Obesity: What Mental Health Professionals Need to Know

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Objective: Obesity is a highly prevalent condition with significant health implications. This report summarizes recent clinically relevant findings concerning the pathogenesis and treatment of obesity and considers their implications for psychiatric diagnosis and management.

Method: The authors conducted selective reviews of the literature from the last 10 years. Topics included the biological and behavioral factors that contribute to the onset and maintenance of obesity, the relationship between obesity and psychiatric illness and treatment, and the questions of whether and how obesity should be treated.

Results: Genetic effects, some mediated by eating behavior, contribute importantly to the potential for obesity, the expression of which is promoted by environmental factors that increase the availability of calorically dense foods and discourage activity. There appear to be behaviorally distinct subsets of obese persons who display particular patterns of disordered eating and elevated rates of psychopathology. Treatment with psychotropic medications may contribute to obesity in ways that are only partly understood. Although successful obesity treatment is associated with clear health benefits and available treatments offer benefit to some, relapse remains the rule.

Conclusions: Although the presence or development of obesity is a daunting problem, it should not be ignored by mental health professionals. Treatment should address not only obesity per se, but also its effects on self-esteem in a hostile cultural climate. Ongoing developments in basic and clinical research are likely to increase the range, efficacy, and acceptability of treatment options in the years ahead.

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Obesity is among the most easy to recognize and the most difficult to treat of medical conditions. Although most mental health practitioners commonly treat obese individuals, there exists no consensus on whether or how to address obesity in the patient's overall treatment plan. This article represents the attempt of the three authors—a psychiatrist (M.J.D.), a clinical psychologist (G.T.W.), and a family physician (S.Z.Y.)—to identify the questions that mental health professionals face when working with obese patients, to address these questions to the extent that the evidence allows, and to direct the interested reader to sources of more detailed information on scientific and clinical aspects of obesity. The article is based on a selective review of the literature from the last 10 years. Recent reviews detailing the basic science of weight regulation (1) and clinical aspects of weight management (2, 3) provide further information for interested readers. Patient-oriented information about obesity is also available through the Weight-Control Information Network of the National Institute of Diabetes and Digestive and Kidney Diseases at http://www.niddk.nih.gov/health/nutrit/nutrit.htm.

What Constitutes Obesity, and How Common Is It?

Obesity is defined best in terms of what is known as the body mass index, calculated by dividing weight (in kilograms) by height (in meters) squared. Recent clinical guidelines developed by the National Heart, Lung, and Blood Institute define "overweight" as a body mass index of 25–29.9 kg/m^2 and obesity as a body mass index of 30 kg/m^2 and greater. Body mass index of 30–34.9 kg/m^2 is classified as class I obesity, 35–39.9 kg/m^2 as class II obesity, and 40 kg/m^2 or more as class III or extreme obesity (2). The institute's definition of overweight is consistent with recommendations of the World Health Organization (3) and health agencies in most countries outside the United States and is grounded in epidemiological data showing increases in morbidity and mortality with body mass indexes above 25 (2). Using this classification, it is estimated that approximately 55% of adults in the U.S. are overweight, and about one-half of these are considered obese (4). Obesity is inversely associated with social class (5). Minority populations, especially African American women, are disproportionately affected. The percentage

of individuals in the "overweight but not obese" category (body mass index of 25.0–29.9 kg/m^2) has remained relatively stable over the past 30 years, whereas the prevalence of obesity has increased more than 50% between 1976 and 1980 (14.5%) and 1988 and 1994 (22.5%), with a consequent reduction in those meeting criteria for "normal weight" (4).

What Is the Impact of Obesity on Individuals and on Society?

Obesity is a chronic disorder and a serious public health problem. The literature has presented well-documented links between obesity and increased mortality and morbidity due to hypertension, dyslipidemia, diabetes mellitus, coronary heart disease, congestive heart failure, stroke, gallstones, osteoarthritis, sleep apnea, certain types of cancer (colon, breast, endometrial, gall bladder), menstrual abnormalities, impaired fertility, and increased pregnancy risks (2). Overall mortality is modestly increased for overweight individuals (body mass index of 25–29.9 kg/m^2) and markedly increased for those with body mass index over 30 kg/m^2, particularly those with severe obesity (6). Among older adults, mortality is less strongly linked to body mass index (7), but body mass index in the obese range remains associated with elevated mortality (8). In addition to degree of obesity, excess fat in the abdominal region, estimated by measuring waist circumference or waist/hip ratio, is independently associated with increased morbidity and mortality, particularly for overweight and class I obese individuals (2).

The adverse effects of obesity are not only medical. In Western societies, negative attitudes toward obesity are prevalent in society at large and among health care professionals. These attitudes translate into tangible disadvantages in several common endeavors, including getting into college, renting a residence, and getting married (2). The economic impact of obesity is also considerable. For an individual, obesity is associated not only with increased health care expenditures but also with decreased job earnings (9). Estimates of the total cost of obesity in the United States, including direct (health care) and indirect (lost productivity) costs, amount to nearly $100 billion per year (10).

