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Committee on Adolescent Health Care

This Committee Opinion was developed by the American College of Obstetricians and Gynecologists' Committee on Adolescent Health Care in collaboration with committee members Bliss Kaneshiro, MD, MPH and Samantha Erin Vilano, MD, MSc.

Obesity in Adolescents

ABSTRACT: Rates of obesity among adolescents in the United States have increased at a dramatic rate along with the prevalence of weight-related diseases. Between the 1980s and 2014, the prevalence of obesity among adolescent females in the United States increased from approximately 10% to 21%. Although the steep increase in the prevalence of obesity in children (2–11 years) has slowed, the prevalence of obesity in adolescents (12–19 years) continues to increase. Because the obese female adolescent faces medical, psychological, and reproductive health challenges, early intervention is imperative in preventing short-term and long-term morbidity. The obstetrician–gynecologist who is knowledgeable about the behavioral and environmental factors that influence obesity may be better able to educate parents, guardians, and adolescents and advocate for programs that increase physical activity and improve nutrition. The obstetrician–gynecologist should be able to identify obese adolescents, particularly those at risk of comorbid conditions. They may have the opportunity to initiate behavioral counseling, participate in multidisciplinary teams that care for overweight and obese adolescents, and advocate for community programs to prevent obesity.

Recommendations

The American College of Obstetricians and Gynecologists (ACOG) offers the following conclusions and recommendations:

- The obstetrician–gynecologist should be knowledgeable about the behavioral and environmental factors that influence obesity and should educate adolescents and their parents about an active lifestyle and healthy caloric intake.
- The obstetrician–gynecologist should be able to identify obese adolescents, particularly those at risk of comorbid conditions. They may have the opportunity to initiate behavioral counseling, participate in multidisciplinary teams that care for overweight and obese adolescents, and advocate for community programs to prevent obesity.
- Oral emergency contraception should not be withheld from adolescents or women who are overweight or obese because no research to date has been powered adequately to evaluate a threshold weight at which it would be ineffective.
- The risks of all contraceptive methods are lower than the risks of pregnancy and the postpartum period for overweight and obese adolescents.

- The obstetrician–gynecologist should screen overweight and obese adolescents for depression, bullying, and peer victimization and appropriately refer to school-based and community-based resources as well as psychiatric services.
- There are currently no evidence-based guidelines for the use of pharmaceutical agents in the management of obesity in adolescents.
- The obstetrician–gynecologist should caution against the use of weight loss supplements.
- A multidisciplinary team, including an experienced bariatric surgeon, dietitian, and psychologist or psychiatrist, should be used to select appropriate candidates for surgical intervention and provide postoperative support.

Introduction

Rates of obesity among adolescents in the United States have increased at a dramatic rate along with the prevalence of weight-related diseases. Because the obese female adolescent faces medical, psychological, and reproductive health challenges, early intervention is imperative in preventing short-term and long-term morbidity. The obstetrician–gynecologist should be knowledgeable about

the behavioral and environmental factors that influence obesity and should educate adolescents and their parents about an active lifestyle and healthy caloric intake. The obstetrician–gynecologist should be able to identify obese adolescents, particularly those at risk of comorbid conditions. Obstetrician–gynecologists may have the opportunity to initiate behavioral counseling, participate in multidisciplinary teams that care for overweight and obese adolescents, and advocate for community programs to prevent obesity.

Definition

Body mass index (BMI) is defined as weight in kilograms divided by height in meters squared. Measuring BMI is inexpensive and identifies adolescents at risk of weight-related diseases (1, 2). The standard BMI weight-status categories used for adults are not dependent on age or sex and, therefore, cannot be used in the interpretation of BMI in adolescents. To account for normal sex-specific changes in weight, height, and adiposity up to the age of 19 years, BMI should be interpreted after it is plotted on growth charts to determine BMI-for-age and BMI-for-sex percentile (<https://nccd.cdc.gov/dnpabmi/calculator.aspx>). Because BMI does not directly measure adiposity, it is not accurate in predicting health risk in athletic adolescents with increased muscle mass or sedentary adolescents with reduced muscle mass. *Overweight* is defined as a BMI at or above the 85th percentile, *obesity* is a BMI at or above the 95th percentile, and *severe obesity* is a BMI greater than or equal to the 99th percentile for age (see Table 1). Extreme obesity has been used to describe adolescents who are at or above 120% of the sex-specific 95th percentile for age (3).

Prevalence and Trends

Between the 1980s and 2014, the prevalence of obesity among adolescent females in the United States increased from approximately 10% to 21% (4). Although the steep increase in the prevalence of obesity in children (2–11 years) has slowed, the prevalence of obesity in adolescents (12–19 years) continues to increase (4). Obesity

Table 1. Weight Categories ⇐

Overweight	Greater than or equal to 85th percentile to less than 95th percentile
Class I	Greater than or equal to 95th percentile to less than 120% of the 95th percentile
Class II	Greater than or equal to 120% to less than 140% of the 95th percentile, or BMI greater than or equal to 35, whichever is lower
Class III	Greater than or equal to 140% of the 95th percentile, or BMI greater than or equal to 40, whichever is lower

Abbreviation: BMI, body mass index.

Data from Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *N Engl J Med* 2015;373:1307–17.

is highly complex in terms of etiology and prevalence (5). Genetic predisposition, race, socioeconomic status, built environment (eg, the presence of sidewalks or community design), accessibility of healthy and affordable foods, sleep habits, and geographic region all play a role. Changes in the “American lifestyle,” which includes consuming foods and beverages with a high glycemic index, increased food portion sizes, decreased structured physical activity, and increased screen-based sedentary behavior, have influenced the prevalence of obesity (6, 7). Intrauterine environment also plays a role; large-for-gestational-age infants have a higher risk of obesity in adolescence compared with normal-weight infants (8). The prevalence of obesity varies by race, with black adolescents having the highest prevalence (24.4%), followed by Hispanic (22.8%), white (20.4%), and Asian (5.7%) adolescents (4). Eight percent of female adolescents have extreme obesity (BMI at or above 120% of the sex-specific 95th percentile for age) (4).

Health Risks

Adolescents affected by obesity are at an increased risk of developing comorbidities. As weight increases, the risk of impaired glucose tolerance increases. The prevalence of impaired glucose tolerance (HgbA_{1c} greater than 5.7 or fasting glucose greater than or equal to 100 mg/dL) is 1.87% in overweight adolescents (12–18 years) and increases progressively to a prevalence of 13.19% in adolescents with class III obesity (see Table 1). Because type 2 diabetes is associated with progressive neuropathy, retinopathy, nephropathy, and cardiovascular disease, prevention, early diagnosis, and treatment are imperative.

Even in the absence of type 2 diabetes, obese adolescents are at increased risk of cardiovascular disease in adulthood because they have an increased risk of hypertension and dyslipidemia (9). As BMI increases so does the risk of hypertension. Class III obese adolescents (12–18 years) have 2.5–7.6 times the risk of high blood pressure (greater than 95th percentile for age, sex, and height) compared with class I obese adolescents (9). Obese adolescents begin to develop cardiovascular damage once seen only in adults, including atherosclerotic changes, increased left ventricular mass, and systolic and diastolic dysfunction (10–12).

