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Pediatric obesity: Parallels with addiction and treatment recommendations

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Abstract

Rates of pediatric obesity have increased dramatically over the past decade. This trend is particularly alarming as obesity is associated with significant medical and psychosocial consequences. Obesity may contribute to cardiovascular, metabolic, and hepatic complications, as well as psychiatric difficulties. The development of obesity appears to be influenced by a complex array of genetic, metabolic, and neural frameworks, as well as behavior, eating habits, and physical activity. Numerous parallels exist between obesity and addictive behaviors, including genetic predisposition, personality, environmental risk factors, and common neurobiological pathways in the brain. Typical treatments for pediatric obesity include behavioral interventions targeting diet and/or exercise. Treatments focusing on diet and exercise have yielded mixed results, and typically have been examined in specialty clinic populations, limiting their generalizability. There are limited medication options for overweight children and adolescents, and no approved medical intervention in children younger than 16. Bariatric surgery may be an option for some adolescents, but due to the risks of surgery it is often seen as a last resort. The parallels between addiction and obesity aid in the development of novel interventions for pediatric obesity. Motivational enhancement and cognitive-behavioral strategies used in addiction treatment may serve to be beneficial.

Keywords

Pediatric obesity; medical co-morbidity; addiction; treatment; review

Pediatric obesity has reached epidemic proportions, with a 200-400% increase in the past 10 years¹⁻³. Alarming, routine screening for obesity in children is very low (0.5% in a recent review of 600 pediatric cases), treatment is only provided to a fraction of children identified as overweight, and the most frequently used treatment (general advice) is known to be ineffective⁴. The current article will review some of the correlates of obesity, examine parallels with addictive behaviors, and review treatment recommendations.

Due to concerns about stigma, the CDC recommends the term “overweight”, rather than obese, when referring to youth⁵. Overweight is a condition where one has excess adipose tissue⁶. Body Mass Index (BMI) is the most accepted parameter for measuring total body fat. A BMI of > 85th percentile indicates mild to moderate overweight, and a BMI > 95th percentile indicates a need for intervention. Based on these criteria, more than 20% of US

children and adolescents are overweight/obese ⁷. Poor minority youth are disproportionately affected: 44% of Mexican-American and 46% of African-American youths are obese compared to 29% of whites ^{8, 9}. Low SES likewise contributes to being overweight by limiting purchase of high quality healthy foods and opportunity for exercise ¹⁰. Finally, children born to overweight parents are at higher risk for obesity ^{11, 12} perpetuating a cycle of obesity.

Medical and psychosocial consequences of overweight in children and adolescents

Being overweight during childhood and adolescence is associated with a myriad of concurrent medical and psychosocial consequences, as well as placing children and teens at higher risk for medical comorbidity and mortality as adults. One of the most widely documented health consequences of overweight is a metabolic syndrome that includes insulin resistance, type 2 diabetes mellitus, polycystic ovary syndrome, dyslipidemia, and hypertension ¹³. This syndrome has been documented in children and adolescents and associated with increased risk for developing cardiovascular disease (CVD) in adulthood ^{14, 15}. In a large, nationally representative sample of American adolescents, metabolic syndrome was present in 6.8% of adolescents with BMI > 85th percentile and 28.7% of adolescents with BMI > 95th percentile ¹⁶. In fact, 89% of overweight adolescents had at least one irregular metabolic indicator ¹⁶. Regarding insulin resistance, overweight is the most common cause of insulin resistance in children ¹⁷ and the strongest predictor of insulin resistance in young adulthood ¹⁸. In a recent trial of almost 500 children and adolescents, the prevalence of metabolic syndrome increased with BMI and was found in nearly 50% of severely overweight children (BMI > 99th percentile), providing further evidence of the link between severity of overweight and prevalence of metabolic problems ¹⁹.

In addition to linking overweight with a metabolic syndrome, there is a literature associating overweight in children and adolescents with the unique aspects and consequences of the metabolic syndrome. For example, there is a strong association between childhood overweight and type 2 diabetes CDC ^{8, 9}, CDC ^{20, 21-23}. Rates of type 2 diabetes have increased significantly in recent years, paralleling the increase in overweight ²⁴; a recent review by Cruz and colleagues ²⁵ reported rates of type 2 diabetes in overweight children between 0-6%. Being overweight is a significant risk factor for developing type 2 diabetes in childhood, perhaps through the mechanism of impaired glucose tolerance ^{2, 14, 15, 20}. In a recent study of 167 overweight children and adolescents, impaired glucose tolerance was detected in 25% of children aged 4 to 10 years and 21% of adolescents aged 11 to 18 years; type 2 diabetes also was identified in 4% of the adolescents ²⁶.

Similar to type 2 diabetes, polycystic ovarian syndrome ²⁷ and dyslipidemia ²⁸ have been associated with overweight in adolescents. Also, overweight in adolescence predicts the development of CVD in young adulthood ²⁹⁻³¹. In children and adolescents, overweight has been shown to increase the chances of hypertension 2-3.7 times ^{32, 33}. In a large epidemiological study of 47,000 teens, there was an increased incidence of hypertension in obese children with a rate of 2:1 once BMI exceeded 38 ³². Another large study reviewed primary care charts for 18,618 children and adolescents aged 2-19 years and found that increased BMI was significantly associated with increased blood pressure, regardless of age ³⁴.