What Causes and Maintains Obesity?

Obesity is best conceptualized as a complex, multifactorial disorder with both genetic and environmental components.

Genetics of Obesity

Single-gene animal models of obesity have led to the identification of several gene products that are associated with obesity, including leptin, the leptin receptor, agouti signaling protein, and carboxypeptidase E (11). Of particular importance, the mutated gene products in these animal models have human homologues that appear to have similar functions, suggesting that these proteins may also play roles in the development or maintenance of human obesity.

Numerous central and peripheral factors that increase or decrease food intake and energy expenditure have been identified, and more such factors are being discovered on a regular basis (1). Uncoupling protein was described decades ago as a ubiquitous component of brown fat, where it serves to dissipate energy as heat. Because adult humans have little brown fat, its role in human energy regulation was assumed to be minimal. The recent discovery of novel uncoupling proteins that are found in many human tissues, including white adipose tissue (uncoupling protein 2) and muscle (uncoupling protein 3) (12), offers a new pathway by which energy regulation might occur, and the potential for therapeutic manipulation has generated an explosion of research. Another target of human and animal research is the β_3 adrenergic receptor. Preliminary human studies suggested that a variant in this receptor might be associated with lower resting metabolic rate, earlier-onset diabetes, and greater degree of obesity (13, 14), and β_3 adrenergic agonists are an active target of drug development.

Although recent discoveries of novel mechanisms for energy regulation are exciting, single-gene mutations are unlikely to account for the majority of human obesity. Cases have been reported of humans with mutations of several of the genes involving animal models of obesity (11). In addition, a recently described mutation in the gene coding for peroxisome-proliferator-activated receptor 2 (a regulator of adipocyte differentiation) may be more common in obese individuals (15). However, the vast majority of obese humans do not show major mutations involving known pathways leading to obesity. For example, leptin levels in most obese humans are high and correlate extremely well with body fatness (16). Although single-gene mutations as causal factors in obesity are probably rare, a number of studies have implicated linkage between certain sites housing candidate genes believed to be involved in animal models of obesity and phenotypic characteristics such as body mass index or percentage of body fat (17–19). Animal models that more closely represent human obesity, such as mice that become obese only when exposed to a high-fat diet, have also been developed (20). Although genetic variants may not lead to major alterations in the protein product of the gene, they may nonetheless, either singly or in combination with other genes, have an impact on the protein product's function.

The complexity of the genetics of obesity means that simple answers about its causes are unlikely. However, basic research findings identifying pathways that regulate food intake, adipocyte differentiation, and energy expenditure are likely to assist in the development of effective strategies for both prevention and treatment of obesity.

Drugs could be developed to block the effects of neuropeptides and receptors stimulating food intake, to enhance the effects on food intake or energy expenditure of factors involved in the catabolic pathway, or to alter the pathways involved in the differentiation or metabolism of adipocytes.

Behavioral Genetics

Often, "genetic" causes of obesity are contrasted with those seen as "behavioral," implying that only factors such as metabolic rate or propensity to gain weight as fat are under genetic control. Behavioral genetics refers to the contribution of genetic variability to relevant behaviors (eating and physical activity). Behavioral factors such as dietary preference for fats, choice of interval between meals, degree of caloric compensation in response to food restriction, or even inclination to engage in physical activity can have strong genetic components (21–23). Genetically determined preferences may interact with environmental factors to yield conditioned eating patterns (24). Unfortunately, research in this area has lagged behind investigations of some of the more easily measured phenotypic characteristics, such as metabolic rate or percentage of body fat (23). The recognition that certain behaviors may have a genetic basis has important implications for targeting behavior change. Environmentally triggered eating behaviors should be much more responsive to environmental modifications than those with a genetic basis. The degree to which certain behaviors are genetically or environmentally based may vary both between individuals and between populations.

Our "Toxic" Environment

Adoption, twin, and family studies have consistently noted that obesity has a strong heritable component, with approximately 30%–70% of variability in body weight or fat mass being genetically determined (25, 26). This is considered a major genetic effect, comparable to the genetic contribution in diseases such as schizophrenia, coronary artery disease, and breast cancer (27). However, it leaves a large component of variation in body weight unexplained by genetic mechanisms. Environmental factors account for this component. Of interest, studies have suggested that most of the familial resemblance for body mass index in adults is due to genetic influences rather than shared family environment (25).

