions used MI.⁵⁵²⁻⁵⁷⁴

Conversely, approximately one-quarter of the ineffective programs used MI.^{123,125,150,151,567,575-611}

Use of MI in high-intensity programs is more difficult to interpret, because many of these studies resulted in several publications at different outcomes points, with discrepant effects. Some used MI in both study arms.^{439,610,612-614} An estimated one-quarter of the effective high-intensity programs used MI, however.^{439,612,613,615-622}

Although much more work is needed to examine the optimal characteristics that might moderate MI's impact, like training, fidelity to the MI process, potential patient characteristics, as well as target behaviors, the success of the studies in which MI was the core treatment supports this KAS.

D. Intensive Health Behavior and Lifestyle Treatment

IHBLT is the foundational approach to achieve body mass reduction or the attenuation of excessive weight gain in children. It involves visits of sufficient frequency and intensity to facilitate sustained healthier eating and physical activity habits.⁷⁹ IHBLT typically involves engagement with, and participation of, families in discussions of necessary treatment based on the severity of disease. It also involves interaction with pediatricians and other PHCPs who are trained in lifestyle-related fields and requires significantly more time and resources than are typically allocated to routine well-child care.⁶²³

TABLE 17 Treatment Intensity and BMI Reduction in Randomized Controlled Trials

Authors	Study Population	BMI Outcomes	Duration
Wilfley (2017) ⁶¹⁵	US; ages 7–11; OW/OB; n = 172	Not reported; “–6.71 percent overweight” at 8 mo	26–51 h per 8 mo
Butte (2017) ⁶⁴²	US; ages 2–12; OW/OB; n = 549	–0.42 at 12 mo; only effective in ages 6–8	>52 h per 12 mo
Nemet (2005) ⁶²¹	Israel; ages 6–16; OB; n = 46	–2.2 at 12 mo	26–51 h per 3 mo
Savoie (2007) ⁴³⁹	US; ages 8–16; OB; n = 174	–3.3 at 12 mo, –2.8 at 24 mo	>52 h per 12 mo
Vos (2011) ⁶¹⁷	Germany; ages 7–15; OB; n = 73	–0.2 (BMIz) at 12 mo	26–51 h per 3 mo
Weigel (2008) ⁶¹²	Germany; ages 7–15; OB; n = 73	–4.3 at 12 mo	>52 h per 12 mo
Reinehr (2010) ⁶⁴³	Germany; ages 7–15; OW/OB; n = 66	–1.5 at 6 mo	>52 h per 6 mo

OW, overweight; OB, obese.

There are known limitations for families to access and participate in IHBLT. These limitations include the relative scarcity and distribution of such treatment programs and pediatricians or other pediatric health care providers with experience and/or training in pediatric obesity treatment, family transportation challenges, loss of school or work time to attend multiple recurring appointments during what are typically working hours, SDoHs, competing health issues for children or family members, and mismatched expectations between the family (who may expect significant weight loss) and pediatricians or other pediatric health care providers.⁶²⁴ IHBLT is appropriate for typically developing children and adolescents as well as CYSHCN, although will require modification based on the patient's unique health conditions and developmental factors.










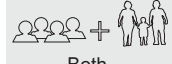


The most consistently effective IHBLT programs deliver 26 or more hours of face-to-face, family-based counseling on nutrition and physical activity over at least a 3- to 12-month period, for children aged for children 6 years and older with overweight and obesity, with more limited evidence for children 2 to 5

years of age.⁶²⁵ Although not universally available, treatment programs that provide engaging, group-based physical activity and nutrition programming are currently available in various forms across the United States.^{222,626} Some IHBLT are housed in academic medical centers or community hospitals, some are in primary care offices or obesity treatment specialty clinics (ie, weight management clinics), and others are delivered through partnerships with local community entities such as the YMCA⁶²⁷ or parks and recreation departments.⁶²⁸ Clinic-community partnerships in particular have demonstrated implementation feasibility and are engaging to low-income and racially diverse populations.⁶²⁹ Pediatricians and other PHCPs should investigate local programs in the area and become familiar with the referral requirements and processes to connect patients with this treatment option (Fig 3).

Each of the components of the KAS below are supported by evidence that is detailed in the technical report on interventions and summarized here:

- **26 or more hours:** The major factor driving the effectiveness of IHBLT is the intensity (or dose) of the intervention, measured in hours of face-to-face patient contact. The number of hours, or “dose,” delivered is directly proportional to the likelihood a child will experience a reduction in BMI (Table 17). Although a threshold effect was observed at 26 or more hours over a 3- to 12-month period, interventions that delivered ≥ 52 hours of contact over the same duration demonstrated the most consistent and significant reduction in BMI and cardiometabolic comorbidity improvement.⁶²⁵ As described in the technical report, 28% of treatments < 5 hours,^{525-527,529, 531-535,537-540,546-549,630} 38% of treatments 5 to 25 hours,^{527,537,554, 555,557,559,560,563,567,568,570,572,573, 575-583,602} and 75% of treatments ≥ 26 hours^{290,554,555,557,559, 560,563,567,568,570,572,573,575-583,602, 604-606,612,617,618,621,631} led to significant improvements in BMI among pediatric participants.⁸⁰ Hence, pediatricians and other PHCPs should look for programs that engage families often and frequently. No studies directly compare the same intervention

Intensive Health Behavior and Lifestyle Treatment (IHBLT)

WHO:	WHEN:	WHAT:	WHERE:	DOSAGE:	FORMAT:	CHANNEL:
 Patient and family in partnership with a multidisciplinary treatment team*	 Promptly for child or adolescent with overweight or obesity	 Health education and skill building on multiple topics  Behavior modification and counseling	 Healthcare setting  Community-based setting with linkage to medical home	 Longitudinal treatment across 3-12 months with ideally ≥ 26 contact hours	 Group,  Individual, or  Both	 Face-to-face (strongest evidence)  Virtual (growing evidence)

* PCPs and/or PHCPs with training in obesity as well as other professionals trained in behavior and lifestyle fields such as dietitians, exercise specialists and behavioral health practitioners

FIGURE 3
Intensive health behavior and lifestyle treatment.

over a shorter versus longer period of time, however.

- **Face-to-face:** Most of the studies included interventions where IHBLT occurred in group settings where families gathered together in a health care or community location, or in a family's home as part of a home visit. Sessions were led by a variety of individuals or combinations of individuals, including community health workers, nutritionists, exercise physiologists, physical therapists, and social workers. Fewer studies evaluated the effectiveness of treatment that did not take place in a face-to-face setting, including mobile health tools for parents or adolescents,^{530,632} telemedicine-delivered counseling sessions,⁵⁵⁷ or

guided self-help for families.⁵⁷⁸ Although there is promising evidence that these strategies may be successful, more research is needed to understand the target population, effectiveness on health outcomes, and implementation potential.

- **Family-based:** In all effective studies, the parent or the family unit was included in the treatment. Prior evidence has demonstrated that parent involvement is associated with early success in child obesity treatment⁶³² and that family-based interventions are more effective in achieving and sustaining child BMI reduction than interventions that target the child without including the family (ie, school-based,

summer camp, after school).⁶³³ For adolescent populations, the evidence and best practices for including parents in obesity treatment is less clear.⁶³⁴ Several studies measure parental BMI as a treatment outcome, along with adolescent BMI, although none included in the technical report have demonstrated significant reduction in parent BMI.^{549,566,568,569,578,563,606,628} Obesity tends to affect families; thus, family-based treatment has the potential to improve the health and weight status of other household members, including siblings, although no data are available to support this outcome.

- **Multicomponent:** Nearly all of the evidence for effective treatment

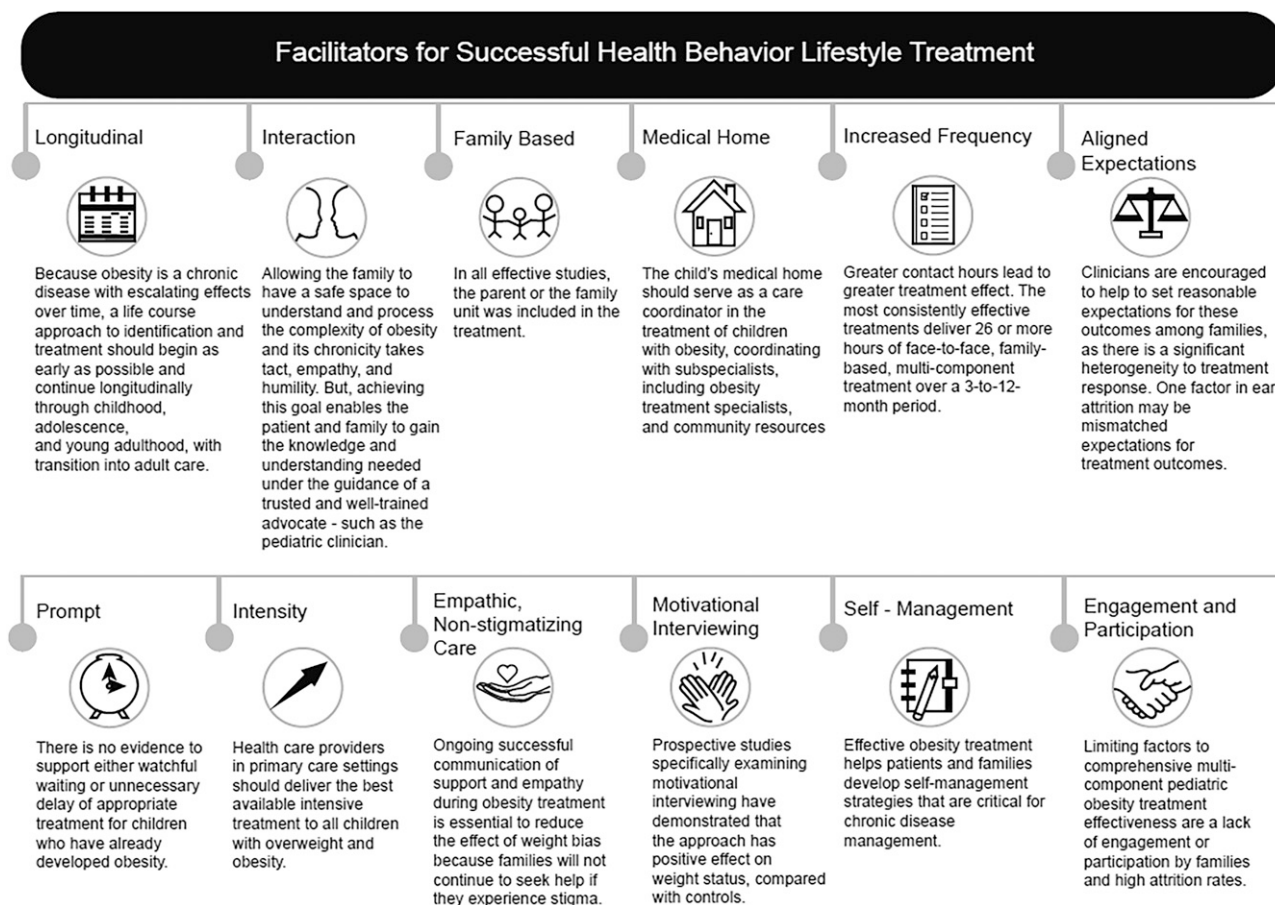


FIGURE 4

Facilitators for successful health behavior lifestyle treatment (this figure highlights some of the factors that are associated with successful health behavior and lifestyle treatment).

of childhood obesity includes components that focus on healthy eating and physical activity. The specific nutritional and exercise content varies widely among studies, and is delivered in different combinations, so no one unified approach has demonstrated superiority over another.

The physical activity component is more effective when children are engaged in a combination of aerobic and nonaerobic physical activity,⁶³⁵ compared with physical activity counseling. Noncompetitive, cooperative, fun activities that build motor skills as well as self-confidence are more engaging for children. Several studies have noted adaptations for children with obesity, including a preference for water-based activities and nonweight-bearing activities (ie, cycling) and considerations for physical therapy or conditioning or training if a child has a low level of fitness.

Nutrition skill-building sessions that involve direct meal preparation or tasting are more effective in increasing children's acceptance of new foods and increasing parent confidence to prepare meals at home, compared with nutrition education.⁶³⁶ Specific nutritional content included in treatment varies as well. Several studies relied on published guidelines (ie, NHLBI-supported CHILd-1diet), whereas others used specific dietary approaches, such as the reduced-glycemic load diet^{553,564,574} or using meal replacements.⁵⁹¹

Several of the more-intensive programs that demonstrate effectiveness also included a focus on mental health and parenting skills; thus, evidence exists to support the addition of these components to increase effectiveness.^{613,618,619,622,637–639}

Programs often address other components of health as well, including getting enough sleep, reducing sedentary screen time, and addressing stigma and weight bias. Although these additional elements are generally recognized as positive and are consistent with anticipatory guidance, they do not have evidence for additional benefit in the context of IHBLT.

- *Over a 3- to 12-month period:* The criteria for the evidence review required a weight-specific outcome at least 3 months after the intervention started. The rationale for excluding shorter-term lifestyle treatments was to ensure that pediatricians and other PHCPs focus on treatments that have a more-sustained treatment effect and that reinforce with parents that obesity does not have a “quick fix,” but requires long-term and ongoing attention. Treatments with duration longer than 12 months are likely to have additional and sustained treatment benefit. There is limited evidence, however, to evaluate the durability of effectiveness and the ability of long-term treatments to retain family engagement.
- *For children 2 to 18 years of age with overweight and obesity:* The USPSTF identified evidence for intensive “lifestyle” treatments starting at age 6 years.⁷⁹ This KAS includes children down to age 2, recognizing that several recent studies show treatment effectiveness in preschoolers 2 to 5 years of age.^{565,566,569,570,618,631,638,640} Treatment studies delivered care differently depending on the child's age. For example, studies targeting preschoolers more often involved home visits and focused on parental skills training. Studies targeting adolescents more often focused on the teen's autonomy,

preferences, and self-image. Intensive behavioral interventions should be tailored to the child's developmental abilities and learning skills.

D.1. Evidence for Effectiveness

The 2017 USPSTF recommendation for pediatric obesity treatment is based on high-quality randomized and nonrandomized studies that demonstrate a significant BMI reduction and were published through January 2016 ($n = 42$).^{79,625} The evidence review to inform this CPG additionally included a systematic evidence review of randomized and nonrandomized interventions leading to BMI reduction (March 2020; 214 total intervention studies of all types; of these, 126 were randomized lifestyle studies).

The evidence review is additive to the USPSTF review, as it includes new studies since January 2016 as well as studies with a comparative effectiveness design. Dose is clearly the factor most strongly correlated with treatment outcomes, as evidenced by a selection of trials that deliver “high intensity” and “comprehensive” contact over a 2- to 12-month⁶²⁵ or 3- to 12-month time period.⁸⁰

In research settings simulating clinical practice, intensive behavioral intervention has evidence for effectiveness in lowering child BMI, reducing comorbidities, and improving quality of life. Interventions that provide more ≥ 26 hours of treatment are associated with a reduction in BMI z-score between -0.10 and -0.50 ^{530,565,569,570,612,618,622,641} or a range of -1.6 to -8.1 kg (3.5 to 18 lb) weight loss over 1 year. Interventions that meet the intensity or “dose” threshold of 26 hours or more over 3 to 12 months can lead to clinically significant

improvements in BMI, systolic and diastolic BP, insulin, and glucose levels and to clinically meaningful improvements in comorbidities such as asthma, obstructive sleep apnea, and NAFLD.⁴⁴⁰⁻⁴⁴⁴ Interventions lasting less than 3 months did not demonstrate effectiveness.

- **High-intensity:** Greater contact hours lead to greater treatment effect. In all of the studies, intervention dose is most strongly associated with weight outcomes. Although weight management interventions above a threshold of 26 contact hours are generally effective in reducing excess weight (mean BMI z-score reduction 0.2), higher-dose interventions with contact time ≥ 52 hours demonstrate a stronger and more consistent BMI reduction effect.⁶²⁵ The mean difference in change of BMI z-score between controls and interventions with ≥ 52 hours of contact is -0.31 (95% CI, -0.16 to -0.46), and absolute BMI z-score reductions in the pooled intervention groups is 0.05 to 0.34.⁶²⁵ This treatment effect is observed in children 2 to 16 years of age but most consistently in children 6 to 12 years of age.⁶⁴² Savoye showed effective treatment in those 8 to 16 years of age but less effectiveness among those 13 to 16 years of age,⁴³⁹ and BMI reduction is driven by the younger age groups in the Weigel⁶¹² and Reinehr⁶⁴³ studies. A high dropout rate in obesity treatment is a known threat to delivering actual treatment hours. In research settings, attrition rates of 40% are common⁴³⁹; in real-world settings working with low-income families, attrition rates can be as high as 60%.⁶⁴⁴
- **Comprehensive:** Interventions that include behavior, physical activity, and nutrition components are associated with child

BMI reduction. In randomized studies, effective *behavior change* uses a family-focused approach.⁶¹⁵ Parents are taught self-management for their own behaviors (eg, role modeling), as well as positive parenting strategies and contingency management. Children learn goal-setting, body acceptance, and strategies to manage bullying. Effective interventions, for example, deliver moderate-to-vigorous *physical activity* for at least 50 minutes twice a week for 6 months and 40 minutes of *nutrition* counseling weekly for 6 months.^{439,645} Nutrition content includes a nondiet, lifestyle modification approach that teaches families to set goals for meal preparation, grocery shopping, and learning skills including portion size and label reading.^{439,612,643}

There is no evidence that obesity treatments harm patients' quality of life. Among the studies that included quality of life measures, none showed worsening; about one-third showed improvement to quality of life.^{535,555,615,642} More study on treatments' impact on mental health is needed, however. Few studies examined mental health impact; although none showed worsening mental health, all of the studies excluded subjects who had established mental health disorders at baseline.^{553,579,646}

The prevalence of eating disorders is not well-characterized in patients participating in obesity treatment,⁶⁴⁷ but disordered eating patterns may be more common among youth with obesity compared with youth at a healthy weight.³⁸⁷ Therefore, pediatricians and other PHCPs should evaluate patients before, during, and after intensive behavioral intervention for the presence of disordered eating (as discussed in the evaluation section) as well as for greater-than-expected weight change.

D.2. Referral Strategies

Limiting factors to IHBLT effectiveness include lack of engagement or participation by families and high attrition rates. Thus, when referring to more intensive treatment, pediatricians and other PHCPs should inform patients and their family members about the reason for the referral, encourage families to actively participate in the treatment, and schedule follow-up visits to monitor progress in the treatment.

One factor in early attrition may be mismatched expectations for weight loss.⁶⁴⁸ Families can best make decisions about IHBLT participation after providers inform them of commitment and likely outcomes. Pediatricians and other PHCPs are encouraged to help to set reasonable expectations for these outcomes among families, as there is a significant heterogeneity to treatment response and there is currently no evidence to predict how individual children will respond. Many children will not experience BMI improvement, particularly if their participation falls below the treatment threshold. As described in the Health Behavior and Lifestyle Treatment section, those who do experience BMI improvement will likely note a modest improvement of 1% to 3% BMI percentile decline.³⁹⁶

D.3. Prompt IHBLT

A key distinction from prior recommendations is for pediatricians and other PHCPs to refer as soon as possible to IHBLT. Current practice patterns involve counseling in primary care practices, often for months to years, before referring to more intensive programs. Although providing patient-centered and nonstigmatizing nutrition and activity counseling is important for children of all weight classifications,

KAS 11. Pediatricians and other PHCPs should provide or refer children 6 y and older (Grade B) and may provide or refer children 2 through 5 y of age (Grade C) with overweight (BMI \geq 85th percentile to <95th percentile) and obesity (BMI \geq 95th percentile) to intensive health behavior and lifestyle treatment. Health behavior and lifestyle treatment is more effective with greater contact hours; the most effective treatment includes 26 or more hours of face-to-face, family-based, multicomponent treatment over a 3- to 12-mo period.

Aggregate Evidence Quality	Grade B: Children \geq 6 y of Age. Grade C: Children 2 Through 5 y of Age.
Benefits	BMI reduction, quality of life improvement, comorbidity improvement or resolution is associated with 26 or more hours of face-to-face, family-based, multicomponent treatment over 3 to 12 mo.
Risks, harms, costs	Minimal risk or harm. Participation is time-intensive and requires repeated visits. Treatment is costly to administer and inconsistently paid.
Benefit-harm assessment	Benefit outweighs risk.
Intentional vagueness	Impact is inconsistent in studies with significant heterogeneity of treatment response. Increasing dose of treatment is associated with more BMI improvement.
Role of patient preference	Patient preference is central.
Exclusions	Patients not responsible for their behavior change, such as children who are young or with developmental or cognitive impairment.
Strength	Moderate.
Key references	625

there is no evidence to support either watchful waiting or unnecessary delay of appropriate treatment of children who have already developed obesity. Many children are only referred to treatment programs when their obesity has become more severe.^{529,626} A delay in care ultimately reduces the likelihood of treatment success for the child.⁶⁴⁹

Similarly, no evidence supports selectively referring patients to obesity treatment programs based on those who meet certain criteria, such as obesity severity, presence of comorbidities, and/or readiness or motivation to change. Although there is currently limited evidence for obesity treatment in children 2 through 5 years of age, excess weight gain in early life predicts future obesity⁶⁵⁰; therefore, future studies should examine treatment in this age group. Pediatricians and other PHCPs are advised to prioritize the most effective treatment available for patients with obesity and encourage patients and families to use these programs at the time of obesity diagnosis.^{555,615,642}

It is necessary to provide IHBLT within various sites of health care delivery. Face-to-face time with pediatricians and other PHCPs cannot realistically achieve the intensity that

is most effective. Thus, RDNs, health behavior specialists, and exercise professionals should be part of the health care team and have critical skills for IHBLT. They can work within a multidisciplinary obesity treatment clinic, be embedded within a medical home organization so that they coordinate with pediatricians and other PHCPs, and participate in care through referrals. Given the number of children who meet criteria for treatment, and the current limitations on number of pediatricians and other PHCPs who deliver IHBLT, a significant effort toward medical home capacity-building will be needed to achieve equitable access for all children. Current programs are generally located in cities, and often in academic centers.⁶⁵¹ An individual center may or may not offer the entire range of intensive treatment, including intensive lifestyle, pharmacotherapy, and surgery. Providers should be familiar with the treatment programs at local centers; their knowledge of the child and family, along with awareness of available options, can guide treatment direction. Rural communities need resources and programs, especially ones that accommodate the distinct challenges of rural living, including transportation and economic and cultural factors.⁶⁵²

On the strength of the literature, the USPSTF gives the evidence for intensive “lifestyle” treatment of childhood obesity a “B” rating, which means that health plans should cover this care.^{79,625} The USPSTF is authorized by Congress to assign grades to the state of the evidence regarding treatment options for diseases. Under the Affordable Care Act, grades of “A” or “B” are mandated to be covered with no deductibles, copayments, or cost-sharing.⁶⁵³ A large gap currently exists, however, between this expectation and the actual policies in state Medicaid and commercial health plans. Health care systems should build the capacity necessary to deliver this evidence-based level of care (see the Implementation Barriers section for more discussion).

E. When Intensive Programs Are Not Available

Pediatricians and other PHCPs are on the frontline of identifying overweight and obesity. Consistent success in behavior-based obesity treatment is highly related to treatment intensity. Both individual and systemic barriers may, however, keep many families from receiving the recommended moderate- to high-intensity multicomponent obesity treatment. In addition, pediatricians and other PHCPs

should be aware that pediatric patients who seek bariatric surgical treatment may encounter significant disparities in access to care based on demographics, SES, and insurance type.⁶⁵⁴

Availability of IHBLT or other treatments is generally poor, as described in a Children's Hospital Association report in 2013.⁶⁵⁵ Differences in access based on demographics and similar factors have not been well studied. A consortium of academic centers with pediatric weight management programs reported a high proportion of publicly insured patients and racial and ethnic diversity⁶²⁶; however, these sites are safety-net medical centers in larger cities and, as such, are a small sample of the children in the United States with obesity. Pediatricians and other PHCPs are encouraged to pay attention to difference in care access and seek mechanisms to mitigate these challenges.⁶⁵⁴

Although the health care and payment systems often limit the time and resources available within the primary care office, families can benefit from guidance outside of intensive programs, including pre and postprogram participation. Several successful studies have been conducted in the primary care setting, with less than 5 hours of treatment and using individual visits rather than group visits. These treatments varied in their approach and included clinical decision support built into electronic health records (EHRs), MI, and a self-guided curriculum for teens and parents.^{525,529,591} Strategies that may help pediatricians and other PHCPs include use of EHRs to remind and streamline care during office visits, MI training to effectively encourage families to take action, and resources for families to use outside of office visits.

When an IHBLT is not available, pediatricians and other PHCPs can increase the intensity of weight management support through collaboration and by connecting families with community resources to support nutrition and address food insecurity (eg, food provision programs), physical activity (eg, local parks, recreation programs), and other SDoHs. Pediatricians and other PHCPs should familiarize themselves with resources and actively collaborate with other specialists and community programs.

For example, pediatricians and other PHCPs should assess the availability and pediatric expertise of local dietitians and offer referrals to patients where possible. RDNs can assess a child's nutritional needs, including appropriate food groups and portion sizes, and provide guidance for specific diet needs and preferences, including cultural patterns. Some RDNs have received special certification in pediatric and adolescent obesity, and the Academy of Nutrition and Dietetics offers RDNs additional learning opportunities in pediatric and adolescent obesity and encourages training in patient-centered counseling techniques. RDNs can complement the care of medical providers and may be the most widely available specialist with whom pediatricians and other PHCPs can work to provide more intensive behavioral intervention. Behavioral health specialists, ideally integrated into primary care, can focus on the process of behavior change, including parenting skills, role modeling, and consistent reinforcement techniques.

Implementation tools can help address actions in low-resourced settings. Exercise specialists can provide counseling and training to engage children and families in

noncompetitive, cooperative, fun, aerobic and nonaerobic activities. Behavior goals related to physical activity include aiming for the physical activity guidelines of 60 minutes per day of moderate to vigorous physical activity^{635,642} and reducing time spent in sedentary behavior.⁶⁵⁶ Physical activity limitations, such as joint pain related to musculoskeletal comorbidities or increased work of breathing related to severe obesity, should be considered, and a stepped care plan for a gradual increase in physical activity can be made. Medicaid and other insurance plans may restrict coverage to specific medical conditions which do not include obesity or risk factor reduction. Providers can search for community programs that follow a philosophy of noncompetitive, fun activities, ideally engaging the whole family. See Figure 4 for facilitators of successful health behavior lifestyle treatment.

Consensus Recommendations

The CPG authors recommend that pediatricians and other PHCPs:

- Deliver the best available intensive treatment to all children with overweight and obesity.
- Build collaborations with other specialists and programs in their communities.

F. Specific Health Behavior Recommendations

Many pediatricians and other PHCPs, especially those in primary care, have an important role in recommending specific health behaviors to improve energy balance. The following specific health behavior recommendations do not form a KAS, because randomized controlled trial (RCTs) that test each in isolation do not exist and are unlikely to be performed.

Effective programs described in the technical report incorporated many nutrition, physical activity, and behavior change strategies simultaneously, an approach that is responsive to the multiple behaviors that contribute to energy imbalance. Single-component interventions, such as physical activity alone, were generally not successful, and the multicomponent studies were not designed to isolate the impact of one component over another. If the study target were too narrow (for example, consumption of 5 fruits and vegetables a day), any impact on weight or BMI would likely be too small to detect.

Most of the successful interventions described in the technical report described nutrition counseling without a structured diet. (Exceptions were a small study that found both low and modified carbohydrate diets were better than control,⁵⁷² and a study that found a focus on only beverages and a focus on multiple nutrition changes were both superior to control but not different from each other.⁵⁷¹) Two effective studies of adolescents implemented caloric restriction of 1300 to 1550 calories per day, but the interventions also included additional components, such as physical activity promotion and behavior change strategies.^{563,591} Two small studies found benefit from interventions that reduced glycemic load.^{564,574}

Despite the lack of evidence for specific strategies on weight outcomes, many of these strategies have clear health benefits and were components in RCTs of intensive behavioral intervention. Many strategies are endorsed by major professional or public health organizations. Therefore, pediatricians and other PHCPs can appropriately encourage families to adopt these strategies.

Pediatricians and other PHCPs should present these specific strategies in the context of MI, helping families to identify their own goals and to determine steps to overcome barriers in making change. The AAP's *Next Steps: A Practitioner's Guide of Themed Follow-up Visits to Help Patients Achieve a Healthy Weight* provides step-by-step strategies for the pediatrician or other PHCP on content and delivery of each theme, including portion sizes, screen time and sleep, meal patterns and snacks, and bullying and teasing.^{657,658}

Table 18 lists specific behavior strategies endorsed by major professional and public health organizations. Some systematic reviews are cited, which include many association studies, but a comprehensive search for studies and reviews was not performed.

Table 19 presents strategies that are common but have not at this time been addressed by the AAP or other major health organizations. For some, rigorous systematic reviews provide information about potential benefit as well as harm or lack of harm. Brief mention of existing literature is included, but extensive searches for publications were not performed.

When actively intervening to treat overweight and obesity in the primary care setting, pediatricians and other PHCPs should also evaluate and address the modifiable risk factors for obesity that are described in the Risk Factors section. These include parenting feeding styles, frequency of dining out and eating fast food, and ACEs, among other household risk factors. Awareness of supports and barriers in the patient's community will also help guide the family to resources outside the home, such as parks and recreation programs, community

gardens, and school wellness policies.

Parents and caregivers have a crucial role to play in obesity treatment through strategies such as parental monitoring, limit setting, reducing barriers, managing family conflict, and modifying the home environment.^{15,684,685} A systematic review of parental involvement in childhood obesity treatment studies found that medium- to high-intensity parental involvement was associated with weight-related measures of treatment effectiveness.⁶⁸⁶ Parents can serve as role models and provide support in obesity treatment. In addition, an enhanced parent-child relationship functions as a mediator in development of healthier behaviors and further weight control.⁶⁸⁷ Parents themselves and family relationships may also benefit from children's obesity treatment.