Besides the medical consequences related to metabolic syndrome, overweight in children and adolescents is associated with other serious conditions such as non-alcoholic fatty liver disease NAFLD; ³⁵⁻³⁸, obstructive sleep apnea ³⁹, and orthopedic problems including joint

pain, impaired mobility, fractures, etc.⁴⁰. Studies have shown between 10–25% of children referred to obesity centers have elevated liver enzymes predictive of future liver disease^{41–43}. Obstructive sleep apnea also is more common among overweight children and adolescents³⁹. Further, while severity of apnea does not differ between overweight and non-overweight children, being overweight significantly decreased the efficacy of treatment; apnea resolved in 78% of non-overweight compared to 45% of overweight children³⁹. In sum, overweight is associated with numerous health consequences in childhood and adolescence.

In addition to a myriad of health problems, obesity also may negatively impact psychiatric and behavioral functioning. While the prevalence of psychiatric disorders among obese youth (other than eating disorders) is not well documented⁴⁴, they have more behavioral and self-esteem problems compared to non-obese children. For instance, one study found that 34% of obese girls had low self-esteem, compared to only 8% of non-obese girls⁴⁵. Longer-term complications of overweight include the development of psychosocial and psychological problems such as depression and low self-esteem in adulthood⁴⁶. Similarly, a recent 20-year prospective study demonstrated an association between obesity in youth and atypical depression and aggression in adulthood⁴⁷.

Eating disorders may also be over-represented in obese adolescent females, with 30% having a binge eating disorder (BED; ^{5, 48}). Although little is known about this subgroup, obese adults with BED are significantly different from those without in that they exhibit more eating disorder pathology (e.g., recurrent episodes of binge eating, body image disparagement, distorted attitudes about eating and weight;⁴⁹, more severe psychopathology (e.g. depression, anxiety, substance abuse;^{50, 51}, and more impaired social functioning⁵². Regarding comorbid substance use disorders, studies have shown that adolescents who binge eat are at increased risk for substance abuse and have more first-degree relatives with addictive disorders^{53–55}.

Causal mechanisms for obesity

Obesity is a complex disorder influenced by genetics, metabolism, neural frameworks as well as behavior, food habits and physical activity. Approximately 200 genes, gene markers and chromosomal regions are associated with obesity⁴⁶. The ‘ob’ gene (mapped to chromosome seven) codes for the hormone leptin, a 167 amino acid protein that influences both energy intake and expenditure; mutation of the ‘ob’ gene leads to improper coding of leptin, contributing to obesity⁴⁶.

In addition to leptin, the neurotransmitter neuropeptide Y (NPY) and hormone ghrelin are involved in weight regulation. The hypothalamus contains key pathways for weight regulation and houses high concentrations of both Leptin and NPY. Higher levels of NPY activity have been found in the hypothalamus of obese rodents. Leptin, which is secreted mainly by adipose tissue, acts directly on the brain, is higher in obese than in lean individuals, and decreases during weight and fat loss⁵⁶. Ghrelin is a recently discovered peripheral peptide hormone produced mainly by the stomach and stimulates food intake⁵⁷. Administration of ghrelin increases food intake in animals⁵⁸ and humans⁵⁹. Ghrelin acts primarily on the hypothalamus⁶⁰, possibly via vagal receptors⁶¹. Surprisingly, fasting ghrelin has been found to be 27% lower in obese than in normal weight individuals⁶². Ghrelin rises following weight loss in obese subjects⁶³. The lower fasting levels in obesity and obese binge eaters⁶⁴ suggest that ghrelin is down-regulated in response to overeating or excess body weight^{62, 65}. Indeed, fasting ghrelin is negatively correlated with % body fat, fasting insulin and leptin, all of which are higher in obesity^{58, 66}. Ghrelin, however, is elevated in Prader-Willi syndrome (PWS), which is associated with severe obesity⁶⁷.

Functional neuroimaging likewise supports a neurobiological explanation for obesity⁵⁵. In one study⁶⁸, brain activity changes following glucose ingestion were compared in non-obese and obese individuals. In normal weight subjects, brain activity changed in approximately 9 minutes; there were significant reductions in hypothalamic activity after glucose ingestion and these reductions were correlated with increased plasma insulin levels. In obese subjects, these changes were significantly delayed and plasma insulin levels failed to correlate with decreases in hypothalamic activity⁶⁸. This response could translate into a delayed awareness of satiety, leading to overeating and obesity⁶⁹⁻⁷¹.

Environmental factors that contribute to obesity include learned lifestyle patterns, an overabundance of energy-dense food choices, and decreased motivation and/or opportunities for physical activity^{46, 72-74}. Level of physical activity consistently has been demonstrated as being inversely related to weight⁷⁵. Individuals who are inactive are at increased risk for obesity, with exercise being the single strongest predictor of long-term maintenance of weight loss⁷⁵. Unfortunately, the popularity of sedentary activities such as TV watching and playing video games may have contributed to the increasing prevalence of childhood obesity⁷⁶.

Parallels between obesity and addiction

Numerous parallels between overeating and addiction have been demonstrated⁷⁷⁻⁷⁹. Volkow and Wise⁷⁹ posit that obesity and drug or alcohol addiction are the result of “ingestion habits” that are powerfully reinforced by the ingested substance and cause significant negative consequences. Obesity and addiction share similar patterns of transmission, including genetic predisposition, environmental risk factors, and common neurobiological pathways in the brain. Understanding these parallels may be helpful in preventing and treating the growing obesity epidemic.