Obesity has increased dramatically over the past decade in the United States, as well as in most of the developed world. The most likely explanation is that our population has been exposed to dramatic environmental changes over the past 30 years and that these changes have acted together to create what has been called an obesity-promoting "toxic" environment (28). Throughout most of human history, starvation, rather than obesity, has been the predominant threat to survival, with a resulting evolution of a "thrifty" phenotype that is energy-efficient and fat-loving (29). What serves as a genetic advantage in a low-energy, high-physical-activity environment can lead to obesity with the ready availability of high-fat, high-calorie foods coupled with significant decreases in physical activity. The case of the Pima Indians provides a good example of environmental effects on a given genetic background (30). Pimas living in Arizona have been compared with distant relatives living in Mexico who are much more physically active and eat a diet 50% lower in fat. The Mexican Pimas averaged 57 pounds lighter than their Arizona counterparts, and few had diabetes (30).

Another more salutary environmental trend contributing to increased rates of overweight and obesity in the United States is the decline in the prevalence of smoking in recent years. Smoking cessation is often accompanied by weight gain that reflects a combination of decreased metabolic rate and increased caloric intake. Possible mediators of the effect of smoking and smoking cessation on weight include 1) elevation of plasma leptin levels due to smoking (31); 2) nicotine-associated increase in hypothalamic leptin receptor sensitivity, which reverses after smoking cessation (32); and 3) compensatory increase in the activity of adipose tissue lipoprotein lipase among smokers, which persists in the short term after quitting (33). One-quarter of the increase in prevalence of overweight for men in the past 10 years and one-sixth of the increase for women is attributable to smoking cessation (34). Nonetheless, the health benefits of smoking cessation far outweigh the negative consequences of weight gain, and weight gain can be minimized if smoking cessation is accompanied by a moderate increase in exercise (35).

Do Obese People Eat More or Are They More Metabolically Efficient?

Although genetic differences in metabolic efficiency are likely to exist, there is little evidence that low metabolic rate plays a major role in the development or maintenance of obesity for the vast majority of overweight persons, suggesting that the development and maintenance of obesity is indeed mediated by the consumption of a greater than normal amount of food.

Eating Behavior and Metabolism

The study of human eating behavior is methodologically difficult. Less intrusive methods, such as patient self-report or 24-hour dietary recall, rely on subjects' often inaccurate estimates, and more objective methods, such as observed eating in a laboratory setting, run the risk of being unrepresentative of the subject's unobserved eating. Early studies that relied on these methods suggested that many overweight individuals ate very little (36). This "diet resistance" was often attributed to slowed metabolism due to frequent dieting or weight cycling (37). Studies using the highly accurate doubly labeled water method in adolescents (38) and in adults (39) confirm that over-

weight individuals tend to underreport their food intake and overreport their physical activity, reporting only 60%–80% of actual caloric intake, in contrast to normal weight subjects, who report 80%–100% (39, 40).

Overweight people generally consume more calories than people of normal weight, but this does not mean they are overeating, as they are consuming an appropriate amount of calories for their higher body weight (assuming weight stability). This distinction is important, as the idea that obese individuals bring about or maintain their obesity by inappropriate overeating underlies many of our culture's negative stereotypes about obesity.

Apart from the amount of food consumed, patterns of food consumption are abnormal in subgroups of obese persons. Stunkard (41) first described a distinctive subgroup of obese patients who reported recurrent uncontrolled binge eating. Many of these patients would meet the criteria for binge eating disorder found in Appendix B (Criteria Sets and Axes Provided for Further Study) of DSM-IV in that they displayed regular binge eating in the absence of the extreme weight loss behaviors characteristic of bulimia nervosa. Compared with non-binge-eaters, obese binge eaters eat significantly more food in laboratory studies when instructed to binge or eat normally, report an earlier onset of obesity and greater percentage of lifetime on a diet, overeat more in response to negative emotional states, report lower levels of self-esteem, and display significantly greater levels of psychopathology, especially depression and personality disorders (42–46).

Another abnormal eating pattern that exists most commonly in obese individuals has become known as the "night-eating syndrome." First described in 1955, its key features are morning anorexia, evening hyperphagia, and insomnia (47). More recent clinical reports have suggested that many of these patients suffer from sleep disorders such as somnambulism, restless legs syndrome, and obstructive sleep apnea (48) and that most patients report some degree of amnesia for the eating episode (49). In addition, night-eating syndrome is associated with neuroendocrine abnormalities including attenuated nocturnal rise in leptin and melatonin and increased plasma cortisol (50). Further research is needed to better characterize the clinical features and treatment responses of these individuals.

Metabolic Factors

Energy regulation in persons who are already obese is not necessarily the same as energy regulation in those who are developing obesity. There is some evidence that certain individuals in a given population may be more metabolically efficient than others, which could lead, in time, to weight gain. Twin studies have shown a strong familial response to overfeeding and underfeeding, both in the amount of weight gained as fat and in distribution of body fat (51, 52). Thus, there are clear metabolic differences in propensity for weight gain in response to caloric excess or deficit. A recent study, in which volunteers were overfed by about 1000 kcal/day, suggested that differences in propensity to gain weight in the presence of caloric excess could be due, in part, to increases in energy expenditure from everyday activities, such as walking, maintenance of posture, and fidgeting (53).