A recent systematic review found that certain common features involving parents in obesity treatment interventions with their preadolescent children were successful in producing nutrition and physical activity behavior change. These features include promotion of intrinsic motivation and self-efficacy through empowerment of parents and children and fostering shared value and whole-family ownership. The activities most commonly associated with positive behavior change included parental leadership in goal setting, problem solving, social support, demonstrating desired behaviors, and restructuring the home environment. It is encouraging that the majority of studies that included low-income populations in this review found favorable results.⁶⁸⁸

Adolescence can present substantial challenges to family-based care, because this period is marked by a developmentally normative period of

TABLE 18 Behavior Strategies

Strategy	Description	References
Reduction of sugar-sweetened beverages (SSBs)	Higher intake of sugar-sweetened beverages (carbonated beverages, sweetened beverages, soda, sports drinks, and fruit drinks) is associated with greater wt gain in adults and children. The American Heart Association (AHA) recommends not more than 25 g (6 tsp) each day of added sugar and not more than 1, 8-oz serving of SSB per week. The AAP discourages the consumption of sports drinks and energy drinks for children and adolescents. The AAP statement on fruit juice notes that it is a poor substitute for whole fruit because of its high sugar and calorie content and pediatricians should advocate for elimination of fruit juice in children with excessive wt gain.	Systematic review ⁶⁵⁹ ; AHA SSB ⁶⁶⁰ ; AAP sports and energy drinks ⁶⁶¹ ; AAP fruit juice ⁶⁶²
Choose My Plate	MyPlate is the US Department of Agriculture's (USDA) broad set of recommendations for healthy eating for Americans. These recommendations include multiple healthy diet goals: low in added sugar, low in concentrated fat, nutrient dense but not calorie dense, within an appropriate calorie range without defined calorie restriction, and with balanced protein and carbohydrate. The principles can be adapted to different food cultures. There is a surprising dearth of literature on the impact of these guidelines on health and BMI outcomes and on the most effective education practices.	USDA choosemyplate.gov
60 min daily of moderate to vigorous physical activity	Aerobic exercise, especially for 60 min at a time, is associated with improved body weight in youth although its effect may be small and variable. It is also associated with better glucose metabolism profiles. High-intensity interval training in youth with obesity may improve body fat, weight, and cardiometabolic risk factors, although the effect is variable. ⁶⁶⁵ The Physical Activity Guidelines for Americans recommends 60 min per day for children and adolescents.	Systematic reviews ^{664–667} ; AAP physical activity; Guidelines for Americans ^{379,655}
Reduction in sedentary behavior	Reduction in sedentary behavior, generally defined as reduced screen time, has consistently shown improvement in BMI measures, although impact is small. Early studies focused on reduced television, a discrete activity that is simpler than current multifunctional electronic devices. The AAP recommends no media use under age 18 mo, a 1-h limit for ages 2–5 y, and a parent-monitored plan for media use in older children, with a goal of appropriate, not-excessive use but without a defined upper limit.	AAP media and young minds ¹⁷⁰ ; systematic review ⁶⁵⁶

increased desire for independence and autonomy, despite continued reliance on parents for many needs. Given these challenges, the research investigating specific clinical

paradigms for parent involvement in adolescent obesity treatment demonstrates mixed findings regarding the ideal level of involvement and the specific parenting strategies that can

optimize treatment. Further research and detailed reporting are needed to inform clinical guidelines for optimizing the role of parents in adolescent obesity treatment.⁶³⁴

TABLE 19 Common Strategies

Strategy	Description	References
Avoidance of breakfast skipping	Breakfast skipping among children is associated with overweight and obesity and with lower quality of dietary intake throughout the day.	Systematic review ⁶⁶⁸
Traffic Light Diet	This approach to teaching healthy eating has shown consistent success within the context of moderate- to high-intensive multicomponent programs, in which experienced providers help families learn and use the diet.	Evidence summary can be found on the Academy of Nutrition and Dietetics Web site: https://www.andean.org/topic.cfm?cat=1429&evidence_summary_id=250033&highlight=traffice%20light%20diet&home=1 .
5 2 1 0	This messaging emerged from a consortium of primary care pediatricians as simple, memorable, and feasible (www.mainehealth.org/Lets-Go/Childrens-Program). Each component of 5-2-1-0 messaging aligns with a major recommendation or guideline: 5 fruits and vegetables a day is consistent with the USDA ChooseMyPlate recommendations, 2 h or less of screen time is consistent with earlier versions of AAP policy; 1 h or more of moderate to vigorous physical activity is consistent with Physical Activity Recommendations for Americans, and 0 (or nearly no) sugar-sweetened beverages aligns with USDA, AHA, and AAP.	Scant literature on weight or BMI impact. ^{669,670} Attainment of 5-2-1-0 behaviors is low. ⁶⁷¹
Use of screen-based physical activity (exergames)	Video games that require physical activity can reduce children's body wt. Players can reach levels of light-to-moderate intensity physical activity during exergame play, especially games that involve whole-body movement. Systematic reviews have shown that children can lose body weight or attenuate weight gain when playing exergames over a sustained period of time. Specific setting in which exergaming resulted in weight, adiposity, or BMI z-score improvement included home, part of a structured physical activity program, and part of a multicomponent obesity treatment. Children experienced modest reductions in weight, adiposity, or BMI z-score when exergames were provided in the home, within a structured physical activity program, and within an obesity treatment program. There is less evidence to date for newer technologies like smartphone apps and wearables, but these are promising tools to engage and sustain youths' interest in healthy behaviors.	645,672–683
Appropriate amount of sleep for age	Obesity is associated with shorter sleep duration, and the association appears to be driven by increased calorie consumption, decreased physical activity from fatigue, and potential hormonal and metabolic alterations such as increased ghrelin and decreased leptin leading to hunger.	Systematic review ^{243–247}

G. Self-Management

Obesity is a complex chronic disease with biologic, environmental, and other causative factors that are systemic and operate at the local, regional, and global level. As with all

chronic disease, the patient and family have to manage the demands of the disease and evidence-based treatment in the context of these factors. This means that individual patients and their families will have

unique challenges to overcome based on the severity of their disease and the adversity of their environments. Effective obesity treatment helps patients and families develop self-management

strategies that are critical for chronic disease management in this context.

Self-management has been defined as “the development of a range of attitudes, health behaviours and skills to help minimize the impact of their condition on all aspects of life for themselves and their families and caregivers.”⁶⁸⁹ Self-management has also been described “as a dynamic, interactive, and daily process in which individuals engage to manage a chronic illness,”⁶⁹⁰ and “the ability of the individual, in conjunction with family, community, and health care professionals, to manage symptoms; treatments; lifestyle changes; and psychosocial, cultural, and spiritual consequences of health conditions.”⁶⁹¹ For example, dietary change is an important treatment strategy in the self-management of many chronic diseases^{692,693} and is about “tailoring support to improve knowledge, skills and confidence”⁶⁹⁴ by promoting facilitators such as “location, language, incentive and

tailored resources”⁶⁹⁵ while mitigating barriers, such as inadequate knowledge or skills or lack of time.⁶⁹⁶ For young children and children with disabilities, “self-management” may apply to caregivers on behalf of or in conjunction with the patient.

The complex environment in which children and adolescents with obesity and their families live needs to be acknowledged in the way providers individualize and tailor self-management supports.

H. Treatment Considerations for Children and Youth With Special Health Care Needs

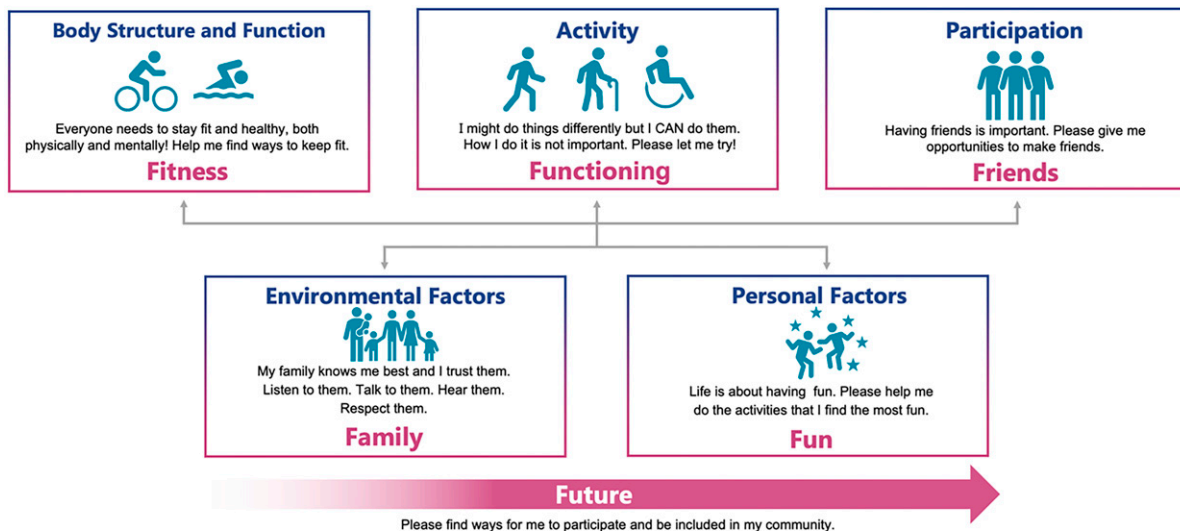
CYSHCN are those who have, or who are at increased risk for, a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally.⁷⁴

It is important that management of obesity for CYSHCN includes similar protocols and processes as those for

children with typical development. This includes assessing health behaviors, identifying community resources and policies, sharing appropriate resources, and promoting healthy behaviors.^{697,698} Pediatricians and other PHCPs are encouraged to assess risks contributing to obesity in collaboration with families and interdisciplinary teams (specialists, psychologists, primary care providers, mental health professionals, social workers, physical therapists, and dietitians), providing their patients (CYSHCN) and their families with essential skills and resources to manage obesity.

In addition, it is critical to recognize that CYSHCN may have physical, emotional, and/or cognitive condition(s) preventing them from engaging in community or clinical activities that are available for their peers with typical development.⁶⁹⁹ Therefore, creative solutions for promoting physical activity and healthy nutrition and behavior change are vital for this special population. Solutions come from

The ICF Framework and the F-Words



For more information visit the F-words Knowledge Hub: www.canchild.ca/f-words



1) World Health Organization. (2001) *International Classification of Functioning, Disability and Health (ICF)*
 2) Rosenbaum P & Gorter JW. (2012). The 'F-words' in childhood disability: I swear this is how we should think! *Child Care Health Dev*; 38.

FIGURE 5
The ICF framework and the F-words.⁷⁰⁵ Used with permission.

guidance and supervision from families, health care providers, and community recreation staff for appropriate physical activities and consumption of healthier food options.⁷⁰⁰⁻⁷⁰²

It is essential to emphasize the numerous benefits of recreational activities for CYSHCN to include improve socialization, fitness level, and motor and movement skills.^{703,704} Additional nutrition, physical activity, and sedentary time recommendations can be found in a recent report from the Healthy Weight Research Network, an interdisciplinary group of clinical investigators and experts providing obesity treatment of children with autism and other intellectual and/or developmental disabilities.⁶⁹⁷

The International Classification of Functioning (ICF), Disability, and Health model and AAP recommendations are appropriate frameworks related to assessing pertinent health conditions and contextual factors affecting CYSHCN to aid in planning to treat obesity, as seen in Fig 5. The ICF framework can be used to organize approaches in the management of childhood obesity.

Children with obesity-related genetic syndromes, behavioral difficulties, developmental disabilities, and hypothalamic

disorders (Table 2) have added obesity risk and may need additional supports in obesity treatment.³¹⁶

Hyperphagia can be particularly challenging to manage in CYSHCN. A combination of specific behavioral techniques within the context of family-based behavioral treatment and the use of pharmacotherapy may be necessary.⁷⁰⁰

Prader Willi syndrome is a complex genetic disorder affecting 1 in 15 000 to 1 in 30 000 people and is associated with obesity and hyperphagia.^{706,707} Specific recommendations for health supervision and multicomponent care can help pediatricians and other PHCPs institute a longitudinal treatment approach to care.

Hypothalamic obesity is a neuroendocrine disorder resulting from damage to the hypothalamus, disrupting the body's energy regulatory system, which requires intensive multicomponent treatment.⁷⁰⁸

ADHD is associated with obesity, and symptoms such as deficits in alertness and attention that are caused by sleep-disordered breathing attributable to obesity can overlap with those of ADHD. Impulsivity in ADHD may contribute to dysregulated eating and weight gain. Effective treatment of ADHD in children with obesity can be

associated with attainment of healthier weight status.⁷⁰⁹

I. Use of Pharmacotherapy

Consensus Recommendation

The CPG authors recommend pediatricians and other PHCPs:

- May offer children ages 8 through 11 years of age with obesity weight loss pharmacotherapy, according to medication indications, risks, and benefits, as an adjunct to health behavior and lifestyle treatment.

Although IHBLT has the largest body of evidence meeting the evidence review's high-quality evidence for effectiveness criteria, it is important to consider the use of pharmacotherapy for children and adolescents who require an additional treatment option to manage their obesity. In particular, children with more immediate and life-threatening comorbidities, those who are older, and those affected by more severe obesity may require additional therapeutic options. For children younger than 12 years, there is insufficient evidence to provide a KAS for use of pharmacotherapy for the sole indication of obesity. There are, however, specific conditions outlined below for which use of medication may be indicated. Additionally, although the evidence is insufficient at the time of this

KAS 12. Pediatricians and other PHCPs should offer adolescents 12 y and older with obesity (BMI ≥ 95th percentile) weight loss pharmacotherapy, according to medication indications, risks, and benefits, as an adjunct to health behavior and lifestyle treatment.

Aggregate Evidence Quality	Grade B
Benefits	BMI reduction as an adjunct to lifestyle treatment.
Risks, harms, costs	Varies by pharmacotherapeutic agent.
Benefit-harm assessment	Benefit and harm are individualized by patient, must weigh the side effects and potential benefit of the medication and patient-specific factors.
Intentional vagueness	None.
Role of patient preference	Significant; must determine appropriate timing and duration of treatment, monitor for side effects.
Exclusions	Medication-dependent exclusions.
Strengths	Moderate.
Key references	710

evidence review, the use of pharmacotherapy to aid BMI reduction in children is a rapidly evolving field; new evidence may lead to additional options for children younger than 12 years in the future.

Studies Included

At the time of the evidence review for this CPG, 27 randomized studies met the inclusion criteria for review, and an additional 8 observational studies were also considered. The majority of the studies evaluated the efficacy of metformin either alone ($n = 16$ randomized, 7 observational) or in combination with other medications ($n = 7$ randomized). Other studies evaluated orlistat ($n = 2$ randomized), exenatide ($n = 2$ randomized), or other medications with only 1 study cited (phentermine, mixed carotenoids, topiramate, ephedrine, and recombinant human growth hormone). Since the evidence review, additional high-quality evidence has been published to define the safety and efficacy of novel agents (setmelanotide, liraglutide, and the combination phentermine or topiramate) and are included in this discussion.

Medication Use and Mechanisms of Action

Metformin is an antidiabetic agent used in T2DM among patients 10 years and older. Metformin also has several indications not approved by the US Food and Drug Administration (FDA), including prediabetes, PCOS, and prevention of weight gain when used with atypical antipsychotic medications. Metformin is a biguanide drug that reduces blood glucose levels by decreasing blood glucose production in the liver, decreasing intestinal absorption, and increasing insulin sensitivity. It comes in immediate- and extended-release formulations; the recommended starting dose is 500 mg, once or twice

daily, with gradual increases up to a maximum total daily dose of 2500 mg. Adverse effects are dose-dependent, and include bloating, nausea, flatulence, and diarrhea. Lactic acidosis is a serious but very rare complication in pediatric populations.⁷¹¹

Metformin has not been approved as a weight-loss drug. A 2020 meta-analysis of metformin studies in adults with obesity showed modest (<5%, or 1 BMI unit) weight reduction with metformin when used as an adjunct to lifestyle; however, the effectiveness is inconsistent across different populations.⁷¹² The evidence for effectiveness of metformin for weight loss in pediatric populations is similarly conflicting. One small study randomized 39 teens 13 to 18 years of age with obesity participating in a lifestyle modification program to either metformin hydrochloride XR 2000 mg, or placebo once daily for 48 weeks.⁷¹³ Adolescents taking metformin reduced BMI by approximately 1 kg/m² as compared with a slight increase in BMI among teens in the lifestyle-only program.

Another well-designed randomized study of 100 children 6 to 12 years of age with severe obesity (mean BMI 34.6 kg/m²) showed a BMI reduction of approximately 1 kg/m² at a dose of 1000 mg twice daily for 6 months, also as an adjunct to lifestyle treatment.⁷¹⁴ Gastrointestinal symptoms limited the tolerated maximum dose in nearly 20% of patients, however, and no additional BMI reduction was noted with treatment beyond 6 months. Of the 16 studies of metformin that met quality inclusion criteria for the evidence review, about two-thirds showed modest BMI reduction.^{713–724} One-third showed no benefit.^{725–728}

The effective studies typically included higher metformin doses, more intensive lifestyle adjunct

treatment, and use in children or adolescents with more severe obesity and/or a secondary diagnosis, such as prediabetes or PCOS. Given the modest and inconsistent effectiveness, metformin may be considered as an adjunct to intensive health behavior and lifestyle treatment and when other indications for use of metformin are present.

Orlistat is an intestinal lipase inhibitor that blocks fat absorption through inhibition of pancreatic and gastric lipase. It is currently approved for children 12 years and older at a dose of 120 mg, 3 times per day. Adverse effects include steatorrhea, fecal urgency, and flatulence; these adverse effects greatly limit tolerability, and thus, orlistat is uncommonly used in pediatric obesity treatment. Orlistat is FDA approved for long-term treatment of obesity in children 12 years and older.^{729,730}

Glucagon-like peptide-1 receptor agonists, such as liraglutide, exenatide, dulaglutide, and semaglutide, decrease hunger by slowing gastric emptying and by acting on targets in the central nervous system. Depending on the medication, the formulation is either oral or a daily or weekly subcutaneous injection. Two small studies of exenatide (weekly injection) among children as young as 8 years showed BMI reduction ranging from 0.9 to 1.18 U but with significant adverse effects. Exenatide is currently approved in children 10 to 17 years of age with T2DM. A recent randomized controlled trial found liraglutide (daily injection) more effective than placebo in weight loss at 1 year among patients 12 years and older with obesity who did not respond to lifestyle treatment.⁷¹⁰ The magnitude of the difference was approximately 4.5 kg body weight lost, or 5% BMI reduction. The starting dose is 0.6 mg

per day up to a maximum dose of 3.0 mg per day, by subcutaneous injection. Adverse effects include nausea and vomiting, and among patients with a family history of multiple endocrine neoplasia, a slightly increased risk of medullary thyroid cancer.⁷¹⁰ Liraglutide is FDA approved for long-term treatment of obesity (with or without T2DM) in children 12 years and older.

Melanocortin 4 receptor (MC4R) agonists, such as setmelanotide, act on the MC4R pathway to restore normal function for appetite regulation that has been disrupted because of genetic deficits upstream of the MC4 receptor. MC4 receptors in the brain regulate hunger, satiety, and energy expenditure. The daily dose is 1 to 3 mg daily, given subcutaneously, and results in weight loss of 12% to 25% over 1 year in a small, uncontrolled study of patients with these rare deficits. Common adverse effects include injection site reaction and nausea. Setmelanotide is FDA approved for patients 6 years and older with proopiomelanocortin (POMC) deficiency, proprotein subtilisin or kexin type 1 deficiency, and leptin receptor deficiency confirmed by genetic testing.

Phentermine is a central norepinephrine uptake inhibitor but also nonselectively inhibits serotonin and dopamine reuptake and reduces appetite. Recommended doses include 7.5 mg, 15 mg, 30 mg, or 37.5 mg, and adverse effects include elevated BP, dizziness, headache, tremor, dry mouth, and stomachache. Adverse effects are dose-dependent; however, effectiveness does not always increase with increasing dose. Phentermine is FDA approved for short-course therapy (3 months) for adolescents 16 years or older.

Topiramate is a carbonic anhydrase inhibitor and suppresses appetite

centrally through largely unknown mechanisms. The major adverse effect is cognitive slowing, which can interfere with academic concentration or other activities of daily living. In addition, topiramate is a potential teratogen and requires counseling and reliable birth control for patients able to become pregnant. Typical dosing for headache prevention ranges from 25 mg a day to 100 mg a day in twice daily doses. Although topiramate has an indication for treatment of binge eating disorder in adults (age ≥ 18), only 1 study has evaluated the use of topiramate in children, and it did not differ from placebo. Topiramate is currently FDA approved for children 2 years and older with epilepsy and for headache prevention in children 12 years and older.

Phentermine and topiramate as a combination medication is approved for weight loss in adults. Recent data show that among adolescents 12 to 17 years of age with documented history of failure to lose sufficient weight or failure to maintain weight loss in a lifestyle modification program (mean age, 14 years; mean BMI, 37.8 kg/m²), BMI percent change at 56 weeks was -10.44 (high dose; 15 mg/92 mg) and -8.11 (mid dose; 7.5 mg/46 mg) as compared with placebo.⁷³¹ Treatment also improved HDL and TG cholesterol profiles. Adverse events reported were not more common than placebo in the high- or mid-dose range.

Lisdexamfetamine is similar in mechanism to phentermine and is a stimulant-class medication approved for children 6 years and older with ADHD. It has an indication for treatment of binge eating disorder in patients 18 years and older; thus, it is used off-label for children with obesity. However, no evidence is available at the time of this review

to demonstrate safety or efficacy for the indication of obesity in children.

Prescriber Qualifications

Weight loss medications require the same oversight and expertise in management as other medications used in pediatric care. To adequately inform patients and parents about the risks and benefits of off-label or experimental use of new therapies, pediatricians and other PHCPs who prescribe weight loss medications should have knowledge of the patient selection criteria, medication efficacy, adverse effects, and follow-up monitoring guidelines. In addition, injectable medications may require additional teaching for families that is not available in all primary care offices. Pediatricians and other PHCPs may choose to refer to pediatric obesity experts or treatment centers for prescribing weight loss medication.

No current evidence supports weight loss medication use as a monotherapy; thus, pediatricians and other PHCPs who prescribe weight loss medication to children should provide or refer to intensive behavioral interventions for patients and families as an adjunct to medication therapy.

J. Pediatric Metabolic and Bariatric Surgery

It is widely accepted that the most severe forms of pediatric obesity (ie, \geq class 2 obesity; BMI ≥ 35 kg/m², or 120% of the 95th percentile for age and sex, whichever is lower) represent an “epidemic within an epidemic.” Moreover, severe obesity is a harbinger of the establishment and cumulative progression of numerous related comorbidities, diminished long-term health status, and shortened life expectancy.^{654,739}

For adults, the evidence supporting the clinical indications and associated recommendations on the use of metabolic and bariatric

Aggregate Evidence Quality	Grade C ^a
Benefits	Referral for evaluation to a comprehensive pediatric metabolic and bariatric surgery center may result in the determination of eligibility for laparoscopic Roux-en-Y gastric bypass or vertical sleeve gastrectomy, which are both associated with significant and sustained weight loss, accompanied by improvements and/or resolution of numerous related comorbid conditions, remission of certain cardiometabolic risk factors, and improvements in weight-related quality of life.
Risks, harms, costs	Minor perioperative risks may occur in up to 15% of patients, and major perioperative risks may occur in up to 8% of patients. Subsequent related procedures may be required in 13% to 25% of patients up to 5 y following surgery. Vitamin deficiencies are common and need long-term monitoring and potential intervention. Health care costs and costs to families (eg, time, recovery).
Benefit-harm assessment	Perioperative risks considered within acceptable tolerances compared with other elective surgical abdominal procedures. Benefit and harm are patient-specific with generally accepted safety profiles within the context of perceived long-term post-procedural benefits (ie, reduction in comorbid disease burden), resulting in improved health status and quality of life.
Intentional vagueness	This action statement does not recommend surgery for all who have severe obesity but rather the opportunity for children, adolescents, and families to consider and undergo evaluation.
Role of patient preference	Significant; must determine appropriate timing of intervention and comprehension of treatment plan.
Exclusions	Not applicable.
Strength	Moderate.
Key references	126, 654, 732–738

^a This KAS was given a Grade C recommendation, as available evidence is from observational and case-controlled studies. As described in the methods section for the evidence review, only randomized and comparative effectiveness studies were included for the CPG. The Evidence Review Panel made the decision to include observational and case-control studies specifically for surgical interventions only, because of ethical considerations and practical challenges to randomization.

surgery is founded on a body of literature that has been expanding since the early 1960s.⁷⁴⁰

Corresponding analyses related to the use of various surgical weight loss procedures in pediatric populations have been primarily established in the last 20 to 30 years. Large contemporary and well-designed prospective observational studies have compared adolescent cohorts undergoing bariatric surgical treatment versus intensive obesity treatment or nonsurgical controls.

These studies suggest that weight loss surgery is safe and effective for pediatric patients in comprehensive metabolic and bariatric surgery settings that have experience working with youth and their families. Laparoscopic Roux-en-Y gastric bypass and vertical sleeve gastrectomy are both commonly performed in the pediatric age group and result in significant and sustained weight loss, accompanied by improvements and/or resolution of numerous related comorbid conditions.⁷³² Laparoscopic

adjustable gastric band procedures, approved by the FDA only for patients 18 years and older, have declined in use in both adults and youth because of worse long-term effects as well as higher-than-expected complication rates.^{647,652,654,655,706,732–736,739–744}

Similar to the adult experience, an expanding body of data shows that pediatric bariatric patients also experience durable reduction in BMI,^{152,744–748} as well as significant improvement and/or complete amelioration of several obesity-related comorbid conditions. These include HTN, T2DM, dyslipidemia, cardiovascular disease risk factors, and weight-related quality of life.^{152,654,734}

Furthermore, recent evidence showing that adolescents had a higher probability of remission of certain cardiometabolic risk factors (T2DM and HTN) compared with adults highlights the argument that earlier surgical intervention may impart specific advantages related to the cumulative impact of chronic obesity-related diseases.⁷³⁶ The

significantly lower magnitude of efficacy of intensive behavioral interventions—compared with larger and more durable improvements in BMI and comorbidity resolution after metabolic and bariatric surgery—has led to a significant increase in pediatric bariatric surgical case volume since the early 2000s.¹²⁶

The majority of complications following metabolic and bariatric surgery in the pediatric population are minor (15%), occur mostly in the early postoperative timeframe, and consist of a combination of postoperative nausea and/or dehydration, although major perioperative (30-day) complications have been reported in 8% of individuals.^{654,733} Subsequent related procedures may be required in 13% to 25% of patients up to 5 years following metabolic and bariatric surgery.^{744,746,747} In addition, recent data showing multiple micronutrient deficiencies following metabolic and bariatric surgery serve to highlight the need for routine and long-term monitoring.

TABLE 20 Criteria for Pediatric Metabolic and Bariatric Surgery⁷³³

Weight Criteria	Criteria for Comorbid Conditions
Class 2 obesity, BMI \geq 35 kg/m ² or 120% of the 95th percentile for age and sex, whichever is lower	Clinically significant disease; examples include but are not limited to T2DM, IIH, NASH, Blount disease, SCFE, GERD, obstructive sleep apnea (AHI $>$ 5), cardiovascular disease risks (HTN, hyperlipidemia, insulin resistance), depressed health-related quality of life.
Class 3 obesity, BMI \geq 40 kg/m ² or 140% of the 95th percentile for age and sex, whichever is lower	Not required but commonly present.

AHI, apnea-hypopnea index.

Although the determination of eligibility for metabolic and bariatric surgery relies heavily on a multicomponent and individualized approach between members of the metabolic and bariatric surgery team, the patient, and the patient's parents or guardians, initial steps toward consideration should be provided, when appropriate, within the medical home. Specifically, pediatricians and other PHCPs should be familiar with recent and clearly defined clinical and anthropometric benchmarks, which serve as a prompt for the initiation of these discussions with the patient and family and ongoing bilateral communication between the medical home and surgical center^{654,733} (Table 20). In addition to knowledge of indications for metabolic and bariatric surgery, pediatricians and other PHCPs should build and maintain skills in discussing this topic with families in a nonbiased and sensitive manner. Pediatricians and other PHCPs should also seek to establish a local and/or regional referral mechanism to qualified facilities that offer pediatric-focused metabolic and bariatric surgical services.

Individual determination of eligibility status at the time of referral to a center that offers metabolic and bariatric surgical intervention for the pediatric population involves a comprehensive and multidisciplinary assessment of longitudinal BMI and comorbidity status as well as physiologic and psychosocial assessment, including the determination of potential contraindications such as correctable causes of obesity, ongoing substance

use disorder, and pregnancy. Important elements include the ability to determine the patient's and family's capacity to understand the risks and benefits of metabolic and bariatric surgery and adhere to required lifestyle modifications leading up to and following such intervention. The evaluative process is rooted in a framework of thoughtful, shared decision making between the patient, parents(s) and/or guardian(s), and medical and surgical providers and ideally includes coordinated and ongoing communication with the patient's medical home.^{654,733}

A referral to a comprehensive metabolic and bariatric surgery center with experience and expertise in treatment of patients younger than 18 years does not necessarily mean the child will ultimately have surgery. This referral provides the family with important information and additional evaluation of risks and benefits for use in making an informed decision. In the case of younger children, recommendations for referral to a comprehensive multidisciplinary obesity treatment center with surgical capability should be considered on a case-by-case basis.

Although data addressing surgical intervention in the younger age group are limited, recent comparative analysis show sustained efficacy and similar safety profiles when compared with adolescents.^{737,738} Additional research is needed before broad

recommendations can be made for children 12 years and younger.

Age is not the sole determinant of eligibility for metabolic and bariatric surgery. The pediatrician or other PHCP should take into account the patient's physical and psychosocial needs. Evaluation for metabolic and bariatric surgery should include a holistic view of the patient and family, including individual and social risk factors. Families should be fully informed of the benefits and risks of metabolic and bariatric surgery, and their preferences are paramount. As highlighted in a recent AAP policy statement, the decision to continue care with a pediatrician or pediatric medical or surgical subspecialist should be made solely by the patient (and the family, as appropriate).⁷⁴⁹

Insurance authorization is a key consideration for individuals considering metabolic and bariatric surgical intervention regardless of age; however, data highlight a significant disparity regarding benefit coverage when comparing pediatric versus adult populations.¹²⁶ Efforts to determine coverage availability, including potential mitigation strategies designed to address coverage gaps, should be the focus of early discussions between the family, medical home, and metabolic and bariatric surgical specialty providers. Children and adolescents who are referred for evaluation for metabolic and bariatric surgery should have this referral visit covered, and those who are deemed eligible for metabolic and bariatric surgery

should have their preparation visits, the surgery itself, hospitalization, postoperative visits, and ongoing care covered.

K. Comprehensive Obesity Treatment (COT) for Children and Adolescents

The essential components of COT of children and adolescents include treatment of the obesity as a chronic disease and evaluation and management of comorbidities. This treatment is delivered by primary care providers and their teams, in collaboration with pediatric obesity specialists, allied health providers, community partners, and metabolic and bariatric surgery teams.

COT: COT includes^{79,80}:

- Providing intensive, longitudinal treatment in the medical home
- Evaluating and monitoring child or adolescent for obesity-related medical and psychological comorbidities
- Identifying and addressing social drivers of health
- Using nonstigmatizing approaches to clinical treatment that honor unique individual qualities of each child and family
- Using MI that addresses nutrition, physical activity, and health behavior change using evidence-based targets for weight reduction and health promotion
- Setting collaborative treatment goals not limited to BMI stabilization or reduction, including goals that reflect improvement or resolution of comorbidities, quality of life, self-image, and other goals related to holistic care
- Integrating weight management components and strategies across appropriate disciplines, which can include intensive health behavior and lifestyle treatment, with pharmacotherapy and metabolic and bariatric surgery if indicated

- Tailoring treatment to the ongoing and changing needs of the individual child or adolescent and the family and community context

Who delivers COT? Ideally, primary care teams and pediatric weight management specialty teams will partner to provide COT for children and adolescents with obesity. Primary care providers evaluate for obesity, evaluate for comorbidities, and provide patient-centered and evidence-based nutrition and physical activity guidance, using MI. Some primary care practices may also be able to provide IHBLT and pharmacotherapeutic options. IHBLT, regardless of where it is delivered, requires the allocation of significantly more time and resources than are typical in the provision of routine well childcare. Coordination in the medical home with additional professionals, such as dietitians, exercise specialists and behavioral health practitioners, will depend on the child's COT plan and available resources. Pediatric health care providers can augment COT with referral to community resources and programs (see algorithm in Appendix 1).

XII. SYSTEMS OF CARE FOR CHILDREN WITH OVERWEIGHT AND OBESITY

Obesity is a chronic disease—similar to asthma and diabetes. Children and adolescents with obesity have the potential to benefit from the foundational standards for systems of care designed for children and youth with special health care needs.^{153,750} CYSHCN are defined as “those who have or are at increased risk for a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally.”⁷⁴ The principles of the chronic care model and the medical home that

can also benefit children with obesity include the following:

A. Provision of Evidence-based Care

All care provided to children and youth with obesity and their families should be evidence based where possible; where evidence-based approaches do not exist, care should be evidence informed and/or based on promising practices.