Genes may contribute 50% of vulnerability to both addiction and obesity⁸⁰⁻⁸². However, genetic influence on the vulnerability to both obesity and addiction is complex, involving both environmental and cultural influences, and may be polygenetic⁷⁹. Regarding environmental influences, both obesity and addiction have increased when food and drug have been cheap and available. The recent increase in obesity may coincide with increased prevalence of low-cost, high-fat, high-carbohydrate food and significantly larger portion sizes, similar to the recent addiction problems with crack cocaine and crystal methamphetamine as they became less costly and more available⁷⁹.

Findings from functional brain imaging studies have shown that the inability to control eating produces changes in neural activity similar to those produced by substance use^{70, 83}. The hypothalamus helps maintain energy homeostasis and alterations in hypothalamic activity could result in delayed awareness of satiety, leading to obesity. In addition, chemical neuromediators such as leptin, ghrelin, melanocortin agonists, neuropeptide Y and opioids have been implicated in alcoholism and other drug dependencies as well as in obesity⁶⁹. Positron emission tomography (PET) scans have shown that there is a reduction in striatal dopamine D2 receptor availability in pathologically obese subjects similar to what has been observed in drug-addicted subjects^{55, 84, 85}. With the anticipation and ingestion of food, there is an increase in the extracellular level of dopamine in the nucleus accumbens, as there is with other substances of abuse. Since dopamine D2 receptors mediate reinforcing responses, obesity can be viewed as a “reward deficiency syndrome”^{85, 86}. In order to compensate for deficiencies in the dopaminergic system, individuals will engage in behaviors that increase brain dopamine levels, namely ingestion of more food or other substances that increase dopamine. This would then stimulate the mesocorticolimbic circuits of the brain, which are involved in behavioral reward and reinforcement⁸⁴.

Appetitive drive and drug craving are hypothesized to share overlapping and interactive pathways⁸⁷. Several sites in the hypothalamus are involved in regulation of food intake and energy expenditure⁸⁸. This circuitry that regulates appetite is under feedback control of hormonal signals, anorexigenic leptin from white adipose tissue and orexigenic ghrelin from the stomach. Studies in rodents have revealed that drugs of abuse share one or more components of the same hypothalamic circuits that affect appetite. Similar pathways may be involved with appetitive drive in response to decreased energy stores in the body as in drug craving during withdrawal in humans⁸⁷. More specifically, some brain signaling proteins that mediate excessive eating and weight gain may also mediate uncontrolled alcohol drinking⁸⁹. Both melanocortin agonists and neuropeptide Y (NPY) have been extensively studied and are prime targets that could link alcoholism and obesity. Melanocortin (MC) agonists, known to inhibit food intake and cause reduction of body weight in animal models, may also be involved in alcohol consumption⁹⁰. Studies with melanotan-II (MT II), a non-selective MC agonist that mimics the effects of melanocortins at specific cellular receptors, have shown to decrease both voluntary alcohol drinking and food intake in mice that habitually drank large amounts of alcohol^{91, 92}. Neuropeptide Y is a signaling protein that stimulates feeding, interacts with the satiety hormone leptin, and appears to be an important regulator of alcohol intake⁹³. In one study, mice lacking one receptor subtype for neuropeptide Y drank significantly more alcohol than normal mice⁹⁴. It has also been hypothesized that NPY is involved in mediating the anxiety that occurs during the alcohol withdrawal syndrome⁹⁵. Other peptides such as corticotropin releasing factor and cholecystokinin (CCK) have been associated with reductions of food intake, while others such as galanin and ghrelin found to stimulate feeding⁸⁹. Current research is directed at determining if these peptides regulate voluntary ethanol intake as well, since both food and ethanol intake are consummatory behaviors and have rewarding properties.

Leptin is another well-studied peptide hormone that may be a shared physiologic substrate of alcohol and food intake⁹⁶. Leptin, which has been implicated in the regulation of appetite and expenditure of energy, is also associated with increased craving for ethanol. Plasma levels of leptin have a positive correlation with lifetime alcohol use in male alcoholics^{96, 97}. In addition, recent research indicates that leptin may act through cross-talk with the endogenous cannabinoid system in the hypothalamus, which in turn regulates energy homeostasis, fluid balance, and stress response⁹⁸.

Endogenous cannabinoid receptors in the brain act primarily to modulate other neurotransmitter systems, including amino acids (e.g., GABA, glutamate) and monoamines (e.g., dopamine, serotonin, and noradrenaline;^{99, 100}. Decreased levels of leptin may lead to increased endocannabinoid activity in the hypothalamus, which may lead to increased eating behavior^{101, 102}. Further, behavioral studies indicate that endocannabinoids may increase motivation to eat see¹⁰³ for review. Endocannabinoids also may indirectly modulate (through GABA and glutamate) dopamine activity in the nucleus accumbens, the brain's "reward system" described above¹⁰⁴. Development of an endocannabinoid receptor antagonist has shown efficacy for weight loss and related cardiovascular health problems compared to placebo in several large phase III clinical trials of overweight adults¹⁰⁵ but no research has examined this in children or adolescents.

The brain's opioid systems also have been implicated as a common neural substrate for food and drug reward. It has been proposed that opioid neurotransmission and stimulation of the mu opiate receptors within the nucleus accumbens and ventral stratum induce a marked increase in food intake, sucrose, salt and ethanol intake¹⁰⁶. The nucleus accumbens and its circuitry are postulated to subserve motivated behaviors, such as feeding, drinking, sexual behavior, and incentive learning¹⁰⁷⁻¹⁰⁹. This system has been frequently implicated in drug addiction¹¹⁰⁻¹¹².