Weight Regain

Why is long-term weight loss so difficult to achieve? The degree to which obese individuals who lose weight become more metabolically efficient remains unresolved. The concept of a "set point for energy balance," first described by Keesey and Hirvonen (54), as a predetermined point at which a given individual's body weight is regulated, is an attractive one. Caloric restriction clearly increases energy efficiency over the short term, decreasing leptin and thyroid hormone levels as well as resting metabolic rate to a level lower than that predicted by decreases in lean body mass (55, 56). Differences may also exist in physical activity-related energy expenditure after weight reduction (57). Over the longer term, however, most evidence of increased conservation of energy dissipates, with resting energy expenditure levels appropriate for the new (lower) level of body weight (58).

Thus, although metabolic factors that promote weight regain may exist, they do not seem to explain satisfactorily the almost inevitable weight regain observed clinically (59). The observed 5%–10% change in energy expenditure (57) is relatively small, and compensation either by caloric restriction or increases in physical activity should be possible. Moreover, it should be noted that increases in feeding lead to increases in energy expenditure, which would be expected to assist in the overfed subjects' return to a lower body weight. Thus, any "set point" is frequently overcome—at least in the direction of allowing weight gain.

It has been suggested that postobese persons—those who have lost weight—are metabolically different from never-obese persons. Potential contributors to weight regain include the presence of fat cell hyperplasia, leading to an increased number of small fat cells (60), increases in lipoprotein lipase (61), which tends to promote fat storage, and other as yet unidentified factors. Some postobese individuals may be more sensitive to the macronutrient composition of their diet than are never-obese persons, and thus they must consistently eat a low-fat diet to maintain their body weight (62). High levels of physical activity have also been found to correlate with successful weight maintenance in a registry of long-term weight maintainers (63), and evidence suggests that the level of physical activity must be substantial to have an impact on weight regain (64). In addition, food intake and physical activity may interact, such that physical activity can mitigate against some effects of a higher-fat diet (65). Combining a lower-fat, lower-energy-density diet with increased physical activity may allow previously obese individuals to decrease the dietary restraint necessary to maintain weight loss in the face of physiologic pressures for regain. It appears that

those who have been significantly obese (i.e., body mass index >30 kg/m^2) need to persist with long-term changes in their diet and physical activity in order to overcome the tendency to regain their lost weight.

Do Psychotropic Medications Play a Role in Obesity?

Weight gain is among the most problematic side effects of psychotropic agents and is one of the most frequent reasons for nonadherence to prescribed medication regimens, particularly when treatment extends beyond the short term (66, 67). Several classes of psychotropic medications are associated with unwanted weight gain, including antipsychotics, antidepressants, mood stabilizers, and, to a lesser degree, anxiolytics (68–70). Weight gain could theoretically be due to increased energy intake, decreased energy expenditure, or a combination of the two; in most cases, we have too little information to distinguish among these possibilities. In general, medications that block histamine H_1, serotonin 5-HT_{2C}, and dopamine D_2 receptors tend to be associated with weight gain (71).

Antipsychotics

Weight gain has been noted as a concomitant of antipsychotic treatment for the past several decades. Among currently available antipsychotics, the low-potency phenothiazines (e.g., chlorpromazine, thioridazine, mesoridazine) and novel antipsychotics (e.g., clozapine, olanzapine, risperidone, quetiapine) are most often associated with clinically problematic weight gain. Molindone is relatively unique in its lack of association with weight gain and its association with weight loss in several studies (70). In terms of relative effects on weight, the novel antipsychotics appear to be the most potent; they are followed by phenothiazines, which have large effects, haloperidol, which has a relatively small effect, and finally drugs that do not promote weight gain, including molindone and possibly loxapine and pimozide (68, 70). When weight gain occurs, it is usually most rapid in the acute phase of treatment and generally plateaus after 1–2 years (70). Although there are some indications of increased appetite in patients treated with antipsychotics (72), no systematic studies of energy intake or expenditure involving such patients have been done.

Mood Stabilizers

Weight gain of at least 5% (and much more in some cases) occurs in an estimated one-third to two-thirds of patients treated with lithium and in one-quarter to one-half of patients treated with anticonvulsants, including valproic acid and carbamazepine (68). In the case of lithium, weight gain appears to be dose-related (73), to occur primarily in the first 2 years of treatment, and to occur more frequently in patients who are already overweight at the onset of treatment (74). Among mood stabilizers that have come into use more recently, gabapentin appears to be associated with weight gain in a substantial minority of patients (75), lamotrigine is not typically associated with weight change, and topiramate is associated with mild, dose-related weight loss (76). The effect of lithium on weight may be mediated by increased intake of calorically dense fluids due to drug-induced thirst, increased carbohydrate and/or lipid storage, or lithium-induced hypothyroidism, but it is as yet unclear which if any of these factors is most clinically relevant (68). The mechanism of anticonvulsant-induced weight gain remains poorly understood.