As pediatric obesity becomes an increasing public health issue and recognizable chronic disease, it becomes critical to rely on evidence-based medicine and or expert recommendations to establish prevention, assessment, and treatment of obesity. To date, several guidelines have been developed and updated to address obesity in children. Examples include “Pediatric Obesity—Assessment, Treatment, and Prevention: An Endocrine Society Clinical Practice Guideline,” and “Expert Committee Recommendations Regarding the Prevention, Assessment, and Treatment of Child and Adolescent Overweight and Obesity.” These guidelines have been critical in moving forward with the prevention, assessment, and treatment of obesity in children as new research is conducted and more new evidence becomes available. This CPG adds to this body of knowledge.^{15,268,685}

B. Partnership With Children and Families

Children and families of children and youth with obesity should be active and core partners in decision making in all levels of care.

Patient-centered care involves not only an understanding of the family's social and cultural context but also an appreciation for their desires as decisions are made about obesity treatment. Family desire for treatment should not be assumed despite attendance at primary care or

specialty weight management visits. Pediatricians' and other PHCPs' use of MI techniques can, however, reinforce the importance of family-driven treatment decisions and foster families' internal motivation that can sustain treatment. As a relationship is built with the family, MI can form the foundation for shared decision making between pediatricians (and other PHCPs) and families about treatment continuation and intensification.⁷⁵¹

The advent of parent and patient EHR portals has enabled bidirectional communication between families and health care teams and can facilitate shared decision making in obesity treatment. Additional facilitators may include tools featuring direct input from families, such as care plans and care coordination agreements.⁷⁵² A patient decision aid to promote shared decision-making about options for adolescent severe obesity treatment was found to be feasible and acceptable by pediatricians (and other pediatric health care providers) and families.⁷⁵³

C. Provision of Health Care That Recognizes Cultural Values

All services and supports for children and youth with obesity and their families should be implemented and delivered in a linguistically appropriate and accessible manner that recognizes cultural values.

All written materials provided to children and youth with obesity and their families should be culturally appropriate and in a manner and format appropriate for children and their parents or caregivers who have limited English proficiency, lower levels of literacy, or sensory impairments. With families with limited English proficiency, vigilance is needed to also provide a trained interpreter and use

growth charts in educating about obesity.⁷⁵⁴

D. Medical Home

Children With Obesity Should be Cared for in a Medical Home

The benefits of a medical home for children and youth with obesity include streamlined care, efficient use of resources (home-, school-, and community-based services), expanded expertise and competence for the involved providers, establishment of a forum for problem solving, and improved satisfaction for the patient, family, and provider. Primary care providers can help identify children early as obesity is developing and base intervention efforts on family dynamics and reduction in high-risk dietary and activity behaviors.⁷⁵⁵ Linkages should also be established between primary care practices and obesity treatment clinics to coordinate care with obesity specialists when necessary (eg, psychologist, dietitian, physician). The medical home also provides an effective model for implementing successful transitions to adult-oriented systems to treat obesity.

E. Transition to Adult Primary Care Providers

Children and adolescents with obesity should have a plan for a transition of care to adult primary care providers.

Transition of care for children with obesity to adult primary care providers is an important field for which evidence and recommendations have been developing. Generally, the concept of transition of care highlights the importance of the collaboration of the primary and/or subspecialty pediatric, adolescent, or family medicine care team, with the adult provider who is preparing to assume the role of primary care provider for a young adult. The importance of a formal transition

plan has been shown in children with diabetes mellitus, sickle cell disease, and congenital heart disease.^{468,756-762} Specifically, transition of care plans have been shown to improve knowledge and self-efficacy and help to integrate the young adult into a new medical home or neighborhood.^{757,761,763,764}

The transition of care literature for childhood obesity is limited but growing.^{752,765,766} In general, transition of care for obesity involves the development of specific goals and a timeline for the transition.⁷⁵⁹ This also includes the development of a registry type system to track and alert pediatricians and other PHCPs when a child reaches the age of 12 years so that outreach to the family to start the transition process can occur.^{468,762,763} It is generally understood that timing of these discussions and the actual transition of care to an adult primary care provider(s) or team depends on the individual needs of the patient and family as well as developmental and neurocognitive abilities. It is also understood that these discussions should be ongoing, at least annually. It is highly recommended that the providers and teams should be in direct communication with each other. Peer support groups are also highly recommended to assist the patient and family and to provide a peer network as part of the medical home.^{752,767}

The complex nature of obesity management and care, including the structured changes involved in behavioral strategies, the connection with community-based programs, and the need for medication therapy and surgery for a number of children and adolescents with obesity highlights the importance of a coordinated transition plan, frequent communication with the

patient and family, and between providers and teams early on.^{752,765,766,768,770}

XIII. BARRIERS AND RECOMMENDATIONS FOR CPG IMPLEMENTATION

Pediatricians and other PHCPs and families face numerous barriers to promoting healthy, active lifestyles and to supporting obesity treatment among children. The successful implementation of this CPG into routine practice requires careful consideration of barriers and facilitators at the policy, community, practice, and provider level that can modify implementation, effectiveness, and sustainability.

A. Policy Level

At the forefront of the policy-level barriers to the implementation of these guidelines are both direct and indirect costs that are associated with recommended evaluation and effective treatment of obesity. In 2010 and again in 2017, the USPSTF designated a grade B classification for evidence of effectiveness of childhood obesity screening and high-intensity, family-based behavioral treatment.⁷⁹ Although a grade B designation should secure reliable insurance payment under the statutes of the Affordable Care Act, the lack of payment by insurers remains a major barrier to childhood obesity treatment.^{769,770} There is currently no consistent coverage of other evidence-based treatment strategies not explicitly included in the USPSTF recommendation.

Direct costs to families include payment for obesity evaluation and treatment that insurers do not pay for, as well as insurance plans with high deductibles or copays. These cost barriers make it difficult for families to access care, obtain laboratory testing, attend follow-up visits, and initiate and/or complete

treatment programs. Differences in payment policies between public and private insurers and restrictive provider networks can make it challenging for pediatricians and other PHCPs to achieve consistent management practices. The overall lack of financial support from either insurance companies or families' inability to pay for treatment programs disincentivizes the expansion of their availability, and there have not been robust studies on best practices for scaling up and sustaining effective treatment programs.

For patients and families, efforts to implement the recommendations for behavior change are also associated with indirect costs (such as time and costs associated with healthier foods and exercise) and are heavily impacted by policies at the federal, state, and local level. These policies include agricultural subsidies, school nutrition, and physical activity standards and curricula, zoning and public spaces to promote safe physical activity, tax deductions for direct advertising of unhealthy foods to minors, and nutrition labeling.⁷⁷¹⁻⁷⁷³ As the recent AAP policy statement on the role of racism in child health and well-being notes, the long-standing structural racism that has plagued these policies manifests in the form of limited access to high-quality education, safe neighborhoods for active play, healthy food, and health care for people of certain races and ethnicities.^{52,774}

One simulation-based cost effectiveness analysis of multiple interventions for childhood obesity found that 3 policies were estimated to be cost-saving; in other words, they would save more in health care costs through reduction in obesity prevalence than it would cost to implement.⁷⁷¹ The policies are: (1) implementing an excise tax on SSBs; (2) elimination of the tax

deduction for companies advertising unhealthy foods to children; and (3) improving nutrition standards for food and beverages sold in schools outside of meals. Another simulation study projected that an excise tax on SSBs, banning child-directed advertising of fast food, and providing after-school physical activity programs would all reduce obesity prevalence while also reducing disparities.⁷⁷³

Implementation Consensus

Recommendation 1: The subcommittee recommends that the AAP and its membership strongly promote supportive payment and public health policies that cover comprehensive obesity prevention, evaluation, and treatment. The medical costs of untreated childhood obesity are well-documented and add urgency to provide payment for treatment.¹²² There is a role for AAP policy and advocacy, in partnership with other organizations, to demand more of our government to accelerate progress in prevention and treatment of obesity for all children through policy change within and beyond the health care sector to improve the health and well-being of children. Furthermore, targeted policies are needed to purposefully address the structural racism in our society that drives the alarming and persistent disparities in childhood obesity and obesity-related comorbidities.

B. Community and Population Level

At the community and population level, SDOHs can limit the implementation and prioritization of health behavior recommendations (including counseling on nutrition, physical activity, sleep, and screen time) for both pediatricians (or other PHCPs) and families. These SDOHs include food security, safe neighborhoods and housing, health

literacy, weight-related parenting skills, household chaos, and access to transportation.

Many communities lack access to evidence-based, high-intensity weight management programs for treatment, either in clinical or community-based settings.^{775,776} Telehealth and mobile technologies are emerging as a potential means to close this access gap, but little is known about the effectiveness of pediatric weight management treatments via these modalities.⁷⁷⁷ The well-described digital divide may also limit their utility among populations disproportionately impacted by obesity.^{778,779} A failure to consider these factors could lead to worsening disparities.

The recent iteration of the Obesity Chronic Care Model proposes an integrated framework for the prevention and treatment of obesity.^{521,780} The framework discusses the importance of coordinating and integrating approaches to address obesity across clinical and community systems as well as stakeholders. In doing so, an integrated approach strives to identify and address barriers to equitable implementation, access to healthier options, and data sharing. An important part of integration includes the identification of an integrator or convener that brings stakeholders together in a collaborative effort to address population health. Integrators are essential to addressing social determinants of health and complex problems that no single stakeholder can address.

Implementation Consensus Recommendation 2: The subcommittee recommends that public health agencies, community organizations, health care systems, health care providers, and community members partner with each other to expand access

to evidence-based pediatric obesity treatment programs and to increase community resources that address social determinants of health in promoting healthy, active lifestyles.⁵²¹

C. Practice and Provider Level

At the practice and provider level, classic barriers to implementation of guidelines include the lack of time, resources, knowledge, awareness, self-efficacy (confidence in one's ability to perform a behavior), and outcome expectancy (the belief that a recommended behavior will lead to a specific effect).⁷⁸¹ Evidence indicates that clinical decision support (CDS) can be delivered through EHR systems to help overcome some of these practice- and provider-level barriers and improve evaluation and effective management. Evidence-based CDS tools include assessment components (ie, flagging abnormal heights, weights, and BMIs) and provision of suggestions for obesity treatment, such as order sets for recommended laboratory tests or other follow-up actions.^{529,782-784} Most pediatric providers currently use EHRs that calculate and plot BMI; however, limitations of EHR systems hinder rapid dissemination and implementation of innovations to support practice.⁷⁸⁵⁻⁷⁸⁷

Implementation Consensus Recommendation 3: The subcommittee recommends that EHR vendors, health systems, and practices implement CDS systems broadly in EHRs to provide prompts and facilitate best practices for managing children and adolescents with obesity.

At the same time, EHRs can only do so much and are ineffective in the absence of a clinical workforce that is knowledgeable about evidence-based obesity treatment and skilled in delivering high-quality, patient-centered

care that will yield improved health outcomes for children with obesity. The recent AAP policy statement on weight stigma highlights the detrimental effects of weight bias and ineffective approaches to the diagnosis and management of obesity.²⁸

Implementation Consensus Recommendation 4: The subcommittee recommends that medical and other health professions schools, training programs, boards, and professional societies improve education and training opportunities related to obesity for both practicing providers and in preprofessional schools and residency and fellowship programs. Such training includes the underlying physiologic basis for weight dysregulation, MI, weight bias, the social and emotional impact of obesity on patients, the need to tailor management to SDoHs that impact weight, and weight-related outcomes and other emerging science.

XIV. EVIDENCE GAPS AND FUTURE RESEARCH DIRECTIONS

Research in the field of pediatric overweight and obesity has rapidly increased over the past decade and supports the evidence-based recommendations and guidelines contained in this CPG on the assessment and management of pediatric overweight and obesity. Although research has progressed in these areas, significant gaps remain and are described in detail in the accompanying technical reports. The gaps and limitations that are most relevant to pediatricians and other PHCPs treating children with obesity include duration and heterogeneity of treatment effects and limits in our understanding of how specific treatment components interact.

- **Duration of treatment effects.** Limited research with long-term follow-up exists to determine:

(1) whether treatment leads to sustained weight improvements, and (2) how comorbidities develop throughout childhood. Longer-term data are needed to establish sufficient weight loss or cardiovascular improvements influencing health into adulthood.

- **Heterogeneity of treatment effects and special populations.**

Current research does not provide sufficient information about the heterogeneity of treatment effects for obesity interventions, limiting our ability to identify which treatment is most likely to be effective for a specific child. Many factors may increase obesity risk and impact treatment course but are poorly isolated in studies to date. These factors may include geographical region, food insecurity, poverty, ACEs, and other social drivers. Perhaps most importantly, severity of obesity has not been clearly considered in most interventions, and treatment is likely to have different effectiveness in children with greater severity of disease. Similar to obesity research, most research on treatment and comorbidities use relatively restrictive inclusion criteria, excluding children with comorbidities (including mental health conditions), children with physical activity limitations, children with disabilities, or those using medications. In clinical practice, these children often have the greatest need for support in addressing obesity.

- **Limited understanding of specific components, dose, and duration.** Published intervention studies often provided limited information about the dose, duration, and specifics of the intervention components.⁸⁰ This limitation makes it difficult to provide detailed information about specific intervention content, behavior change

techniques, and approaches to improve retention and family motivation. Further, the limited research on potential synergies among lifestyle intervention components as well as pharmaceutical and surgical interventions prevents the development of individualized treatment plans tailored to a child's weight and health status, motivation, and readiness.

Despite these limitations, half of the lifestyle randomized-control trials reviewed were effective in reducing adiposity. Reports of future studies detailing specific treatment components, implementation of behavioral approaches, provider involvement in clinical practice, and the socioeconomic and cultural context of the family and community are needed to better understand which interventions work, for whom, and in what situations.

In addition to these critical areas for future research, there are other limitations that should be considered in the context of the guidelines.

For comorbidity assessment, studies were mostly cross-sectional, limiting the ability to assess within-individual changes in comorbidity prevalence across age and obesity class and, therefore, guidance on the appropriate age to begin laboratory evaluation for cardiometabolic comorbidities. Epidemiologic studies intended for specific age ranges and that examine comorbidity prevalence across different levels of overweight and obesity would help identify specific ages and BMI classes with increased prevalence of cardiometabolic comorbidities to focus further research. Longitudinal studies would also help identify the optimal age and BMI ranges to begin evaluation and are needed to monitor progression of comorbidity related to age and BMI level and provide guidance on the recommended frequency of

comorbidity reevaluation. Consistent thresholds of laboratory values across studies are also needed.

Although many treatment studies examined change in biomedical outcomes as markers of secondary prevention of comorbidities, few studies in primary care evaluated outcomes other than BMI. Additionally, studies that assess if comorbidity assessment and/or diagnosis influences patient and family engagement in weight management treatment are lacking. The role of SDOHs and culture in the treatment of obesity comorbidities is also limited. Studies examining motivation for behavior change related to health outcomes and inclusion of SDOH factors in treatment outcomes are needed. Finally, most studies provided no or very limited assessment of harms or unintended consequences. In general, behavioral interventions carry low-risk of harms; this is not well-documented in the existing literature, however, as few studies report adverse events.

The scope of the evidence review for this CPG did not include primary prevention of obesity or assessment and treatment of children 0 to 2 years of age. Early prevention of obesity is important, as 1 in 7 preschool-aged children already have obesity, and disparities in obesity are evident in the first years of life.³ Identification and guidance on treatment strategies in this population are needed. Resources for primary prevention in children of all ages and treatment of children younger than 2 years can be found on the AAP Institute for Healthy Childhood Weight's Web site (www.ihcw.aap.org) and are also provided in the implementation materials. Future CPGs should incorporate the voices of caregivers, children or adolescents, and organizations that represent families to lend important context.

TABLE 21 List of Gaps

Type of Gap	Example of Gap
Epidemiology	<ul style="list-style-type: none"> • Key drivers of reducing obesity prevalence • Predictors of severe obesity • Factors associated with obesity among racial and ethnic groups, including impact of SDoHs on disparities
Definition or measurement	<ul style="list-style-type: none"> • Identification of medical costs associated with obesity and comorbidities • Alternative, accurate measurements of adiposity in primary care; • BMI trajectories in clinical practice and response to treatment over time; • BMI trajectories and development of comorbid conditions; • BMI status and trajectories among race/ethnic groups and the impact of social drivers on BMI status and trajectories
Risk factors	<ul style="list-style-type: none"> • Mechanism by which maternal obesity predisposes to adverse outcomes in the offspring • Impact of sedentary behavior alone on BMI and comorbidities • Improved understanding of associations between obesity and food insecurity
Comorbidities	<ul style="list-style-type: none"> • Age to begin evaluation for cardiometabolic comorbidities • Frequency of evaluation to monitor progression of comorbidity • Comorbidity identification as motivation for behavior change • Role of social determinants of health in obesity comorbidities, especially among minority populations • Role of social determinants of health in obesity comorbidities
Treatment	<ul style="list-style-type: none"> • Tailored age-based treatment approaches • Evidence-based treatment options for 0–5 = year-olds • Optimal level of parent involvement among adolescents in weight management • Optimizing MI use with respect to training, fidelity to the MI process, and potential patient characteristics • Evidence for specific components of intensive lifestyle intervention on BMI trajectory in clinical practice • Approaches to intensive lifestyle intervention that are most effective, including clustering of behavioral recommendations, messaging, delivery, and implementation, especially in primary care • Optimal duration of treatment, including strategies to address attrition and sustainability • Adverse events of treatment • Treatment outcomes by age, degree of obesity, and social determinants of health • Studies reporting long term outcomes are limited • Paucity of published studies reporting negative outcomes • Evaluation of intervention on quality of life and mental health • Studies of EHR tools, including clinical decision support, to improve attention to weight, counseling, and referral • Telemedicine and electronic and mobile health approaches • Robust community programs with clinical linkages • Cultural considerations in wt management
Systems of care	<ul style="list-style-type: none"> • Feasibility of application and benefits of recommended systems of care for CYSHCN in obesity, including care transition strategies on the adolescents' and young adult's improvements in the treatment of obesity and health outcomes
Barriers to and facilitators of CPG implementation	<ul style="list-style-type: none"> • Evidence informing best practices for rapid, cost-effective, and sustainable scale up of effective treatment program for childhood obesity that balance fidelity with adaptability to unique contexts • Research addressing the inconclusive evidence around technology-based interventions for obesity prevention⁷⁷⁷

CPG, clinical practice guideline; CYSHCN, children and youth with special health care needs; EHR, electronic health record; MI, motivational interviewing.

In addition to the above, a list of other gaps and considerations for further research are provided in Table 21.

XV. CONCLUSION—PUTTING IT ALL TOGETHER

Pediatricians and other PHCPs now have more evidence-based tools than

ever before that support obesity treatment that is effective, provides ongoing health benefits, supports children and families longitudinally, and reduces potential harms for disordered eating. In contrast to previous recommendations, these clinical guidelines highlight the urgency of providing immediate, intensive obesity

treatment to each patient as soon as they receive the diagnosis of obesity.

As highlighted in the previous sections, there are Key Action Statements (KASs) that, collectively, comprise a holistic patient-centered approach to COT that should be coordinated within the context of

the medical home. These strategies form the basis for applying evidence-based approaches that take the child's health status, family system, community context, and resources for treatment into consideration to create the best evidence-based treatment plan for each individual child. Obesity is a complex chronic disease but societal stigma around obesity results in pervasive weight bias. This makes compassionate and sensitive communication with patients and families even more imperative (see the Communication of BMI and Weight Status to Children and Parents section).

It is important to recognize that treatment of obesity is integral to the treatment of its comorbidities and overweight or obesity and comorbidities should be treated concurrently (KAS 4). It is also important to consider that a child with overweight and obesity and their family require longitudinal and coordinated care in a medical home (KAS 9) (see algorithm in Appendix 1).

Measuring BMI and assessing weight classification (KAS 1) is a screening step that is applied to a practice population of children, which allows the pediatrician and other PHCP's to initiate obesity evaluation. This evaluation is guided by a comprehensive history, physical examination, and diagnostic studies, including those for SDoHs, disordered eating, and mental and behavioral health (KAS 2). Overall guidance for evaluation of comorbidities is that in children 10 years and older, pediatricians and other PHCPs should evaluate for lipid abnormalities, abnormal glucose metabolism, and abnormal liver function in children and adolescents with obesity (BMI \geq 95th percentile) and for lipid

abnormalities in children and adolescents with overweight (BMI 85th to <95th percentile).

For younger children or children who are overweight, the recommendations are more conservative. In KAS 3.1, for children 10 years and older with overweight (BMI 85th to <95th percentile), pediatricians and other PHCPs may evaluate for abnormal glucose metabolism and liver function in the presence of risk factors for T2DM or NAFLD. In children 2 through 9 years of age with obesity (BMI \geq 95th percentile), pediatricians and other PHCPs may evaluate for lipid abnormalities. In addition to laboratory evaluation, pediatricians and other PHCPs should evaluate for hypertension by measuring blood pressure at every visit starting at 3 years of age in children and adolescents with overweight and obesity (KAS 8). More specific guidance for further evaluation for each comorbidity is included in the Appendices, along with discussion of other obesity-related comorbidities.

The purpose of the evaluation is to determine the child's individual health status, including the presence and extent of obesity related comorbidities, obesity risk factors present in the child's history and environment, and the resources available to the family to conduct obesity treatment. Most importantly, comorbidities must be addressed concurrently with treatment of obesity (KAS 4).

MI is a collaborative approach to conversation about change that is a core component of delivering of COT, including engaging patients and families in addressing overweight and obesity (KAS 10), setting goals, and promoting participation in available resources and programs.

Providing or referring to intensive IHBLT, as described in KAS 11, is a foundational aspect of COT. Pediatricians and other PHCPs should provide or refer children 6 years and older and may provide or refer children 2 through 5 years with overweight and obesity to IHBLT. IHBLT is more effective with greater contact hours; the most effective programs include 26 or more hours of face-to-face, family-based, multicomponent treatment over a 2- to 12-month period. IHBLT provides ongoing behavioral and lifestyle support to the child and family and treatment of obesity-related comorbidities.

Delivering IHBLT requires being able to address nutrition and activity in a holistic manner, using a family-centered and nonstigmatizing approach that acknowledges structural and contextual drivers of obesity and follows the principles of the chronic care model and medical home, in the same manner as other special health care needs (KAS 9). IHBLT should be delivered by primary care providers and their teams, in collaboration with pediatric obesity specialists, allied health providers, and community partners. If IHBLT is not available, the pediatrician or other PHCP should deliver the highest-intensity HBLT possible. In addition, the pediatrician or other PHCP serves as a medical home for the patient, coordinating care, advocating for the patient and family, and supporting transition to adult care.

Concurrent treatment of obesity and obesity-related comorbidities is crucial as is the provision of weight loss pharmacologic treatment and metabolic and bariatric surgery to patients according to indications, risks, and benefits of these modalities. Weight loss pharmacotherapy and metabolic bariatric surgery are evidence-based obesity treatment modalities that

should be available and offered to patients when indicated and should always occur along with IHBLT. Pediatricians and other PHCPs should offer adolescents 12 years and older with obesity weight loss pharmacotherapy, according to medication indications, risks, and benefits, as an adjunct to IHBLT (KAS 12). Pediatricians and other PHCPs should offer referral for adolescents 13 and older with severe obesity for evaluation for metabolic and bariatric surgery to local or regional comprehensive multidisciplinary pediatric metabolic and bariatric surgery centers (KAS 13; see Table 22).

Pediatricians and other PHCPs play a crucial role in providing COT as primary treatment providers, in coordinating care with subspecialists and in the community, and in advocating for obesity treatment resources and elimination of weight bias and stigma. Because obesity is a chronic disease with exacerbations and remissions, children and adolescents with obesity need appropriate reassessments of medical and psychological risks and comorbidities with appropriate modifications to their treatment plan. COT also requires ongoing evaluation and capacity building of both practice and community resources that can aid the family in addressing SDoHs as needed.

Obesity in children and adolescents is a complex, multifactorial, and treatable disease. Evidence for successful treatment, despite stated gaps and complexities, gives hope to patients and families that pediatricians and PHCPs can successfully assess and address the disease of obesity with an individualized and compassionate approach. In contrast to earlier practices of watchful waiting or following a staged approach to intensifying treatment, this CPG supports early treatment at the highest level of intensity appropriate for and available to the child. It is

TABLE 22 Role of the Pediatrician or PCHP

The Continuum of Obesity Care and the Role of PCP or PHCP	Role of the Pediatrician or PHCP
Focus	
Diagnosis and measurement	<ul style="list-style-type: none"> ✓Measure height and weight ✓Calculate BMI and assess BMI Percentile
Risk factors Evaluation	<ul style="list-style-type: none"> ✓Communicate BMI and weight status to patient and family ✓Assess individual, structural, and contextual risk factors ✓Perform comprehensive patient history ✓Conduct physical exam ✓Evaluate for comorbidities⁰ ✓Order relevant diagnostic studies and laboratories ✓Assess readiness to change ✓Treat obesity and comorbidities concurrently
Treat comorbidities	<ul style="list-style-type: none"> ✓Manage children with overweight & obesity following principles of chronic care model and medical home
Treat obesity	<ul style="list-style-type: none"> ✓Deliver nonstigmatizing care ✓Use MI to engage patient and families in addressing overweight and obesity, set goals and promote participation or utilization of local resources or programs ✓Promptly engage and refer children to intensive HBL T treatment, if available. If intensive HBL T treatment is not available in your area, deliver highest intensity HBL T treatment possible. ✓Foster self-management strategies ✓Refer to subspecialists if needed ✓Serve as medical home, coordinate care, advocate for family, and support transition to adult care. ✓Offer weight loss pharmacotherapy to eligible patients, according to medication indications, risks, and benefits, as an adjunct to HBL T. ✓For eligible patients with severe obesity, offer referral to a local or regional comprehensive multidisciplinary pediatric metabolic and bariatric surgery center for surgical evaluation.

This CPG uses "evaluation" to describe patient assessment, and "test" to describe specific tests conducted as part of the evaluation.

hoped that pediatricians and other PHCPs, health systems, community partners, payers, and policy makers will recognize the significance and urgency outlined by this CPG to advance the equitable and universal provision of treatment of the chronic disease of obesity in children and adolescents.

SUBCOMMITTEE ON OBESITY

Sarah E. Hampl, MD, FAAP
Sandra G. Hassink, MD, FAAP
Asheley C. Skinner, PhD
Sarah C. Armstrong, MD, FAAP

Sarah E. Barlow, MD, MPH, FAAP
Christopher F. Bolling, MD, FAAP
Kimberly C. Avila Edwards, MD, FAAP
Ihuoma Eneli, MD, MS, FAAP
Robin Hamre, MPH
Madeline M. Joseph, MD, FAAP
Doug Lunsford, MEd
Eneida Mendonca, MD, PhD, FAAP
Marc P. Michalsky, MD, MBA, FAAP
Nazrat Mirza, MD, ScD, FAAP
Eduardo R. Ochoa, Jr, MD, FAAP
Mona Sharifi, MD, MPH, FAAP
Amanda E. Staiano, PhD, MPP
Ashley E. Weedn, MD, MPH, FAAP

Susan K. Flinn, MA
Jeanne Lindros, MPH
Kymika Okechukwu, MPA

ACKNOWLEDGMENTS

We thank Pia Daniels, MPH, PMP, Program Manager, Evidence-Based Medicine Initiatives and Jeremiah Salmon, MPH, Manager, Clinical Initiatives, Institute for Healthy Childhood Weight. We also thank Brook Belay, MD, and Alyson B. Goodman, CDC liaisons to the writing committee, for their technical input.

filed conflict of interest statements with the American Academy of Pediatrics. Any conflicts have been resolved through a process approved by the Board of Directors. The American Academy of Pediatrics has neither solicited nor accepted any commercial involvement in the development of the content of this publication.

The guidance in this report does not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

All clinical practice guidelines from the American Academy of Pediatrics automatically expire 5 years after publication unless reaffirmed, revised, or retired at or before that time.

DOI: <https://doi.org/10.1542/peds.2022-060640>

Address correspondence to Sarah Hampl, MD. E-mail: shampl@cmh.edu

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

Copyright © 2023 by the American Academy of Pediatrics

FUNDING: No external funding.

FINANCIAL/CONFLICT OF INTEREST DISCLOSURES: An Independent review for bias was completed by the American Academy of Pediatrics. Dr Barlow has disclosed a financial relationship with the Eunice Kennedy Shriver National Institute of Child Health and Human Development as a co-investigator.