There also is overlap in the theoretical underpinnings of addiction and obesity in terms of impulsivity, decision-making ability, and negative self-awareness/avoidance¹¹³⁻¹¹⁵. In both conditions, researchers have demonstrated elevated levels of trait impulsivity, defined as novelty and sensation seeking¹¹⁶. Studies of reward sensitivity have shown that, just as alcohol abusers demonstrate more sensitivity to the reinforcing effects of alcohol, adolescent girls who engage in binge eating shown greater sensitivity to the rewarding qualities of food when compared to girls who restrict food intake¹¹⁶. Additionally, a study of 41 adult women that utilized a gambling task to assess decision-making ability found that women with higher BMI's showed more decision-making impairment compared to those with healthy BMI's; specifically, the choices made were based on rewards with negative consequences having little effect¹¹⁴. Research using this same gambling task with substance abusers shows similar impairments in judgment¹¹⁴. It is hypothesized that individuals who show poor decision-making skills will be more likely to engage in behaviors that provide immediate reward and show a disregard for future consequences of behaviors¹¹⁴. While the construct of impulsivity has been explored and established in both addiction and obesity, the distinction between compulsive versus impulsive behaviors regarding obesity and addiction has not been fully explored.

Some researchers have proposed a unidimensional continuum with compulsivity and impulsivity at opposite ends as a way to help characterize psychiatric disorders^{117, 118}. For example, "compulsive" disorders (such as obsessive-compulsive disorder) are characterized by harm avoidance and risk aversiveness, while "impulsive" disorders (such as pathological gambling and bulimia nervosa; BN) are characterized by increased risk taking, increased sensation-seeking, and weak harm avoidance^{119, 120} employed this conceptualize to differentiate self-injurious behaviors (SIB) into "compulsive" behaviors (i.e., hair pulling, skin picking, severe nail biting), which are habitual and repetitive, and "impulsive" behaviors (e.g., skin cutting and burning) which are episodic, gratifying, impulsive responses to stressful triggers. Other researchers conceptualize impulsivity and compulsivity as two separate and distinct, but often co-existing, traits¹²¹⁻¹²³. Supporting this conceptualization is the high comorbidity for obsessive-compulsive and impulse-control disorders^{124, 125}.

Eating disorders are an example of this comorbidity, as patients with BN and binge eating disorders may exhibit both obsessive-compulsive and impulsive traits^{121, 126-129}. However, findings regarding correlates of compulsivity and impulsivity in this population are mixed. For example, Engel and colleagues were able to classify their sample of 204 females with BN into 4 groups based on high versus low scores on compulsivity and impulsivity. They found high impulsivity was related to substance abuse severity, regardless of level of compulsivity. Conversely, compulsivity was associated with more severe eating disorder behavior and depression¹²⁹. In contrast, Favaro and colleagues¹³⁰ reported that, in a sample of 554 patients with eating disorders, impulsivity was associated with higher levels of eating disordered behavior and psychiatric symptoms (including depression).

Neurobiological studies support the notion that impulsivity and compulsivity are a unidimensional construct in patients with BN. For example, the link between lower serotonergic activity has been associated with impulsivity^{131, 132} as well as compulsivity^{133, 134}. However, it is unlikely that serotonin is the sole neurobiological pathway through with compulsivity and impulsivity function. For example, dopamine also may play a role, as treatment with a dopamine agonist increases the risk of developing an impulse control disorder (gambling, shopping, sexual behavior) in patients with Parkinson disease¹³⁵.

The clarification of compulsivity versus impulsivity is not well defined in the substance abuse literature. A key diagnostic symptom of drug or alcohol addiction is compulsive drug

use, including losing control of use despite negative consequences of using¹³⁶. Volkow and Fowler¹³⁷ outline a model of addiction that defines addiction as compulsive behavior. However, while addiction is commonly diagnosed and recognized as a problem with compulsivity, impulsivity is often the trait that is associated with addiction in humans. For example, there is a large literature establishing that impulsivity predicts current and future substance use problems of all types^{113, 138-140}. Interestingly, there is very little exploration in the literature of compulsivity versus impulsivity and how they relate to type of substance use or patterns of substance use. Regardless of this lack of exploration, there is ample evidence to demonstrate shared neurobiologic and psychosocial commonalities associated with both food and drug reward. This overlap supports the notion that over eating (the behavior that leads to obesity and overweight) may respond to the same type of interventions that have demonstrated effectiveness in the treatment of substance abuse.

Treatments for pediatric obesity

In March 1997, professionals from the Maternal and Child Health Bureau, Health Resources and Services Administration, and the Department of Health and Human Services established guidelines for medical professionals caring for obese/overweight youth¹⁴¹. Guidelines state that treatment should focus on reducing caloric intake, increasing overall activity level, behavior modification, inclusion of parents and family members as active treatment participants, parent skill-training, and a focus on motivation and readiness to make behavior/dietary changes¹⁴¹. Despite these recommendations, there have been few large randomized clinical trials targeting pediatric obesity¹⁴². Furthermore, most of the research conducted has been in specialty obesity clinics with treatment-seekers¹⁴³, limiting the generalizability of findings. The vast majority of current treatment strategies for pediatric obesity have consisted of behavioral interventions for children and/or their families targeting one or more of the following: (1) changes in diet; (2) increases in physical activity; and (3) decreases in sedentary behavior. Less research has been conducted examining medical treatments for obesity, including medication and surgical intervention.