Antidepressants

According to conventional wisdom about the relationship between antidepressants and weight gain, treatment with tricyclic antidepressants or with monoamine oxidase inhibitors (MAOIs) is associated with marked weight gain, particularly during the continuation and maintenance phases of treatment. However, as reviewed by Ackerman and Nolan (68), more recent studies have reported relatively modest mean weight gains, even over the long term, and much larger increases in a relatively small number of patients. Of the tricyclics, amitriptyline is thought to induce the most weight gain (67, 69). The mechanism of tricyclic antidepressant-induced weight gain appears to involve decreased basal metabolic energy expenditure and diet-induced thermogenesis. Shifts in food preference toward calorically dense foods occur in a minority of patients but do not seem to be associated with significant weight gain (69). Among the MAOIs, the reversible inhibitors of MAO_A such as moclobemide (not yet available in this country) appear to be least likely to induce weight gain (77). Newer antidepressants, including bupropion, venlafaxine, and nefazodone, are generally not associated with weight gain (68). An exception is mirtazapine, which, like the tricyclic antidepressants, is associated with weight increase (78).

The selective serotonin reuptake inhibitors (SSRIs), including fluoxetine, sertraline, paroxetine, fluvoxamine, and citalopram, have generally been thought not to be associated with weight gain. In fact, in a large multicenter trial, patients treated with 60 mg/day of fluoxetine lost a significant amount of weight, with maximum weight loss occurring at week 20, but on average had regained most of their lost weight by the end of 1 year of treatment (79). Why do SSRIs differ from tricyclic antidepressants and MAOIs in their effects on weight? Proposed mechanisms include SSRI-induced increases in metabolic rate (69) or serotonin-mediated effects on appetite and food intake. However, although the SSRI fluoxetine is known to decrease binge eating in patients with bulimia nervosa (80) and to decrease meal size but not frequency in overweight women (81), the effects of SSRIs on food intake have not yet been well characterized.

Recent observations of patients receiving long-term treatment with SSRIs have suggested that the effect on

weight may vary over time. Clinicians have reported that long-term treatment with SSRIs (i.e., several months) is associated with regain of lost weight and overshoot to higher-than-baseline weight in certain patients, an effect which may be mediated by serotonin 5-HT_{2C} receptors (82). However, a recent large-scale study found that patients receiving continuation treatment with fluoxetine gained no more weight over 1 year than those receiving placebo (83). Systematic studies are needed to determine whether the pattern of weight regain in patients who have lost weight during fluoxetine treatment differs from the general pattern of weight regain in patients who lose weight intentionally.

Should Obese Individuals Attempt to Lose Weight?

Does Weight Loss Reduce Medical Risks?

Most successful weight loss treatments yield reduced blood pressure, reduced serum triglycerides, increased HDL cholesterol, reduced total cholesterol and LDL cholesterol, and reduced blood glucose and hemoglobin A_{1c} in some patients with type 2 diabetes. Medical benefits begin to accrue with weight losses of as little as 5%–10% of initial weight (84, 85), and successful weight loss may play an important role in prevention of these illnesses among those at risk (2, 86). These findings suggest that physicians should treat not just the complications of obesity, but obesity itself, even in the absence of recognized sequelae. NIH guidelines recommend weight loss for individuals with a body mass index >30 kg/m^2 and for individuals with a body mass index >25 kg/m^2 and two or more obesity-related risk factors (2). For those with a body mass index between 25 and 30 kg/m^2, but without additional risk factors, prevention of further weight gain, rather than weight loss, is the goal.

Critiques of Weight Loss

Critics have called for a moratorium on weight loss programs, arguing that dieting is not only ineffective but harmful. According to this view, the pattern of recurrent weight loss followed by regain—so-called "yo-yo dieting"—demoralizes patients, makes future weight loss even more difficult, and even increases the risk of disease (37).

Available evidence provides little empirical support for these objections. Weight cycling has been found to be associated with increased morbidity and mortality in several studies. However, these studies have not looked specifically at the effects of intentional weight loss among obese individuals. A review examining weight cycling concluded that concerns about its adverse health effects should not preclude efforts at sensible, moderate weight loss by obese persons, but stressed the need for further research in this area (87). The authors also underscored the importance of commitment to long-term behavioral change in enhancing the likelihood of weight stability.

Critics have also argued that the dietary treatment of obesity may trigger or exacerbate binge eating in obese patients. This concern is based on the link between dieting and the onset of bulimia nervosa. However, it is important to distinguish between normal or underweight women with bulimia nervosa and obese binge eaters. The latter show significantly less dietary restraint than the former (88). In contrast to patients with bulimia nervosa in whom the onset of dieting almost always precedes the onset of binge eating (89), obese binge eaters are just as likely to diet after the onset of binge eating as before it (90–92). Studies have indicated that behavioral treatments of obesity using either moderate or severe caloric restriction appear to be effective in reducing binge eating disorder (93–95).