REFERENCES

- Centers for Disease Control and Prevention. Prevalence of childhood obesity in the United States. 2021. Available at: <https://www.cdc.gov/obesity/data/childhood.html>. Accessed October 5, 2022
- Fryar CD, Carroll MD, Afful J. Prevalence of overweight, obesity, and severe obesity among children and adolescents aged 2–19 years: United States, 1963–1965 through 2017–2018. *NCHS E-Health Stats*. 2020. Available at: <https://www.cdc.gov/nchs/data/hestat/obesity-child-17-18/obesity-child.htm>. Accessed October 5, 2022
- Hales CM, Fryar CD, Carroll MD, Freedman DS, Ogden CL. Trends in obesity and severe obesity prevalence in US youth and adults by sex and age, 2007–2008 to 2015–2016. *JAMA*. 2018;319(16):1723–1725
- Ogden CL, Fryar CD, Martin CB, et al. Trends in obesity prevalence by race and Hispanic origin—1999–2000 to 2017–2018. *JAMA*. 2020;324(12):1208–1210
- Centers for Disease Control and Prevention. Prevalence of childhood obesity in the United States. 2021. Available at: <https://www.cdc.gov/obesity/data/childhood.html>. Accessed October 5, 2022
- National Institutes of Health. *Obesity Research Task Force. Strategic Plan for NIH Obesity Research*. Bethesda, MD: US Department of Health and Human Services, National Institutes of Health; 2011
- Kyle TK, Dhurandhar EJ, Allison DB. Regarding obesity as a disease: evolving policies and their implications. *Endocrinol Metab Clin North Am*. 2016; 45(3):511–520
- Centers for Disease Control and Prevention. About social determinants of health (SDOH). Available at: <https://www.cdc.gov/socialdeterminants/about.html>. Accessed October 5, 2022
- Medvedyuk S, Ahmednur A, Raphael D. Ideology, obesity and the social determinants of health: a critical analysis of the obesity and health relationship. *Crit Public Health*. 2018;28(5):573–585
- Kaiser Permanente Research Affiliates, Evidence-based Practice Center. *Multi-component Behavioral Interventions for Weight Management in Children and Adolescents Who Are Overweight or With Obesity: A Systematic Evidence Review for the American Psychological Association*, Portland, OR: Kaiser

- Permanente Research Affiliates Evidence-based Practice Center; 2016
11. O'Connor EA, Evans CV, Burda BU, et al. *Screening for Obesity and Interventions for Weight Management in Children and Adolescents: A Systematic Evidence Review for the U.S. Preventive Services Task Force*. Rockville, MD: Agency for Healthcare Research and Quality; 2017
 12. Barlow SE, Dietz WH. Obesity evaluation and treatment: expert committee recommendations. *Pediatrics*. 1998; 102(3):E29
 13. Davis MM, Gance-Cleveland B, Hassink S, Johnson R, Paradis G, Resnicow K. Recommendations for prevention of childhood obesity. *Pediatrics*. 2007; 120(Suppl 4):S229–S253
 14. Krebs NF, Himes JH, Jacobson D, Nicklas TA, Guilday P, Styne D. Assessment of child and adolescent overweight and obesity. *Pediatrics*. 2007;120(Suppl 4):S193–S228
 15. Spear BA, Barlow SE, Ervin C, et al. Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics*. 2007;120 (Suppl 4):S254–S288
 16. American Academy of Pediatrics, Institute for Healthy Childhood Weight. Algorithm for the assessment and management of childhood obesity in patients 2 years and older. Available at: https://downloads.aap.org/AAP/PDF/algorithm_brightfutures_032819.pdf?_ga=2.59652141.1052480977.1664918331-1880284146.1663164182. Accessed October 5, 2022
 17. Timper K, Brüning JC. Hypothalamic circuits regulating appetite and energy homeostasis: pathways to obesity. *Dis Model Mech*. 2017; 10(6):679–689
 18. Baumer Y, Powell-Wiley TM. Interdisciplinary approaches are fundamental to decode the biology of adversity. *Cell*. 2021;184(11):2797–2801
 19. Gao W, Liu J-L, Lu X, Yang Q. Epigenetic regulation of energy metabolism in obesity. *J Mol Cell Biol*. 2021; 13(7):480–499
 20. Browne NT, Hodges EA, Small L, et al. Childhood obesity within the lens of racism. *Pediatr Obes*. 2022;17(5):e12878
 21. Pratt CA, Loria CM, Arteaga SS, et al. A systematic review of obesity disparities research. *Am J Prev Med*. 2017;53(1):113–122
 22. Trust for America's Health. State of obesity 2018: better policies for a healthier America. Available at: <https://www.tfah.org/report-details/the-state-of-obesity-2018/>. Accessed October 5, 2022
 23. Alvarado SE. Neighborhood disadvantage and obesity across childhood and adolescence: evidence from the NLSY children and young adults cohort (1986-2010). *Soc Sci Res*. 2016;57:80–98
 24. Linton JM, Green A; Council on Community Pediatrics. Providing care for children in immigrant families. *Pediatrics*. 2019;144(3):e20192077
 25. Ogden CL, Carroll MD, Fakhouri TH, et al. Prevalence of obesity among youths by household income and education level of head of household—United States, 2011-2014. *MMWR Morb Mortal Wkly Rep*. 2018;67(6):186–189
 26. Eales L, Reynolds AJ, Ou S-R. Childhood predictors of adult obesity in the Chicago Longitudinal Study. *Prev Med*. 2020;132:105993
 27. Ortega Hinojosa AM, MacLeod KE, Balmes J, Jerrett M. Influence of school environments on childhood obesity in California. *Environ Res*. 2018; 166:100–107
 28. Pont SJ, Puhl R, Cook SR, Slusser W; Section on Obesity; Obesity Society. Obesity Society. Stigma experienced by children and adolescents with obesity. *Pediatrics*. 2017;140(6):e20173034
 29. Kivimäki M, Vahtera J, Tabák AG, et al. Neighbourhood socioeconomic disadvantage, risk factors, and diabetes from childhood to middle age in the Young Finns Study: a cohort study. *Lancet Public Health*. 2018;3(8): e365–e373
 30. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999;896:173–188
 31. Marmot M. Social determinants of health inequalities. *Lancet*. 2005; 365(9464):1099–1104
 32. Vargas CM, Stines EM, Granado HS. Health-equity issues related to childhood obesity: a scoping review. *J Public Health Dent*. 2017;77(Suppl 1):S32–S42
 33. Byrd AS, Toth AT, Stanford FC. Racial disparities in obesity treatment. *Curr Obes Rep*. 2018;7(2):130–138
 34. Rossen LM, Talih M. Social determinants of disparities in weight among US children and adolescents. *Ann Epidemiol*. 2014;24(10):705–713.e2
 35. Raphael JL, Cooley WC, Vega A, et al. Outcomes for children with chronic conditions associated with parent- and provider-reported measures of the medical home. *J Health Care Poor Underserved*. 2015;26(2):358–376
 36. Ward ZJ, Long MW, Resch SC, Giles CM, Craddock AL, Gortmaker SL. Simulation of growth trajectories of childhood obesity into adulthood. *N Engl J Med*. 2017;377(22):2145–2153
 37. Australian Institute of Health and Welfare. Chronic diseases 2016. Available at: www.aihw.gov.au/chronic-diseases/. Accessed September 16, 2020
 38. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. *Obes Rev*. 2016;17(2):95–107
 39. Jung RT. Obesity as a disease. *Br Med Bull*. 1997;53(2):307–321
 40. Jastreboff AM, Kotz CM, Kahan S, Kelly AS, Heymsfield SB. Obesity as a disease: The Obesity Society 2018 position statement. *Obesity (Silver Spring)*. 2019;27(1):7–9
 41. Dixon B, Peña M-M, Taveras EM. Life-course approach to racial/ethnic disparities in childhood obesity. *Adv Nutr*. 2012;3(1):73–82
 42. Council on Community Pediatrics and Committee on Native American Child Health. Policy statement—health equity and children's rights. *Pediatrics*. 2010; 125(4):838–849
 43. Puhl R, Suh Y. Health consequences of weight stigma: implications for obesity prevention and treatment. *Curr Obes Rep*. 2015;4(2):182–190
 44. Kirk SFL, Ramos Salas X, Alberga AS, Russell-Mayhew S. *Canadian Adult Obesity Clinical Practice Guidelines*:

- Reducing Weight Bias in Obesity Management, Practice and Policy*. Edmonton, Alberta: Canadian Association of Bariatric Physicians and Surgeons, Obesity Canada; 2020
45. Keating DP, Hertzman C, eds. *Developmental Health and the Wealth of Nations: Social, Biological, and Educational Dynamics*. New York, NY: Guilford Press; 1999
 46. Krieger N. A glossary for social epidemiology. *J Epidemiol Community Health*. 2001;55(10):693–700
 47. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med*. 1993;22(2):167–177
 48. Whitehead M, Dahlgren G. What can be done about inequalities in health? *Lancet*. 1991;338(8774):1059–1063
 49. American Academy of Pediatrics, Committee on Community Pediatrics, Committee on Native American Child Health. Policy statement—health equity and children’s rights. *Pediatrics*. 2010;125(4):838–849
 50. Jones CP, Truman BI, Elam-Evans LD, et al. Using “socially assigned race” to probe white advantages in health status. *Ethn Dis*. 2008;18(4):496–504
 51. Paradies Y, Ben J, Denson N, et al. Racism as a determinant of health: a systematic review and meta-analysis. *PLoS One*. 2015;10(9):e0138511
 52. Trent M, Dooley DG, Dougé J; Section on Adolescent Health; Council on Community Pediatrics; Committee on Adolescence. The impact of racism on child and adolescent health. *Pediatrics*. 2019;144(2):e20191765
 53. Bell CN, Kerr J, Young JL. Associations between obesity, obesogenic environments, and structural racism vary by county-level racial composition. *Int J Environ Res Public Health*. 2019;16(5):861
 54. Cozier YCYJ, Yu J, Coogan PF, Bethea TN, Rosenberg L, Palmer JR. Racism, segregation, and risk of obesity in the Black Women’s Health Study. *Am J Epidemiol*. 2014;179(7):875–883
 55. Bucchianeri MEM, Eisenberg ME, Neumark-Sztainer D. Weightism, racism, classism, and sexism: shared forms of harassment in adolescents. *J Adolesc Health*. 2013;53(1):47–53
 56. Hunte HER. Association between perceived interpersonal everyday discrimination and waist circumference over a 9-year period in the Midlife Development in the United States cohort study. *Am J Epidemiol*. 2011;173(11):1232–1239
 57. Hunte HER. Association between perceived interpersonal everyday discrimination and waist circumference over a 9-year period in the Midlife Development in the United States cohort study. *Am J Epidemiol*. 2011;173(11):1232–1239
 58. Lewis TT, Kravitz HM, Janssen I, Powell LH. Self-reported experiences of discrimination and visceral fat in middle-aged African-American and Caucasian women. *Am J Epidemiol*. 2011;173(11):1223–1231
 59. Molina KM, Estrella ML, Rivera-Olmedo N, Frisard C, Lemon S, Rosal MC. It weigh(t)s on you: everyday discrimination and adiposity among Latinos. *Obesity (Silver Spring)*. 2018;26(9):1474–1480
 60. Gmeiner MS, Warschburger P. Intrapersonal predictors of weight bias internalization among elementary school children: a prospective analysis. *BMC Pediatr*. 2020;20(1):408
 61. Palad CJ, Yarlaga S, Stanford FC. Weight stigma and its impact on paediatric care. *Curr Opin Endocrinol Diabetes Obes*. 2019;26(1):19–24
 62. Rubino F, Puhl RM, Cummings DE, et al. Joint international consensus statement for ending stigma of obesity. *Nat Med*. 2020;26(4):485–497
 63. Gundersen C, Mahatmya D, Garasky S, Lohman B. Linking psychosocial stressors and childhood obesity. *Obes Rev*. 2011;12(5):e54–e63
 64. Slopen N, Goodman E, Koenen KC, Kubzansky LD. Socioeconomic and other social stressors and biomarkers of cardiometabolic risk in youth: a systematic review of less studied risk factors. *PLoS One*. 2013;8(5):e64418
 65. Halfon N, Larson K, Son J, Lu M, Bethell C. Income inequality and the differential effect of adverse childhood experiences in US children. *Acad Pediatr*. 2017;17(7S):S70–S78
 66. Eisenburg LK, van Wijk KJE, Liefbroer AC, Smidt N. Accumulation of adverse childhood events and overweight in children: a systematic review and meta-analysis. *Obesity (Silver Spring)*. 2017;25(5):820–832
 67. Suglia SF, Koenen KC, Boynton-Jarrett R, et al; American Heart Association Council on Epidemiology and Prevention; Council on Cardiovascular Disease in the Young; Council on Functional Genomics and Translational Biology; Council on Cardiovascular and Stroke Nursing; and Council on Quality of Care and Outcomes Research. Childhood and adolescent adversity and cardiometabolic outcomes: a scientific statement from the American Heart Association. *Circulation*. 2018;137(5):e15–e28
 68. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am J Prev Med*. 1998;14(4):245–258
 69. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA*. 1999;282(17):1652–1658
 70. Schuler BR, Vazquez C, Kobulsky JM, Schroeder K, Tripicchio GL, Wildfeuer R. The early effects of cumulative and individual adverse childhood experiences on child diet: examining the role of socioeconomic status. *Prev Med*. 2021;145:106447
 71. Wiss DA, Brewerton TD. Adverse childhood experiences and adult obesity: a systematic review of plausible mechanisms and meta-analysis of cross-sectional studies. *Physiol Behav*. 2020;223:112964
 72. Centers for Disease Control and Prevention. Body mass index (BMI). Available at: <https://www.cdc.gov/healthyweight/assessing/bmi/index.html>. Accessed October 5, 2022
 73. Health Resources and Services Administration. National organizations of state and local officials: public health capacity. Available at: <https://www.hrsa.gov/>

- hrsa.gov/grants/find-funding/hrsa-20-084. Accessed October 5, 2022
74. McPherson M, Arango P, Fox H, et al. A new definition of children with special health care needs. *Pediatrics*. 1998; 102(1 Pt 1):137–140
 75. Health Resources and Services Administration. Chronic care model. Available at: <https://www.hrsa.gov/behavioral-health/chronic-care-model>. Accessed October 5, 2022
 76. National Center for Chronic Disease Prevention and Health Promotion. About chronic diseases. Available at: <https://www.cdc.gov/chronicdisease/about/index.htm>. Accessed October 5, 2022
 77. American Academy of Pediatrics. COVID-19 interim guidance. Obesity management and treatment during COVID-19. Available at: <https://services.aap.org/en/pages/2019-novel-coronavirus-covid-19-infections/clinicalguidance/obesity-management-and-treatment-during-covid-19/>. Accessed October 5, 2022
 78. Cuda S, Censani M, O'Hara V, Browne N, Paisley J. *Pediatric Obesity Algorithm*. Centennial, CO: Obesity Medicine Association; 2020–2022
 79. Grossman DC, Bibbins-Domingo K, Curry SJ, et al; US Preventive Services Task Force. Screening for obesity in children and adolescents: US Preventive Services Task Force recommendation statement. *JAMA*. 2017;317(23):2417–2426
 80. Skinner AC. Appraisal of clinical care practices for child obesity prevention and treatment to inform quality improvement. Part I: interventions. *Pediatrics*. 2023;151(2):e2022060642
 81. American Academy of Pediatrics, National Resource Center for Patient/Family-Centered Medical Home. What is a medical home? Available at: <https://medicalhomeinfo.aap.org/overview/Pages/Whatisthemedicalhome.aspx>. Accessed October 5, 2022
 82. National Center on Birth Defects and Developmental Disabilities. Communicating with and about people with disabilities. Available at: <https://www.cdc.gov/ncbddd/disabilityandhealth/materials/factsheets/fs-communicating-withpeople.html>. Accessed October 5, 2022
 83. Skinner AC, Skelton JA. Prevalence and trends in obesity and severe obesity among children in the United States, 1999–2012. *JAMA Pediatr*. 2014;168(6):561–566
 84. Sokol R, Austin A, Chandler C, et al. Screening children for social determinants of health: a systematic review. *Pediatrics*. 2019;144(4):e20191622
 85. US Department of Health and Human Services, Office of Disease Prevention and Health Promotion. Healthy people 2030. Available at: <https://health.gov/healthypeople/objectives-and-data/social-determinants-health>. Accessed October 5, 2022
 86. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents; National Heart, Lung, and Blood Institute. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128(Suppl 5):S213–S256
 87. Flynn JT, Kaelber DC, Baker-Smith CM, et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 2017;140(6):e20173035
 88. Vos MB, Abrams SH, Barlow SE, et al. NASPGHAN clinical practice guideline for the diagnosis and treatment of nonalcoholic fatty liver disease in children: recommendations from the Expert Committee on NAFLD (ECON) and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN). *J Pediatr Gastroenterol Nutr*. 2017;64(2):319–334
 89. Marcus CL, Brooks LJ, Draper KA, et al; American Academy of Pediatrics. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics*. 2012;130(3):e714–e755
 90. American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of medical care in diabetes-2021. *Diabetes Care*. 2021; 44(Suppl 1):S15–S33
 91. World Health Organization. *WHO Child Growth Standards: Length/Height-for-Age, Weight-for-Age, Weight-for-Length, Weight-for-Height and Body Mass Index-for-Age: Methods and Development*. Geneva, Switzerland: World Health Organization; 2006
 92. Barlow SE; Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*. 2007;120(Suppl 4):S164–S192
 93. American Academy of Pediatrics. *Evidence-Based Clinical Practice Guidelines Development and Implementation Manual*. Itasca, IL: American Academy of Pediatrics; 2019
 94. Centers for Disease Control and Prevention. Childhood obesity causes and consequences. Available at: <https://www.cdc.gov/obesity/childhood/causes.html>. Accessed October 5, 2022
 95. Centers for Disease Control and Prevention. Defining childhood obesity. Available at: <https://www.cdc.gov/obesity/childhood/defining.html>. Accessed October 5, 2022
 96. Skinner AC, Ravanbakht SN, Skelton JA, Perrin EM, Armstrong SC. Prevalence of obesity and severe obesity in US children, 1999–2016. *Pediatrics*. 2018;141(3):e20173459
 97. Lange SJ, Kompaniyets L, Freedman DS, et al; DNP3. Longitudinal trends in body mass index before and during the COVID-19 pandemic among persons aged 2–19 years—United States, 2018–2020. *MMWR Morb Mortal Wkly Rep*. 2021;70(37):1278–1283
 98. Lobstein T, Neveux M, Brown T, et al; STOP project consortium. Social disparities in obesity treatment for children age 3–10 years: a systematic review. *Obes Rev*. 2021;22(2):e13153
 99. Vazquez CE, Cubbin C. Socioeconomic status and childhood obesity: a review of literature from the past decade to inform intervention research. *Curr Obes Rep*. 2020;9(4):562–570
 100. Buoncristiano M, Williams J, Simmonds P, et al. Socioeconomic inequalities in overweight and obesity among 6- to 9-year-old children in 24 countries from the World Health Organization European region. *Obes Rev*. 2021;22(Suppl 6):e13213

101. Rogers R, Eagle TF, Sheetz A, et al. The relationship between childhood obesity, low socioeconomic status, and race/ethnicity: lessons from Massachusetts. *Child Obes.* 2015;11(6):691–695
102. Maiano C, Hue O, Morin AJ, Moullec G. Prevalence of overweight and obesity among children and adolescents with intellectual disabilities: a systematic review and meta-analysis. *Obes Rev.* 2016;17(7):599–611
103. Lynch BA, Agunwamba A, Wilson PM, et al. Adverse family experiences and obesity in children and adolescents in the United States. *Prev Med.* 2016;90:148–154
104. Bullock A, Sheff K, Moore K, Manson S. Obesity and overweight in American Indian and Alaska Native children, 2006–2015. *Am J Public Health.* 2017;107(9):1502–1507
105. Centers for Disease Control and Prevention. Obesity among young children enrolled in WIC. Available at: <https://www.cdc.gov/obesity/data/obesity-among-WIC-enrolled-young-children.html>. Accessed October 5, 2022
106. Ghandour RM, Grason HA, Schempf AH, et al. Healthy people 2010 leading health indicators: how children with special health care needs fared. *Am J Public Health.* 2013;103(6):e99–e106
107. Rimmer JH, Yamaki K, Lowry BM, Wang E, Vogel LC. Obesity and obesity-related secondary conditions in adolescents with intellectual/developmental disabilities. *J Intellect Disabil Res.* 2010;54(9):787–794
108. Must A, Curtin C, Hubbard K, Sikich L, Bedford J, Bandini L. Obesity prevention for children with developmental disabilities. *Curr Obes Rep.* 2014;3(2):156–170
109. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes Rev.* 2008;9(5):474–488
110. Freedman DSLH, Lawman HG, Galuska DA, Goodman AB, Berenson GS. Tracking and variability in childhood levels of BMI: The Bogalusa Heart Study. *Obesity (Silver Spring).* 2018;26(7):1197–1202
111. Li X, Keown-Stoneman CDG, Lebovic G, et al; TARGet Kids! Collaboration. The association between body mass index trajectories and cardiometabolic risk in young children. *Pediatr Obes.* 2020;15(8):e12633
112. Kelly B, West J, Yang TC, Mason D, Hasan T, Wright J. The association between body mass index, primary healthcare use and morbidity in early childhood: findings from the Born In Bradford cohort study. *Public Health.* 2019;167:21–27
113. Umer A, Kelley GA, Cottrell LE, Giacobbi P Jr, Innes KE, Lilly CL. Childhood obesity and adult cardiovascular disease risk factors: a systematic review with meta-analysis. *BMC Public Health.* 2017;17(1):683
114. Andes LJ, Cheng YJ, Rolka DB, Gregg EW, Imperatore G. Prevalence of pre-diabetes among adolescents and young adults in the United States, 2005–2016. *JAMA Pediatr.* 2020;174(2):e194498
115. Moradi M, Mozaffari H, Askari M, Azadbakht L. Association between overweight/obesity with depression, anxiety, low self-esteem, and body dissatisfaction in children and adolescents: a systematic review and meta-analysis of observational studies. *Crit Rev Food Sci Nutr.* 2022;62(2):555–570
116. Schwartz BS, Glass TA, Pollak J, et al. Depression, its comorbidities and treatment, and childhood body mass index trajectories. *Obesity (Silver Spring).* 2016;24(12):2585–2592
117. Akhabue E, Perak AM, Chan C, Greenland P, Allen NB. Racial differences in rates of change of childhood body mass index and blood pressure percentiles. *J Pediatr.* 2018;202:98–105.e6
118. de Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW, Rifai N. Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. *Circulation.* 2004;110(16):2494–2497
119. Martos-Moreno GÁ, Martínez-Villanueva J, González-Leal R, Chowen JA, Argente J. Sex, puberty, and ethnicity have a strong influence on growth and metabolic comorbidities in children and adolescents with obesity: report on 1300 patients (the Madrid Cohort). *Pediatr Obes.* 2019;14(12):e12565
120. Messiah SE, Arheart KL, Luke B, Lipshultz SE, Miller TL. Relationship between body mass index and metabolic syndrome risk factors among US 8- to 14-year-olds, 1999 to 2002. *J Pediatr.* 2008;153(2):215–221
121. Kompaniyets L, Lundeen EA, Belay B, Goodman AB, Tangka F, Blanck HM. Hospital length of stay, charges, and costs associated with a diagnosis of obesity in US children and youth, 2006–2016. *Med Care.* 2020;58(8):722–726
122. Finkelstein EA, Graham WC, Malhotra R. Lifetime direct medical costs of childhood obesity. *Pediatrics.* 2014;133(5):854–862
123. Sherwood NE, Levy RL, Seburg EM, et al. The Healthy Homes/Healthy Kids 5-10 Obesity Prevention Trial: 12 and 24-month outcomes. *Pediatr Obes.* 2019;14(8):e12523
124. Sepúlveda AR, Solano S, Blanco M, Lacruz T, Veiga O. Feasibility, acceptability, and effectiveness of a multidisciplinary intervention in childhood obesity from primary care: Nutrition, physical activity, emotional regulation, and family. *Eur Eat Disord Rev.* 2020;28(2):184–198
125. Kozioł-Kozakowska A, Wójcik M, Furtak A, Januś D, Starzyk JB. A comparison of the impact of two methods of nutrition-behavioral intervention on selected auxological and biochemical parameters in obese prepubertal children-crossover preliminary study. *Int J Environ Res Public Health.* 2019;16(16):2841
126. Inge TH, Coley RY, Bazzano LA, et al; PCORnet Bariatric Study Collaborative. Comparative effectiveness of bariatric procedures among adolescents: the PCORnet bariatric study. *Surg Obes Relat Dis.* 2018;14(9):1374–1386
127. Gulati AK, Kaplan DW, Daniels SR. Clinical tracking of severely obese children: a new growth chart. *Pediatrics.* 2012;130(6):1136–1140
128. Racette SB, Yu L, DuPont NC, Clark BR. BMI-for-age graphs with severe obesity percentile curves: tools for plotting

- cross-sectional and longitudinal youth BMI data. *BMC Pediatr*. 2017;17(1):130
129. Chambers M, Tanamas SK, Clark EJ, et al. Growth tracking in severely obese or underweight children. *Pediatrics*. 2017;140(6):e20172248
 130. Tucker JM, DeFrang R, Orth J, Wakefield S, Howard K. Evaluation of a primary care weight management program in children aged 2-5 years: changes in feeding practices, health behaviors, and body mass index. *Nutrients*. 2019;11(3):498
 131. Freedman DS, Sherry B. The validity of BMI as an indicator of body fatness and risk among children. *Pediatrics*. 2009;124(Suppl 1):S23–S34
 132. Javed A, Jumean M, Murad MH, et al. Diagnostic performance of body mass index to identify obesity as defined by body adiposity in children and adolescents: a systematic review and meta-analysis. *Pediatr Obes*. 2015;10(3):234–244
 133. Lohman TG. *Advances in Body Composition Assessment*. Champaign, IL: Human Kinetics Publishers; 1992
 134. Lazarus R, Baur L, Webb K, Blyth F. Body mass index in screening for adiposity in children and adolescents: systematic evaluation using receiver operating characteristic curves. *Am J Clin Nutr*. 1996;63(4):500–506
 135. Ryder JR, Kaizer AM, Rudser KD, Daniels SR, Kelly AS. Utility of body mass index in identifying excess adiposity in youth across the obesity spectrum. *J Pediatr*. 2016;177:255–261.e2
 136. Wilkes M, Thornton J, Horlick M, et al. Relationship of BMI z score to fat percent and fat mass in multiethnic prepubertal children. *Pediatr Obes*. 2019;14(1):10.1111/ijpo12463
 137. Horlick M, Hediger ML. Measurement matters. *J Pediatr*. 2010;156(2):178–179
 138. Dioum A, Gartner A, Maire B, Delpeuch F, Wade S. Body composition predicted from skinfolds in African women: a cross-validation study using air-displacement plethysmography and a black-specific equation. *Br J Nutr*. 2005;93(6):973–979
 139. Wells JCK, Fuller NJ, Dewit O, Fewtrell MS, Elia M, Cole TJ. Four-component model of body composition in children: density and hydration of fat-free mass and comparison with simpler models. *Am J Clin Nutr*. 1999;69(5):904–912
 140. Weber DR, Moore RH, Leonard MB, Zemel BS. Fat and lean BMI reference curves in children and adolescents and their utility in identifying excess adiposity compared with BMI and percentage body fat. *Am J Clin Nutr*. 2013;98(1):49–56
 141. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*. 2004;363(9403):157–163
 142. Freedman DS, Wang J, Maynard LM, et al. Relation of BMI to fat and fat-free mass among children and adolescents. *Int J Obes*. 2005;29(1):1–8
 143. Centers for Disease Control and Prevention, National Center for Health Statistics. Clinical growth charts. Available at: https://www.cdc.gov/growthcharts/clinical_charts.htm. Accessed October 5, 2022
 144. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat 11*. 2002;(246):1–190
 145. Freedman DS, Butte NF, Taveras EM, Goodman AB, Ogden CL, Blanck HM. The limitations of transforming very high body mass indexes into z-scores among 8.7 million 2- to 4-year-old children. *J Pediatr*. 2017;188:50–56.e1
 146. Grummer-Strawn LM, Reinold C, Krebs NF; Centers for Disease Control and Prevention (CDC). Use of World Health Organization and CDC growth charts for children aged 0-59 months in the United States. *MMWR Recomm Rep*. 2010;59(RR-9):1–15
 147. World Health Organization. Weight-for-length/height charts. Available at: <https://www.who.int/tools/child-growth-standards/standards/weight-for-length-height>. Accessed October 5, 2022
 148. Zemel BS, Pipan M, Stallings VA, et al. Growth charts for children with Down syndrome in the United States. *Pediatrics*. 2015;136(5):e1204–e1211
 149. Bailey-Davis L, Kling SMR, Wood GC, et al. Feasibility of enhancing well-child visits with family nutrition and physical activity risk assessment on body mass index. *Obes Sci Pract*. 2019;5(3):220–230
 150. Fedele DA, Janicke DM, McQuaid EL, et al. A behavioral family intervention for children with overweight and asthma. *Clin Pract Pediatr Psychol*. 2018;6(3):259–269
 151. Forsell C, Gronowitz E, Larsson Y, Kjellberg B-M, Friberg P, Mårild S. Four-year outcome of randomly assigned lifestyle treatments in primary care of children with obesity. *Acta Paediatr*. 2019;108(4):718–724
 152. Inge TH, Laffel LM, Jenkins TM, et al; Teen-Longitudinal Assessment of Bariatric Surgery (Teen-LABS) and Treatment Options of Type 2 Diabetes in Adolescents and Youth (TODAY) Consortia. Comparison of surgical and medical therapy for type 2 diabetes in severely obese adolescents. *JAMA Pediatr*. 2018;172(5):452–460
 153. Hagan JF, Shaw JS, Duncan PM, eds. *Bright Futures: Guidelines for Health Supervision of Infants, Children, and Adolescents*, 4th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2017
 154. O'Connor SG, Maher JP, Belcher BR, et al. Associations of maternal stress with children's weight-related behaviours: a systematic literature review. *Obes Rev*. 2017;18(5):514–525
 155. McPherson AC, Hamilton J, Kingsnorth S, et al. Communicating with children and families about obesity and weight-related topics: a scoping review of best practices. *Obes Rev*. 2017;18(2):164–182
 156. Knierim SD, Rahm AK, Haemer M, et al. Latino parents' perceptions of weight terminology used in pediatric weight counseling. *Acad Pediatr*. 2015;15(2):210–217
 157. McPherson AC, Knibbe TJ, Oake M, et al. "Fat is really a four-letter word": Exploring weight-related communication best practices in children with and without disabilities and their caregivers. *Child Care Health Dev*. 2018;44(4):636–643
 158. American Medical Association. *Person-first Language For Obesity*. Policy H-

- 440.821. Chicago, IL: American Medical Association; 2017
159. Ames H, Mosdøl A, Blaasvær N, Nøkleby H, Berg RC, Langøien LJ. Communication of children's weight status: what is effective and what are the children's and parents' experiences and preferences? A mixed methods systematic review. *BMC Public Health*. 2020;20(1):574
 160. Karnik S, Kanekar A. Childhood obesity: a global public health crisis. *Int J Prev Med*. 2012;3(1):1–7
 161. McFarland MR, Wehbe-Alamah HB. Leininger's theory of culture care diversity and universality: an overview with a historical retrospective and a view toward the future. *J Transcult Nurs*. 2019;30(6):540–557
 162. Swinburn B, Egger G, Raza F. Dissecting obesogenic environments: the development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med*. 1999;29(6 Pt 1):563–570
 163. Boyland EJ, Halford JC. Television advertising and branding. Effects on eating behaviour and food preferences in children. *Appetite*. 2013;62:236–241
 164. Boyland EJ, Nolan S, Kelly B, et al. Advertising as a cue to consume: a systematic review and meta-analysis of the effects of acute exposure to unhealthy food and nonalcoholic beverage advertising on intake in children and adults. *Am J Clin Nutr*. 2016; 103(2):519–533
 165. Jenkin G, Madhvani N, Signal L, Bowers S. A systematic review of persuasive marketing techniques to promote food to children on television. *Obes Rev*. 2014;15(4):281–293
 166. Smith R, Kelly B, Yeatman H, Boyland E. Food marketing influences children's attitudes, preferences and consumption: a systematic critical review. *Nutrients*. 2019;11(4):875
 167. Villegas-Navas V, Montero-Simo MJ, Araque-Padilla RA. The effects of foods embedded in entertainment media on children's food choices and food intake: a systematic review and meta-analyses. *Nutrients*. 2020;12(4):964
 168. Sadeghirad B, Duhaney T, Motaghipisheh S, Campbell NR, Johnston BC. Influence of unhealthy food and beverage marketing on children's dietary intake and preference: a systematic review and meta-analysis of randomized trials. *Obes Rev*. 2016;17(10):945–959
 169. Strasburger VC; Committee on Communications, American Academy of Pediatrics. Children, adolescents, and advertising. *Pediatrics*. 2006; 118(6):2563–2569
 170. Council on Communications and Media. Media and young minds. *Pediatrics*. 2016;138(5):e20162591
 171. Poulsen MN, Glass TA, Pollak J, et al. Associations of multidimensional socioeconomic and built environment factors with body mass index trajectories among youth in geographically heterogeneous communities. *Prev Med Rep*. 2019;15:100939
 172. Jia P, Dai S, Rohli KE, et al. Natural environment and childhood obesity: a systematic review. *Obes Rev*. 2021; 22(Suppl 1):e13097
 173. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA*. 2010; 303(3):242–249
 174. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity in the United States, 2009-2010. *NCHS Data Brief*. 2012;82:1–8
 175. Ogden CL, Lamb MM, Carroll MD, Flegal KM. Obesity and socioeconomic status in children and adolescents: United States, 2005-2008. *NCHS Data Brief*. 2010;51(51):1–8
 176. Babey SH, Hastert TA, Wolstein J, Diamant AL. Income disparities in obesity trends among California adolescents. *Am J Public Health*. 2010;100(11): 2149–2155
 177. Children's Defense Fund. *The State of America's Children*. Washington, DC: Children's Defense Fund; 2020
 178. Lee H, Andrew M, Gebremariam A, Lumeng JC, Lee JM. Longitudinal associations between poverty and obesity from birth through adolescence. *Am J Public Health*. 2014;104(5):e70–e76
 179. Li M, Mustillo S, Anderson J. Childhood poverty dynamics and adulthood overweight/obesity: unpacking the black box of childhood. *Soc Sci Res*. 2018; 76:92–104
 180. Isong IA, Rao SR, Bind M-A, Avendaño M, Kawachi I, Richmond TK. Racial and ethnic disparities in early childhood obesity. *Pediatrics*. 2018;141(1): e20170865
 181. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr*. 2004;79(1):6–16
 182. Harvard University, Center on the Developing Child. Key concepts. Toxic stress. Available at: <https://developingchild.harvard.edu/science/key-concepts/toxic-stress/>. Accessed October 5, 2022
 183. Baker EH, Rendall MS, Weden MM. Epidemiological paradox or immigrant vulnerability? Obesity among young children of immigrants. *Demography*. 2015;52(4):1295–1320
 184. Gilbert PA, Khokhar S. Changing dietary habits of ethnic groups in Europe and implications for health. *Nutr Rev*. 2008;66(4):203–215
 185. Gualdi-Russo E, Zaccagni L, Manzon VS, Masotti S, Rinaldo N, Khyatti M. Obesity and physical activity in children of immigrants. *Eur J Public Health*. 2014; 24(Suppl 1):40–46
 186. Buttenheim AM, Pebley AR, Hsieh K, Chung CY, Goldman N. The shape of things to come? Obesity prevalence among foreign-born vs. US-born Mexican youth in California. *Soc Sci Med*. 2013;78:1–8
 187. Gualdi-Russo E, Manzon VS, Masotti S, et al. Weight status and perception of body image in children: the effect of maternal immigrant status. *Nutr J*. 2012;11:85
 188. Flores G, Fuentes-Afflick E, Barbot O, et al. The health of Latino children: urgent priorities, unanswered questions, and a research agenda. *JAMA*. 2002; 288(1):82–90
 189. Dubois L, Francis D, Burnier D, et al. Household food insecurity and childhood overweight in Jamaica and Québec: a gender-based analysis. *BMC Public Health*. 2011;11:199
 190. Burke MP, Martini LH, Çayör E, Hartline-Grafton HL, Meade RL. Severity of household food insecurity is