Attaining a healthy weight can dramatically improve the cardiovascular health of adolescents as they transition into adulthood. Data from four large longitudinal studies noted obese and overweight children who are not obese as adults have similar cardiovascular risks as individuals who were never obese (13).

Metabolic syndrome has been used to describe the coexistence of risk factors for type 2 diabetes and cardiovascular disease, including abdominal obesity, hyperglycemia, dyslipidemia, and hypertension. The prevalence of metabolic syndrome among adolescents is estimated to be approximately 9% (14), although pubertal growth results in instability in the diagnosis of metabolic

syndrome in adolescents. One half of all adolescents initially classified with metabolic syndrome will no longer meet the criteria after 3 years, whereas others will acquire the diagnosis (15). In adolescents, the long-term cardiovascular and type 2 diabetes risk for individuals with metabolic syndrome is not well defined.

Obesity is associated with nonalcoholic fatty liver disease in adolescents. The prevalence of fatty liver is estimated to be 38% in obese children and adolescents (2–19 years), with the highest risk noted in Hispanic youths followed by Asian, white, and black youth (16). Because nonalcoholic fatty liver disease is associated with insulin resistance, dyslipidemia, and hypertension, adolescents with nonalcoholic fatty liver disease should be evaluated for each of these conditions. The natural history of nonalcoholic fatty liver disease in adolescents is not well-described, although it may lead to fibrosis, cirrhosis, and liver failure in some cases (17).

The prevalence of disordered breathing is increased in obese adolescents. Obesity hypoventilation syndrome is characterized by poor alveolar air exchange during wakefulness secondary to excess weight and typically occurs in cases of extreme obesity (18). Obstructive sleep apnea is a more common condition in obese adolescents. In a study of obese children and adolescents, 11% had mild obstructive sleep apnea and 8% had moderate to severe obstructive sleep apnea (18). The association between sleep and obesity is complex. Obese adolescents are at increased risk of obstructive sleep apnea, and shortened sleep duration or fragmented sleep promotes obesity. A study in children demonstrated they consumed more food and gained weight when deficient of sleep for 1 week (19).

Obese children and adolescents are at an increased risk of orthopedic conditions, including slipped capital femoral epiphysis, tibia vara, genu valgum, and musculoskeletal pain (20, 21). Obese adolescents also are more susceptible to fractures because they have reduced bone mass when adjusted for body size (21).

Adolescence and early adulthood is a period of concern for the development of eating disorders; these disorders should be screened for regardless of BMI. Greater-than-expected weight dissatisfaction, large weight fluctuations, and depressive symptoms in adolescents can be signs of a binge eating disorder (22).

Gynecologic Health Risks

Abnormal uterine bleeding is common in obese adolescents who report amenorrhea, heavy menstrual bleeding, or other menstrual abnormalities. In addition to anovulation because of immaturity of the hypothalamic–pituitary–ovarian axis, abnormal uterine bleeding in obese adolescents can result from elevated levels of free estrogens due to increased peripheral aromatization of androgens to estrogens, decreased sex hormone binding globulin, and increased insulin levels that can stimulate ovarian stromal tissue production of androgens (23, 24).

Elevated peripheral estrogen disrupts normal ovulation, which results in abnormal uterine bleeding. In the rare case reports of adolescents with endometrial cancer, the clinical history typically includes 2–3 years of abnormal bleeding and obesity (25, 26). Endometrial evaluation should be performed if medical treatment of abnormal bleeding has failed after a thorough investigation of all potential other causes and comorbid disorders (27).

Polycystic ovary syndrome (PCOS), characterized by ovulatory dysfunction and hyperandrogenism, frequently presents during adolescence. Because PCOS has lifelong implications, including an increased risk of type 2 diabetes, metabolic syndrome, cardiovascular disease, and endometrial carcinoma, a diagnosis of PCOS should be considered in any adolescent female with obesity accompanied by hirsutism or menstrual irregularity (28).

Different organizations have endorsed different diagnostic criteria for PCOS in adult women (29–31). Caution should be taken when diagnosing PCOS in adolescents because the features of PCOS overlap with normal pubertal development. Anovulatory cycles can be typical for several years after menarche. Signs of hyperandrogenism, such as hair growth and acne vulgaris, are common in this age group. Table 2 outlines the Pediatric Endocrine Society's recommendations to assist in the diagnosis of PCOS in adolescents.

Even if a definitive diagnosis of PCOS cannot be made during adolescence, treatment may be indicated (32). Treatment of PCOS in adolescents should address the symptoms that the adolescent finds bothersome. Weight reduction can improve menstrual cycle regularity, but does not have a significant effect upon hirsutism. Metformin can be used for abnormal glucose tolerance, although it provides no advantage over lifestyle modification with regard to weight reduction or menstrual regulation in adolescents (33, 34). Combined hormonal contraceptives can regulate menstrual cycles and normalize serum androgens, thereby improving acne; they also provide protection against unintended pregnancy and decrease the risk of developing endometrial cancer. The progestin intrauterine device (IUD), implant, and depot medroxyprogesterone acetate (DMPA) decrease heavy bleeding and provide highly effective contraception; however, they are associated with irregular bleeding and will not improve the cutaneous manifestations of hyperandrogenism (35).

Obstetric Health Risks

Obese adolescents who become pregnant are at increased risk of cesarean delivery, labor induction, preeclampsia, and gestational diabetes compared with their normal-weight peers (36, 37). However, obesity also has a protective effect on preterm birth in adolescents. This has been noted in white adolescents and black adolescents who have a higher baseline rate of preterm birth (36, 37).

Epidemiologic data suggest exercise may be beneficial in preventing gestational diabetes in pregnant

Table 2. Recommendations by the Pediatric Endocrine Society to Assist in the Diagnosis of Polycystic Ovary Syndrome in Adolescents ⇄