Even modest weight loss can reduce health risks in obese patients in the short term at least. The long-term effects of weight loss have yet to be systematically evaluated. Nevertheless, although obese individuals must be evaluated for their specific risks and likelihood of success, for most, weight loss would be beneficial. Clearly, the patient's motivation must be assessed, and interventions must be geared to his or her readiness to undertake the difficult tasks involved in losing weight.

How Well Do Currently Existing Treatments Work?

Behavioral Weight Control

A comprehensive behavioral weight control program, comprising components of improved eating habits, lifestyle change, and increased exercise, is widely viewed as the treatment of choice for overweight and moderately obese individuals. With 5 months of treatment, behavioral treatment combined with moderate dietary restriction (e.g., 1000–1500 kcal/day of self-selected foods) results in a mean weight loss of 15–20 pounds (96). Behavioral weight loss programs are also associated with significant decreases in depression and body image dissatisfaction, together with increases in self-esteem and interpersonal functioning (97). The problem is that these treatment effects are not maintained over time.

At 1-year follow-up, patients who have received behavioral treatment with dietary restriction regain 35%–50% of their weight loss, both in research clinics and in the general population. Five-year follow-ups have revealed that the vast majority of patients regained all of the weight they had lost. A large and diverse literature is strikingly consistent in showing the same inexorable pattern, namely, gradual regain of weight over time (98). Viewing obesity as a chronic condition, it is not surprising that improvement recedes once treatment has stopped. Accordingly, a continuous care model has been proposed (2). Extended maintenance sessions have improved persistence of weight loss, but attendance declines after 6 months (99).

Behavioral treatment combined with a very low calorie diet of up to 800 kcal/day, often in the form of a liquid nu-

tritional supplement, was highly touted and widely used several years ago. Typical programs used a very low calorie diet for 12–16 weeks, followed by reintroduction of a self-selected 1000–1500 kcal/day diet. This approach produces more rapid weight loss initially, but relapse occurs more quickly, so that treated individuals, after 1 year, show similar weight regain to those on more moderate calorie restriction (100).

A notable contrast to the high relapse rate among obese adults is the outcome in children. Weight loss has been maintained over a 10-year follow-up in children, even though their parents showed the predictable relapse rate (101). It may be easier to teach children healthy eating and activity habits. Moreover, the parents in these studies provided a structured environment that supported weight control by regulating access to food (102). This structure had the effect of lessening reliance on self-control that may be insufficient in the long term to cope with the pressures of a toxic environment (28).

Predictors of weight loss have proven elusive. Personality traits, measures of psychopathology, presence of binge eating, dietary restraint, and history of weight cycling have all proven unreliable (103). The process variables of early weight loss and compliance with self-monitoring are the most useful predictors (103, 104). Patients who do neither present very poor risk for treatment. Adherence to an exercise regimen is a reliable correlate of maintenance of weight loss (105, 106).

Pharmacotherapy

Adding weight loss medications to the behavioral treatment of obesity results in weight loss 5–20 lbs. greater than that seen with behavioral treatment alone (107). The majority of weight loss occurs over the initial 6 months, with most studies showing relative stability of weight over the ensuing 6 months of medication treatment. There is little information on the safety or efficacy of drug treatment for more than 1 year, but studies of longer treatment indicate that gradual regain occurs in many patients (108, 109). Weight loss medications are similar to medications used to treat other chronic medical conditions; that is, they don't work when they are not taken. The vast majority of studies have found that weight is quickly regained when medication is discontinued (107). Therefore, it makes little sense to use obesity medications short term, in the hope that they will provide a "jump start" for patient motivation.

After the 1992 publication of the Weintraub study (110), showing the efficacy of long-term use of fenfluramine/phentermine therapy, an exponential increase in the use of these agents occurred (111). The subsequent finding that the serotonergic reuptake and releasing agents fenfluramine and dexfenfluramine were associated with valvular insufficiency led to their withdrawal from the market (112). The lesson learned is that treatments have potential risks. Where the risk-to-benefit ratio of longer-term treatment is largely unknown, as is the case with many weight loss drugs, the prescribing physician should have a high threshold for instituting treatment.

For obesity, current recommendations include prescription of weight loss medications only for patients with a body mass index >30 kg/m^2 without or >27 kg/m^2 with associated comorbid conditions such as type 2 diabetes (2). Two medications are currently approved for long-term use. Sibutramine is a selective serotonin and norepinephrine reuptake inhibitor whose efficacy is similar to that of other single-drug treatments (113). In short-term studies, it has not been associated with valvular heart disease (114). The primary limiting factors for its use are elevations in pulse and blood pressure, which are usually modest, but can be significant in some patients (115). Another new weight-loss medication, orlistat, is a gastrointestinal lipase inhibitor, which leads to approximately one-third of ingested dietary fat being excreted in the stool (116). Its efficacy for weight loss and weight maintenance is similar to that of other obesity drugs, and it appears to have favorable impact on obesity-related comorbidity (109). Side effects are gastrointestinal, such as loose stools and oily spotting, and are related to the amount of dietary fat ingested, which may aid in adherence to a low-fat diet. Fat-soluble vitamin absorption is also decreased, and multivitamin supplementation is recommended. Other medications, including leptin, are in earlier stages of development, and are likely to be years away from approval.