- positively associated with mental disorders among children and adolescents in the United States. *J Nutr*. 2016;146(10):2019–2026
191. Gross RS, Mendelsohn AL, Fierman AH, Racine AD, Messito MJ. Food insecurity and obesogenic maternal infant feeding styles and practices in low-income families. *Pediatrics*. 2012;130(2):254–261
 192. Coleman-Jensen A, Rabbitt MP, Gregory CA, Singh A. *Household Food Security in the United States in 2018*. Washington, DC: US Department of Agriculture, Economic Research Service; 2019
 193. Powell LM, Chaloupka FJ. Food prices and obesity: evidence and policy implications for taxes and subsidies. *Milbank Q*. 2009;87(1):229–257
 194. Sturm R, Datar A. Body mass index in elementary school children, metropolitan area food prices and food outlet density. *Public Health*. 2005;119(12):1059–1068
 195. Sturm R, Datar A. Food prices and weight gain during elementary school: 5-year update. *Public Health*. 2008;122(11):1140–1143
 196. Lee J, Kubik MY, Fulkerson JA. Diet quality and fruit, vegetable, and sugar-sweetened beverage consumption by household food insecurity among 8- to 12-year-old children during summer months. *J Acad Nutr Diet*. 2019;119(10):1695–1702
 197. Adams EL, Caccavale LJ, Smith D, Bean MK. Food insecurity, the home food environment, and parent feeding practices in the era of COVID-19. *Obesity (Silver Spring)*. 2020;28(11):2056–2063
 198. Kakarala M, Keast DR, Hoerr S. Schoolchildren's consumption of competitive foods and beverages, excluding à la carte. *J Sch Health*. 2010;80(9):429–435, quiz 461–463
 199. Matsuzaki M, Sánchez BN, Acosta ME, Botkin J, Sanchez-Vaznaugh EV. Food environment near schools and body weight—A systematic review of associations by race/ethnicity, gender, grade, and socio-economic factors. *Obes Rev*. 2020;21(4):e12997
 200. Williams J, Scarborough P, Matthews A, et al. A systematic review of the influence of the retail food environment around schools on obesity-related outcomes. *Obes Rev*. 2014;15(5):359–374
 201. Fiechtner L, Kleinman K, Melly SJ, et al. Effects of proximity to supermarkets on a randomized trial studying interventions for obesity. *Am J Public Health*. 2016;106(3):557–562
 202. Griffiths C, Frearson A, Taylor A, Radley D, Cooke C. A cross sectional study investigating the association between exposure to food outlets and childhood obesity in Leeds, UK. *Int J Behav Nutr Phys Act*. 2014;11:138
 203. Zhou Q, Zhao L, Zhang L, et al. Neighborhood supermarket access and childhood obesity: a systematic review. *Obes Rev*. 2021;22(Suppl 1):e12937
 204. Kim Y, Cubbin C, Oh S. A systematic review of neighbourhood economic context on child obesity and obesity-related behaviours. *Obes Rev*. 2019;20(3):420–431
 205. Yang S, Zhang X, Feng P, et al. Access to fruit and vegetable markets and childhood obesity: a systematic review. *Obes Rev*. 2021;22(Suppl 1):e12980
 206. Demory-Luce D. Fast food and children and adolescents: implications for practitioners. *Clin Pediatr (Phila)*. 2005;44(4):279–288
 207. Rosenheck R. Fast food consumption and increased caloric intake: a systematic review of a trajectory towards weight gain and obesity risk. *Obes Rev*. 2008;9(6):535–547
 208. Cobb LK, Appel LJ, Franco M, Jones-Smith JC, Nur A, Anderson CA. The relationship of the local food environment with obesity: a systematic review of methods, study quality, and results. *Obesity (Silver Spring)*. 2015;23(7):1331–1344
 209. Jia P, Luo M, Li Y, Zheng JS, Xiao Q, Luo J. Fast-food restaurant, unhealthy eating, and childhood obesity: a systematic review and meta-analysis. *Obes Rev*. 2021;22(Suppl 1):e12944
 210. Islam MZ, Johnston J, Sly PD. Green space and early childhood development: a systematic review. *Rev Environ Health*. 2020;35(2):189–200
 211. Malacarne D, Handakas E, Robinson O, et al. The built environment as determinant of childhood obesity: a systematic literature review. *Obes Rev*. 2022;23(Suppl 1):e13385
 212. Lovasi GS, Schwartz-Soicher O, Quinn JW, et al. Neighborhood safety and green space as predictors of obesity among preschool children from low-income families in New York City. *Prev Med*. 2013;57(3):189–193
 213. Ghassabian A, Vandenberg L, Kannan K, Trasande L. Endocrine-disrupting chemicals and child health. *Annu Rev Pharmacol Toxicol*. 2022;62:573–594
 214. Kassotis CD, Vandenberg LN, Demeneix BA, Porta M, Slama R, Trasande L. Endocrine-disrupting chemicals: economic, regulatory, and policy implications. *Lancet Diabetes Endocrinol*. 2020;8(8):719–730
 215. Kahn LG, Philippat C, Nakayama SF, Slama R, Trasande L. Endocrine-disrupting chemicals: implications for human health. *Lancet Diabetes Endocrinol*. 2020;8(8):703–718
 216. Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. *Lancet*. 2002;360(9331):473–482
 217. Kral TVE, Moore RH, Chittams J, Jones E, O'Malley L, Fisher JO. Identifying behavioral phenotypes for childhood obesity. *Appetite*. 2018;127:87–96
 218. Baumrind D. Effects of authoritative parental control on child behavior. *Child Dev*. 1966;37(4):887–907
 219. Maccoby EE, Martin JA. *Socialization in the Context of the Family: Parent-Child Interaction*. New York, NY: Wiley; 1983
 220. Sleddens EF, Gerards SM, Thijs C, de Vries NK, Kremers SP. General parenting, childhood overweight and obesity-inducing behaviors: a review. *Int J Pediatr Obes*. 2011;6(2-2):e12–e27
 221. Sokol RL, Qin B, Poti JM. Parenting styles and body mass index: a systematic review of prospective studies among children. *Obes Rev*. 2017;18(3):281–292
 222. Frankel LA, Hughes SO, O'Connor TM, Power TG, Fisher JO, Hazen NL. Parental influences on children's self-regulation of energy intake: insights from developmental literature on emotion regulation. *J Obes*. 2012;2012:327259
 223. Patrick H, Hennessy E, McSpadden K, Oh A. Parenting styles and practices in children's obesogenic behaviors: scientific gaps and future research

- directions. *Child Obes.* 2013;9(Suppl): S73–S86
224. Kakinami L, Barnett TA, Séguin L, Paradis G. Parenting style and obesity risk in children. *Prev Med.* 2015;75:18–22
 225. Bates CR, Buscemi J, Nicholson LM, Cory M, Jagpal A, Bohnert AM. Links between the organization of the family home environment and child obesity: a systematic review. *Obes Rev.* 2018;19(5):716–727
 226. Luger M, Lafontan M, Bes-Rastrollo M, Winzer E, Yumuk V, Farpour-Lambert N. Sugar-sweetened beverages and weight gain in children and adults: a systematic review from 2013 to 2015 and a comparison with previous studies. *Obes Facts.* 2017;10(6):674–693
 227. Muth ND, Dietz WH, Magge SN, Johnson RK; American Academy of Pediatrics; Section on Obesity; Committee on Nutrition; American Heart Association. Public policies to reduce sugary drink consumption in children and adolescents. *Pediatrics.* 2019;143(4): e20190282
 228. Birch LL, Savage JS, Fisher JO. Right sizing prevention. Food portion size effects on children's eating and weight. *Appetite.* 2015;88:11–16
 229. Costa CS, Del-Ponte B, Assunção MCF, Santos IS. Consumption of ultra-processed foods and body fat during childhood and adolescence: a systematic review. *Public Health Nutr.* 2018; 21(1):148–159
 230. Lachat C, Nago E, Verstraeten R, Roberfroid D, Van Camp J, Kolsteren P. Eating out of home and its association with dietary intake: a systematic review of the evidence. *Obes Rev.* 2012;13(4):329–346
 231. Nago ES, Lachat CK, Dossa RA, Kolsteren PW. Association of out-of-home eating with anthropometric changes: a systematic review of prospective studies. *Crit Rev Food Sci Nutr.* 2014;54(9): 1103–1116
 232. Hammons AJ, Fiese BH. Is frequency of shared family meals related to the nutritional health of children and adolescents? *Pediatrics.* 2011;127(6):e1565–e1574
 233. Dallacker M, Hertwig R, Mata J. The frequency of family meals and nutritional health in children: a meta-analysis. *Obes Rev.* 2018;19(5):638–653
 234. Poitras VJ, Gray CE, Janssen X, et al. Systematic review of the relationships between sedentary behaviour and health indicators in the early years (0–4 years). *BMC Public Health.* 2017; 17(Suppl 5):868
 235. Stiglic N, Viner RM. Effects of screen-time on the health and well-being of children and adolescents: a systematic review of reviews. *BMJ Open.* 2019; 9(1):e023191
 236. Tripathi M, Mishra SK. Screen time and adiposity among children and adolescents: a systematic review. *J Public Health (Germany).* 2019; 28:227–244
 237. Viner RM, Cole TJ. Television viewing in early childhood predicts adult body mass index. *J Pediatr.* 2005;147(4): 429–435
 238. Poorolajal J, Sahraei F, Mohamdadi Y, Doosti-Irani A, Moradi L. Behavioral factors influencing childhood obesity: a systematic review and meta-analysis. *Obes Res Clin Pract.* 2020;14(2):109–118
 239. Coombs NA, Stamatakis E. Associations between objectively assessed and questionnaire-based sedentary behaviour with BMI-defined obesity among general population children and adolescents living in England. *BMJ Open.* 2015;5(6):e007172
 240. Strasburger VC; Council on Communications and Media. Children, adolescents, obesity, and the media. *Pediatrics.* 2011;128(1):201–208
 241. Biddle SJ, García Bengoechea E, Wiesner G. Sedentary behaviour and adiposity in youth: a systematic review of reviews and analysis of causality. *Int J Behav Nutr Phys Act.* 2017; 14(1):43
 242. Magee L, Hale L. Longitudinal associations between sleep duration and subsequent weight gain: a systematic review. *Sleep Med Rev.* 2012;16(3): 231–241
 243. Reilly JJ, Armstrong J, Dorosty AR, et al; Avon Longitudinal Study of Parents and Children Study Team. Early life risk factors for obesity in childhood: cohort study. *BMJ.* 2005; 330(7504):1357
 244. Ruan H, Xun P, Cai W, He K, Tang Q. Habitual sleep duration and risk of childhood obesity: systematic review and dose-response meta-analysis of prospective cohort studies. *Sci Rep.* 2015;5:16160
 245. Nedeltcheva AV, Kilkus JM, Imperial J, Kasza K, Schoeller DA, Penev PD. Sleep curtailment is accompanied by increased intake of calories from snacks. *Am J Clin Nutr.* 2009;89(1): 126–133
 246. Weiss A, Xu F, Storfer-Isser A, Thomas A, levers-Landis CE, Redline S. The association of sleep duration with adolescents' fat and carbohydrate consumption. *Sleep.* 2010;33(9): 1201–1209
 247. Larqué E, Labayen I, Flodmark CE, et al. From conception to infancy - early risk factors for childhood obesity. *Nat Rev Endocrinol.* 2019;15(8): 456–478
 248. Nadhiroh SR, Djokosujono K, Utari DM. The association between secondhand smoke exposure and growth outcomes of children: a systematic literature review. *Tob Induc Dis.* 2020;18:12
 249. Tate EB, Wood W, Liao Y, Dunton GF. Do stressed mothers have heavier children? A meta-analysis on the relationship between maternal stress and child body mass index. *Obes Rev.* 2015;16(5):351–361
 250. Kumar S, Kelly AS. Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc.* 2017;92(2):251–265
 251. Solmi M, Köhler CA, Stubbs B, et al. Environmental risk factors and non-pharmacological and nonsurgical interventions for obesity: an umbrella review of meta-analyses of cohort studies and randomized controlled trials. *Eur J Clin Invest.* 2018;48(12): e12982
 252. Jackson DB, Chilton M, Johnson KR, Vaughn MG. Adverse childhood experiences and household food insecurity: findings from the 2016 National Survey of Children's Health. *Am J Prev Med.* 2019;57(5):667–674
 253. Harvard University, Center on the Developing Child. Key concepts. Toxic stress. Available at: <https://>

developingchild.harvard.edu/science/key-concepts/toxic-stress/. Accessed October 5, 2022

254. Chapman DP, Whitfield CL, Felitti VJ, Dube SR, Edwards VJ, Anda RF. Adverse childhood experiences and the risk of depressive disorders in adulthood. *J Affect Disord*. 2004;82(2):217–225
255. Dube SR, Anda RF, Felitti VJ, Chapman DP, Williamson DF, Giles WH. Childhood abuse, household dysfunction, and the risk of attempted suicide throughout the life span: findings from the Adverse Childhood Experiences Study. *JAMA*. 2001;286(24):3089–3096
256. Isohookana R, Marttunen M, Hakko H, Riiipinen P, Riala K. The impact of adverse childhood experiences on obesity and unhealthy weight control behaviors among adolescents. *Compr Psychiatry*. 2016;71:17–24
257. Schilling EA, Aseltine RH Jr, Gore S. Adverse childhood experiences and mental health in young adults: a longitudinal survey. *BMC Public Health*. 2007;7:30
258. Burke NJ, Hellman JL, Scott BG, Weems CF, Carrion VG. The impact of adverse childhood experiences on an urban pediatric population. *Child Abuse Negl*. 2011;35(6):408–413
259. Benyshek DC. The “early life” origins of obesity-related health disorders: new discoveries regarding the intergenerational transmission of developmentally programmed traits in the global cardiometabolic health crisis. *Am J Phys Anthropol*. 2013;152(Suppl 57):79–93
260. McEwen CA, McEwen BS. Social structure, adversity, toxic stress, and intergenerational poverty: an early childhood model. *Annu Rev Sociol*. 2017;43:445–472
261. Maes HH, Neale MC, Eaves LJ. Genetic and environmental factors in relative body weight and human adiposity. *Behav Genet*. 1997;27(4):325–351
262. Stunkard AJ, Foch TT, Hrubec Z. A twin study of human obesity. *JAMA*. 1986;256(1):51–54
263. Elks CE, den Hoed M, Zhao JH, et al. Variability in the heritability of body mass index: a systematic review and meta-regression. *Front Endocrinol (Lausanne)*. 2012;3:29
264. Speliotes EK, Willer CJ, Berndt SI, et al; MAGIC; Procardis Consortium. Association analyses of 249,796 individuals reveal 18 new loci associated with body mass index. *Nat Genet*. 2010;42(11):937–948
265. Vaisse C, Clement K, Durand E, Hercberg S, Guy-Grand B, Froguel P. Melanocortin-4 receptor mutations are a frequent and heterogeneous cause of morbid obesity. *J Clin Invest*. 2000;106(2):253–262
266. Farooqi S, O’Rahilly S. Genetics of obesity in humans. *Endocr Rev*. 2006;27(7):710–718
267. Heymsfield SB, Avena NM, Baier L, et al. Hyperphagia: current concepts and future directions proceedings of the 2nd international conference on hyperphagia. *Obesity (Silver Spring)*. 2014;22(Suppl 1):S1–S17
268. Styne DM, Arslanian SA, Connor EL, et al. pediatric obesity-assessment, treatment, and prevention: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2017;102(3):709–757
269. Cordero P, Li J, Oben JA. Epigenetics of obesity: beyond the genome sequence. *Curr Opin Clin Nutr Metab Care*. 2015;18(4):361–366
270. van Dijk SJ, Molloy PL, Varinli H, Morrison JL, Muhlhauser BS; Members of EpiSCOPE. Epigenetics and human obesity. *Int J Obes*. 2015;39(1):85–97
271. Bell CG, Walley AJ, Froguel P. The genetics of human obesity. *Nat Rev Genet*. 2005;6(3):221–234
272. Bouret S, Levin BE, Ozanne SE. Gene-environment interactions controlling energy and glucose homeostasis and the developmental origins of obesity. *Physiol Rev*. 2015;95(1):47–82
273. Gnawali A. Prematurity and the risk of development of childhood obesity: piecing together the pathophysiological puzzle. A literature review. *Cureus*. 2021;13(12):e20518
274. Uthaya S, Thomas EL, Hamilton G, Doré CJ, Bell J, Modi N. Altered adiposity after extremely preterm birth. *Pediatr Res*. 2005;57(2):211–215
275. Ou-Yang MC, Sun Y, Liebowitz M, et al. Accelerated weight gain, prematurity, and the risk of childhood obesity: a meta-analysis and systematic review. *PLoS One*. 2020;15(5):e0232238
276. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337(13):869–873
277. Lima NMS, Leal VS, Oliveira JS, et al. Overweight among adolescents and nutritional status of their parents: a systematic review. *Cien Saude Colet*. 2017;22(2):627–636
278. Isganaitis E, Suehiro H, Cardona C. Who’s your daddy?: paternal inheritance of metabolic disease risk. *Curr Opin Endocrinol Diabetes Obes*. 2017;24(1):47–55
279. Rasmussen KM, Yaktine AL, eds. *Weight Gain During Pregnancy: Reexamining the Guidelines*. Washington, DC: National Academies Press; 2009
280. Lau EY, Liu J, Archer E, McDonald SM, Liu J. Maternal weight gain in pregnancy and risk of obesity among offspring: a systematic review. *J Obes*. 2014;2014:524939
281. Matusiak K, Barrett HL, Callaway LK, Nitert MD. Periconception weight loss: common sense for mothers, but what about for babies? *J Obes*. 2014;2014:204295
282. Buckley AJ, Jaquiere AL, Harding JE. Nutritional programming of adult disease. *Cell Tissue Res*. 2005;322(1):73–79
283. Logan KM, Gale C, Hyde MJ, Santhakumaran S, Modi N. Diabetes in pregnancy and infant adiposity: systematic review and meta-analysis. *Arch Dis Child Fetal Neonatal Ed*. 2017;102(1):F65–F72
284. Kim SY, Sharma AJ, Callaghan WM. Gestational diabetes and childhood obesity: what is the link? *Curr Opin Obstet Gynecol*. 2012;24(6):376–381
285. Nehring I, Chmitorz A, Reulen H, von Kries R, Ensenauer R. Gestational diabetes predicts the risk of childhood overweight and abdominal circumference independent of maternal obesity. *Diabet Med*. 2013;30(12):1449–1456

286. Lawlor DA, Lichtenstein P, Långström N. Association of maternal diabetes mellitus in pregnancy with offspring adiposity into early adulthood: sibling study in a prospective cohort of 280 866 men from 248 293 families. *Circulation*. 2011;123(3):258–265
287. Catalano PM, Hauguel-De Mouzon S. Is it time to revisit the Pedersen hypothesis in the face of the obesity epidemic? *Am J Obstet Gynecol*. 2011;204(6):479–487
288. Gibson KS, Waters TP, Catalano PM. Maternal weight gain in women who develop gestational diabetes mellitus. *Obstet Gynecol*. 2012;119(3):560–565
289. Heerwagen MJ, Miller MR, Barbour LA, Friedman JE. Maternal obesity and fetal metabolic programming: a fertile epigenetic soil. *Am J Physiol Regul Integr Comp Physiol*. 2010;299(3):R711–R722
290. Dabelea D, Mayer-Davis EJ, Saydah S, et al; SEARCH for Diabetes in Youth Study. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. *JAMA*. 2014;311(17):1778–1786
291. Ge ZJ, Zhang CL, Schatten H, Sun QY. Maternal diabetes mellitus and the origin of non-communicable diseases in offspring: the role of epigenetics. *Biol Reprod*. 2014;90(6):139
292. Rayfield S, Plugge E. Systematic review and meta-analysis of the association between maternal smoking in pregnancy and childhood overweight and obesity. *J Epidemiol Community Health*. 2017;71(2):162–173
293. Ino T. Maternal smoking during pregnancy and offspring obesity: meta-analysis. *Pediatr Int*. 2010;52(1):94–99
294. Oken E, Levitan EB, Gillman MW. Maternal smoking during pregnancy and child overweight: systematic review and meta-analysis. *Int J Obes*. 2008;32(2):201–210
295. Qureshi R, Jadotte Y, Zha P, et al. The association between prenatal exposure to environmental tobacco smoke and childhood obesity: a systematic review. *JBI Database Syst Rev Implementation Reports*. 2018;16(8):1643–1662
296. von Kries R, Toschke AM, Koletzko B, Slikker W Jr. Maternal smoking during pregnancy and childhood obesity. *Am J Epidemiol*. 2002;156(10):954–961
297. Trandafir LM, Temneanu OR. Pre and post-natal risk and determination of factors for child obesity. *J Med Life*. 2016;9(4):386–391
298. Symonds ME, Garnder DS. *The Developmental Environment and the Development of Obesity*. Cambridge, United Kingdom: Cambridge University Press; 2006
299. Oken E, Gillman MW. Fetal origins of obesity. *Obes Res*. 2003;11(4):496–506
300. Dean SV, Lassi ZS, Imam AM, Bhutta ZA. Preconception care: nutritional risks and interventions. *Reprod Health*. 2014;11(Suppl 3):S3
301. Taveras EM, Scanlon KS, Birch L, Rifas-Shiman SL, Rich-Edwards JW, Gillman MW. Association of breastfeeding with maternal control of infant feeding at age 1 year. *Pediatrics*. 2004;114(5):e577–e583
302. Azad MB, Vehling L, Chan D, et al; CHILD Study Investigators. Infant feeding and weight gain: separating breast milk from breastfeeding and formula from food. *Pediatrics*. 2018;142(4):e20181092
303. Appleton J, Russell CG, Laws R, Fowler C, Campbell K, Denney-Wilson E. Infant formula feeding practices associated with rapid weight gain: a systematic review. *Matern Child Nutr*. 2018;14(3):e12602–e12602
304. Pearce J, Langley-Evans SC. The types of food introduced during complementary feeding and risk of childhood obesity: a systematic review. *Int J Obes*. 2013;37(4):477–485
305. Stettler N, Iotova V. Early growth patterns and long-term obesity risk. *Curr Opin Clin Nutr Metab Care*. 2010;13(3):294–299
306. Zheng M, Lamb KE, Grimes C, et al. Rapid weight gain during infancy and subsequent adiposity: a systematic review and meta-analysis of evidence. *Obes Rev*. 2018;19(3):321–332
307. Woo Baidal JA, Locks LM, Cheng ER, Blake-Lamb TL, Perkins ME, Taveras EM. Risk factors for childhood obesity in the first 1,000 days: a systematic review. *Am J Prev Med*. 2016;50(6):761–779
308. Pearce J, Taylor MA, Langley-Evans SC. Timing of the introduction of complementary feeding and risk of childhood obesity: a systematic review. *Int J Obes*. 2013;37(10):1295–1306
309. Cox LM, Blaser MJ. Antibiotics in early life and obesity. *Nat Rev Endocrinol*. 2015;11(3):182–190
310. Miller SA, Wu RKS, Oremus M. The association between antibiotic use in infancy and childhood overweight or obesity: a systematic review and meta-analysis. *Obes Rev*. 2018;19(11):1463–1475
311. Poulsen MN, Pollak J, Bailey-Davis L, Hirsch AG, Glass TA, Schwartz BS. Associations of prenatal and childhood antibiotic use with child body mass index at age 3 years. *Obesity (Silver Spring)*. 2017;25(2):438–444
312. Aghaali M, Hashemi-Nazari SS. Association between early antibiotic exposure and risk of childhood weight gain and obesity: a systematic review and meta-analysis. *J Pediatr Endocrinol Metab*. 2019;32(5):439–445
313. Li DK, Chen H, Ferber J, Odouli R. Infection and antibiotic use in infancy and risk of childhood obesity: a longitudinal birth cohort study. *Lancet Diabetes Endocrinol*. 2017;5(1):18–25
314. Rasmussen SH, Shrestha S, Bjerregaard LG, et al. Antibiotic exposure in early life and childhood overweight and obesity: a systematic review and meta-analysis. *Diabetes Obes Metab*. 2018;20(6):1508–1514
315. Karam JG, McFarlane SI. Secondary causes of obesity. *Therapy*. 2007;4(5):641–650
316. Bandini L, Danielson M, Esposito LE, et al. Obesity in children with developmental and/or physical disabilities. *Disabil Health J*. 2015;8(3):309–316
317. Institute of Medicine, Committee on Prevention of Obesity in Children and Youth. In: Koplan JP, Liverman CT, Kraak VA, eds. *Preventing Childhood Obesity: Health in the Balance*. Washington, DC: National Academies Press; 2005
318. Cassidy SB, Driscoll DJ. Prader-Willi syndrome. *Eur J Hum Genet*. 2009;17(1):3–13
319. McPheeters ML, Warren Z, Sathe N, et al. A systematic review of medical

- treatments for children with autism spectrum disorders. *Pediatrics*. 2011; 127(5):e1312–e1321
320. Tardieu S, Micallef J, Gentile S, Blin O. Weight gain profiles of new anti-psychotics: public health consequences. *Obes Rev*. 2003;4(3):129–138
321. Cermak SA, Curtin C, Bandini LG. Food selectivity and sensory sensitivity in children with autism spectrum disorders. *J Am Diet Assoc*. 2010;110(2):238–246
322. Puder JJ, Munsch S. Psychological correlates of childhood obesity. *Int J Obes (Lond)*. 2010;34(Suppl 2):S37–S43
323. Eichstaedt CB, Lavay BW. *Physical Activity for Individuals with Mental Retardation: Infancy through Adulthood*. Champaign, IL: Human Kinetics Books; 1992
324. Rimmer JA, Rowland JL. Physical activity for youth with disabilities: a critical need in an underserved population. *Dev Neurorehabil*. 2008;11(2):141–148
325. Kahathuduwa CN, West BD, Blume J, Dharavath N, Moustaid-Moussa N, Mastergeorge A. The risk of overweight and obesity in children with autism spectrum disorders: a systematic review and meta-analysis. *Obes Rev*. 2019;20(12):1667–1679
326. Maillard AM, Ruef A, Pizzagalli F, et al; 16p11.2 European Consortium. The 16p11.2 locus modulates brain structures common to autism, schizophrenia and obesity. *Mol Psychiatry*. 2015;20(1):140–147
327. Shinawi M, Sahoo T, Maranda B, et al. 11p14.1 microdeletions associated with ADHD, autism, developmental delay, and obesity. *Am J Med Genet A*. 2011;155A(6):1272–1280
328. Atladóttir HO, Henriksen TB, Schendel DE, Parner ET. Autism after infection, febrile episodes, and antibiotic use during pregnancy: an exploratory study. *Pediatrics*. 2012;130(6):e1447–e1454
329. Mueller NT, Whyatt R, Hoepner L, et al. Prenatal exposure to antibiotics, cesarean section and risk of childhood obesity. *Int J Obes*. 2015;39(4):665–670
330. Jung Y, Lee AM, McKee SA, Picciotto MR. Maternal smoking and autism spectrum disorder: meta-analysis with population smoking metrics as moderators. *Sci Rep*. 2017;7(1):4315
331. Raz R, Levine H, Pinto O, Broday DM, Yuval Weisskopf MG. Traffic-related air pollution and autism spectrum disorder: a population-based nested case-control study in Israel. *Am J Epidemiol*. 2018;187(4):717–725
332. Wan H, Zhang C, Li H, Luan S, Liu C. Association of maternal diabetes with autism spectrum disorders in offspring: a systemic review and meta-analysis. *Medicine (Baltimore)*. 2018;97(2):e9438
333. Sanchez CE, Barry C, Sabhlok A, et al. Maternal pre-pregnancy obesity and child neurodevelopmental outcomes: a meta-analysis. *Obes Rev*. 2018;19(4):464–484
334. Hack M, Taylor HG, Schluchter M, Andreias L, Drotar D, Klein N. Behavioral outcomes of extremely low birth weight children at age 8 years. *J Dev Behav Pediatr*. 2009;30(2):122–130
335. Lindberg J, Norman M, Westrup B, Öhrman T, Domellöf M, Berglund SK. Overweight, obesity, and body composition in 3.5- and 7-year-old Swedish children born with marginally low birth weight. *J Pediatr*. 2015;167(6):1246–52.e3
336. Dominick KC, Davis NO, Lainhart J, Tager-Flusberg H, Folstein S. Atypical behaviors in children with autism and children with a history of language impairment. *Res Dev Disabil*. 2007;28(2):145–162
337. Sharp WG, Berry RC, McCracken C, et al. Feeding problems and nutrient intake in children with autism spectrum disorders: a meta-analysis and comprehensive review of the literature. *J Autism Dev Disord*. 2013;43(9):2159–2173
338. Fournier KA, Hass CJ, Naik SK, Lodha N, Cauraugh JH. Motor coordination in autism spectrum disorders: a synthesis and meta-analysis. *J Autism Dev Disord*. 2010;40(10):1227–1240
339. Shetreat-Klein M, Shinnar S, Rapin I. Abnormalities of joint mobility and gait in children with autism spectrum disorders. *Brain Dev*. 2014;36(2):91–96
340. Rendeli C, Kuczynska E, Giuliano AC, Chiaretti A, Ausili E. Dietary approach to prevent obesity risk in Spina Bifida patients. *Childs Nerv Syst*. 2020;36(7):1515–1520
341. Littlewood RA, Trocki O, Shepherd RW, Shepherd K, Davies PSW. Resting energy expenditure and body composition in children with myelomeningocele. *Pediatr Rehabil*. 2003;6(1):31–37
342. Caminiti C, Saure C, Weglinski J, de Castro F, Campmany L. Body composition and energy expenditure in a population of children and adolescents with myelomeningocele. *Arch Argent Pediatr*. 2018;116(1):e8–e13
343. Mueske NM, Ryan DD, Van Speybroeck AL, Chan LS, Wren TAL. Fat distribution in children and adolescents with myelomeningocele. *Dev Med Child Neurol*. 2015;57(3):273–278
344. van den Berg-Emons HJG, Bussmann JBJ, Meyerink HJ, Roebroek ME, Stam HJ. Body fat, fitness and level of everyday physical activity in adolescents and young adults with meningomyelocele. *J Rehabil Med*. 2003;35(6):271–275
345. Cortese S, Moreira-Maia CR, St Fleur D, Morcillo-Peñalver C, Rohde LA, Faraone SV. Association between ADHD and obesity: a systematic review and meta-analysis. *Am J Psychiatry*. 2016; 173(1):34–43
346. Cortese S, Ramos Olazagasti MA, Klein RG, Castellanos FX, Proal E, Mannuzza S. Obesity in men with childhood ADHD: a 33-year controlled, prospective, follow-up study. *Pediatrics*. 2013; 131(6):e1731–e1738
347. Khalife N, Kantomaa M, Glover V, et al. Childhood attention-deficit/hyperactivity disorder symptoms are risk factors for obesity and physical inactivity in adolescence. *J Am Acad Child Adolesc Psychiatry*. 2014;53(4):425–436
348. Kooij JJS. ADHD and obesity. *Am J Psychiatry*. 2016;173(1):1–2
349. Ohkuma T, Hirakawa Y, Nakamura U, Kiyohara Y, Kitazono T, Ninomiya T. Association between eating rate and obesity: a systematic review and meta-analysis. *Int J Obes*. 2015;39(11):1589–1596
350. Berkowitz RI, Moore RH, Faith MS, Stallings VA, Kral TV, Stunkard AJ. Identification of an obese eating style in 4-year-old children born at high and