Clinical Finding	Recommendation
Hirsutism	<ul style="list-style-type: none"> Isolated mild hirsutism should not be considered clinical evidence of hyperandrogenism in the early postmenarcheal years Moderate-to-severe hirsutism constitutes clinical evidence of hyperandrogenism Adolescents with acne that is persistent and poorly responsive to topical dermatologic therapy should be evaluated for the presence of hyperandrogenism before initiating medical therapy
Biochemical hyperandrogenism	<ul style="list-style-type: none"> Hyperandrogenism should be defined based on the detailed characteristics of the testosterone assay Biochemical evidence of hyperandrogenism, as indicated by persistent elevation of serum total or free testosterone levels, or both, and determined in a reliable reference laboratory, provides the clearest support of the presence of hyperandrogenism in an adolescent girl with symptoms of PCOS A single androgen level greater than 2 SDs above the mean for a specific assay should not be considered to be evidence of hyperandrogenism in an otherwise asymptomatic adolescent girl
Oligo-anovulation	<ul style="list-style-type: none"> Menstrual intervals persistently shorter than 20 days or more than 45 days in individuals 2 or more years after menarche are evidence of oligo-anovulation A menstrual interval greater than 90 days is unusual even in the first year after menarche and requires further investigation regardless of year after menarche Lack of onset of menses by age 15 years or more than 2 years after thelarche regardless of chronologic age is uncommon and warrants further investigation
Polycystic ovarian morphology	<ul style="list-style-type: none"> No compelling criteria to define polycystic ovarian morphology has been established for adolescents An ovarian volume of greater than 12.0 cm³ can be considered enlarged Follicle counts should not be used to define polycystic ovarian morphology in adolescents Multifollicular pattern is more common in adolescents, does not have a relationship with hyperandrogenism, and should not be considered a pathologic finding In healthy girls with regular menstrual cycles and no evidence of hyperandrogenism, polycystic ovarian morphology does not indicate a diagnosis of PCOS Antimüllerian hormone concentration is elevated in adolescents with PCOS and is being explored as a surrogate for antral follicle count. Although currently it has no role in the diagnosis of PCOS, it has potential as a screening tool.* Abdominal ultrasonography, particularly in obese adolescents, may yield inadequate information Ovarian imaging can be deferred during the diagnostic evaluation of PCOS until better quality or consistent data are available
Insulin resistance or hyperinsulinemia	<ul style="list-style-type: none"> Insulin resistance and hyperinsulinemia should not be used as diagnostic criteria for PCOS Insulin resistance and hyperinsulinemia can be considered as indications to investigate and treat potential comorbidities

Abbreviation: PCOS, polycystic ovarian syndrome.

*Dumont A, Robin G, Catteau-Jonard S, Dewailly D. Role of Anti-Müllerian Hormone in pathophysiology, diagnosis and treatment of polycystic ovary syndrome: a review. *Reprod Biol Endocrinol* 2015;13:137.

Data from Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. *Endocrine Society. J Clin Endocrinol Metab* 2013; 98:4565–92.

women with a BMI greater than 33. The American Diabetes Association has endorsed physical activity as a safe and effective therapy for glucose management in women with gestational diabetes (38).

Contraception

Although no evidence suggests that any contraceptive method is ineffective in obese adolescents or women, concerns have been raised about the diminished effec-

tiveness of certain methods because of physiologic differences in those who are obese versus normal weight. This is a particular concern for methods whose mechanism of action relies on the systemic distribution of steroid hormones (eg, pills, the patch, the vaginal ring, DMPA, and implants) (39–42).

Although several studies have explored the effect of obesity on the pharmacokinetics and pharmacodynamics of the oral contraceptive pill, studies specific to

adolescents are nonexistent (43). In adult women, studies have demonstrated altered contraceptive steroid half-life (44, 45), although follicular development did not differ significantly in obese versus normal-weight women who were taking oral contraceptives (44–46). A large prospective postmarketing study of more than 52,000 women who contributed 73,000 woman-years of oral contraceptive exposure did demonstrate a slight increased risk of failure (hazard ratio, 1.5; CI 1.3–1.8) in obese women compared with normal-weight women (47). Although women younger than 20 years of age were included in this study, they were not analyzed separately.

No data specific to adolescents have been published to analyze the effect of BMI on the efficacy of the contraceptive patch or ring. The contraceptive patch package label states that the patch may be less effective in women weighing more than 90 kg (48). Two small studies of the vaginal ring reported follicular development was minimal in obese women (49), and hormone levels remained in the therapeutic range up to 35 days after ring insertion in obese women (50).

The efficacy of the intramuscular formulation of DMPA (150 mg) is not decreased in obese women, and the efficacy of the lower-dose subcutaneous DMPA formulation (104 mg) has been confirmed in obese adult women (51). Throughout a period of 26 weeks, median DMPA levels remained above a level needed to prevent ovulation (200 pg/mL) even in women with class III obesity (BMI greater than or equal to 40), and there was no evidence of ovulation (51).

Although not directly studied, BMI should not have an effect on the effectiveness of the copper or hormonal IUD for either adults or adolescents as the contraceptive effect is local (43). A prospective cohort study that included IUD users 14 years of age and older found no difference in contraceptive failure rate by BMI for the first 2–3 years among copper and hormonal IUD users (52).

The efficacy of the etonogestrel implant does not appear to be affected by weight. A 6-month pharmacokinetic study of the etonogestrel implant in obese adult women reported circulating levels of hormone lower than that of normal-weight historical controls. However, the 2-year and 3-year projected serum levels remained above the minimum needed to suppress ovulation (53).

Levonorgestrel and ulipristal acetate-based oral emergency contraception may be less effective as weight increases (54, 55). Data specific to adolescents are lacking, although one study did include females who were younger than 18 years (55). Oral emergency contraception should not be withheld from adolescents or women who are overweight or obese because no research to date has been powered adequately to evaluate a threshold weight at which it would be ineffective (56). Not only does the copper IUD have superior efficacy compared with oral forms of emergency contraception, but its efficacy is not affected by body weight and it is a

highly effective method of ongoing contraception. Consideration should be given to use of a copper IUD as an alternative to oral emergency contraception in obese adolescents.

Safety of Hormonal Contraceptives in Overweight or Obese Adolescents

The risks of all contraceptive methods are lower than the risks of pregnancy and the postpartum period for overweight and obese adolescents. The main concern with the use of estrogen-containing contraceptives in obese adolescents is the risk of venous thromboembolism (VTE) given that obesity is an independent risk factor for VTE (57). However, VTE is an exceedingly rare event in children and adolescents, even among those with significant risk factors for clotting. For example, patients with hypercoagulable conditions do not typically present with a clot until after the age of 20 years (58). The *U.S. Medical Eligibility Criteria for Contraceptive Use* gives all contraceptives a classification of either “safe for use with no restrictions” (category 1) or “advantages generally outweigh theoretical or proven risks” (category 2) for obese adolescents from menarche to 18 years without other medical conditions (59).

Weight Gain With Hormonal Contraceptives

Depot medroxyprogesterone acetate injection is classified as category 2 for obese adolescent females because some studies show certain adolescents may be more susceptible to weight gain with DMPA (59), although studies in this area are challenged by poor methodology, specifically, the lack of an appropriate control group. An observational study compared change in body fat and lean body mass in adolescents (12–18 years old) who were using DMPA with those who were using a nonhormonal method (60). Over 6 months, adolescents using DMPA had a 10.3% increase in total body fat compared with a decrease of 0.7% in adolescents who were using a nonhormonal method (mean difference, 11.00%; 95% CI, 2.64–19.36). Another observational study noted racial differences in weight gain, with black adolescents who were using DMPA experiencing a higher increase in weight (4.2% versus 1.2%) and body fat (12.5% versus 1.2%) compared with white adolescents using DMPA (61). Adolescents who gain weight during the first few doses of DMPA may have a propensity for weight gain with DMPA; adolescents who have a 5% increase in body weight in the first 6 months of DMPA use will gain more weight than those who do not (62).

Adolescents who use contraceptive pills, the patch, the vaginal ring, implant, or IUD do not experience an increase in body weight or a change in body composition (63, 64). A 12-month study in females 14 years of age and older found no difference in weight gain with the etonogestrel implant, levonorgestrel IUD, or DMPA compared with copper IUD users (65).