Behavioral Treatment Strategies

In a recent review, Stuart et al.¹⁴⁴ provide an in-depth analysis of treatment studies that targeted adolescent obesity and reviewed studies that met the following criteria: 1) were published between 1980 to 2003; 2) employed a minimum of 2 comparison groups, with an intervention designed to effect weight loss, and 3) enrolled participants between ages of 11 and 19 years. Initially, the authors intended to conduct a meta-analysis. However, similar to the conclusions reached by Summerbell et al.¹⁴³, a meta-analytic approach was rejected because the wide variability in independent variables in the studies. A total of 152 studies were identified and reviewed but most did not include a comparison or control group, included participants younger or older than the inclusion criteria or were not interventions for weight loss. For the seventeen studies that met the inclusion criteria, there were 831 participants, with 44% Caucasian, 38% African-American, 2.5% Latino/Hispanic, and 0.5% Asian American, and 15% other. The studies ranged from 12 to 20 weeks. The interventions varied from lifestyle changes with/without family member involvement, lifestyle changes with and without exercise, the use of monetary reward structures, telephone and mail-based behavioral intervention, and an intervention with metformin as an adjunct to behavioral therapy, diet and exercise¹⁴⁴. Most of the studies used group interventions (13 out of the 17), 2 studies used an individual, nongroup intervention for behavioral change^{145, 146} and 2 studies used physiologic interventions (metformin or low-carbohydrate/low fat diet;^{147, 148}. Another recent systematic review of approximately 100 articles by the American Dietetic Association⁶ organized the literature by dividing studies into treatments based at home (or with the individual child/adolescent) and school-based treatments. They concluded that the research showed a positive effects for multi-component, family-based programs in school-

aged children (5-12 years old), and a positive effect for multi-component, school-based programs for adolescents. Multi-component programs were defined as incorporating behavioral counseling, dietary counseling/nutrition education, enhancing physical activity and/or parent training ⁶.

Varying success has been found for diet regimens, such as the Stoplight Diet, the diabetic exchange system, and individual diet plans ¹⁴⁹. The ADA ⁶ reviewed 47 studies that used dietary counseling and/or as part of a multi-component intervention including behavior modification and/or physical activity. Overall, comprehensive approaches that focus on eating behaviors as well as physical activity are more effective than either waitlist or nutrition education control groups ^{6, 142}. However, in a study combining nutrition education with cognitive behavioral counseling, Kirschenbaum and colleagues ¹⁵⁰ reported significant decreases in weight and percent body fat in 9-13 year olds up to a year follow-up. Few studies have assessed the impact of dietary modifications alone and those few have reported mixed findings, making the unique contribution of diet (above and beyond the other components of treatment), difficult to establish. The unique contribution of diet was examined in 2 studies by Epstein and colleagues ^{151, 152}, but the results were inconsistent. In the first study, dietary counseling plus increased daily activity was compared to dietary counseling alone for 6 months. Both treatment groups demonstrated significant weight loss and reduction in percent body fat at discharge and 6 month follow-up, though there were no differences between the 2 conditions. In contrast, a subsequent study showed that dietary counseling plus structured exercise resulted in a significantly greater weight loss and reduction in percent body fat than dietary counseling alone (Epstein et al., 1985).

Exercise also may contribute to weight loss in children and adolescents ^{6, 153-156}. Of 24 studies reviewed by the ADA ⁶, only 1 ¹⁵⁷ failed to demonstrate significant benefits from adding an exercise or increased activity component to the treatment. Interestingly, programs that emphasize “life style” exercise (i.e., aimed at increasing energy expenditure in everyday activities) show greater long-term effectiveness than those with prescribed eating/activity ¹⁵⁸. Again, most studies have examined the impact of exercise/increased activity within the context of a larger, multi-component intervention, making it difficult to discern the impact of exercise alone. One study comparing exercise plus diet and behavior modification and exercise alone to a control group found that both treatment groups showed significant weight loss and reduction in percent body fat compared to the control group, though no differences were found between treatment groups ¹⁵⁹.

Regarding sedentary behavior, a 4-month plus 1-year follow-up study by Epstein and colleagues ¹⁶⁰, demonstrated that reducing sedentary behavior (television watching) was superior to increasing physical activity in helping children maintain weight loss. In addition, three primary prevention studies targeting decreased television watching resulted in decreases in BMI ¹⁶¹⁻¹⁶³. No studies were identified that addressed reduction in other sedentary behaviors, such as homework, reading, and/or computer use, as a prevention strategy for reduction of overweight in children.

The extent of parental involvement in treatment has varied across studies and findings have been mixed. Family-based interventions are those that target the behavior of multiple family members (usually parents and children; ¹⁶⁴. Kitman and Beech ¹⁶⁵ reviewed 31 family-based interventions and concluded that family-based interventions were effective at reducing weight and percent body fat in children and adolescents, relative to controls. In addition, 27 of 33 family-based interventions reviewed by the ADA ⁶ demonstrated significant reductions in children’s weight. In addition, parental weight loss was related to child weight loss in two studies ^{166, 167}. Kitzman and Beech ¹⁶⁵ also caution that most families that are able to engage in family-based interventions are likely to be more motivated to change and

have the resources to attend regularly scheduled appointments for a lengthy period of time. Indeed, higher family support have been associated with greater child weight loss, while higher levels of family conflict have been associated with decreased retention in treatment ¹⁵⁰. These findings suggest that stressed families with limited resources and/or poor parenting skills may need a more tailored approach including parent training and stress management ¹⁶⁵. In addition, Kitman and Beech ¹⁶⁵ point out that family-based interventions may need to be tailored for ethnic or racial minority families, as well. They cite differential effects for similar family-based treatments in a sample of White adolescent females ¹⁶⁸, compared with African American adolescent females ¹⁶⁹ of comparable SES; in White families, treatment groups in which mother and daughter remained separate lost significant more weight than those where they were together ¹⁶⁸, whereas the reverse was true in African American families ¹⁶⁹.