- low risk for obesity. *Obesity (Silver Spring)*. 2010;18(3):505–512
351. Llewellyn CH, van Jaarsveld CH, Boniface D, Carnell S, Wardle J. Eating rate is a heritable phenotype related to weight in children. *Am J Clin Nutr*. 2008;88(6):1560–1566
 352. Wood AC, Blissett JM, Brunstrom JM, et al; American Heart Association Council on Lifestyle and Cardiometabolic Health; Council on Epidemiology and Prevention; Council on Lifelong Congenital Heart Disease and Heart Health in the Young; Council on Cardiovascular and Stroke Nursing; and Stroke Council. Caregiver influences on eating behaviors in young children: a scientific statement from the American Heart Association. *J Am Heart Assoc*. 2020;9(10):e014520
 353. Karam JG, McFarlane SI. Prevention of type 2 diabetes: evidence and strategies. *J Clin Outcomes Manag*. 2017;24(4)
 354. Eneli IU, Wang W, Kelleher K. Identification and counseling for obesity among children on psychotropic medications in ambulatory settings. *Obesity (Silver Spring)*. 2013;21(8):1656–1661
 355. Bak M, Fransen A, Janssen J, van Os J, Drukker M. Almost all antipsychotics result in weight gain: a meta-analysis. *PLoS One*. 2014;9(4):e94112
 356. Tarricone I, Ferrari Gozzi B, Serretti A, Grieco D, Berardi D. Weight gain in antipsychotic-naïve patients: a review and meta-analysis. *Psychol Med*. 2010;40(2):187–200
 357. Pillay J, Boylan K, Carrey N, et al. *First- and Second-Generation Antipsychotics in Children and Young Adults: Systematic Review Update. Comparative Effectiveness Reviews, No. 184*. Rockville, MD: Agency for Healthcare Research and Quality; 2017
 358. Galling B, Roldán A, Nielsen RE, et al. Type 2 diabetes mellitus in youth exposed to antipsychotics: a systematic review and meta-analysis. *JAMA Psychiatry*. 2016;73(3):247–259
 359. Sweeney B, Kelly AS, San Giovanni CB, Kelsey MM, Skelton JA. Clinical approaches to minimize iatrogenic weight gain in children and adolescents. *Clin Obes*. 2021;11(1):e12417
 360. Quek YH, Tam WWS, Zhang MWB, Ho RCM. Exploring the association between childhood and adolescent obesity and depression: a meta-analysis. *Obes Rev*. 2017;18(7):742–754
 361. Esposito M, Gallai B, Roccella M, et al. Anxiety and depression levels in prepubertal obese children: a case-control study. *Neuropsychiatr Dis Treat*. 2014;10:1897–1902
 362. Lindberg L, Hagman E, Danielsson P, Marcus C, Persson M. Anxiety and depression in children and adolescents with obesity: a nationwide study in Sweden. *BMC Med*. 2020;18(1):30
 363. Berk M, Williams LJ, Jacka FN, et al. So depression is an inflammatory disease, but where does the inflammation come from? *BMC Med*. 2013; 11:200
 364. Reeves GM, Postolache TT, Snitker S. Childhood depression: connection between these growing problems in growing children. *Int J Child Health Hum Dev*. 2008;1(2):103–114
 365. Armstrong S, Lazoric S, Hampl S, et al. Physical examination findings among children and adolescents with obesity: an evidence-based review. *Pediatrics*. 2016;137(2): e20151766
 366. Visscher TLS, Lakerveld J, Olsen N, et al. Perceived health status: is obesity perceived as a risk factor and disease? *Obes Facts*. 2017;10(1):52–60
 367. Spurrier NJ, Magarey A, Wong C. Recognition and management of childhood overweight and obesity by clinicians. *J Paediatr Child Health*. 2006;42(7-8):411–418
 368. Robinson E, Sutin AR. Parental perception of weight status and weight gain across childhood. *Pediatrics*. 2016; 137(5):e20153957
 369. He M, Evans A. Are parents aware that their children are overweight or obese? Do they care? *Can Fam Physician*. 2007;53(9):1493–1499
 370. Uy MJA, Pereira MA, Berge JM, Loth KA. How should we approach and discuss children's weight with parents? A qualitative analysis of recommendations from parents of preschool-aged children to physicians. *Clin Pediatr (Phila)*. 2019;58(2):226–237
 371. Neumark-Sztainer D, Story M, Hannan PJ, Perry CL, Irving LM. Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders. *Arch Pediatr Adolesc Med*. 2002;156(2):171–178
 372. Puhl RM, Peterson JL, Luedicke J. Parental perceptions of weight terminology that providers use with youth. *Pediatrics*. 2011;128(4):e786–e793
 373. Knierim SD, Newcomer S, Castillo A, et al. Latino parents' perceptions of pediatric weight counseling terms. *Acad Pediatr*. 2018;18(3):342–353
 374. Valente TW, Fujimoto K, Chou C-P, Spruijt-Metz D. Adolescent affiliations and adiposity: a social network analysis of friendships and obesity. *J Adolesc Health*. 2009;45(2):202–204
 375. Eismann EA, Theuerling J, Maguire S, Hente EA, Shapiro RA. Integration of the Safe Environment for Every Kid (SEEK) model across primary care settings. *Clin Pediatr (Phila)*. 2019;58(2): 166–176
 376. Center for Medicare and Medicaid Innovation. The accountable health communities health-related social needs screening tool. Available at: <https://innovation.cms.gov/files/worksheets/ahcm-screeningtool.pdf>. Accessed October 5, 2022
 377. Goddard A. Adverse childhood experiences and trauma-informed care. *J Pediatr Health Care*. 2021;35(2): 145–155
 378. Forkey H, Szilagyi M, Kelly ET, Duffee J; Council on Foster Care, Adoption, and Kinship Care; Council on Community Pediatrics; Council on Child Abuse and Neglect; Committee on Psychosocial Aspects of Child and Family Health. Trauma-informed care. *Pediatrics*. 2021;148(2):e2021052580
 379. Lobelo F, Muth ND, Hanson S, Nemeth BA; Council on Sports Medicine and Fitness; Section on Obesity. Physical activity assessment and counseling in pediatric clinical settings. *Pediatrics*. 2020;145(3):e20193992
 380. Rankin J, Matthews L, Copley S, et al. Psychological consequences of childhood obesity: psychiatric comorbidity and prevention. *Adolesc Health Med Ther*. 2016;7:125–146

381. Jellinek M, Murphy M. *Pediatric Symptom Checklist*. Boston, MA: Massachusetts General Hospital; 1988
382. Johnson JG, Harris ES, Spitzer RL, Williams JB. The patient health questionnaire for adolescents: validation of an instrument for the assessment of mental disorders among adolescent primary care patients. *J Adolesc Health*. 2002;30(3):196–204
383. Albert Pérez E, Mateu Olivares V, Martínez-Espinosa RM, Molina Vila MD, Reig García-Galbis M. New insights about how to make an intervention in children and adolescents with metabolic syndrome: diet, exercise vs. changes in body composition. A systematic review of RCT. *Nutrients*. 2018;10(7):878
384. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med*. 2006;166(10):1092–1097
385. Wolraich ML, Lambert W, Doffing MA, Bickman L, Simmons T, Worley K. Psychometric properties of the Vanderbilt ADHD diagnostic parent rating scale in a referred population. *J Psychiatr Psychol*. 2003;28(8):559–567
386. Wolraich ML, Hagan JFJ Jr, Allan C, et al; Subcommittee on Children and Adolescents With Attention-Deficit/Hyperactive Disorder. Clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Pediatrics*. 2019;144(4):e20192528
387. Golden NH, Schneider M, Wood C; Committee on Nutrition; Committee on Adolescence; Section on Obesity. Preventing obesity and eating disorders in adolescents. *Pediatrics*. 2016;138(3):e20161649
388. Hornberger LL, Lane MA; Committee on Adolescence. Identification and management of eating disorders in children and adolescents. *Pediatrics*. 2021;147(1):e2020040279
389. Butler MG, Miller JL, Forster JL. Prader-Willi syndrome—clinical genetics, diagnosis and treatment approaches: an update. *Curr Pediatr Rev*. 2019;15(4):207–244
390. Fernandez CJ, Chacko EC, Pappachan JM. Male obesity-related secondary hypogonadism—pathophysiology, clinical implications and management. *Eur Endocrinol*. 2019;15(2):83–90
391. O'Malley GC, Shultz SP, Thivel D, Tsiros MD. Neuromusculoskeletal health in pediatric obesity: incorporating evidence into clinical examination. *Curr Obes Rep*. 2021;10(4):467–477
392. Miller WR, Rollnick S. *Motivational Interviewing: Helping People Change*, 3rd ed. New York, NY: Guilford Press; 2013
393. Baker JL, Olsen LW, Sørensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. *N Engl J Med*. 2007;357(23):2329–2337
394. Kelly AS, Barlow SE, Rao G, et al; American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee of the Council on Cardiovascular Disease in the Young; Council on Nutrition, Physical Activity and Metabolism; and Council on Clinical Cardiology. Severe obesity in children and adolescents: identification, associated health risks, and treatment approaches: a scientific statement from the American Heart Association. *Circulation*. 2013;128(15):1689–1712
395. Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *N Engl J Med*. 2015;373(14):1307–1317
396. Skinner AC. Appraisal of clinical care practices for child obesity prevention and treatment to inform quality improvement. Part II: comorbidities. *Pediatrics*. 2023;151(2):e2022060643
397. Schwimmer JB, Deutsch R, Kahen T, Lavine JE, Stanley C, Behling C. Prevalence of fatty liver in children and adolescents. *Pediatrics*. 2006;118(4):1388–1393
398. Rehm JL, Connor EL, Wolfgram PM, Eickhoff JC, Reeder SB, Allen DB. Predicting hepatic steatosis in a racially and ethnically diverse cohort of adolescent girls. *J Pediatr*. 2014;165(2):319–325.e1
399. Divers J, Mayer-Davis EJ, Lawrence JM, et al. Trends in incidence of type 1 and type 2 diabetes among youths - selected counties and Indian reservations, United States, 2002-2015. *MMWR Morb Mortal Wkly Rep*. 2020;69(6):161–165
400. Copeland KC, Zeitler P, Geffner M, et al; TODAY Study Group. Characteristics of adolescents and youth with recent-onset type 2 diabetes: the TODAY cohort at baseline. *J Clin Endocrinol Metab*. 2011;96(1):159–167
401. McCarthy MI. Genomics, type 2 diabetes, and obesity. *N Engl J Med*. 2010;363(24):2339–2350
402. Commodore-Mensah Y, Matthie N, Wells J, et al. African Americans, African immigrants, and Afro-Caribbeans differ in social determinants of hypertension and diabetes: evidence from the National Health Interview Survey. *J Racial Ethn Health Disparities*. 2018;5(5):995–1002
403. Rughani A, Friedman JE, Tryggvæstad JB. Type 2 diabetes in youth: the role of early life exposures. *Curr Diab Rep*. 2020;20(9):45
404. Duncan DT, Sharifi M, Melly SJ, et al. Characteristics of walkable built environments and BMI z-scores in children: evidence from a large electronic health record database. *Environ Health Perspect*. 2014;122(12):1359–1365
405. Sharifi M, Sequist TD, Rifas-Shiman SL, et al. The role of neighborhood characteristics and the built environment in understanding racial/ethnic disparities in childhood obesity. *Prev Med*. 2016;91:103–109
406. Gaston SA, Atere-Roberts J, Ward J, et al. Experiences with everyday and major forms of racial/ethnic discrimination and type 2 diabetes risk among White, Black, and Hispanic/Latina women: findings from the Sister Study. *Am J Epidemiol*. 2021;190(12):2552–2562
407. Jaffiol C, Thomas F, Spira A, Pannier B, Danchin N. Prediabetes and deprivation: a couple at high risk of diabetes. *Rev Epidemiol Sante Publique*. 2021;69(6):361–365
408. Butler AM. Social determinants of health and racial/ethnic disparities in type 2 diabetes in youth. *Curr Diab Rep*. 2017;17(8):60

409. Kelsey MM, Zeitler PS. Insulin resistance of puberty. *Curr Diab Rep*. 2016;16(7):64
410. Rajjo T, Almasri J, Al Nofal A, et al. The association of weight loss and cardiometabolic outcomes in obese children: systematic review and meta-regression. *J Clin Endocrinol Metab*. 2017;102(3):758–762
411. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2019;73(24):3168–3209
412. National Institutes of Health, National Heart Lung and Blood Institute. *Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents: Summary Report. NIH Publication 12-7486A*. Washington, DC: National Heart Lung and Blood Institute; 2012
413. Juonala M, Magnussen CG, Berenson GS, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med*. 2011;365(20):1876–1885
414. Arslanian S, Bacha F, Grey M, Marcus MD, White NH, Zeitler P. Evaluation and management of youth-onset type 2 diabetes: a position statement by the American Diabetes Association. *Diabetes Care*. 2018;41(12):2648–2668
415. American Diabetes Association. 13. Children and adolescents: standards of medical care in diabetes-2019. *Diabetes Care*. 2019;42(Suppl 1):S148–S164
416. American Diabetes Association. 2.0 Classification and diagnosis of diabetes. *Diabetes Care*. 2017;40(suppl 1):S11–S24
417. Nguyen D, Kit B, Carroll M. Abnormal cholesterol among children and adolescents in the United States, 2011-2014. *NCHS Data Brief*. 2015;(228):1–8
418. Astudillo M, Tosur M, Castillo B, et al. Type 2 diabetes in prepubertal children. *Pediatr Diabetes*. 2021;22(7):946–950
419. Panagiotopoulos C, Hadjiyannakis S, Henderson M; Diabetes Canada Clinical Practice Guidelines Expert Committee. Type 2 diabetes in children and adolescents. *Can J Diabetes*. 2018;42(Suppl 1):S247–S254
420. Peña AS, Curran JA, Fuery M, et al. Screening, assessment and management of type 2 diabetes mellitus in children and adolescents: Australasian Paediatric Endocrine Group guidelines. *Med J Aust*. 2020;213(1):30–43
421. Harlow KE, Africa JA, Wells A, et al; Nonalcoholic Steatohepatitis Clinical Research Network (NASH CRN). Clinically actionable hypercholesterolemia and hypertriglyceridemia in children with nonalcoholic fatty liver disease. *J Pediatr*. 2018;198:76–83.e2
422. Newton KP, Hou J, Crimmins NA, et al; Nonalcoholic Steatohepatitis Clinical Research Network. Prevalence of prediabetes and type 2 diabetes in children with nonalcoholic fatty liver disease. *JAMA Pediatr*. 2016;170(10):e161971
423. Andes LJ, Cheng YJ, Rolka DB, Gregg EW, Imperatore G. Prevalence of prediabetes among adolescents and young adults in the United States, 2005-2016. *JAMA Pediatr*. 2020;174(2):e194498
424. Holterman AX, Guzman G, Fantuzzi G, et al. Nonalcoholic fatty liver disease in severely obese adolescent and adult patients. *Obesity (Silver Spring)*. 2013;21(3):591–597
425. Bacha F, Gungor N, Lee S, Arslanian SA. Progressive deterioration of β -cell function in obese youth with type 2 diabetes. *Pediatr Diabetes*. 2013;14(2):106–111
426. Kahn SE, Lachin JM, Zinman B, et al; ADOPT Study Group. Effects of rosiglitazone, glyburide, and metformin on β -cell function and insulin sensitivity in ADOPT. *Diabetes*. 2011;60(5):1552–1560
427. Sam S, Edelstein SL, Arslanian SA, et al; RISE Consortium; RISE Consortium Investigators. Baseline predictors of glycemic worsening in youth and adults with impaired glucose tolerance or recently diagnosed type 2 diabetes in the Restoring Insulin Secretion (RISE) Study. *Diabetes Care*. 2021;44(9):1938–1947
428. Welsh JA, Karpen S, Vos MB. Increasing prevalence of nonalcoholic fatty liver disease among United States adolescents, 1988-1994 to 2007-2010. *J Pediatr*. 2013;162(3):496–500.e1
429. Anderson EL, Howe LD, Jones HE, Higgins JP, Lawlor DA, Fraser A. The prevalence of non-alcoholic fatty liver disease in children and adolescents: a systematic review and meta-analysis. *PLoS One*. 2015;10(10):e0140908
430. Drobac S, Brickman W, Smith T, Binns HJ. Evaluation of a type 2 diabetes screening protocol in an urban pediatric clinic. *Pediatrics*. 2004;114(1):141–148
431. Cook S, Weitzman M, Auinger P, Barlow SE. Screening and counseling associated with obesity diagnosis in a national survey of ambulatory pediatric visits. *Pediatrics*. 2005;116(1):112–116
432. Eneli IU, Keast DR, Rappley MD, Camargo CA Jr. Adequacy of two ambulatory care surveillance systems for tracking childhood obesity practice patterns. *Public Health*. 2008;122(7):700–707
433. Xanthakos SA, Lavine JE, Yates KP, et al; NASH Clinical Research Network. Progression of fatty liver disease in children receiving standard of care lifestyle advice. *Gastroenterology*. 2020;159(5):1731–1751.e10
434. Vajravelu ME, Lee JM, Shah R, Shults J, Amaral S, Kelly A. Association between prediabetes diagnosis and body mass index trajectory of overweight and obese adolescents. *Pediatr Diabetes*. 2020;21(5):743–746
435. Bolling GF, Armstrong SC, Reichard KW, Michalsky MP; Section on Obesity; Section on Surgery. Metabolic and bariatric surgery for pediatric patients with severe obesity. *Pediatrics*. 2019;144(6):e20193224
436. Derderian SC, Patten L, Kaizer AM, et al. Influence of weight loss on obesity-associated complications after metabolic and bariatric surgery in adolescents. *Obesity (Silver Spring)*. 2020;28(12):2397–2404
437. de Ferranti SD, Steinberger J, Ameduri R, et al. Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American

- Heart Association. *Circulation*. 2019;139(13):e603–e634
438. Ryder JR, Xu P, Inge TH, et al. Thirty-year risk of cardiovascular disease events in adolescents with severe obesity. *Obesity (Silver Spring)*. 2020;28(3):616–623
439. Savoye M, Shaw M, Dziura J, et al. Effects of a weight management program on body composition and metabolic parameters in overweight children: a randomized controlled trial. *JAMA*. 2007;297(24):2697–2704
440. Akinbami LJRL, Rossen LM, Fakhouri THI, Simon AE, Kit BK. Contribution of weight status to asthma prevalence racial disparities, 2-19 year olds, 1988-2014. *Ann Epidemiol*. 2017;27(8):472–478.e3
441. Andersen IG, Holm J-C, Hornøe P. Obstructive sleep apnea in children and adolescents with and without obesity. *Eur Arch Otorhinolaryngol*. 2019;276(3):871–878
442. Hadjiyannakis S, Ibrahim Q, Li J, et al. Obesity class versus the Edmonton Obesity Staging System for Pediatrics to define health risk in childhood obesity: results from the CANPWR cross-sectional study. *Lancet Child Adolesc Health*. 2019;3(6):398–407
443. Kim SJ, Lee J, Nam CM, Lee SY. Impact of obesity on metabolic syndrome among adolescents as compared with adults in Korea. *Yonsei Med J*. 2011;52(5):746–752
444. Reinehr T, Kleber M, Toschke AM. Lifestyle intervention in obese children is associated with a decrease of the metabolic syndrome prevalence. *Atherosclerosis*. 2009;207(1):174–180
445. Ciemins EL, Joshi V, Cuddeback JK, Kushner RF, Horn DB, Garvey WT. Diagnosing obesity as a first step to weight loss: an observational study. *Obesity (Silver Spring)*. 2020;28(12):2305–2309
446. Banerjee ES, Gambler A, Fogleman C. Adding obesity to the problem list increases the rate of providers addressing obesity. *Fam Med*. 2013;45(9):629–633
447. Gopalan A, Lorincz IS, Wirtalla C, Marcus SC, Long JA. Awareness of pre-diabetes and engagement in diabetes risk-reducing behaviors. *Am J Prev Med*. 2015;49(4):512–519
448. Murillo R, Katic BJ, Gonzalez T, Vasquez E, Echeverria S. The association of pre-diabetes and diabetes risk perception with leisure-time physical activity and weight loss. *Am J Health Promot*. 2019;33(4):534–540
449. Owei I, Umekwe N, Ceesay F, Dagogo-Jack S. Awareness of prediabetes status and subsequent health behavior, body weight, and blood glucose levels. *J Am Board Fam Med*. 2019;32(1):20–27
450. Doshi N, Perrin EM, Lazorick S, Esserman D, Steiner MJ. Short-term change in body mass index in overweight adolescents following cholesterol screening. *Arch Pediatr Adolesc Med*. 2009;163(9):812–817
451. Silva DFO, Sena-Evangelista KCM, Lyra CO, Pedrosa LFC, Arrais RF, Lima SCVC. Motivations for weight loss in adolescents with overweight and obesity: a systematic review. *BMC Pediatr*. 2018;18(1):364
452. Ayer J, Charakida M, Deanfield JE, Celermajer DS. Lifetime risk: childhood obesity and cardiovascular risk. *Eur Heart J*. 2015;36(22):1371–1376
453. Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. Cardiovascular disease risk in healthy children and its association with body mass index: systematic review and meta-analysis. *BMJ*. 2012;345:e4759
454. Li L, Pérez A, Wu LT, Ranjit N, Brown HS, Kelder SH. Cardiometabolic risk factors among severely obese children and adolescents in the United States, 1999-2012. *Child Obes*. 2016;12(1):12–19
455. Kretsoulas C, Flegler EW, Kubzansky LD, McGorrian CM, Subramanian SV. Young adults and adverse childhood events: a potent measure of cardiovascular risk. *Am J Med*. 2019;132(5):605–613
456. Berglund L, Brunzell JD, Goldberg AC, et al; Endocrine society. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2012;97(9):2969–2989
457. McCrindle BW, Tyrrell PN, Kavey R-EW. Will obesity increase the proportion of children and adolescents recommended for a statin? *Circulation*. 2013;128(19):2162–2165
458. Mayer-Davis EJ, Lawrence JM, Dabelea D, et al; SEARCH for Diabetes in Youth Study. Incidence trends of type 1 and type 2 diabetes among youths, 2002-2012. *N Engl J Med*. 2017;376(15):1419–1429
459. Klingensmith GJ, Connor CG, Ruedy KJ, et al; Pediatric Diabetes Consortium. Presentation of youth with type 2 diabetes in the Pediatric Diabetes Consortium. *Pediatr Diabetes*. 2016;17(4):266–273
460. Hutchins J, Barajas RA, Hale D, Escaname E, Lynch J. Type 2 diabetes in a 5-year-old and single center experience of type 2 diabetes in youth under 10. *Pediatr Diabetes*. 2017;18(7):674–677
461. Love-Osborne KA, Sheeder JL, Nadeau KJ, Zeitler P. Longitudinal follow up of dysglycemia in overweight and obese pediatric patients. *Pediatr Diabetes*. 2018;19(2):199–204
462. Hubbard RA, Xu J, Siegel R, Chen Y, Eneli I. Studying pediatric health outcomes with electronic health records using Bayesian clustering and trajectory analysis. *J Biomed Inform*. 2021;113:103654
463. Piccolo RS, Pearce N, Araujo AB, McKinlay JB. The contribution of biogeographical ancestry and socioeconomic status to racial/ethnic disparities in type 2 diabetes mellitus: results from the Boston Area Community Health Survey. *Ann Epidemiol*. 2014;24(9):648–654, 654.e1
464. Magge SN, Silverstein J, Elder D, Nadeau K, Hannon TS. Evaluation and treatment of prediabetes in youth. *J Pediatr*. 2020;219:11–22
465. Zeitler P, Arslanian S, Fu J, et al. ISPAD Clinical Practice Consensus Guidelines 2018: type 2 diabetes mellitus in youth. *Pediatr Diabetes*. 2018;19(Suppl 27):28–46
466. Libman IM, Barinas-Mitchell E, Bartucci A, Robertson R, Arslanian S. Reproducibility of the oral glucose tolerance test in overweight children. *J Clin Endocrinol Metab*. 2008;93(11):4231–4237
467. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and high risk

- for diabetes using A1C criteria in the U.S. population in 1988-2006. *Diabetes Care*. 2010;33(3):562–568
468. Peters A, Laffel L; American Diabetes Association Transitions Working Group. Diabetes care for emerging adults: recommendations for transition from pediatric to adult diabetes care systems: a position statement of the American Diabetes Association. *Diabetes Care*. 2011;34(11):2477–2485
469. Wu EL, Kazzi NG, Lee JM. Cost-effectiveness of screening strategies for identifying pediatric diabetes mellitus and dysglycemia. *JAMA Pediatr*. 2013; 167(1):32–39
470. Peter A, Fritsche A, Stefan N, Heni M, Häring HU, Schleicher E. Diagnostic value of hemoglobin A1c for type 2 diabetes mellitus in a population at risk. *Exp Clin Endocrinol Diabetes*. 2011;19(4):234–237
471. Love-Osborne KA, Sheeder J, Svircev A, Chan C, Zeitler P, Nadeau KJ. Use of glycosylated hemoglobin increases diabetes screening for at-risk adolescents in primary care settings. *Pediatr Diabetes*. 2013;14(7):512–518
472. Vijayakumar P, Nelson RG, Hanson RL, Knowler WC, Sinha M. HbA1c and the prediction of type 2 diabetes in children and adults. *Diabetes Care*. 2017;40(1):16–21
473. Wallace AS, Wang D, Shin JI, Selvin E. Screening and diagnosis of prediabetes and diabetes in US children and adolescents. *Pediatrics*. 2020;146(3): e20200265
474. Lee JM, Wu EL, Tarini B, Herman WH, Yoon E. Diagnosis of diabetes using hemoglobin A1c: should recommendations in adults be extrapolated to adolescents? *J Pediatr*. 2011;158(6): 947–952.e1-3
475. Herman WH, Ma Y, Uwaifo G, et al; Diabetes Prevention Program Research Group. Differences in A1C by race and ethnicity among patients with impaired glucose tolerance in the Diabetes Prevention Program. *Diabetes Care*. 2007;30(10):2453–2457
476. Rao LV, Pratt GW, Bi C, Kroll MH. Large-scale retrospective analyses of the effect of iron deficiency anemia on hemoglobin A1c concentrations. *Clin Chim Acta*. 2022;529:21–24
477. Levy-Marchal C, Arslanian S, Cutfield W, et al; ESPE-LWPES-ISPAD-APPES-APEG-SLEP-JSPE; Insulin Resistance in Children Consensus Conference Group. Insulin resistance in children: consensus, perspective, and future directions. *J Clin Endocrinol Metab*. 2010;95(12): 5189–5198
478. International Expert Committee. International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes. *Diabetes Care*. 2009;32(7):1327–1334
479. Feldstein AE, Charatcharoenwithaya P, Treeprasertsuk S, Benson JT, Enders FB, Angulo P. The natural history of non-alcoholic fatty liver disease in children: a follow-up study for up to 20 years. *Gut*. 2009;58(11):1538–1544
480. Lazo M, Bilal U, Perez-Escamilla R. Epidemiology of NAFLD and type 2 diabetes: health disparities among persons of Hispanic origin. *Curr Diab Rep*. 2015;15(12):116
481. Schwimmer JB, Newton KP, Awai HI, et al. Paediatric gastroenterology evaluation of overweight and obese children referred from primary care for suspected non-alcoholic fatty liver disease. *Aliment Pharmacol Ther*. 2013;38(10):1267–1277
482. Macumber IR, Weiss NS, Halbach SM, Hanevold CD, Flynn JT. The association of pediatric obesity with nocturnal non-dipping on 24-hour ambulatory blood pressure monitoring. *Am J Hypertens*. 2016;29(5):647–652
483. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008; 117(25):3171–3180
484. Hao G, Wang X, Treiber FA, Harshfield G, Kapuku G, Su S. Blood pressure trajectories from childhood to young adulthood associated with cardiovascular risk: results from the 23-year Longitudinal Georgia Stress and Heart Study. *Hypertension*. 2017;69(3): 435–442
485. Theodore RF, Broadbent J, Nagin D, et al. Childhood to early-midlife systolic blood pressure trajectories: early-life predictors, effect modifiers, and adult cardiovascular outcomes. *Hypertension*. 2015;66(6):1108–1115
486. Köchli S, Endes K, Steiner R, et al. Obesity, high blood pressure, and physical activity determine vascular phenotype in young children. *Hypertension*. 2019; 73(1):153–161
487. Zhang T, Li S, Bazzano L, He J, Whelton P, Chen W. Trajectories of childhood blood pressure and adult left ventricular hypertrophy: the Bogalusa Heart Study. *Hypertension*. 2018;72(1):93–101
488. Hagman E, Danielsson P, Elimam A, Marcus C. The effect of weight loss and weight gain on blood pressure in children and adolescents with obesity. *Int J Obes*. 2019;43(10):1988–1994
489. Falkner B, Gidding SS, Portman R, Rosner B. Blood pressure variability and classification of prehypertension and hypertension in adolescence. *Pediatrics*. 2008;122(2):238–242
490. Jackson SL, Zhang Z, Wiltz JL, et al. Hypertension among youths—United States, 2001-2016. *MMWR Morb Mortal Wkly Rep*. 2018;67(27):758–762
491. Ruiz LD, Zuelch ML, Dimitratos SM, Scherr RE. Adolescent obesity: diet quality, psychosocial health, and cardiometabolic risk factors. *Nutrients*. 2019;12(1):43
492. Au CT, Ho CK, Wing YK, Lam HS, Li AM. Acute and chronic effects of sleep duration on blood pressure. *Pediatrics*. 2014;133(1):e64–e72
493. Jiang W, Hu C, Li F, Hua X, Zhang X. Association between sleep duration and high blood pressure in adolescents: a systematic review and meta-analysis. *Ann Hum Biol*. 2018;45(6-8):457–462
494. American Academy of Pediatrics. *Section on Obesity. 5210 Pediatric Obesity Clinical Decision Support Chart*, 3rd ed. Itasca, IL: American Academy of Pediatrics; 2019
495. Hansen ML, Gunn PW, Kaelber DC. Underdiagnosis of hypertension in children and adolescents. *JAMA*. 2007; 298(8):874–879
496. American Academy of Sleep Medicine. *International Classification of Sleep Disorders*, 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014
497. El Hayek S, Bitar L, Hamdar LH, Mirza FG, Daoud G. Poly cystic ovarian syndrome: an updated overview. *Front Physiol*. 2016;7:124