Psychosocial Risks

Adolescents who are overweight or obese are at higher risk of low self-esteem, distorted body image, depression, anxiety, discrimination, and strained peer relationships. Psychosocial morbidity is higher in girls than boys and tends to increase as children transition into adolescence and adulthood (66–68). Data from the National Longitudinal Survey of Youth reported that women who were obese in late adolescence and early adulthood complete fewer years of advanced education, have a lower family income, lower rates of marriage, and higher rates of poverty compared with their nonobese counterparts (69). This association was not noted in men who were obese during adolescence (69).

Bullying (aggressive behavior, characterized by repetition and power imbalance) and peer victimization (the experience of being the target of aggressive behavior) occurs commonly in overweight and obese adolescents, particularly in those with severe obesity (70). For girls in particular, peer victimization occurs through social exclusion (71). Bullying and peer victimization can lead to a range of adjustment difficulties, anxiety, and depression (72). The obstetrician–gynecologist should screen overweight and obese adolescents for depression, bullying, and peer victimization and appropriately refer to school-based and community-based resources as well as psychiatric services.

Preventing Obesity

The obstetrician–gynecologist who is knowledgeable about the behavioral and environmental factors that influence obesity may be better able to educate parents, guardians, and adolescents and advocate for programs that increase physical activity and improve nutrition. Behavioral modification strategies include self-monitoring, stimulus control, goal setting, and positive reinforcement. Parents are the main role models for adolescent eating and physical activity and have a direct effect on the adolescent's food and activity environment (73). Parents are encouraged to focus on healthy eating behavior and exercising to be fit rather than talking about dieting or exercising for weight loss (74). Parents who encourage their adolescent to diet or speak about their own dieting are more likely to have adolescents who engage in unhealthy weight-control behaviors or binge eating (75).

As children transition into adolescence, sports and favorite childhood activities may be replaced by sedentary activities like social networking. Girls, specifically, have a significant drop in physical activity during adolescence (76). As adolescents transition into adulthood, they experience additional changes that affect weight (77). Girls who were active in high school sports may stop these activities as they enter college or the workforce. Food availability, cooking skills, and alcohol intake can affect the nutrition of young adults as they enter college or leave the home. During this time, the obstetrician–gynecologist can encourage a healthy lifestyle, including

healthy body image, physical activity, and balanced nutrition. The American Academy of Pediatrics has developed a multifaceted program to prevent obesity and engage families and communities in promoting healthy behavior (78).

Management of Adolescent Obesity

Lifestyle Interventions

The American Academy of Pediatrics recommends a four-stage approach to obesity in adolescents with a weight loss goal of no more than 2 pounds per week, depending on BMI percentile (79) (see Table 3). Interventions include encouraging healthy eating and physical activity and behavioral counseling by health care providers trained in weight management. Familial involvement has been critical in most studies (80, 81). However, the optimal behavioral interventions that will result in long-term weight alterations in adolescents have yet to be identified.

Medical Therapies

There are currently no evidence-based guidelines for the use of pharmaceutical agents in the management of obesity in adolescents. Medical therapies are most often employed for adolescents who have not responded to lifestyle changes or for those with medical comorbidities. Only tetrahydrolipstatin is approved by the U.S. Food and Drug Administration for use in adolescents and it is considered the first-line adjunct to behavioral interventions (82). Sibutramine was voluntarily withdrawn from the market in 2010 because of cardiovascular concerns (82).

Tetrahydrolipstatin is available over-the-counter and is indicated for adolescents 12 years and older with a BMI greater than or equal to two units above the 95th percentile. Tetrahydrolipstatin inhibits digestive lipases to block approximately 25–30% of dietary fat absorption (82, 83). Adverse effects include fatty or oily stools, abdominal pain, fecal urgency, and diarrhea. Dietary fat reduction can decrease gastrointestinal disturbance and increase tetrahydrolipstatin acceptance in adolescents (82, 84). Studies of tetrahydrolipstatin report modest reductions in weight. A 2009 meta-analysis of randomized clinical trials in adolescents reported a mean BMI reduction of 0.83 with tetrahydrolipstatin (83).

Metformin is not recommended for adolescents for weight loss alone. Adolescents with PCOS diagnosed with insulin resistance may be considered as candidates for this medication. Metformin can result in modest reductions in weight when used with a behavioral weight reduction program (33, 85). Gastrointestinal adverse effects (eg, abdominal pain, diarrhea, vomiting) are dose related and improve with continued use. A meta-analysis of nine randomized clinical trials compared metformin with placebo or other lifestyle interventions in obese adolescents without comorbidities (86). Study duration averaged 6 months, with a range of 2–12 months. The metformin group had a mean BMI reduction of 1.21–1.42

Table 3. Suggested Staged Approach to Weight Management for Children and Adolescents ↵

Stage	Components	Where Implemented	Implemented by Whom and Skills Needed	Frequency of Visits/Duration Before Moving to Next Stage
1. Prevention Plus	<p>Recommend 5 or more servings of fruits and vegetables per day, 2 or fewer hours of screen time per day, no television in room where child sleeps, and no television if less than 2 years of age. Minimize or eliminate sugar-sweetened beverages. Address eating behaviors (eg, eating away from home, daily breakfast, family dinners, and skipping meals). Recommend 1 hour or more of physical activity per day. Amount of physical activity may need to be graded for children who are sedentary; they may not achieve 1 hour per day initially. Involve whole family in lifestyle changes. Acknowledge cultural differences.</p>	Primary care office	Primary care provider or trained professional staff member (eg, registered nurse)	<p>Visit frequency should be based on accepted readiness to change or behavioral counseling techniques and tailored to patient and family. Provider should encourage more frequent visits when obesity is more severe. Advance to more intensive level of intervention depending on responses to treatment, age, health risks, and motivation. A child in this stage whose BMI has tracked in same percentile over time with no medical risks may have low risk for excess body fat. Clinicians can continue obesity prevention strategies and not advance treatment stages.</p>
2. Structured weight management	<p>Develop plan with family for balanced macronutrient diet emphasizing small amounts of energy-dense foods. Because diet provides less energy, ensure that protein is high quality and sufficient to prevent loss of muscle mass. Increase structure of daily meals and snacks. Reduce screen time to 1 hour per day or less. Increase time spent in physical activity (60 minutes or more of supervised activity play per day). Instruct patient and/or parent in monitoring (eg, screen time, physical activity, dietary intake, and restaurant logs) to improve adherence. Perform medical screening (eg, vital signs, assessment tools, and laboratory tests).</p>	Referral to dietitian; primary care office	Registered dietitian or physician or nurse practitioner with additional training, including assessment techniques, motivational interviewing, or behavioral counseling (may need to provide specific information with environmental change and reward examples), parenting skills and managing family conflict, food planning (including energy density and macronutrient knowledge), physical activity counseling, and resources or referrals.	<p>Monthly visits should be tailored to patient and family, based on family's readiness to change. Advance to more-intensive level of intervention depending on responses to treatment, age, health risks, and motivation</p>

(continued)

Table 3. Suggested Staged Approach to Weight Management for Children and Adolescents (*continued*) ↩