Support for long-term weight loss is less clear, with some family-based interventions demonstrating weight loss maintenance as far out as 5 and 10 year follow-ups ^{142, 160, 170-173}, and other studies not ¹⁷⁴; DISC Writing Group, 1995; ¹⁷⁵. In addition, a meta-analysis of 41 family-based interventions showed that parental participation in treatment did not increase the effectiveness of multi-component interventions in overweight children and adolescents ¹⁷⁶.

Not surprisingly, most of the interventions that have been assessed in a controlled fashion were delivered in specialty clinics, thus limiting treatment the generalizability for teen minorities of low SES, who are disproportionately affected by obesity. Several studies have addressed this issue by delivering treatment in settings that teens frequent. For example, several studies have shown that teen weight loss interventions can be feasibly delivered within school settings, though the impact of these programs on weight loss for children and teens is mixed ^{148, 153, 177-181}. In addition, it may not be feasible for many schools to continue offer these interventions due to the high cost of hiring specialized instructors from the community or training current physical education instructors in these interventions. Additionally, many of these interventions take place at least partly within designated physical education periods. Given the significant decreases in time and funding allocated to physical education in public schools, it is unlikely that these limited resources will be used to support a specialized program for a relatively small portion of students. It also is important to note that there exists potential for stigmatization of overweight youth who participate in school-based interventions, as they are singled-out for special treatment due to their weight ⁶.

Enhancements to Behavioral Treatment Strategies

Problem-Solving—Results for incorporating problem-solving strategies into treatment are promising, but few. One study demonstrated that the addition of problem-solving to a family-based, multi-component intervention for 5-12 year olds significantly enhanced weight loss and maintenance at 3 and 6 month follow-up ¹⁸². However, a subsequent study by Epstein and colleagues ¹⁸³ found no differences in weight loss at 6 month follow-up when problem solving was added to parent/child behavioral counseling or child-only behavioral counseling. Interestingly, children's BMI scores were significantly lower in the parent/child problem-solving compared to the child-only problem-solving and no problem-solving groups at a 2-year follow-up. These findings suggest that problem-solving may have long-term benefits, but have only shown efficacy when parents are included in the intervention.

Motivation—Motivational Interviewing (MI) is a style of behavior counseling that is designed to help increase one's motivation to change. Initially developed for treatment with

alcohol abusers, MI has been shown to be effective in addressing drug abuse, smoking, HIV risk, eating disorders, Type II Diabetes, and obesity in adults^{184, 185}. However, few studies using MI have focused specifically on weight loss, and none have done so for youth. Motivational interventions have been shown to be effective in reducing cigarette smoking¹⁸⁶, alcohol use¹⁸⁷, and cannabis use in teens¹⁸⁸. Given that teens are difficult to retain in treatment, MI may be effective at engaging them and reducing drop out. In addition, using an MI platform may allow us to increase the potency of the traditional CBT techniques. Project MATCH, the largest, multi-site, NIH clinical trial to date¹⁸⁹ concluded that 4 sessions of MI were as effective as 12 sessions of traditional CBT or 12-step facilitation in reducing addictive behavior (all treatment groups showed significant improvement; Project MATCH^{190, 191}). Such results highlight the ability of MI to deliver therapeutic effects in a time limited and cost efficient manner, as this treatment is equally powerful to other longer, traditional treatment approaches.

Technology—Computer-delivered interventions have been shown to be as effective as therapist-delivered interventions¹⁹²⁻¹⁹⁵. These interventions can be used repeatedly, with the majority of costs going towards a one-time purchase of hardware or software; in contrast, clinician-delivered interventions have a fixed cost per use, with additional costs associated with therapist training, etc., thereby making computerized interventions cheaper relative to therapist/ educator-delivered interventions¹⁹⁶. In addition, technology is easily exportable and, when exported, the intervention's fidelity is assured. Such programs may be less threatening to teens and also may be viewed as providing greater anonymity, particularly important among teens when sensitive issues are addressed. Another advantage of computer-based interventions is that they require active responding and learning by the user, as opposed to passive learning associated with listening to an educator/therapist, watching a video, or reading educational materials. Furthermore, computer-based interventions can be modified to accommodate new information as it becomes available. Finally, computer-based interventions may allow patients to engage in educational activities and interventions for a greater period of time than would be possible with an educator or counselor alone¹⁹⁶. In sum, a computer driven intervention is sparing of both money and staff time, and is an individual intervention on a platform with a natural appeal to teenagers. Furthermore, adolescents may prefer a technology-based intervention to traditional interventions that involve a doctor or counselor 'telling them what to do'. In a sample of over 3,000 adolescents, 89% reported that they preferred computerized assessment and intervention when compared with paper-based or face-to-face interview assessment and intervention for health behaviors¹⁹⁷.