498. Bremer AA. Polycystic ovary syndrome in the pediatric population. *Metab Syndr Relat Disord*. 2010;8(5):375–394
499. Ibáñez L, Oberfield SE, Witchel S, et al. An international consortium update: pathophysiology, diagnosis, and treatment of polycystic ovarian syndrome in adolescence. *Horm Res Paediatr*. 2017;88(6):371–395
500. Witchel SF, Oberfield S, Rosenfield RL, et al. The diagnosis of polycystic ovary syndrome during adolescence. *Horm Res Paediatr*. 2015;83(6):376–389. DOI: 10.1159/000375530
501. Naz MSG, Tehrani FR, Majd HA, et al. The prevalence of polycystic ovary syndrome in adolescents: a systematic review and meta-analysis. *Int J Reprod Biomed (Yazd)*. 2019;17(8):533–542
502. Legro RS, Arslanian SA, Ehrmann DA, et al; Endocrine Society. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2013;98(12):4565–4592
503. Estrada E, Eneli I, Hampl S, et al; Children's Hospital Association. Children's Hospital Association consensus statements for comorbidities of childhood obesity. *Child Obes*. 2014;10(4):304–317
504. Sutaria S, Devakumar D, Yasuda SS, Das S, Saxena S. Is obesity associated with depression in children? Systematic review and meta-analysis. *Arch Dis Child*. 2019;104(1):64–74
505. Jebeile H, Gow ML, Baur LA, Garnett SP, Paxton SJ, Lister NB. Association of pediatric obesity treatment, including a dietary component, with change in depression and anxiety: a systematic review and meta-analysis. *JAMA Pediatr*. 2019;173(11):e192841–e192841
506. Zuckerbrot RA, Cheung A, Jensen PS, Stein REK, Laraque D; GLAD-PC Steering Group. Guidelines for Adolescent Depression in Primary Care (GLAD-PC): part I. Practice preparation, identification, assessment, and initial management. *Pediatrics*. 2018;141(3):e20174081
507. Lehmann CL, Arons RR, Loder RT, Vitale MG. The epidemiology of slipped capital femoral epiphysis: an update. *J Pediatr Orthop*. 2006;26(3):286–290
508. Novais EN, Millis MB. Slipped capital femoral epiphysis: prevalence, pathogenesis, and natural history. *Clin Orthop Relat Res*. 2012;470(12):3432–3438
509. Castillo C, Mendez M. Slipped capital femoral epiphysis: a review for pediatricians. *Pediatr Ann*. 2018;47(9):e377–e380
510. Janoyer M. Blount disease. *Orthop Traumatol Surg Res*. 2019;105(1S):S111–S121
511. Sabharwal S. Blount disease: an update. *Orthop Clin North Am*. 2015;46(1):37–47
512. Banwarie RR, Hollman F, Meijs N, et al. Insight into the possible aetiologies of Blount's disease: a systematic review of the literature. *J Pediatr Orthop B*. 2020;29(4):323–336
513. Jansen N, Hollman F, Bovendeert F, Moh P, Stegmann A, Staal HM. Blount disease and familial inheritance in Ghana, area cross-sectional study. *BMJ Paediatr Open*. 2021;5(1):e001052
514. Markey KA, Mollan SP, Jensen RH, Sinclair AJ. Understanding idiopathic intracranial hypertension: mechanisms, management, and future directions. *Lancet Neurol*. 2016;15(1):78–91
515. Wall M. Update on idiopathic intracranial hypertension. *Neurol Clin*. 2017;35(1):45–57
516. Ko MW, Chang SC, Ridha MA, et al. Weight gain and recurrence in idiopathic intracranial hypertension: a case-control study. *Neurology*. 2011;76(18):1564–1567
517. Kilgore KP, Lee MS, Leavitt JA, et al. Re-evaluating the incidence of idiopathic intracranial hypertension in an era of increasing obesity. *Ophthalmology*. 2017;124(5):697–700
518. Daniels AB, Liu GT, Volpe NJ, et al. Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension (pseudotumor cerebri). *Am J Ophthalmol*. 2007;143(4):635–641
519. Glueck CJ, Golnik KC, Aregawi D, Goldenberg N, Sieve L, Wang P. Changes in weight, papilledema, headache, visual field, and life status in response to diet and metformin in women with idiopathic intracranial hypertension with and without concurrent polycystic ovary syndrome or hyperinsulinemia. *Transl Res*. 2006;148(5):215–222
520. Kilic K, Korsbæk JJ, Jensen RH, Cveticovic VV. Diagnosis of idiopathic intracranial hypertension - the importance of excluding secondary causes: a systematic review. *Cephalalgia*. 2022;42(6):524–541
521. Dietz WH, Belay B, Bradley D, et al. *A Model Framework That Integrates Community and Clinical Systems for the Prevention and Management of Obesity and Other Chronic Diseases*. Washington, DC: National Academy of Medicine; 2017
522. Medical Home Initiatives for Children With Special Needs Project Advisory Committee; American Academy of Pediatrics. The medical home. *Pediatrics*. 2002;110(1 Pt 1):184–186
523. Cardel MI, Newsome FA, Pearl RL, et al. Patient-centered care for obesity: how health care providers can treat obesity while actively addressing weight stigma and eating disorder risk. *J Acad Nutr Diet*. 2022;122(6):1089–1098
524. Resnicow K, Davis R, Rollnick S. Motivational interviewing for pediatric obesity: conceptual issues and evidence review. *J Am Diet Assoc*. 2006;106(12):2024–2033
525. Resnicow K, McMaster F, Bocian A, et al. Motivational interviewing and dietary counseling for obesity in primary care: an RCT. *Pediatrics*. 2015;135(4):649–657
526. Taylor RW, Cox A, Knight L, et al. A tailored family-based obesity intervention: a randomized trial. *Pediatrics*. 2015;136(2):281–289
527. Broccoli S, Davoli AM, Bonvicini L, et al. Motivational interviewing to treat overweight children: 24-month follow-up of a randomized controlled trial. *Pediatrics*. 2016;137(1)
528. Gourlan M, Sarrazin P, Trouilloud D. Motivational interviewing as a way to promote physical activity in obese adolescents: a randomised-controlled trial using self-determination theory as an explanatory framework. *Psychol Health*. 2013;28(11):1265–1286

529. Taveras EM, Marshall R, Kleinman KP, et al. Comparative effectiveness of childhood obesity interventions in pediatric primary care: a cluster-randomized clinical trial. *JAMA Pediatr*. 2015;169(6):535–542
530. Chen JL, Guedes CM, Cooper BA, Lung AE. Short-term efficacy of an innovative mobile phone technology-based intervention for weight management for overweight and obese adolescents: pilot study. *Interact J Med Res*. 2017; 6(2):e12
531. Love-Osborne K, Fortune R, Sheeder J, Federico S, Haemer MA. School-based health center-based treatment for obese adolescents: feasibility and body mass index effects. *Child Obes*. 2014;10(5):424–431
532. McCallum Z, Wake M, Gerner B, et al. Outcome data from the LEAP (Live, Eat and Play) trial: a randomized controlled trial of a primary care intervention for childhood overweight/mild obesity. *Int J Obes*. 2007;31(4):630–636
533. Sherwood NE, JaKa MM, Crain AL, Martinson BC, Hayes MG, Anderson JD. Pediatric primary care-based obesity prevention for parents of preschool children: a pilot study. *Child Obes*. 2015;11(6):674–682
534. Taveras EM, Gortmaker SL, Hohman KH, et al. Randomized controlled trial to improve primary care to prevent and manage childhood obesity: the High Five for Kids study. *Arch Pediatr Adolesc Med*. 2011;165(8):714–722
535. Rifas-Shiman SL, Taveras EM, Gortmaker SL, et al. Two-year follow-up of a primary care-based intervention to prevent and manage childhood obesity: the High Five for Kids study. *Pediatr Obes*. 2017;12(3):e24–e27
536. Fonseca H, Prioste A, Sousa P, Gaspar P, Machado MC. Effectiveness analysis of an internet-based intervention for overweight adolescents: next steps for researchers and clinicians. *BMC Obes*. 2016;3:15
537. Davoli AM, Broccoli S, Bonvicini L, et al. Pediatrician-led motivational interviewing to treat overweight children: an RCT. *Pediatrics*. 2013; 132(5):e1236–e1246
538. van Grieken A, Veldhuis L, Renders CM, et al. Population-based childhood overweight prevention: outcomes of the 'be active, eat right' study. *PLoS One*. 2013;8(5):e65376
539. Small L, Bonds-McClain D, Melnyk B, Vaughan L, Gannon AM. The preliminary effects of a primary care-based randomized treatment trial with overweight and obese young children and their parents. *J Pediatr Health Care*. 2014;28(3):198–207
540. Stovitz SD, Berge JM, Wetzsteon RJ, Sherwood NE, Hannan PJ, Himes JH. Stage 1 treatment of pediatric overweight and obesity: a pilot and feasibility randomized controlled trial. *Child Obes*. 2014;10(1):50–57
541. Looney SM, Raynor HA. Examining the effect of three low-intensity pediatric obesity interventions: a pilot randomized controlled trial. *Clin Pediatr (Phila)*. 2014;53(14):1367–1374
542. Macdonell K, Brogan K, Naar-King S, Ellis D, Marshall S. A pilot study of motivational interviewing targeting weight-related behaviors in overweight or obese African American adolescents. *J Adolesc Health*. 2012;50(2):201–203
543. Walpole B, Dettmer E, Morrongiello BA, McCrindle BW, Hamilton J. Motivational interviewing to enhance self-efficacy and promote weight loss in overweight and obese adolescents: a randomized controlled trial. *J Pediatr Psychol*. 2013;38(9):944–953
544. Crespo NC, Talavera GA, Campbell NR, et al. A randomized controlled trial to prevent obesity among Latino paediatric patients. *Pediatr Obes*. 2018;13(11): 697–704
545. Moschonis G, Michalopoulou M, Tsoutsouloupoulou K, et al. Assessment of the effectiveness of a computerised decision-support tool for health professionals for the prevention and treatment of childhood obesity. Results from a randomised controlled trial. *Nutrients*. 2019;11(3):706
546. Davis AM, James RL, Boles RE, Goetz JR, Belmont J, Malone B. The use of TeleMedicine in the treatment of paediatric obesity: feasibility and acceptability. *Matern Child Nutr*. 2011;7(1): 71–79
547. Novotny R, Nigg CR, Li F, Wilkens LR. Pacific kids DASH for health (PacDASH) randomized, controlled trial with DASH eating plan plus physical activity improves fruit and vegetable intake and diastolic blood pressure in children. *Child Obes*. 2015;11(2):177–186
548. Parra-Medina D, Mojica C, Liang Y, Ouyang Y, Ramos AI, Gomez I. Promoting weight maintenance among overweight and obese Hispanic children in a rural practice. *Child Obes*. 2015; 11(4):355–363
549. Wake M, Baur LA, Gerner B, et al. Outcomes and costs of primary care surveillance and intervention for overweight or obese children: the LEAP 2 randomised controlled trial. *BMJ*. 2009;339:b3308
550. Armstrong S, Mendelsohn A, Bennett G, Taveras EM, Kimberg A, Kemper AR. Texting motivational interviewing: a randomized controlled trial of motivational interviewing text messages designed to augment childhood obesity treatment. *Child Obes*. 2018;14(1):4–10
551. Bean MK, Ingersoll KS, Powell P, et al. Impact of motivational interviewing on outcomes of an adolescent obesity treatment: results from the MI Values randomized controlled pilot trial. *Clin Obes*. 2018;8(5):323–326
552. Akgöl Gundogdu N, Sevig EU, Guler N. The effect of the solution-focused approach on nutrition-exercise attitudes and behaviours of overweight and obese adolescents: randomised controlled trial. *J Clin Nurs*. 2018;27(7-8):e1660–e1672
553. Berkowitz RI, Wadden TA, Gehrman CA, et al. Meal replacements in the treatment of adolescent obesity: a randomized controlled trial. *Obesity (Silver Spring)*. 2011;19(6):1193–1199
554. Crabtree VM, Moore JB, Jacks DE, Cerrito P, Topp RV. A transtheoretical, case management approach to the treatment of pediatric obesity. *J Prim Care Community Health*. 2010;1(1):4–7
555. DeBar LL, Stevens VJ, Perrin N, et al. A primary care-based, multicomponent lifestyle intervention for overweight adolescent females. *Pediatrics*. 2012;129(3):e611–e620
556. Ek A, Lewis Chamberlain K, Sorjonen K, et al. A parent treatment program for preschoolers with obesity: a

- randomized controlled trial. *Pediatrics*. 2019;144(2):e20183457
557. Fleischman A, Hourigan SE, Lyon HN, et al. Creating an integrated care model for childhood obesity: a randomized pilot study utilizing telehealth in a community primary care setting. *Clin Obes*. 2016;6(6):380–388
558. Garipağaoğlu M, Sahip Y, Darendeliler F, Akdikmen O, Kopuz S, Sut N. Family-based group treatment versus individual treatment in the management of childhood obesity: randomized, prospective clinical trial. *Eur J Pediatr*. 2009;168(9):1091–1099
559. Hofsteenge GH, Chinapaw MJ, Delemarre-van de Waal HA, Weijs PJ. Long-term effect of the Go4it group treatment for obese adolescents: a randomized controlled trial. *Clin Nutr*. 2014;33(3):385–391
560. Kalavainen MP, Korppi MO, Nuutinen OM. Clinical efficacy of group-based treatment for childhood obesity compared with routinely given individual counseling. *Int J Obes*. 2007;31(10):1500–1508
561. Njardvik U, Gunnarsdottir T, Olafsdottir AS, Craighead LW, Boles RE, Bjarnason R. Incorporating appetite awareness training within family-based behavioral treatment of pediatric obesity: a randomized controlled pilot study. *J Pediatr Psychol*. 2018;43(9):1017–1027
562. Norman G, Huang J, Davila EP, et al. Outcomes of a 1-year randomized controlled trial to evaluate a behavioral 'stepped-down' weight loss intervention for adolescent patients with obesity. *Pediatr Obes*. 2016;11(1):18–25
563. Nova A, Russo A, Sala E. Long-term management of obesity in paediatric office practice: experimental evaluation of two different types of intervention. *Ambul Child Health*. 2001;7(3-4):239–247
564. Parillo M, Licenziati MR, Vacca M, De Marco D, Iannuzzi A. Metabolic changes after a hypocaloric, low-glycemic-index diet in obese children. *J Endocrinol Invest*. 2012;35(7):629–633
565. Quattrin T, Roemmich JN, Paluch R, Yu J, Epstein LH, Ecker MA. Treatment outcomes of overweight children and parents in the medical home. *Pediatrics*. 2014;134(2):290–297
566. Quattrin T, Cao Y, Paluch RA, Roemmich JN, Ecker MA, Epstein LH. Cost-effectiveness of family-based obesity treatment. *Pediatrics*. 2017;140(3):e20162755
567. Saelens BE, Sallis JF, Wilfley DE, Patrick K, Cella JA, Buchta R. Behavioral weight control for overweight adolescents initiated in primary care. *Obes Res*. 2002;10(1):22–32
568. Shelton D, Le Gros K, Norton L, Stanton-Cook S, Morgan J, Masterman P. Randomised controlled trial: a parent-based group education programme for overweight children. *J Paediatr Child Health*. 2007;43(12):799–805
569. Stark LJ, Clifford LM, Towner EK, et al. A pilot randomized controlled trial of a behavioral family-based intervention with and without home visits to decrease obesity in preschoolers. *J Pediatr Psychol*. 2014;39(9):1001–1012
570. Stark LJ, Spear Filigno S, Bolling C, et al. Clinic and home-based behavioral intervention for obesity in preschoolers: a randomized trial. *J Pediatr*. 2018;192:115–121.e1
571. Stettler N, Wrotniak BH, Hill DL, et al. Prevention of excess weight gain in paediatric primary care: beverages only or multiple lifestyle factors. The Smart Step Study, a cluster-randomized clinical trial. *Pediatr Obes*. 2015;10(4):267–274
572. Truby H, Baxter K, Ware RS, et al. A randomized controlled trial of two different macronutrient profiles on weight, body composition and metabolic parameters in obese adolescents seeking weight loss. *PLoS One*. 2016;11(3):e0151787
573. Verbeken S, Braet C, Goossens L, van der Oord S. Executive function training with game elements for obese children: a novel treatment to enhance self-regulatory abilities for weight-control. *Behav Res Ther*. 2013;51(6):290–299
574. Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med*. 2003;157(8):773–779
575. Arauz Boudreau AD, Kurowski DS, Gonzalez WI, Dimond MA, Oreskovic NM. Latino families, primary care, and childhood obesity: a randomized controlled trial. *Am J Prev Med*. 2013;44(3 Suppl 3):S247–S257
576. Flodmark CE, Ohlsson T, Rydén O, Sveger T. Prevention of progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics*. 1993;91(5):880–884
577. Martínez-Andrade GO, Cespedes EM, Rifas-Shiman SL, et al. Feasibility and impact of Creciendo Sanos, a clinic-based pilot intervention to prevent obesity among preschool children in Mexico City. *BMC Pediatr*. 2014;14:77
578. Boutelle KN, Norman GJ, Rock CL, Rhee KE, Crow SJ. Guided self-help for the treatment of pediatric obesity. *Pediatrics*. 2013;131(5):e1435–e1442
579. Croker H, Viner RM, Nicholls D, et al. Family-based behavioural treatment of childhood obesity in a UK National Health Service setting: randomized controlled trial. *Int J Obes*. 2012;36(1):16–26
580. Deforche B, De Bourdeaudhuij I, Tanghe A, Deboode P, Hills AP, Bouckaert J. Post-treatment phone contact: a weight maintenance strategy in obese youngsters. *Int J Obes*. 2005;29(5):543–546
581. Kalavainen M, Korppi M, Nuutinen O. Long-term efficacy of group-based treatment for childhood obesity compared with routinely given individual counselling. *Int J Obes*. 2011;35(4):530–533
582. Wright JA, Phillips BD, Watson BL, Newby PK, Norman GJ, Adams WG. Randomized trial of a family-based, automated, conversational obesity treatment program for underserved populations. *Obesity (Silver Spring)*. 2013;21(9):E369–E378
583. Yackobovitch-Gavan M, Wolf Linhard D, Nagelberg N, et al. Intervention for childhood obesity based on parents only or parents and child compared with follow-up alone. *Pediatr Obes*. 2018;13(11):647–655
584. Bohlin A, Hagman E, Klaesson S, Danielsson P. Childhood obesity treatment: telephone coaching is as good as usual care in maintaining weight loss - a randomized controlled trial. *Clin Obes*. 2017;7(4):199–205

585. Ford AL, Bergh C, Södersten P, et al. Treatment of childhood obesity by re-training eating behaviour: randomised controlled trial. *BMJ*. 2009;340:b5388
586. Hills AP, Parker AW. Obesity management via diet and exercise intervention. *Child Care Health Dev*. 1988;14(6):409–416
587. Hughes AR, Stewart L, Chapple J, et al. Randomized, controlled trial of a best-practice individualized behavioral program for treatment of childhood overweight: Scottish Childhood Overweight Treatment Trial (SCOTT). *Pediatrics*. 2008;121(3):e539–e546
588. Pedrosa C, Oliveira BM, Albuquerque I, Simões-Pereira C, Vaz-de-Almeida MD, Correia F. Markers of metabolic syndrome in obese children before and after 1-year lifestyle intervention program. *Eur J Nutr*. 2011;50(6):391–400
589. Banks J, Sharp DJ, Hunt LP, Shield JP. Evaluating the transferability of a hospital-based childhood obesity clinic to primary care: a randomised controlled trial. *Br J Gen Pract*. 2012;62(594):e6–e12
590. Bathrellou E, Yannakoulia M, Papanikolaou K, et al. Parental involvement does not augment the effectiveness of an intense behavioral program for the treatment of childhood obesity. *Hormones (Athens)*. 2010;9(2):171–175
591. Berkowitz RI, Rukstalis MR, Bishop-Gilyard CT, et al. Treatment of adolescent obesity comparing self-guided and group lifestyle modification programs: a potential model for primary care. *J Pediatr Psychol*. 2013;38(9):978–986
592. Casazza K, Cardel M, Dulin-Keita A, et al. Reduced carbohydrate diet to improve metabolic outcomes and decrease adiposity in obese peripubertal African American girls. *J Pediatr Gastroenterol Nutr*. 2012;54(3):336–342
593. de Ferranti SD, Milliren CE, Denhoff ER, et al. Providing food to treat adolescents at risk for cardiovascular disease. *Obesity (Silver Spring)*. 2015;23(10):2109–2117
594. de Niet J, Timman R, Bauer S, et al. The effect of a short message service maintenance treatment on body mass index and psychological well-being in overweight and obese children: a randomized controlled trial. *Pediatr Obes*. 2012;7(3):205–219
595. Demol S, Yackobovitch-Gavan M, Shalitin S, Nagelberg N, Gillon-Keren M, Phillip M. Low-carbohydrate (low & high-fat) versus high-carbohydrate low-fat diets in the treatment of obesity in adolescents. *Acta Paediatr*. 2009;98(2):346–351
596. Yackobovitch-Gavan M, Nagelberg N, Demol S, Phillip M, Shalitin S. Influence of weight-loss diets with different macronutrient compositions on health-related quality of life in obese youth. *Appetite*. 2008;51(3):697–703
597. Larsen LM, Hertel NT, Mølgaard C, Christensen RD, Husby S, Jarbøl DE. Early intervention for childhood overweight: a randomized trial in general practice. *Scand J Prim Health Care*. 2015;33(3):184–190
598. Mirza NM, Palmer MG, Sinclair KB, et al. Effects of a low glycemic load or a low-fat dietary intervention on body weight in obese Hispanic American children and adolescents: a randomized controlled trial. *Am J Clin Nutr*. 2013;97(2):276–285
599. Partsalaki I, Karvela A, Spiliotis BE. Metabolic impact of a ketogenic diet compared to a hypocaloric diet in obese children and adolescents. *J Pediatr Endocrinol Metab*. 2012;25(7-8):697–704
600. Baños RM, Oliver E, Navarro J, et al. Efficacy of a cognitive and behavioral treatment for childhood obesity supported by the ETIOBE web platform. *Psychol Health Med*. 2019;24(6):703–713
601. Stark LJ, Filigno SS, Kichler JC, et al. Maintenance following a randomized trial of a clinic and home-based behavioral intervention of obesity in preschoolers. *J Pediatr*. 2019;213:128–136.e3
602. Davis AM, Sampilo M, Gallagher KS, Landrum Y, Malone B. Treating rural pediatric obesity through telemedicine: outcomes from a small randomized controlled trial. *J Pediatr Psychol*. 2013;38(9):932–943
603. Davis AM, Sampilo M, Gallagher KS, et al. Treating rural paediatric obesity through telemedicine vs. telephone: outcomes from a cluster randomized controlled trial. *J Telemed Telecare*. 2016;22(2):86–95
604. O'Connor TM, Hilmers A, Watson K, Baranowski T, Giardino AP. Feasibility of an obesity intervention for paediatric primary care targeting parenting and children: helping HAND. *Child Care Health Dev*. 2013;39(1):141–149
605. Wilfley DE, Stein RI, Saelens BE, et al. Efficacy of maintenance treatment approaches for childhood overweight: a randomized controlled trial. *JAMA*. 2007;298(14):1661–1673
606. Wake M, Lycett K, Clifford SA, et al. Shared care obesity management in 3-10 year old children: 12 month outcomes of HopSCOTCH randomised trial. *BMJ*. 2013;346:f3092
607. Krebs NF, Gao D, Gralla J, Collins JS, Johnson SL. Efficacy and safety of a high protein, low carbohydrate diet for weight loss in severely obese adolescents. *J Pediatr*. 2010;157(2):252–258
608. Williams CL, Strobino BA, Brotanek J. Weight control among obese adolescents: a pilot study. *Int J Food Sci Nutr*. 2007;58(3):217–230
609. Kumar S, Croghan IT, Biggs BK, et al. Family-based mindful eating intervention in adolescents with obesity: a pilot randomized clinical trial. *Children (Basel)*. 2018;5(7):93
610. Kokkvoll A, Grimsgaard S, Ødegaard R, Flægstad T, Njølstad I. Single versus multiple-family intervention in childhood overweight—Finnmark Activity School: a randomised trial. *Arch Dis Child*. 2014;99(3):225–231
611. Tjønnå AE, Stølen TO, Bye A, et al. Aerobic interval training reduces cardiovascular risk factors more than a multitreatment approach in overweight adolescents. *Clin Sci (Lond)*. 2009;116(4):317–326
612. Weigel C, Kokocinski K, Lederer P, Dötsch J, Rascher W, Knerr I. Childhood obesity: concept, feasibility, and interim results of a local group-based, long-term treatment program. *J Nutr Educ Behav*. 2008;40(6):369–373
613. Savoye M, Nowicka P, Shaw M, et al. Long-term results of an obesity program in an ethnically diverse pediatric population. *Pediatrics*. 2011;127(3):402–410

614. Kokkvoll A, Grimsgaard S, Steinsbekk S, Flægstad T, Njølstad I. Health in overweight children: 2-year follow-up of Finnmark Activity School—a randomised trial. *Arch Dis Child*. 2015; 100(5):441–448
615. Wilfley DE, Saelens BE, Stein RI, et al. Dose, content, and mediators of family-based treatment for childhood obesity: a multisite randomized clinical trial. *JAMA Pediatr*. 2017;171(12): 1151–1159
616. Lisón JF, Real-Montes JM, Torró I, et al. Exercise intervention in childhood obesity: a randomized controlled trial comparing hospital-versus home-based groups. *Acad Pediatr*. 2012; 12(4):319–325
617. Vos RC, Huisman SD, Houdijk EC, Pijl H, Wit JM. The effect of family-based multidisciplinary cognitive behavioral treatment on health-related quality of life in childhood obesity. *Qual Life Res*. 2012;21(9):1587–1594
618. Bocca G, Corpeleijn E, Stolk RP, Sauer PJ. Results of a multidisciplinary treatment program in 3-year-old to 5-year-old overweight or obese children: a randomized controlled clinical trial. *Arch Pediatr Adolesc Med*. 2012; 166(12):1109–1115
619. Bocca G, Ongering EC, Stolk RP, Sauer PJ. Insulin resistance and cardiovascular risk factors in 3- to 5-year-old overweight or obese children. *Horm Res Paediatr*. 2013;80(3):201–206
620. Nemet D, Oren S, Pantanowitz M, Eliakim A. Effects of a multidisciplinary childhood obesity treatment intervention on adipocytokines, inflammatory and growth mediators. *Horm Res Paediatr*. 2013;79(6):325–332
621. Nemet D, Barkan S, Epstein Y, Friedland O, Kowen G, Eliakim A. Short- and long-term beneficial effects of a combined dietary-behavioral-physical activity intervention for the treatment of childhood obesity. *Pediatrics*. 2005; 115(4):e443–e449
622. van der Baan-Slootweg O, Benninga MA, Beelen A, et al. Inpatient treatment of children and adolescents with severe obesity in the Netherlands: a randomized clinical trial. *JAMA Pediatr*. 2014;168(9):807–814
623. Kirk S, Armstrong S, King E, et al. Establishment of the Pediatric Obesity Weight Evaluation Registry: a national research collaborative for identifying the optimal assessment and treatment of pediatric obesity. *Child Obes*. 2017; 13(1):9–17
624. Skelton JA, Irby MB, Beech BM, Rhodes SD. Attrition and family participation in obesity treatment programs: clinicians' perceptions. *Acad Pediatr*. 2012;12(5):420–428
625. O'Connor EA, Evans CV, Burda BU, Walsh ES, Eder M, Lozano P. Screening for obesity and intervention for weight management in children and adolescents: evidence report and systematic review for the US Preventive Services Task Force. *JAMA*. 2017;317(23): 2427–2444
626. Jasik CB, King EC, Rhodes E, et al. Characteristics of youth presenting for weight management: retrospective national data from the POWER Study Group. *Child Obes*. 2015;11(5):630–637
627. Liu S, Weismiller J, Strange K, et al. Evaluation of the scale-up and implementation of mind, exercise, nutrition ... do it! (MEND) in British Columbia: a hybrid trial type 3 evaluation. *BMC Pediatr*. 2020;20(1):392
628. Hoffman J, Frerichs L, Story M, et al. An integrated clinic-community partnership for child obesity treatment: a randomized pilot trial. *Pediatrics*. 2018;141(1):e20171444
629. Andrews M, Sawyer C, Frerichs L, et al. Feasibility of a clinic-community partnership to treat childhood obesity. *Clin Pediatr (Phila)*. 2018;57(7): 783–791
630. Kong AS, Sussman AL, Yahne C, Skipper BJ, Burge MR, Davis SM. School-based health center intervention improves body mass index in overweight and obese adolescents. *J Obes*. 2013;2013:575016
631. Bocca G, Corpeleijn E, van den Heuvel ER, Stolk RP, Sauer PJ. Three-year follow-up of 3-year-old to 5-year-old children after participation in a multidisciplinary or a usual-care obesity treatment program. *Clin Nutr*. 2014; 33(6):1095–1100
632. Heinberg LJ, Kutchman EM, Berger NA, et al. Parent involvement is associated with early success in obesity treatment. *Clin Pediatr (Phila)*. 2010;49(5):457–465
633. Berge JM, Everts JC. Family-based interventions targeting childhood obesity: a meta-analysis. *Child Obes*. 2011; 7(2):110–121
634. Bean MK, Caccavale LJ, Adams EL, et al. Parent involvement in adolescent obesity treatment: a systematic review. *Pediatrics*. 2020;146(3):e20193315
635. Piercy KL, Troiano RP. Physical activity guidelines for Americans from the US Department of Health and Human Services. *Circ Cardiovasc Qual Outcomes*. 2018;11(11):e005263
636. van der Horst K, Ferrage A, Rytz A. Involving children in meal preparation. Effects on food intake. *Appetite*. 2014; 79:18–24
637. Vos RC, Wit JM, Pijl H, Houdijk EC. Long-term effect of lifestyle intervention on adiposity, metabolic parameters, inflammation and physical fitness in obese children: a randomized controlled trial. *Nutr Diabetes*. 2011;1(10):e9
638. Stark LJ, Spear S, Boles R, et al. A pilot randomized controlled trial of a clinic and home-based behavioral intervention to decrease obesity in preschoolers. *Obesity (Silver Spring)*. 2011;19(1):134–141
639. Makkes S, Renders CM, Bosmans JE, van der Baan-Slootweg OH, Hoekstra T, Seidell JC. One-year effects of two intensive inpatient treatments for severely obese children and adolescents. *BMC Pediatr*. 2016;16:120
640. Quattrin T, Roemmich JN, Paluch R, Yu J, Epstein LH, Ecker MA. Efficacy of family-based weight control program for preschool children in primary care. *Pediatrics*. 2012;130(4):660–666
641. Bocca G, Kuitert MW, Sauer PJ, Stolk RP, Flapper BC, Corpeleijn E. A multidisciplinary intervention programme has positive effects on quality of life in overweight and obese preschool children. *Acta Paediatr*. 2014;103(9): 962–967
642. Butte NF, Hoelscher DM, Barlow SE, et al. Efficacy of a community- versus primary care-centered program for childhood obesity: TX CORD RCT. *Obesity (Silver Spring)*. 2017;25(9): 1584–1593