Stage	Components	Where Implemented	Implemented by Whom and Skills Needed	Frequency of Visits/Duration Before Moving to Next Stage
3. Comprehensive multidisciplinary intervention	Distinguished from stage 2 by more frequent patient or provider contact, more active use of behavioral strategies, more formal monitoring, and feedback regarding progress to improve adherence. Multidisciplinary approach is essential. Components of multidisciplinary behavioral weight control programs include (1) moderate/strong parental involvement for children younger than 12 years of age; parental involvement should decrease gradually as adolescents increase in age; (2) assessment of diet, physical activity, and weight (body fat) before treatment and at specified intervals thereafter to evaluate progress; (3) structured behavioral program that includes at least food monitoring, short-term diet and activity goal setting, and contingency management; (4) parent/caregiver training to improve home food and activity environments; and (5) structured dietary and physical activity interventions that improve dietary quality and result in negative energy balance.	Primary care office can coordinate multidisciplinary care; weight management program (community), pediatric weight management center, or commercial programs with the following components: age-appropriate and culturally appropriate treatments; nutrition, exercise, and behavioral counseling provided by trained professionals; and weight loss goals of 2 pounds or less per week. Use primer 1* to evaluate commercial programs.	Multidisciplinary team with expertise in childhood obesity, including behavioral counselor (eg, social worker, psychologist, trained nurse practitioner, or other mental health care provider), registered dietitian, and exercise specialist. Alternative could be dietitian and behavioral counselor based in primary care office, along with outside, structured, physical activity program (eg, team sports, YMCA, or Boys and Girls Club program). For areas without services, consider innovative programs (eg, telemedicine).	Frequent follow-up visits (weekly for a minimum of 8–12 weeks is most efficacious) and then monthly follow-up visits. If not feasible, then telephone or other modalities could be used, with weight checks no less than once per month in local health care provider office (eg, primary care provider or health department). Advance to more intensive level of intervention depending on responses to treatment, age, health risks, and motivation.

(continued)

Table 3. Suggested Staged Approach to Weight Management for Children and Adolescents (*continued*) ↔

Stage	Components	Where Implemented	Implemented by Whom and Skills Needed	Frequency of Visits/Duration Before Moving to Next Stage
4. Tertiary care intervention	Continued diet and activity counseling plus consideration of meal replacement, very-low-energy diet, medication, and surgery.	Pediatric weight management center operating under established protocols (eg, clinical or research) to assess and to monitor risks and outcomes; residential settings (camps or boarding facilities with appropriate medical supervision). Use primer 2 to evaluate centers.*	Multidisciplinary team with expertise in childhood obesity, including behavioral counselor (eg, social worker, psychologist, trained nurse practitioner, or other mental health care provider), or registered dietitian, and exercise specialist. For areas without services, consider innovative programs (eg, telemedicine).	According to protocol

Abbreviation: BMI, body mass index.

*Two primers have been developed to help primary care providers and other practitioners assess the ability of commercial weight loss programs and bariatric surgery centers to treat pediatric obesity patients; they utilize a question-and-answer format. See Spear BA, Barlow SE, Ervin C, Ludwig DS, Saelens BE, Schetzina KE, et al. Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics* 2007;120(suppl 4):S254–88.

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with no effect on fasting glucose levels. There was no difference in adverse effects between the metformin group and the placebo group and the withdrawal rate because adverse effects in those receiving metformin was low (2.7%) (86).

Dietary supplements are widely marketed as weight loss agents, but evidence to support their safety and efficacy is lacking. In addition to having no proven effect on weight, dietary supplements can have stimulant-like cardiovascular effects. The obstetrician–gynecologist should caution against the use of weight loss supplements.

Surgical Interventions

Based on expert opinion, the American Society for Metabolic and Bariatric Surgery has recommended surgery be considered for adolescents who meet the following criteria: BMI greater than or equal to 35 and a severe comorbidity (eg, type 2 diabetes) or BMI greater than or equal to 40 with minor comorbidities; Tanner stage IV or attainment of 95% of predicted adult stature based on bone age; and a history of sustained efforts to lose weight through changes in diet and physical activity (87). Long-term outcome data after gastric bypass surgery in adolescents are lacking, but cohort studies are ongoing. Depressive symptoms and quality of life have been shown to improve in adolescents after surgery (88, 89). Designated Centers of Excellence for weight loss surgery for adolescents can be accessed at www.facs.org/search/

[bariatric-surgery-centers](#). See Table 4 for a list of surgical procedures for weight loss.

A multidisciplinary team, including an experienced bariatric surgeon, dietitian, and psychologist or psychiatrist, should be used to select appropriate candidates for surgical intervention and provide postoperative support. Good candidates are those with mature decision-making abilities, appropriate understanding of the risks and benefits of surgery, and support but not coercion from family members. Patients and their families need to have the ability and motivation to adhere to postoperative treatments, including consistent use of micronutrient supplements.

Surgically induced weight loss will lead to resumption of ovulation in some anovulatory women. Although the effects of bariatric surgery on menstrual irregularities and ovulation in adolescents have not been described, in a series of 47 adolescents who had bariatric surgery, seven pregnancies occurred in the first 2 years after surgery (90). Although women with a history of gastric bypass can have healthy pregnancies, pregnancy should be avoided for at least 12–18 months after gastric bypass because of rapid weight loss and micronutrient deficiencies (91). The *U.S. Medical Eligibility Criteria for Contraceptive Use* categories are presented in Table 4. Limited data have been published on the placement of an IUD at the time of bariatric surgery; however, it may be considered in adolescents who wish to use it as a long-term contraceptive method (92).

Table 4. Types of Surgical Procedures ⇄

Type	Description	Effectiveness	U.S. Medical Eligibility Criteria Category for Contraceptive Use*
Adjustable gastric banding	<ul style="list-style-type: none"> Restrictive procedure Band is placed around the entrance to the stomach 	<ul style="list-style-type: none"> Prospective, randomized controlled trial of 50 adolescents (14–18 years) showed gastric banding was more effective at achieving weight loss than lifestyle modification (mean weight loss in gastric banding group was 34.6 kg compared with 3.0 kg in the lifestyle group)[†] Gastric banding results in resolution of weight-related comorbidities[†] 	All contraceptives category 1
Sleeve gastrectomy (partial gastrectomy, vertical sleeve gastrectomy)	<ul style="list-style-type: none"> Restrictive procedure Resection of the greater curvature of the stomach Most common procedure performed in adolescents Weight loss results from appetite suppression and early satiety 	Cohort study of 67 adolescents (45 females) (mean age 17.0 ± 1.7 years) reported BMI reduction from a mean of 50 to 37 over 3 years (26% weight change) [‡]	All contraceptives category 1
Roux-en-Y gastric bypass	<ul style="list-style-type: none"> Restrictive and malabsorptive procedure Proximal gastric pouch is divided and separated from the distal stomach and anastomosed to a roux limb of small bowel Associated with a higher risk of nutritional deficiencies Associated with decreased bone density although levels do not fall below normal for age and sex[§] 	<ul style="list-style-type: none"> A meta-analysis of six adolescent Roux-en-Y studies found a 35% reduction in BMI and dramatic resolution of hypertension and sleep apnea morbidities Cohort study of 161 adolescents (126 females) (mean age 17.0 ± 1.5 years) reported BMI reduction from a mean of 54 to 39 over 3 years (28% weight change)[‡] 	All contraceptives category 1 except oral contraceptive pills, which are category 3

*Curtis KM, Tepper NK, Jatlaoui TC, Berry-Bibee E, Horton LG, Zapata LB, et al. *U.S. Medical Eligibility Criteria for Contraceptive Use*, 2016. *MMWR Recomm Rep* 2016; 65:1–103.