Despite these obvious advantages, few technology interventions have addressed behavioral health problems in youth, including overeating and sedentary behavior. One study examined brief multi-media computer-delivered CBT intervention for teens with disordered eating showed a reduction in binge/purge behavior^{198, 199}. In addition, three clinical trials have examined technology to promote weight loss in teens. The first employed the internet to teach CBT techniques and provide education to overweight African-American adolescent females and their parents. Investigators found that teens in the CBT group accessed the website more frequently and lost more weight than teens randomized to an education only condition²⁰⁰. The intervention was completed at the teens' home, making it accessible. However, each teen was provided with a personal computer and internet access for the duration of the study, greatly limiting the feasibility of the treatment in real world settings. The second and third studies examined a combined internet/video intervention to deliver either a 4 session²⁰¹ or 8-session²⁰² health promotion/ transtheoretical (HP/TM) intervention to low SES, primarily minority middle-school students. The HP/TM combined some motivational techniques, including personalized feedback tailored to a teen's stage of change, with psychoeducation. The intervention was primarily video-based, with

personalized feedback provided via e-mail and on on-line workbook. Sessions were completed during a computer class at school. For teens who completed at least 4 of 8 sessions, the intervention was effective in increasing activity, decreasing dietary fat intake, and enhancing motivation to change ²⁰². Unfortunately, no measures of weight or percent body fat were completed on these teens, so it is unclear whether these changes translate to decreased obesity in the sample. While the evidence for technology interventions to promote weight loss in teens is promising, the few existing studies have significant limitations including lack of real-world feasibility and lack of assessment of obesity.

Medical treatments for obesity

Aside from behavioral treatments, options for youth are limited. Currently, no weight loss medications have been approved by the Food and Drug Administration for use in children under the age of 16 ²⁰³, although several studies have employed medications previously shown to be efficacious in overweight adults. In addition, although no studies of gastric bypass surgery have been conducted in adolescents, reviews of the database of bariatric surgeries performed on adolescents over the past 20 years suggest outcomes similar to those found for adults ^{204, 205}. While gastric bypass surgery remains an option, experts state that, for children, it should be considered a “last resort” for those who have failed other treatment modalities ^{204, 205}.

Medication—To date, few drugs have been approved by the Food and Drug Administration to treat obesity in adults and the 2 most widely used in clinical practice are sibutramine and orlistat ²⁰⁶. In addition, a recent Cochran review ²⁰⁷ noted that the only two medications with adequately controlled trials to support their efficacy thus far were sibutramine and orlistat. While no weight loss medications have been approved by the FDA for use in children under the age of 16 ²⁰³, several studies employing medications efficacious in overweight adults have also shown promise for use in adolescents. The research examining the impact of medication on weight loss in overweight children is minimal ²⁰⁸.

Medication in overweight adults—Sibutramine is an appetite suppressant that functions by inhibiting the reuptake of noradrenaline and serotonin ^{209, 210}. Sibutramine contribute to significant weight loss within the first 6 months and effects have been documented as far out as 2 years ^{211, 212}. Sibutramine also shows promise for decreasing related cardiovascular risk factors and improving glucose control in adults with Type II diabetes ^{213, 214}. Unfortunately, because sibutramine may increase heart rate and blood pressure, its use is contraindicated for patients with some medical co-morbidities of obesity – uncontrolled hypertension, cardiac dysrhythmias, congestive heart failure, and stroke ²¹⁵.

Orlistat reduces the amount of fat absorbed during digestion by inhibiting gastric and pancreatic carboxylester lipases, reducing the hydrolysis of the digested triglycerides ²¹⁶. Orlistat has been shown to contribute to significant weight loss and decreases weight regain at 2-year follow-up ²¹⁷. In addition, like sibutramine, it has been shown to help decrease high cholesterol and blood pressure ^{218, 219}. The most common side effects of Orlistat are related to fat malabsorption and include loose stools, fecal urgency, and fat soluble vitamin deficiencies ²¹⁷.

Medication in overweight children and adolescents—Two well-controlled (placebo-controlled double-blind) studies have shown promise for sibutramine in overweight adolescents. The first study of 82 adolescents receiving behavior therapy plus sibutramine versus placebo showed a significantly greater reduction in percent weight loss for the sibutramine group ²⁰³. However, the medication was reduced or discontinued in

approximately 40% of the teen participants due to hypertension and other side effects. In addition, the adolescent participants in the study did not demonstrate the other medical gains found in adults; no differences were found between the groups on cholesterol, triglycerides, serum insulin, glucose, or insulin sensitivity²⁰³. A more recent study of 60 adolescents demonstrated significantly greater weight loss in the sibutramine group versus placebo, with no adverse effects reported²²⁰. Reisler and colleagues²²¹ examined sibutramine as an adjuvant to diet and exercise in a sample of 20 adolescent females with morbid obesity. Most patients showed early weight loss (at 3 and 6 months), but stopped losing weight in the following 6 months. During the follow-up period, 17 patients discontinued treatment, primarily due to the slow in weight loss in the second 6 months of treatment. A recent, large, multicenter trial examined sibutramine versus placebo in 498 obese adolescents, 12-16 years old, enrolled in a behavior therapy program²²². Adolescents treated with sibutramine showed significant decreases in BMI at month 12, compared to those treated with placebo. Unfortunately, all of the trials conducted thus far have been of limited duration that has not allowed for the examination of long-term weight maintenance or health effects. In addition, three of four trials demonstrated poor feasibility for maintaining adolescents on the medication long-term. Finally, the few extant medication trials have examined only adolescents; none have looked at safety or efficacy in overweight children.