Older medications used to treat obesity, including phentermine (half of the "fen/phen" combination), phendimetrazine, mazindol, and other adrenergic agents are not approved for long-term use. The use of SSRIs as weight control agents has shown disappointing results. Regarding combination therapy, there have been few controlled studies demonstrating the safety or efficacy of any drug combination for the treatment of obesity, including fluoxetine/phentermine, orlistat/phentermine, or others. The combined use of medications, except in the context of clinical studies with full informed consent, should be discouraged (107).

The NIH Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults provides guidance on acceptable use of weight loss medications in the treatment of obesity. The guidelines may be downloaded from http://www.nhlbi.nih.gov/guidelines/obesity/ob_home.htm.

Surgery

Gastrointestinal surgery appears to be the most effective treatment for some severely obese individuals with class III obesity (body mass index of 40 kg/m^2 or more) or class II obesity (body mass index of 35–39.9 kg/m^2) with comorbid medical conditions such as sleep apnea or type 2 diabetes. The most commonly used procedures are gastric bypass (e.g., Roux-en-Y gastrojejunostomy) and gastric restriction (e.g., vertical banded gastroplasty). On average, patients maintain a weight loss of 25%–40% of their

preoperative body weight after these procedures (117). Lipectomy and liposuction are cosmetic procedures that do not yield medically significant weight loss (118).

It is important for prospective patients to realize a number of things. As with any major surgery, there are operative mortality risks estimated at 0.3%–1.6% in specialized centers, short-term complications such as peritonitis and anastomotic leaks in 1.6%–2.3% of patients, and long-term complications such as cholelithiasis and vitamin and mineral deficiencies, which can be minimized with careful management (119). In addition, after surgery, patients are no longer able to eat in the way they were accustomed to. Those who have undergone gastric bypass experience "dumping syndrome" (sweating, palpitations, lightheadedness, nausea) if they ingest significant amounts of calorically and osmotically dense food, and therefore become conditioned not to eat these foods. Patients who have had gastric restriction surgery are unable to eat more than a limited amount of food at a single sitting without vomiting, and thus must eat several small meals per day to maintain adequate nutrition. Patients also must understand that following surgery, lifelong medical surveillance is a necessity (2).

The Swedish Obese Subjects study, a large-scale nonrandomized study of surgery versus conventional treatment, provided the most compelling evidence for the benefits of surgery. In that study, 1,600 patients followed for a mean duration of 4 years after surgery experienced much greater sustained weight loss and a lower incidence of diabetes mellitus compared to control subjects. Unlike patients receiving conventional treatment, the surgical patients also reported dramatic short-term and sometimes long-term improvements in health-related quality of life (86). However, most reports suggest that 20%–30% of the patients were unimproved in the long term (119, 120). It is possible for patients to eat in a way that maintains their preoperative weight or to be unable to adapt to the limitations imposed by surgery, e.g., to repeatedly eat to the point of vomiting. We cannot yet predict with confidence which patients are likely to have poor outcomes, although some evidence has suggested that the presence of an eating disorder before surgery (121, 122) or history of inpatient psychiatric treatment (123) predict less favorable outcome.

Psychotherapy

Psychotherapy should not be considered a primary treatment for obesity. However, this does not mean that psychotherapy has no role. Both cognitive behavioral therapy and interpersonal therapy have been found to be effective in normalizing eating and reducing distress in obese patients with binge eating disorder, although neither intervention is associated with significant weight loss (124). Psychotherapy may be helpful in enhancing self-acceptance in obese patients who have learned to feel ashamed about their weight and may help patients to cope with the effects of prejudice and "weightism" that are pervasive in our culture. Often, greater self-acceptance and the resulting increase in overall self-esteem are key steps in developing motivation for working toward a healthier lifestyle and/or for undertaking weight control treatment (125). Body image therapy programs have been developed to help obese individuals alter the way they perceive and evaluate their bodies (126). These interventions are crucial for many obese patients, because most such patients, even after successful weight loss treatment, remain at a higher-than-normal weight. Self-help organizations that promote size acceptance provide recognition and support for obese individuals and serve as a forum for addressing discrimination and altering harmful cultural stereotypes.

Integrated Treatment

Behavioral treatment to improve diet and increase physical activity is the cornerstone of any weight loss treatment program regardless of the degree of obesity. If additional treatment is indicated, because of more severe obesity, medical complications, or lack of response to behavioral treatment, other modalities, such as medications or surgery, may be added. Effective treatment may be provided in various settings. Although university and hospital-based programs often provide the most comprehensive care, commercial, work-site, and self-help programs that incorporate behavioral principles may also be appropriate for many individuals. It is likely that there will be increasing use of brief physician or nurse visits to manage obesity, using materials developed by federal agencies and other organizations (2).