[†]O'Brien PE, Sawyer SM, Laurie C, Brown WA, Skinner S, Veit F, et al. Laparoscopic adjustable gastric banding in severely obese adolescents: a randomized trial [published erratum appears in *JAMA* 2010;303:2357]. *JAMA* 2010;303:519–26.

[‡]Inge TH, Courcoulas AP, Jenkins TM, Michalsky MP, Helmuth MA, Brandt ML, et al. Weight loss and health status 3 years after bariatric surgery in adolescents. Teen-LABS Consortium. *N Engl J Med* 2016;374:113–23.

[§]Kaulfers AM, Bean JA, Inge TH, Dolan LM, Kalkwarf HJ. Bone loss in adolescents after bariatric surgery. *Pediatrics* 2011;127:e956–61.

^{||}Treadwell JR, Sun F, Schoelles K. Systematic review and meta-analysis of bariatric surgery for pediatric obesity. *Ann Surg* 2008;248:763–76.

References

- Clarke WR, Woolson RF, Lauer RM. Changes in ponderosity and blood pressure in childhood: the Muscatine Study. *Am J Epidemiol* 1986;124:195–206. ⇄
- Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics* 1999;103:1175–82. ⇄
- Centers for Disease Control and Prevention. About child and teen BMI. Available at: https://www.cdc.gov/healthy-weight/assessing/bmi/childrens_bmi/about_childrens_bmi.html. Retrieved March 23, 2017. ⇄
- Ogden CL, Carroll MD, Lawman HG, Fryar CD, Kruszon-Moran D, Kit BK, et al. Trends in obesity prevalence among children and adolescents in the United States, 1988–1994 through 2013–2014. *JAMA* 2016;315:2292–9. ⇄

5. Challenges for overweight and obese women. Committee Opinion No. 591. American College of Obstetricians and Gynecologists [published erratum appears in *Obstet Gynecol* 2016;127:166]. *Obstet Gynecol* 2014;123:726–30. ↩
6. Taber DR, Chiqui JF, Powell L, Chaloupka FJ. Association between state laws governing school meal nutrition content and student weight status: implications for new USDA school meal standards. *JAMA Pediatr* 2013;167:513–9. ↩
7. Anderson SE, Whitaker RC. Household routines and obesity in US preschool-aged children. *Pediatrics* 2010;125:420–8. ↩
8. Gillman MW, Rifas-Shiman S, Berkey CS, Field AE, Colditz GA. Maternal gestational diabetes, birth weight, and adolescent obesity. *Pediatrics* 2003;111:e221–6. ↩
9. Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardio-metabolic risks and severity of obesity in children and young adults. *N Engl J Med* 2015;373:1307–17. ↩
10. Hanevold C, Waller J, Daniels S, Portman R, Sorof J. The effects of obesity, gender, and ethnic group on left ventricular hypertrophy and geometry in hypertensive children: a collaborative study of the International Pediatric Hypertension Association [published erratum appears in *Pediatrics* 2005;115:1118]. *Pediatrics* 2004;113:328–33. ↩
11. Chinali M, de Simone G, Roman MJ, Lee ET, Best LG, Howard BV, et al. Impact of obesity on cardiac geometry and function in a population of adolescents: the Strong Heart Study. *J Am Coll Cardiol* 2006;47:2267–73. ↩
12. Cote AT, Harris KC, Panagiotopoulos C, Sandor GG, Devlin AM. Childhood obesity and cardiovascular dysfunction. *J Am Coll Cardiol* 2013;62:1309–19. ↩
13. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med* 2011;365:1876–85. ↩
14. 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;110:2494–7. ↩
15. Goodman E, Daniels SR, Meigs JB, Dolan LM. Instability in the diagnosis of metabolic syndrome in adolescents. *Circulation* 2007;115:2316–22. ↩
16. Schwimmer JB, Deutsch R, Kahen T, Lavine JE, Stanley C, Behling C. Prevalence of fatty liver in children and adolescents. *Pediatrics* 2006;118:1388–93. ↩
17. Feldstein AE, Charatcharoenwitthaya 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;58:1538–44. ↩
18. Verhulst SL, Schrauwen N, Haentjens D, Suys B, Rooman RP, Van Gaal L, et al. Sleep-disordered breathing in overweight and obese children and adolescents: prevalence, characteristics and the role of fat distribution. *Arch Dis Child* 2007;92:205–8. ↩
19. Hart CN, Carskadon MA, Considine RV, Fava JL, Lawton J, Raynor HA, et al. Changes in children's sleep duration on food intake, weight, and leptin. *Pediatrics* 2013;132:e1473–80. ↩
20. Taylor ED, Theim KR, Mirch MC, Ghorbani S, Tanofsky-Kraff M, Adler-Wailes DC, et al. Orthopedic complications of overweight in children and adolescents. *Pediatrics* 2006;117:2167–74. ↩
21. Chan G, Chen CT. Musculoskeletal effects of obesity. *Curr Opin Pediatr* 2009;21:65–70. ↩
22. Devlin MJ. Binge-eating disorder comes of age. *Ann Intern Med* 2016;165:445–6. ↩
23. Nestler JE, Powers LP, Matt DW, Steingold KA, Plymate SR, Rittmaster RS, et al. A direct effect of hyperinsulinemia on serum sex hormone-binding globulin levels in obese women with the polycystic ovary syndrome. *J Clin Endocrinol Metab* 1991;72:83–9. ↩
24. Pugeat M, Nader N, Hogeveen K, Raverot G, Dechaud H, Grenot C. Sex hormone-binding globulin gene expression in the liver: drugs and the metabolic syndrome. *Mol Cell Endocrinol* 2010;316:53–9. ↩
25. Howlader N, Noone AM, Krapcho M, Miller D, Bishop K, Altekruse SF, et al, editors. SEER cancer statistics review, 1975–2013. Bethesda (MD): National Cancer Institute; 2016. Available at: http://seer.cancer.gov/csr/1975_2013. Retrieved March 23, 2017. ↩
26. Stovall DW, Anderson RJ, De Leon FD. Endometrial adenocarcinoma in teenagers. *J Pediatr Adolesc Gynecol* 1989;2:157–9. ↩
27. Management of abnormal uterine bleeding associated with ovulatory dysfunction. Practice Bulletin No. 136. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2013;122:176–85. ↩
28. Legro RS, Arslanian SA, Ehrmann DA, Hoeger KM, Murad MH, Pasquali R, et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. Endocrine Society. *J Clin Endocrinol Metab* 2013;98:4565–92. ↩
29. Dunaif A, Chang RJ, Franks S, Legro RS, editors. Polycystic ovary syndrome: current controversies, from the ovary to the pancreas. Totowa (NJ): Humana Press; 2008. ↩
30. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. *Fertil Steril* 2004;81:19–25. ↩
31. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. Criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. Androgen Excess Society. *J Clin Endocrinol Metab* 2006;91:4237–45. ↩
32. Witchel SF, Oberfield S, Rosenfield RL, Codner E, Bonny A, Ibanez L, et al. The diagnosis of polycystic ovary syndrome during adolescence. *Horm Res Paediatr* 2015;83:376–89. ↩
33. Naderpoor N, Shorakae S, de Courten B, Misso ML, Moran LJ, Teede HJ. Metformin and lifestyle modification in polycystic ovary syndrome: systematic review and meta-analysis. *Hum Reprod Update* 2015;21:560–74. ↩
34. Hoeger K, Davidson K, Kochman L, Cherry T, Kopin L, Guzik DS. The impact of metformin, oral contraceptives, and lifestyle modification on polycystic ovary syndrome in obese adolescent women in two randomized,