643. Reinehr T, Schaefer A, Winkel K, Finne E, Toschke AM, Kolip P. An effective lifestyle intervention in overweight children: findings from a randomized controlled trial on "Obeldicks light". *Clin Nutr*. 2010;29(3):331–336
644. Skelton JA, Beech BM. Attrition in paediatric weight management: a review of the literature and new directions. *Obes Rev*. 2011;12(5):e273–e281
645. Butte NF, Watson KB, Ridley K, et al. A youth compendium of physical activities: activity codes and metabolic intensities. *Med Sci Sports Exerc*. 2018;50(2):246–256
646. Anderson YC, Wynter LE, Grant CC, et al. A novel home-based intervention for child and adolescent obesity: the results of the Whanau Pakari randomized controlled trial. *Obesity (Silver Spring)*. 2017;25(11):1965–1973
647. Jebeile H, Lister NB, Baur LA, Garnett SP, Paxton SJ. Eating disorder risk in adolescents with obesity. *Obes Rev*. 2021;22(5):e13173
648. Rhodes ET, Boles RE, Chin K, et al. Expectations for treatment in pediatric weight management and relationship to attrition. *Child Obes*. 2017;13(2):120–127
649. Kumar S, King EC, Christison AL, et al; POWER Work Group. Health outcomes of youth in clinical pediatric weight management programs in POWER. *J Pediatr*. 2019;208:57–65.e4
650. Lycett K, Juonala M, Magnussen CG, et al. Body mass index from early to late childhood and cardiometabolic measurements at 11 to 12 years. *Pediatrics*. 2020;146(2):e20193666
651. Haemer M, Cluett S, Hassink SG, et al. Building capacity for childhood obesity prevention and treatment in the medical community: call to action. *Pediatrics*. 2011;128(Suppl 2):S71–S77
652. Long KA, Weinert C. Rural nursing: developing the theory base. *Sch Inq Nurs Pract*. 1989;3(2):113–127
653. Tolbert J. *The Coverage Provisions in the Affordable Care Act: An Update*. San Francisco, CA: Kaiser Family Foundation; 2015
654. Armstrong SC, Bolling CF, Michalsky MP, Reichard KW; Section on Obesity; Section on Surgery. Pediatric metabolic and bariatric surgery: evidence, barriers, and best practices. *Pediatrics*. 2019;144(6):e20193223
655. Children's Hospital Association. *2013 Survey Findings on Children's Hospitals Obesity Services*. Overland Park, KS: Children's Hospital Association; 2014
656. Azevedo LB, Ling J, Soos I, Robalino S, Ells L. The effectiveness of sedentary behaviour interventions for reducing body mass index in children and adolescents: systematic review and meta-analysis. *Obes Rev*. 2016;17(7):623–635
657. Fanburg JT, Rogers VW, Dedekian MA; National Initiative for Children's Healthcare Quality; The Barbara Bush Children's Hospital; American Academy of Pediatrics Maine Chapter. *Next Steps Activity Book! Activity Book for Caregivers and Children Aged 5 to 12 Years*. Elk Grove Village, IL: American Academy of Pediatrics; 2016
658. Fanburg JT, Rogers VW, Dedekian MA; National Initiative for Children's Healthcare Quality; The Barbara Bush Children's Hospital; American Academy of Pediatrics Maine Chapter. *Pasos Próximos ¡Libro de Actividades! Libro de Actividades para Cuidadores y Niños de Entre 5 y 12 Años*. Elk Grove Village, IL: American Academy of Pediatrics; 2016
659. Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. *Am J Clin Nutr*. 2013;98(4):1084–1102
660. Vos MB, Kaar JL, Welsh JA, et al; American Heart Association Nutrition Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; Council on Epidemiology and Prevention; Council on Functional Genomics and Translational Biology; and Council on Hypertension. Added sugars and cardiovascular disease risk in children: a scientific statement from the American Heart Association. *Circulation*. 2017;135(19):e1017–e1034
661. Committee on Nutrition and the Council on Sports Medicine and Fitness. Sports drinks and energy drinks for children and adolescents: are they appropriate? *Pediatrics*. 2011;127(6):1182–1189
662. Heyman MB, Abrams SA; Section on Gastroenterology, Hepatology, and Nutrition; Committee on Nutrition. Fruit juice in infants, children, and adolescents: current recommendations. *Pediatrics*. 2017;139(6):e20170967
663. Thivel D, Masurier J, Baquet G, et al. High-intensity interval training in overweight and obese children and adolescents: systematic review and meta-analysis. *J Sports Med Phys Fitness*. 2019;59(2):310–324
664. García-Hermoso A, Sánchez-López M, Martínez-Vizcaíno V. Effects of aerobic plus resistance exercise on body composition related variables in pediatric obesity: a systematic review and meta-analysis of randomized controlled trials. *Pediatr Exerc Sci*. 2015;27(4):431–440
665. Kelley GA, Kelley KS. Effects of exercise in the treatment of overweight and obese children and adolescents: a systematic review of meta-analyses. *J Obes*. 2013;2013:783103
666. Ruotsalainen H, Kyngäs H, Tammelin T, Kääriäinen M. Systematic review of physical activity and exercise interventions on body mass indices, subsequent physical activity and psychological symptoms in overweight and obese adolescents. *J Adv Nurs*. 2015;71(11):2461–2477
667. Marson EC, Delevatti RS, Prado AKG, Netto N, Krue LFM. Effects of aerobic, resistance, and combined exercise training on insulin resistance markers in overweight or obese children and adolescents: a systematic review and meta-analysis. *Prev Med*. 2016;93:211–218
668. Monzani A, Ricotti R, Caputo M, et al. A systematic review of the association of skipping breakfast with weight and cardiometabolic risk factors in children and adolescents. What should we better investigate in the future? *Nutrients*. 2019;11(2):387
669. Tucker SJ, Ytterberg KL, Lenocho LM, et al. Reducing pediatric overweight: nurse-delivered motivational interviewing in primary care. *J Pediatr Nurs*. 2013;28(6):536–547

670. Gortmaker SL, Polacsek M, Letourneau L, et al. Evaluation of a primary care intervention on body mass index: the Maine Youth Overweight Collaborative. *Child Obes.* 2015;11(2):187–193
671. Khalsa AS, Kharofa R, Ollberding NJ, Bishop L, Copeland KA. Attainment of '5-2-1-0' obesity recommendations in preschool-aged children. *Prev Med Rep.* 2017;8:79–87
672. LeBlanc AG, Chaput JP, McFarlane A, et al. Active video games and health indicators in children and youth: a systematic review. *PLoS One.* 2013; 8(6):e65351
673. Madsen KA, Thompson HR, Wlasiuk L, Queliza E, Schmidt C, Newman TB. After-school program to reduce obesity in minority children: a pilot study. *J Child Health Care.* 2009;13(4):333–346
674. Oliveira CB, Pinto RZ, Saraiva BTC, et al. Effects of active video games on children and adolescents: a systematic review with meta-analysis. *Scand J Med Sci Sports.* 2020;30(1):4–12
675. Staiano AE, Abraham AA, Calvert SL. Adolescent exergame play for weight loss and psychosocial improvement: a controlled physical activity intervention. *Obesity (Silver Spring).* 2013; 21(3):598–601
676. Staiano AE, Webster EK, Allen AT, Jarrell AR, Martin CK. Screen-time policies and practices in early care and education centers in relationship to child physical activity. *Child Obes.* 2018;14(6):341–348
677. Trost SG, O'Neil M. Clinical use of objective measures of physical activity. *Br J Sports Med.* 2014;48(3):178–181
678. Lu AS, Kharrazi H, Gharghabi F, Thompson D. A systematic review of health videogames on childhood obesity prevention and intervention. *Games Health J.* 2013;2(3):131–141
679. Maddison R, Foley L, Ni Mhurchu C, et al. Effects of active video games on body composition: a randomized controlled trial. *Am J Clin Nutr.* 2011; 94(1):156–163
680. Staiano AE, Marker AM, Beyl RA, Hsia DS, Katzmarzyk PT, Newton RL. A randomized controlled trial of dance exergaming for exercise training in overweight and obese adolescent girls. *Pediatr Obes.* 2017;12(2):120–128
681. Staiano AE, Beyl RA, Guan W, Hendrick CA, Hsia DS, Newton RL Jr. Home-based exergaming among children with overweight and obesity: a randomized clinical trial. *Pediatr Obes.* 2018;13(11): 724–733
682. Trost SG, Sundal D, Foster GD, Lent MR, Vojta D. Effects of a pediatric weight management program with and without active video games a randomized trial. *JAMA Pediatr.* 2014; 168(5):407–413
683. Staiano AE. Exergames, energy expenditure, and obesity. In: van den Bulck J, ed. *The International Encyclopedia of Media Psychology.* Hoboken, NJ: John Wiley and Sons; 2020
684. American Psychological Association. *Clinical Practice Guideline Panel. Clinical Practice Guideline for Multicomponent Behavioral Treatment of Obesity and Overweight in Children and Adolescents: Current State of the Evidence and Research Needs.* Washington, DC: American Psychological Association; 2018
685. Rosenkranz RR, Bauer A, Dziewaltowski DA. Mother-daughter resemblance in BMI and obesity-related behaviors. *Int J Adolesc Med Health.* 2010;22(4): 477–489
686. van der Kruk JJ, Kortekaas F, Lucas C, Jager-Wittenaar H. Obesity: a systematic review on parental involvement in long-term European childhood weight control interventions with a nutritional focus. *Obes Rev.* 2013;14(9):745–760
687. Van Ryzin MJ, Nowicka P. Direct and indirect effects of a family-based intervention in early adolescence on parent-youth relationship quality, late adolescent health, and early adult obesity. *J Fam Psychol.* 2013;27(1):106–116
688. Enright G, Allman-Farinelli M, Redfern J. Effectiveness of family-based behavior change interventions on obesity-related behavior change in children: a realist synthesis. *Int J Environ Res Public Health.* 2020;17(11):4099
689. Modi AC, Pai AL, Hommel KA, et al. Pediatric self-management: a framework for research, practice, and policy. *Pediatrics.* 2012;129(2):e473–e485
690. Ruggiero L, Glasgow R, Dryfoos JM, et al. Diabetes self-management. Self-reported recommendations and patterns in a large population. *Diabetes Care.* 1997;20(4):568–576
691. Richard AA, Shea K. Delineation of self-care and associated concepts. *J Nurs Scholarsh.* 2011;43(3):255–264
692. Welch JL, Johnson M, Zimmerman L, Russell CL, Perkins SM, Decker BS. Self-management interventions in stages 1 to 4 chronic kidney disease: an integrative review. *West J Nurs Res.* 2015;37(5):652–678
693. Ory MG, Ahn S, Jiang L, et al. National study of chronic disease self-management: six-month outcome findings. *J Aging Health.* 2013;25(7):1258–1274
694. Havas K, Douglas C, Bonner A. Meeting patients where they are: improving outcomes in early chronic kidney disease with tailored self-management support (the CKD-SMS study). *BMC Nephrol.* 2018;19(1):279
695. Pugh P, Hemingway P, Christian M, Higginbottom G. Children's, parents', and other stakeholders' perspectives on the factors influencing the initiation of early dietary change in the management of childhood chronic disease: a mixed studies systematic review using a narrative synthesis. *Patient Educ Couns.* 2021;104(4):844–857
696. Jabs J, Devine CM. Time scarcity and food choices: an overview. *Appetite.* 2006;47(2):196–204
697. Curtin C, Hyman SL, Boas DD, et al. Weight management in primary care for children with autism: expert recommendations. *Pediatrics.* 2020;145(Suppl 1):S126–S139
698. Zablotsky B, Black LI, Maenner MJ, et al. Prevalence and trends of developmental disabilities among children in the United States: 2009-2017. *Pediatrics.* 2019;144(4):e20190811
699. Feehan K, O'Neil ME, Abdalla D, et al. Factors influencing physical activity in children and youth with special health care needs: a pilot study. *Int J Pediatr.* 2012;2012:583249
700. Abeyssekara P, Turchi R, O'Neil M. Obesity and children with special healthcare needs: special considerations for a special population. *Curr Opin Pediatr.* 2014;26(4):508–515

701. Ptomey LT, Washburn RA, Goetz JR, et al. Weight loss interventions for adolescents with intellectual disabilities: an RCT. *Pediatrics*. 2021;148(3):e2021050261
702. Ptomey LT, Wittenbrook W. Position of the Academy of Nutrition and Dietetics: nutrition services for individuals with intellectual and developmental disabilities and special health care needs. *J Acad Nutr Diet*. 2015;115(4):593–608
703. Haney K, Messiah SE, Arheart KL, et al. Park-based afterschool program to improve cardiovascular health and physical fitness in children with disabilities. *Disabil Health J*. 2014;7(3):335–342
704. Carbone PS, Smith PJ, Lewis C, LeBlanc C. Promoting the participation of children and adolescents with disabilities in sports, recreation, and physical activity. *Pediatrics*. 2021;148(6):e2021054664
705. CanChild. Research in practice: F words knowledge hub. Available at: <https://www.canchild.ca/en/research-in-practice/f-words-in-childhood-disability>. Accessed October 5, 2022
706. Duis J, van Wattum PJ, Scheimann A, et al. A multidisciplinary approach to the clinical management of Prader-Willi syndrome. *Mol Genet Genomic Med*. 2019;7(3):e514
707. McCandless SE; Committee on Genetics. Clinical report—health supervision for children with Prader-Willi syndrome. *Pediatrics*. 2011;127(1):195–204
708. Haliloglu B, Bereket A. Hypothalamic obesity in children: pathophysiology to clinical management. *J Pediatr Endocrinol Metab*. 2015;28(5-6):503–513
709. Cortese S, Vincenzi B. Obesity and ADHD: clinical and neurobiological implications. *Curr Top Behav Neurosci*. 2012;9:199–218
710. Kelly AS, Auerbach P, Barrientos-Perez M, et al; NN8022-4180 Trial Investigators. A randomized, controlled trial of liraglutide for adolescents with obesity. *N Engl J Med*. 2020;382(22):2117–2128
711. Corcoran C, Jacobs TF. Metformin. StatPearls. Available at: <https://pubmed.ncbi.nlm.nih.gov/30085525/>. Accessed May 2, 2022
712. Pu R, Shi D, Gan T, et al. Effects of metformin in obesity treatment in different populations: a meta-analysis. *Ther Adv Endocrinol Metab*. 2020;11:2042018820926000
713. Wilson DM, Abrams SH, Aye T, et al; Glaser Pediatric Research Network Obesity Study Group. Metformin extended release treatment of adolescent obesity: a 48-week randomized, double-blind, placebo-controlled trial with 48-week follow-up. *Arch Pediatr Adolesc Med*. 2010;164(2):116–123
714. Yanovski JA, Krakoff J, Salaita CG, et al. Effects of metformin on body weight and body composition in obese insulin-resistant children: a randomized clinical trial. *Diabetes*. 2011;60(2):477–485
715. van der Aa MP, Elst MA, van de Garde EM, van Mil EG, Knibbe CA, van der Vorst MM. Long-term treatment with metformin in obese, insulin-resistant adolescents: results of a randomized double-blinded placebo-controlled trial. *Nutr Diabetes*. 2016;6(8):e228
716. Akcam M, Boyaci A, Pirgon O, Kaya S, Uysal S, Dundar BN. Therapeutic effect of metformin and vitamin E versus prescriptive diet in obese adolescents with fatty liver. *Int J Vitam Nutr Res*. 2011;81(6):398–406
717. Atabek ME, Pirgon O. Use of metformin in obese adolescents with hyperinsulinemia: a 6-month, randomized, double-blind, placebo-controlled clinical trial. *J Pediatr Endocrinol Metab*. 2008;21(4):339–348
718. Burgert TS, Duran EJ, Goldberg-Gell R, et al. Short-term metabolic and cardiovascular effects of metformin in markedly obese adolescents with normal glucose tolerance. *Pediatr Diabetes*. 2008;9(6):567–576
719. Casteels K, Fieuws S, van Helvoirt M, et al. Metformin therapy to reduce weight gain and visceral adiposity in children and adolescents with neurogenic or myogenic motor deficit. *Pediatr Diabetes*. 2010;11(1):61–69
720. Clarson CL, Mahmud FH, Baker JE, et al. Metformin in combination with structured lifestyle intervention improved body mass index in obese adolescents, but did not improve insulin resistance. *Endocrine*. 2009;36(1):141–146
721. Freemark M, Bursley D. The effects of metformin on body mass index and glucose tolerance in obese adolescents with fasting hyperinsulinemia and a family history of type 2 diabetes. *Pediatrics*. 2001;107(4):E55
722. Kendall D, Vail A, Amin R, et al. Metformin in obese children and adolescents: the MOCA trial. *J Clin Endocrinol Metab*. 2013;98(1):322–329
723. Rynders C, Weltman A, Delgiorno C, et al. Lifestyle intervention improves fitness independent of metformin in obese adolescents. *Med Sci Sports Exerc*. 2012;44(5):786–792
724. Srinivasan S, Ambler GR, Baur LA, et al. Randomized, controlled trial of metformin for obesity and insulin resistance in children and adolescents: improvement in body composition and fasting insulin. *J Clin Endocrinol Metab*. 2006;91(6):2074–2080
725. Allen HF, Mazzoni C, Heptulla RA, et al. Randomized controlled trial evaluating response to metformin versus standard therapy in the treatment of adolescents with polycystic ovary syndrome. *J Pediatr Endocrinol Metab*. 2005;18(8):761–768
726. Evia-Viscarra ML, Rodea-Montero ER, Apolinar-Jiménez E, et al. The effects of metformin on inflammatory mediators in obese adolescents with insulin resistance: controlled randomized clinical trial. *J Pediatr Endocrinol Metab*. 2012;25(1-2):41–49
727. Maura N, DelGiorno C, Hossain J, et al. Metformin use in children with obesity and normal glucose tolerance—effects on cardiovascular markers and intrahepatic fat. *J Pediatr Endocrinol Metab*. 2012;25(1-2):33–40
728. Pastor-Villaescusa B, Cañete MD, Caballero-Villarraso J, et al. Metformin for obesity in prepubertal and pubertal children: a randomized controlled trial. *Pediatrics*. 2017;140(1):e20164285
729. Chanoine JP, Hampl S, Jensen C, Boldrin M, Hauptman J. Effect of orlistat on weight and body composition in obese adolescents: a randomized controlled trial. *JAMA*. 2005;293(23):2873–2883
730. Ozkan B, Bereket A, Turan S, Keskin S. Addition of orlistat to conventional

- treatment in adolescents with severe obesity. *Eur J Pediatr*. 2004;163(12):738–741
731. Kelly AS, Bensignor MO, Hsia DS, et al Varghese for the Trial Investigators. Phentermine/topiramate for the treatment of adolescent obesity. *N Engl J Med Evid*. 2022;1(6)
732. Olbers T, Beamish AJ, Gronowitz E, et al. Laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity (AMOS): a prospective, 5-year, Swedish nationwide study. *Lancet Diabetes Endocrinol*. 2017;5(3):174–183
733. Pratt JSA, Browne A, Browne NT, et al. ASMBS pediatric metabolic and bariatric surgery guidelines, 2018. *Surg Obes Relat Dis*. 2018;14(7):882–901
734. Inge TH, Courcoulas AP, Jenkins TM, et al; Teen-LABS Consortium. Weight loss and health status 3 years after bariatric surgery in adolescents. *N Engl J Med*. 2016;374(2):113–123
735. Michalsky MP, Inge TH, Jenkins TM, et al; Teen-LABS Consortium. Cardiovascular risk factors after adolescent bariatric surgery. *Pediatrics*. 2018;141(2):e20172485
736. Inge TH, Courcoulas AP, Jenkins TM, et al; Teen-LABS Consortium. Five-year outcomes of gastric bypass in adolescents as compared with adults. *N Engl J Med*. 2019;380(22):2136–2145
737. Alqahtani A, Elahmedi M, Qahtani AR. Laparoscopic sleeve gastrectomy in children younger than 14 years: refuting the concerns. *Ann Surg*. 2016;263(2):312–319
738. Alqahtani AR, Elahmedi M, Abdurabu HY, Alqahtani S. Ten-year outcomes of children and adolescents who underwent sleeve gastrectomy: weight loss, comorbidity resolution, adverse events, and growth velocity. *J Am Coll Surg*. 2021;233(6):657–664
739. Olshansky SJ, Passaro DJ, Hershov RC, et al. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med*. 2005;352(11):1138–1145
740. Chang S-H, Stoll CRT, Song J, Varela JE, Eagon CJ, Colditz GA. The effectiveness and risks of bariatric surgery: an updated systematic review and meta-analysis, 2003-2012. *JAMA Surg*. 2014;149(3):275–287
741. Dewberry LC, Jalivand A, Gupta R, et al. Weight loss and health status 5 years after adjustable gastric banding in adolescents. *Obes Surg*. 2020;30(6):2388–2394
742. Himpens J, Cadière GB, Bazi M, Vouche M, Cadière B, Dapri G. Long-term outcomes of laparoscopic adjustable gastric banding. *Arch Surg*. 2011;146(7):802–807
743. Zitsman JLD, DiGiorgi MF, Fennoy I, Kopchinski JS, Sysko R, Devlin MJ. Adolescent laparoscopic adjustable gastric banding (LAGB): prospective results in 137 patients followed for 3 years. *Surg Obes Relat Dis*. 2015;11(1):101–109
744. Göthberg G, Gronowitz E, Flodmark CE, et al. Laparoscopic Roux-en-Y gastric bypass in adolescents with morbid obesity—surgical aspects and clinical outcome. *Semin Pediatr Surg*. 2014;23(1):11–16
745. O'Brien PE, Sawyer SM, Laurie C, et al. Laparoscopic adjustable gastric banding in severely obese adolescents: a randomized trial. *JAMA*. 2010;303(6):519–526
746. Olbers T, Gronowitz E, Werling M, et al. Two-year outcome of laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity: results from a Swedish Nationwide Study (AMOS). *Int J Obes*. 2012;36(11):1388–1395
747. Olbers T, Beamish AJ, Gronowitz E, et al. Laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity (AMOS): a prospective, 5-year, Swedish nationwide study. *Lancet Diabetes Endocrinol*. 2017;5(3):174–183
748. Ryder JR, Gross AC, Fox CK, et al. Factors associated with long-term weight-loss maintenance following bariatric surgery in adolescents with severe obesity. *Int J Obes*. 2018;42(1):102–107
749. Hardin AP, Hackell JM; Committee on Practice and Ambulatory Medicine. Age limit of pediatrics. *Pediatrics*. 2017;140(3):e20172151
750. Association of Maternal and Child Health Programs. *Standards for Systems of Care for Children*. Washington, DC: Association of Maternal and Child Health Programs; 2014
751. Kebbe M, Perez A, Ball GDC. Is there a role for shared decision-making in pediatric weight management? *Obes Res Clin Pract*. 2018;12(2):246–248
752. Christison AL, Vaidya S, Tinajero-Deck L, Hampl SE. Application of the medical neighborhood to children with severe obesity. *Child Obes*. 2018;14(7):461–467
753. Moore J, Haemer M, Mirza N, et al. Pilot testing of a patient decision aid for adolescents with severe obesity in us pediatric weight management programs within the COMPASS Network. *Int J Environ Res Public Health*. 2019;16(10):1776
754. Turer CB, Montaña S, Lin H, Hoang K, Flores G. Pediatricians' communication about weight with overweight Latino children and their parents. *Pediatrics*. 2014;134(5):892–899
755. Daniels SR, Hassink SG; Committee on Nutrition. The role of the pediatrician in primary prevention of obesity. *Pediatrics*. 2015;136(1):e275–e292
756. Hart LC, Patel-Nguyen SV, Merkley MG, Jonas DE. An evidence map for interventions addressing transition from pediatric to adult care: a systematic review of systematic reviews. *J Pediatr Nurs*. 2019;48:18–34
757. American Academy of Pediatrics; American Academy of Family Physicians; American College of Physicians-American Society of Internal Medicine. A consensus statement on health care transitions for young adults with special health care needs. *Pediatrics*. 2002;110(6 Pt 2):1304–1306
758. Schultz AT, Smaldone A. Components of interventions that improve transitions to adult care for adolescents with type 1 diabetes. *J Adolesc Health*. 2017;60(2):133–146
759. Shrewsbury VA, Baur LA, Nguyen B, Steinbeck KS. Transition to adult care in adolescent obesity: a systematic review and why it is a neglected topic. *Int J Obes*. 2014;38(4):475–479
760. Uzark K, Yu S, Lowery R, et al. Transition readiness in teens and young adults with congenital heart disease: can we make a difference? *J Pediatr*. 2020;221:201–206.e1
761. Hankins JS, Osarogiagbon R, Adams-Graves P, et al. A transition

- pilot program for adolescents with sickle cell disease. *J Pediatr Health Care*. 2012;26(6):e45–e49
762. Saulsberry AC, Hodges JR, Cole A, Porter JS, Hankins J. Web-based technology to improve disease knowledge among adolescents with sickle cell disease: pilot study. *JMIR Pediatr Parent*. 2020;3(1):e15093
763. Blum RW, Garell D, Hodgman CH, et al. Transition from child-centered to adult health-care systems for adolescents with chronic conditions. A position paper of the Society for Adolescent Medicine. *J Adolesc Health*. 1993;14(7):570–576
764. Chu PY, Maslow GR, von Isenburg M, Chung RJ. Systematic review of the impact of transition interventions for adolescents with chronic illness on transfer from pediatric to adult healthcare. *J Pediatr Nurs*. 2015;30(5):e19–e27
765. Cairo SB, Majumdar I, Pryor A, Posner A, Harmon CM, Rothstein DH; Delivery of Surgical Care Committee of the American Academy of Pediatrics Section on Surgery. Challenges in transition of care for pediatric patients after weight-reduction surgery: a systematic review and recommendations for comprehensive care. *Obes Surg*. 2018;28(4):1149–1174
766. Leyser A, Taube-Schiff M, Daniel KS, Toulany A. Transition of care in obesity management: bridging the gap between pediatric and adult health care services. *Can J Diabetes*. 2015;39(1):S15
767. White PH, Cooley WC; Transitions Clinical Report Authoring Group; American Academy of Pediatrics; American Academy of Family Physicians; American College of Physicians. Supporting the health care transition from adolescence to adulthood in the medical home. *Pediatrics*. 2018;142(5):e20182587
768. Yanovski SZ, Yanovski JA. Long-term drug treatment for obesity: a systematic and clinical review. *JAMA*. 2014;311(1):74–86
769. Department of the Treasury, Internal Revenue Service; Department of Labor, Employee Benefits Security Administration; Department of Health and Human Services. Coverage of Certain Preventive Services Under the Affordable Care Act. Final rules. *Fed Regist*. 2015;80(134):41317–41347
770. Staiano A, Marker A, Liu M, Hayden E, Hsia D, Broyles S. Childhood obesity screening and treatment practices of pediatric healthcare providers. *J La State Med Soc*. 2017;169(1):2–10
771. Gortmaker SL, Wang YC, Long MW, et al. Three interventions that reduce childhood obesity are projected to save more than they cost to implement. *Health Aff (Millwood)*. 2015;34(11):1932–1939
772. Kenney EL, Barrett JL, Bleich SN, Ward ZJ, Cradock AL, Gortmaker SL. Impact of the healthy, hunger-free kids act on obesity trends. *Health Aff (Millwood)*. 2020;39(7):1122–1129
773. Kristensen AH, Flottemesch TJ, Maciosek MV, et al. Reducing childhood obesity through U.S. federal policy: a microsimulation analysis. *Am J Prev Med*. 2014;47(5):604–612
774. Mackey ER, Burton ET, Cadieux A, et al. Addressing structural racism is critical for ameliorating the childhood obesity epidemic in black youth. *Child Obes*. 2022;18(2):75–83
775. Odulana A, Basco WT, Bishu KG, Egede LE. Dietary and physical activity counseling trends in U.S. children, 2002-2011. *Am J Prev Med*. 2017;53(1):9–16
776. Imoisili OE, Goodman AB, Dooyema CA, Harrison MR, Belay B, Park S. Screening and referral for childhood obesity: adherence to the U.S. Preventive Services Task Force recommendation. *Am J Prev Med*. 2019;56(2):179–186
777. Fowler LA, Grammer AC, Staiano AE, et al. Harnessing technological solutions for childhood obesity prevention and treatment: a systematic review and meta-analysis of current applications. *Int J Obes*. 2021;45(5):957–981
778. Ramsetty A, Adams C. Impact of the digital divide in the age of COVID-19. *J Am Med Inform Assoc*. 2020;27(7):1147–1148
779. Dorsey ER, Topol EJ. State of telehealth. *N Engl J Med*. 2016;375(2):154–161
780. Boyd RW, Lindo EG, Weeks LD, McLemore MR. On racism: a new standard for publishing on racial health inequities. Available at: <https://www.healthaffairs.org/doi/10.1377/forefront.20200630.939347/>. Accessed March 1, 2021
781. Cabana MD, Rand CS, Powe NR, et al. Why don't physicians follow clinical practice guidelines? A framework for improvement. *JAMA*. 1999;282(15):1458–1465
782. Ayash CR, Simon SR, Marshall R, et al. Evaluating the impact of point-of-care decision support tools in improving diagnosis of obese children in primary care. *Obesity (Silver Spring)*. 2013;21(3):576–582
783. Sharifi M, Franz C, Horan CM, et al. Cost-effectiveness of a clinical childhood obesity intervention. *Pediatrics*. 2017;140(5):e20162998
784. Agency for Healthcare Research and Quality. The healthy weight care assistant. Available at: <https://cda.ahrq.gov/cdsconnect/artifact/healthy-weight-care-assistant>. Accessed October 5, 2022
785. Bronder KL, Dooyema CA, Onufrak SJ, Foltz JL. Electronic health records to support obesity-related patient care: results from a survey of United States physicians. *Prev Med*. 2015;77:41–47
786. Harrison MR, Lundeen EA, Belay B, Goodman AB. Clinical decision supports in electronic health records to promote childhood obesity-related care: results from a 2015 survey of healthcare providers. *Clin Nutr Res*. 2019;8(4):255–264
787. Temple MW, Sisk B, Krams LA, Schneider JH, Kirkendall ES, Lehmann CU. Trends in use of electronic health records in pediatric office settings. *J Pediatr*. 2019;206:164–171.e2
788. Marcus MD, Foster GD, El Ghormli L. Stability of relative weight category and cardiometabolic risk factors among moderately and severely obese middle school youth. *Obesity (Silver Spring)*. 2014;22(4):1118–1125
789. American Diabetes Association. 3. Prevention or delay of type 2 diabetes: standards of medical care in diabetes-2019. *Diabetes Care*. 2019;42(Suppl 1):S29–S33
790. Hannon TS, Janosky J, Arslanian SA. Longitudinal study of physiologic

- insulin resistance and metabolic changes of puberty. *Pediatr Res*. 2006;60(6):759–763
791. Moran A, Jacobs DR Jr, Steinberger J, et al. Insulin resistance during puberty: results from clamp studies in 357 children. *Diabetes*. 1999;48(10):2039–2044
792. Kleber M, Lass N, Papcke S, Wabitsch M, Reinehr T. One-year follow-up of untreated obese white children and adolescents with impaired glucose tolerance: high conversion rate to normal glucose tolerance. *Diabet Med*. 2010;27(5):516–521
793. Weiss R, Taksali SE, Tamborlane WV, Burgert TS, Savoye M, Caprio S. Predictors of changes in glucose tolerance status in obese youth. *Diabetes Care*. 2005;28(4):902–909
794. Tamborlane WV, Barrientos-Pérez M, Fainberg U, et al; Ellipse Trial Investigators. Liraglutide in children and adolescents with type 2 diabetes. *N Engl J Med*. 2019;381(7):637–646
795. Sheka AC, Adeyi O, Thompson J, Hameed B, Crawford PA, Ikramuddin S. Nonalcoholic steatohepatitis: a review. *JAMA*. 2020;323(12):1175–1183
796. Corey KE, Rinella ME. Medical and surgical treatment options for nonalcoholic steatohepatitis. *Dig Dis Sci*. 2016;61(5):1387–1397
797. Lavine JE, Schwimmer JB, Van Natta ML, et al; Nonalcoholic Steatohepatitis Clinical Research Network. Effect of vitamin E or metformin for treatment of nonalcoholic fatty liver disease in children and adolescents: the TONIC randomized controlled trial. *JAMA*. 2011;305(16):1659–1668
798. Schwimmer JB, Ugalde-Nicalo P, Welsh JA, et al. Effect of a low free sugar diet vs usual diet on nonalcoholic fatty liver disease in adolescent boys: a randomized clinical trial. *JAMA*. 2019;321(3):256–265
799. Burrello J, Erhardt EM, Saint-Hilary G, et al. Pharmacological treatment of arterial hypertension in children and adolescents: a network meta-analysis. *Hypertension*. 2018;72(2):306–313
800. Cheung AH, Zuckerbrot RA, Jensen PS, Laraque D, Stein REK; GLAD-PC Steering Group. Guidelines for adolescent depression in primary care (GLAD-PC): part II. treatment and ongoing management. *Pediatrics*. 2018;141(3):e20174082
801. Wall M, McDermott MP, Kiebertz KD, et al; NORDIC Idiopathic Intracranial Hypertension Study Group Writing Committee. Effect of acetazolamide on visual function in patients with idiopathic intracranial hypertension and mild visual loss: the idiopathic intracranial hypertension treatment trial. *JAMA*. 2014;311(16):1641–1651