Several studies support the use of orlistat to significant reduce weight in overweight children (Norgren et al., 2003) and adolescents²²³⁻²²⁵, though one study of 40 overweight adolescents found no difference between teens receiving orlistat and those receiving placebo²²⁶. Unfortunately, these studies only examined outcomes as far out as 6 months. In a large (n=539 with 349 treatment completers), well-controlled trial in overweight adolescents, those receiving orlistat showed significant decreases in weight, BMI, and waist circumference compared to those receiving placebo at 54 weeks²²⁷, suggesting that long-term weight loss in adolescents may be possible with orlistat. It is important to note that orlistat reduces the absorption of fat-soluble vitamins (e.g., Vitamin D); therefore adolescents prescribed orlistat should be monitored and receive vitamin supplements as part of their treatments.

Metformin may be another medication that holds promise for teens. Metformin is a hypoglycemic agent used commonly in the treatment of type 2 diabetes in adults. Metformin has demonstrated effectiveness in treating many of the medical problems associated with obesity in pediatric populations, including diabetes and insulin resistance^{228, 229}, polycystic ovary disease²³⁰⁻²³², and fatty liver disease²³³. Four randomized, controlled trials in overweight adolescents indicate that metformin contributed to significant reductions in BMI and improvement in related medical problems, including serum glucose, insulin and lipid profile up to 6 months^{147, 234-236}. Unfortunately, poor adherence was reported in several of the trials.

Surgical intervention—Bariatric surgery may be a treatment option for some adolescents with morbid obesity or overweight adolescents with weight-related medical problems^{237, 238}, AAP²³⁹. The gastric bypass is the most frequently used surgical intervention for morbid obesity, commonly leading to a 25–35% loss of body weight and showing promise for long-term maintenance of clinically significant weight loss²³⁸. In addition, gastric bypass is often performed using minimally invasive laparoscopic techniques²³⁸. Although few controlled trials of gastric bypass surgery have been conducted in adolescents, reviews of the literature on overweight adolescents suggest outcomes similar to those found for adults^{204, 205, 240}. Sugerman and colleagues²⁰⁴ reported that 20 morbidly obese adolescents demonstrated an average 36% reduction in BMI at 5 years post-surgery, and maintained their weight loss for up to 10 years following surgery. Similarly, Rand and McGregor²⁴¹ conducted a 6-year follow-up with 34 adolescents who had bariatric surgery

and found that they lost an average of 66% of their excess weight. However, the disadvantages of gastric bypass surgery include potential deficiencies of iron, calcium, and B vitamins as well as a 0.5% risk of death following operation. These disadvantages may be significant in young people post gastric bypass surgery; in Rand and McGregor's ²⁴¹ sample, medical non-adherence at follow-up was prevalent. They reported that only 13% of patients were taking their nutritional supplements as instructed, while 97% were snacking between meals and 76% exercised once a month or less. In addition, 15% of patients were unable to maintain initial weight loss due to poor dietary behaviors.

Given the post-surgical risks, bariatric surgery for overweight adolescents should be considered carefully. Inge ²³⁸ recommends that adolescents should be evaluated by an experienced, multidisciplinary bariatric team who are able to provide long-term follow-up. He recommends that teams include specialists in pediatric psychology, nutrition, exercise, and bariatric surgery, as well as adolescent medicine, endocrinology, pulmonology, and gastroenterology.

Treatment Recommendations

Over the past several decades, pediatric overweight has emerged as a major public health concern ²⁴². Being overweight is associated with a host of medical, behavioral health, and psychological problems and is more prevalent among economically disadvantaged and minority children and adolescents. While these demographics suggest the need for community outreach in settings frequented by low SES and minority youth, most treatment occurs within the confines of specialty clinics that are inaccessible to those who need them most. Given the need to reach a larger portion of overweight children and adolescents, we recommend that services be provided in more accessible settings. For example, pediatricians' offices would be a logical setting in which to deliver a pediatric obesity intervention.

Current reviews of the literature show that multi-component treatments are effective for overweight children and adolescents. Effective components of treatment generally include dietary counseling, increasing physical activity, decreasing sedentary behavior. Unfortunately, multi-component interventions are lengthy, expensive, and require extensive physician time and expertise making implementation in many settings impractical. Providing concrete, realistic strategies to parents and their children may be beneficial. For example, Kirk and colleagues ²⁴³ suggest helping children and teens modify their diet by limiting the availability, frequency, and amount of high-fat or high-sugar foods and drinks consumed; helping children and teens to eat smaller portions at regularly scheduled times, limiting seconds to fresh fruits and non starchy vegetables, drinking more water and sugar-free beverages, and choosing food that has been baked, broiled, grilled or boiled instead of fried. They also recommend between 30-60 minutes of physical activity, which may be more easily achieved if the activity is fun, noncompetitive, and part of their daily routine. In addition, decreasing sedentary activities, like TV watching or video game playing may be helpful. In addition, there is strong evidence to suggest that parental involvement is important in helping overweight children and teens lose weight. Because of this, it may be important to engage parents and provide education and support about using these strategies to change the dietary and activity habits of the entire family.

The parallels between obesity and addiction also may provide guidance in effective treatments. For example, employing motivational strategies to engage children and families in treatment may be helpful. Similarly, cognitive-behavioral therapy techniques that teach children and families to identify the triggers to their bad eating habits may help them to problem-solve ways to eat healthier or exercise.

In addition to behavioral intervention, there is evidence that medication may serve as a helpful adjuvant to treatment for overweight children and adolescents. If medication is used, it is important to inform parents and children about potential side effects and monitor children and adolescents closely. Gastric bypass surgery also may be an option for morbidly obese adolescents or for adolescents with weight-related medical problems. When considering surgical intervention, it is important to consider the risks and adolescents should be carefully screened and evaluated by an experienced, multi-disciplinary team before proceeding.

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