- placebo-controlled clinical trials. *J Clin Endocrinol Metab* 2008;93:4299–306. ↩
35. Kaunitz AM, Inki P. The levonorgestrel-releasing intrauterine system in heavy menstrual bleeding: a benefit-risk review. *Drugs* 2012;72:193–215. ↩
 36. Sukalich S, Mingione MJ, Glantz JC. Obstetric outcomes in overweight and obese adolescents. *Am J Obstet Gynecol* 2006;195:851–5. ↩
 37. Haeri S, Guichard I, Baker AM, Saddlemire S, Boggess KA. The effect of teenage maternal obesity on perinatal outcomes. *Obstet Gynecol* 2009;113:300–4. ↩
 38. Colberg SR, Sigal RJ, Fernhall B, Regensteiner JG, Blissmer BJ, Rubin RR, et al. Exercise and type 2 diabetes: the American College of Sports Medicine and the American Diabetes Association: joint position statement. American College of Sports Medicine; American Diabetes Association. *Diabetes Care* 2010;33:e147–67. ↩
 39. Brunner Huber LR, Toth JL. Obesity and oral contraceptive failure: findings from the 2002 National Survey of Family Growth. *Am J Epidemiol* 2007;166:1306–11. ↩
 40. Dinger JC, Cronin M, Mohner S, Schellschmidt I, Minh TD, Westhoff C. Oral contraceptive effectiveness according to body mass index, weight, age, and other factors. *Am J Obstet Gynecol* 2009;201:263.e1–9. ↩
 41. Holt VL, Scholes D, Wicklund KG, Cushing-Haugen KL, Daling JR. Body mass index, weight, and oral contraceptive failure risk. *Obstet Gynecol* 2005;105:46–52. ↩
 42. Kaneshiro B, Edelman A, Carlson N, Nichols M, Jensen J. The relationship between body mass index and unintended pregnancy: results from the 2002 National Survey of Family Growth. *Contraception* 2008;77:234–8. ↩
 43. Grimes DA, Shields WC. Family planning for obese women: challenges and opportunities. *Contraception* 2005;72:1–4. ↩
 44. Edelman AB, Carlson NE, Cherala G, Munar MY, Stouffer RL, Cameron JL, et al. Impact of obesity on oral contraceptive pharmacokinetics and hypothalamic-pituitary-ovarian activity. *Contraception* 2009;80:119–27. ↩
 45. Westhoff CL, Torgal AH, Mayeda ER, Pike MC, Stanczyk FZ. Pharmacokinetics of a combined oral contraceptive in obese and normal-weight women. *Contraception* 2010;81:474–80. ↩
 46. Edelman AB, Cherala G, Munar MY, Dubois B, McInnis M, Stanczyk FZ, et al. Prolonged monitoring of ethinyl estradiol and levonorgestrel levels confirms an altered pharmacokinetic profile in obese oral contraceptives users. *Contraception* 2013;87:220–6. ↩
 47. Dinger J, Minh TD, Buttman N, Bardenheuer K. Effectiveness of oral contraceptive pills in a large U.S. cohort comparing progestogen and regimen. *Obstet Gynecol* 2011;117:33–40. ↩
 48. Ziemann M, Guillebaud J, Weisberg E, Shangold GA, Fisher AC, Creasy GW. Contraceptive efficacy and cycle control with the Ortho Evra/Evra transdermal system: the analysis of pooled data. *Fertil Steril* 2002;77:S13–8. ↩
 49. Westhoff CL, Torgal AH, Mayeda ER, Petrie K, Thomas T, Dragoman M, et al. Pharmacokinetics and ovarian suppression during use of a contraceptive vaginal ring in normal-weight and obese women [published erratum appears in *Am J Obstet Gynecol* 2013;208:326]. *Am J Obstet Gynecol* 2012;207:39.e1–6. ↩
 50. Dragoman M, Petrie K, Torgal A, Thomas T, Cremers S, Westhoff CL. Contraceptive vaginal ring effectiveness is maintained during 6 weeks of use: a prospective study of normal BMI and obese women. *Contraception* 2013;87:432–6. ↩
 51. Segall-Gutierrez P, Taylor D, Liu X, Stanczyk F, Azen S, Mishell DR Jr. Follicular development and ovulation in extremely obese women receiving depo-medroxyprogesterone acetate subcutaneously. *Contraception* 2010;81:487–95. ↩
 52. Xu H, Wade JA, Peipert JF, Zhao Q, Madden T, Secura GM. Contraceptive failure rates of etonogestrel subdermal implants in overweight and obese women. *Obstet Gynecol* 2012;120:21–6. ↩
 53. Mornar S, Chan LN, Mistretta S, Neustadt A, Martins S, Gilliam M. Pharmacokinetics of the etonogestrel contraceptive implant in obese women. *Am J Obstet Gynecol* 2012;207:110.e1–6. ↩
 54. Kapp N, Abitbol JL, Mathe H, Scherrer B, Guillard H, Gainer E, et al. Effect of body weight and BMI on the efficacy of levonorgestrel emergency contraception. *Contraception* 2015;91:97–104. ↩
 55. Glasier AF, Cameron ST, Fine PM, Logan SJ, Casale W, Van Horn J, et al. Ulipristal acetate versus levonorgestrel for emergency contraception: a randomised non-inferiority trial and meta-analysis [published erratum appears in *Lancet* 2014;384:1504]. *Lancet* 2010;375:555–62. ↩
 56. Emergency contraception. Practice Bulletin No. 152. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2015;126:e1–11. ↩
 57. Abdollahi M, Cushman M, Rosendaal FR. Obesity: risk of venous thrombosis and the interaction with coagulation factor levels and oral contraceptive use. *Thromb Haemost* 2003;89:493–8. ↩
 58. Lensen RP, Rosendaal FR, Koster T, Allaart CF, de Ronde H, Vandenbroucke JP, et al. Apparent different thrombotic tendency in patients with factor V Leiden and protein C deficiency due to selection of patients. *Blood* 1996;88:4205–8. ↩
 59. Curtis KM, Tepper NK, Jatlaoui TC, Berry-Bibee E, Horton LG, Zapata LB, et al. U.S. medical eligibility criteria for contraceptive use, 2016. *MMWR Recomm Rep* 2016;65:1–103. ↩
 60. Bonny AE, Sedic M, Cromer BA. A longitudinal comparison of body composition changes in adolescent girls receiving hormonal contraception. *J Adolesc Health* 2009;45:423–5. ↩
 61. Bonny AE, Britto MT, Huang B, Succop P, Slap GB. Weight gain, adiposity, and eating behaviors among adolescent females on depot medroxyprogesterone acetate (DMPA). *J Pediatr Adolesc Gynecol* 2004;17:109–15. ↩
 62. Le YC, Rahman M, Berenson AB. Early weight gain predicting later weight gain among depot medroxyprogesterone acetate users. *Obstet Gynecol* 2009;114:279–84. ↩
 63. Reubinoff BE, Grubstein A, Meirou D, Berry E, Schenker JG, Brzezinski A. Effects of low-dose estrogen oral contraceptives on weight, body composition, and fat distribution in young women. *Fertil Steril* 1995;63:516–21. ↩