Do Strategies Exist for Evaluation and Treatment of Psychiatrically Ill Obese Patients?

Initial Evaluation

Because some studies of clinical groups of obese patients report unexpectedly high rates of depressive and anxiety disorders (127, 128), the clinician should be particularly careful to screen for these disorders, even if they are not suggested by the initial complaint. Obese individuals, even those who are seen for treatment of a condition unrelated to their weight, are at increased risk of suffering from binge eating disorder. Therefore, it is important to inquire about problematic eating patterns, loss of control related to eating, and weight control practices, including dieting, exercise, diet pills (prescribed, over-the-counter, and illicit), or various forms of purging after eating. Is the patient caught in a cycle of "crash dieting" followed by weight regain and further demoralization? If the patient is exercising, is the need to exercise interfering in any way with occupational or interpersonal functioning? It is crucial to assess the patient's attitudes toward his or her weight and shape. Is the patient's obesity a source of shame, and does the patient avoid particular activities

(e.g., parties, shopping for clothes)? Has the patient experienced discrimination in social or occupational settings? Finally, it is important to ascertain whether the patient is receiving adequate medical care (129). Obese patients may avoid doctors because of fear of being criticized or humiliated about their weight. It is particularly important to inquire about gynecological care; obese women may experience particular anxiety related to gynecological exams and may therefore avoid routine care. A discussion of the patient's anxiety and referral to an understanding practitioner may be invaluable.

Cognitively Impaired Patients

Although weight control strategies have not been systematically evaluated in obese patients with impaired cognition and/or psychosis, a small number of open studies and case reports suggest that weight-reducing diets (130), behavioral programs (131–134), and even surgery (135) can be successful in some individuals. The risk/benefit calculation of weight loss treatment in this population is even more complex than for psychiatrically well persons, but these studies make the important point that the possibility of weight loss in cognitively impaired individuals should not be dismissed out of hand.

Patients Receiving Psychotropic Agents

An open discussion of the possibility of weight gain and, should significant weight gain occur, a discussion of the pros and cons of continued treatment and the options for management are crucial in promoting collaboration rather than treatment nonadherence. Overall strategies include dose adjustment or a switch to an alternative medication in the same class that is less associated with weight gain. If these maneuvers are impossible or ineffective, the stepped-care approach to obesity management outlined above should be considered, and the clinician should take into account the patient's ability to adhere to recommendations, e.g., exercise or dietary regimens. Education and consciousness raising, e.g., the importance of drinking noncaloric or low-calorie rather than high-calorie fluids, is a relatively simple and potentially helpful intervention. For patients with antidepressant-induced weight gain, some clinicians report success with adding SSRIs, bupropion, or psychostimulants to the primary antidepressant. This addition may serve as an antidepressant augmentation strategy as well as a weight control maneuver (136).

Obesity Treatment: Where Are We Headed?

Given the lack of effective treatments for adults, prevention is all the more a priority. Primary prevention of obesity is likely to require significant environmental manipulations that could be brought about only through changes in public policy (137). If we cannot do this, we must intervene as early as possible. In striking contrast to the discouraging long-term results of treatment studies with adults, studies have shown lasting maintenance of weight loss in the treatment of obese children (101). Care must be taken, however, to ensure that obese children receive adequate nutrition and do not engage in the dysfunctional type of dieting that has been linked to the development of bulimia nervosa. It will be important to encourage sensible eating habits, increased physical activity, and development of healthy body-related attitudes (102).

Another response to the poor long-term outcome of treatment has been the call to reassess therapy goals. Brownell and Wadden (138) have proposed that we abandon traditional weight loss goals based on weight tables in favor of what they describe as "reasonable weight." Significant health benefits are associated with relatively modest weight losses that fall far short of the healthy ideal and patients' own aesthetic ideals (84). Unfortunately, many if not most patients will find such modest weight loss goals unsatisfying, even unacceptable (139). Appearance-driven concerns, rather than health needs, continue to motivate obese individuals to lose weight. Societal pressures reinforce these appearance-driven concerns by portraying obese individuals in a negative manner. In addition, some patients believe that they need to lose far more than 10% of their weight to attain significant health benefits.

The challenge to health care providers, particularly mental health professionals, is clear. It is important to help obese people build self-esteem and encourage them to lead as full a life as possible, regardless of their weight and of whether they succeed in efforts at weight control. "Nondieting" programs have demonstrated that significant gains in self-acceptance are possible even in the absence of weight loss (T.A. Wadden, personal communication, 1998). Enhancing self-acceptance may not only provide a more compassionate approach to what has proved a refractory problem, but might also lead to more lasting reductions in weight by virtue of helping patients to accept only modest weight loss and improve compliance with health-relevant eating and exercise behaviors (125, 140, 141).

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