64. Lloyd T, Lin HM, Matthews AE, Bentley CM, Legro RS. Oral contraceptive use by teenage women does not affect body composition. *Obstet Gynecol* 2002;100:235–9. ↩
65. Vickery Z, Madden T, Zhao Q, Secura GM, Allsworth JE, Peipert JF. Weight change at 12 months in users of three progestin-only contraceptive methods. *Contraception* 2013;88:503–8. ↩
66. Erickson SJ, Robinson TN, Haydel KF, Killen JD. Are overweight children unhappy? Body mass index, depressive symptoms, and overweight concerns in elementary school children. *Arch Pediatr Adolesc Med* 2000;154:931–5. ↩
67. Strauss RS. Childhood obesity and self-esteem. *Pediatrics* 2000;105:e15. ↩
68. Swallen KC, Reither EN, Haas SA, Meier AM. Overweight, obesity, and health-related quality of life among adolescents: the National Longitudinal Study of Adolescent Health. *Pediatrics* 2005;115:340–7. ↩
69. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993;329:1008–12. ↩
70. Curtis P. The experiences of young people with obesity in secondary school: some implications for the healthy school agenda. *Health Soc Care Community* 2008;16:410–8. ↩
71. Pearce MJ, Boergers J, Prinstein MJ. Adolescent obesity, overt and relational peer victimization, and romantic relationships. *Obes Res* 2002;10:386–93. ↩
72. Hawker DS, Boulton MJ. Twenty years' research on peer victimization and psychosocial maladjustment: a meta-analytic review of cross-sectional studies. *J Child Psychol Psychiatry* 2000;41:441–55. ↩
73. Daniels SR, Hassink SG. The role of the pediatrician in primary prevention of obesity. Committee on Nutrition. *Pediatrics* 2015;136:e275–92. ↩
74. Golden NH, Schneider M, Wood C. Preventing obesity and eating disorders in adolescents. Committee on Nutrition; Committee on Adolescence; Section on Obesity. *Pediatrics* 2016;138(3). ↩
75. Loth KA, Neumark-Sztainer D, Croll JK. Informing family approaches to eating disorder prevention: perspectives of those who have been there. *Int J Eat Disord* 2009;42:146–52. ↩
76. Kimm SY, Glynn NW, Kriska AM, Barton BA, Kronsberg SS, Daniels SR, et al. Decline in physical activity in black girls and white girls during adolescence. *N Engl J Med* 2002;347:709–15. ↩
77. Gordon-Larsen P, The NS, Adair LS. Longitudinal trends in obesity in the United States from adolescence to the third decade of life. *Obesity (Silver Spring)* 2010;18:1801–4. ↩
78. American Academy of Pediatrics. Institute for Healthy Childhood Weight. Available at: <https://ihcw.aap.org/programs/Pages/default.aspx>. Retrieved March 23, 2017. ↩
79. Spear BA, Barlow SE, Ervin C, Ludwig DS, Saelens BE, Schetzina KE, et al. Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics* 2007;120(suppl 4):S254–88. ↩
80. Savoye M, Nowicka P, Shaw M, Yu S, Dziura J, Chavent G, et al. Long-term results of an obesity program in an ethnically diverse pediatric population. *Pediatrics* 2011;127:402–10. ↩
81. Faith MS, Van Horn L, Appel LJ, Burke LE, Carson JA, Franch HA, et al. Evaluating parents and adult caregivers as “agents of change” for treating obese children: evidence for parent behavior change strategies and research gaps: a scientific statement from the American Heart Association. *Circulation* 2012;125:1186–207. ↩
82. Greydanus DE, Bricker LA, Feucht C. Pharmacotherapy for obese adolescents. *Pediatr Clin North Am* 2011;58:139–53, xi. ↩
83. Catoira N, Nagel M, Di Girolamo G, Gonzalez CD. Pharmacological treatment of obesity in children and adolescents: current status and perspectives. *Expert Opin Pharmacother* 2010;11:2973–83. ↩
84. Dunican KC, Desilets AR, Montalbano JK. Pharmacotherapeutic options for overweight adolescents. *Ann Pharmacother* 2007;41:1445–55. ↩
85. Gambineri A, Patton L, Vaccina A, Cacciari M, Morselli-Labate AM, Cavazza C, et al. Treatment with flutamide, metformin, and their combination added to a hypocaloric diet in overweight–obese women with polycystic ovary syndrome: a randomized, 12-month, placebo-controlled study. *J Clin Endocrinol Metab* 2006;91:3970–80. ↩
86. Bouza C, Lopez-Cuadrado T, Gutierrez-Torres LF, Amate J. Efficacy and safety of metformin for treatment of overweight and obesity in adolescents: an updated systematic review and meta-analysis. *Obes Facts* 2012;5:753–65. ↩
87. Michalsky M, Reichard K, Inge T, Pratt J, Lenders C. ASMBS pediatric committee best practice guidelines. American Society for Metabolic and Bariatric Surgery. *Surg Obes Relat Dis* 2012;8:1–7. ↩
88. Ratcliff MB, Reiter-Purtill J, Inge TH, Zeller MH. Changes in depressive symptoms among adolescent bariatric candidates from preoperative psychological evaluation to immediately before surgery. *Surg Obes Relat Dis* 2011;7:50–4. ↩
89. Loux TJ, Haricharan RN, Clements RH, Kolotkin RL, Bledsoe SE, Haynes B, et al. Health-related quality of life before and after bariatric surgery in adolescents. *J Pediatr Surg* 2008;43:1275–9. ↩
90. Roehrig HR, Xanthakos SA, Sweeney J, Zeller MH, Inge TH. Pregnancy after gastric bypass surgery in adolescents. *Obes Surg* 2007;17:873–7. ↩
91. Bariatric surgery and pregnancy. ACOG Practice Bulletin No. 105. American College of Obstetricians and Gynecologists. *Obstet Gynecol* 2009;113:1405–13. ↩
92. Hillman JB, Miller RJ, Inge TH. Menstrual concerns and intrauterine contraception among adolescent bariatric surgery patients. *J Womens Health (Larchmt)* 2011;20:533–8. ↩

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