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Review article

Barriers to obesity treatment

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Abstract

Obesity, one of the most prevalent health problems in the Western world, is a chronic and progressive condition. Therefore, as with other chronic diseases, patients with obesity require lifelong treatment. Long-term efficacy and effectiveness of obesity treatments is notoriously poor. This may in part be attributable to the substantial barriers that undermine long-term obesity management strategies. These can include lack of recognition of obesity as a chronic condition, low socioeconomic status, time constraints, intimate saboteurs, and a wide range of comorbidities including mental health, sleep, chronic pain, musculoskeletal, cardiovascular, respiratory, digestive and endocrine disorders. Furthermore, medications used to treat some of these disorders may further undermine weight-loss efforts. Lack of specific obesity training of health professionals, attitudes and beliefs as well as coverage and availability of obesity treatments can likewise pose important barriers. Health professionals need to take care to identify, acknowledge and address these barriers where possible to increase patient success as well as compliance and adherence with treatments. Failure to do so may further undermine the sense of failure, low self esteem and self efficacy already common among obese individuals. Addressing treatment barriers can save resources and increase the prospect of long-term success. © 2007 European Federation of Internal Medicine. Published by Elsevier B.V. All rights reserved.

Keywords: Obesity therapy; Barriers; Counseling/education; Medical education; Medication; Surgery; Lifestyle

1. Introduction

Obesity is one of the most prevalent health problems in the Western world [1]. Obesity increases the risk of medical illness and premature death [2] and thus imposes an enormous economic burden on the health care system [3]. Obesity is also associated with a reduced quality of life resulting from substantial limitations and restrictions in activities of daily living [4]. Obese individuals are less likely to obtain insurance, employment or promotion or enjoy personal relationships [5]. Prevention and treatment of obesity is therefore now widely recognized as a chief priority for most health care systems.

Once established, obesity becomes a chronic and progressive condition. There is currently no accepted “cure” for obesity in that patients, once obese, seldom succeed in achieving or maintaining a healthy weight in the long-term. Although maintaining a weight reduction of as little as 5% can lead to substantial reduction in risk for comorbidities [6], even such modest goals are difficult to sustain in clinical practice [7]. The few individuals able to sustain a significant weight reduction do so by chronically and often drastically restricting caloric intake in addition to maintaining markedly increased physical activity levels [8]. Thus, as with other chronic conditions (such as hypertension or type 2 diabetes), obese patients require lifelong intervention.

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Patients seeking obesity treatment generally present with a myriad of issues that need to be considered. Even in patients who are clearly ready for change and fully recognize that they will need to make considerable lifestyle adjustments, it is important to recognize potential barriers that will make weight loss difficult and/or undermine efforts at weight-loss maintenance [9]. Fortunately, many of these barriers can be modified in order to facilitate adherence to a weight management plan.

Early identification and management of treatment barriers can save resources and increase the prospect of long-term success, thereby protecting the patient from the emotional and physical consequences of weight cycling [10]. In cases where objective barriers to obesity management are insurmountable, the focus may need to be on the prevention of further weight gain rather than on weight loss.

This review will focus on common barriers that should be identified, addressed and, if possible, modified to reduce the failure rate in weight loss and maintenance (Table 1).

2. Recognition of obesity as a chronic condition

Recent physician surveys have identified poor education on obesity during medical school and residency as the most prevalent barriers to obesity care [11]. Both patients and

health care providers need to clearly recognize and acknowledge the chronicity of obesity to fully appreciate the importance of long-term treatment strategies [9]. There is a common misconception that the simple solution to obesity is weight loss. However, weight loss is only the first phase of obesity management [12]. Without successful strategies to prevent weight regain, weight loss is meaningless and may be counter productive. While patients are generally prepared to go through the initial stage of weight loss they are often unprepared to accept the reality that maintaining weight loss requires lifelong lifestyle changes, and in some cases, medication or surgery to counteract the complex neurobiological and endocrine changes that seek to restore body weight to pre-loss weights. Although medications have been shown to significantly promote weight loss, their real value perhaps lies in their ability to limit weight regain [13]. Unfortunately, long-term compliance with obesity medications is notoriously poor, with 1-year persistence rates <10% and 2-year persistence rates of 2% [14]. Even in the case of bariatric surgery, arguably the most effective treatment for patients with severe obesity [15], some patients may regain weight and most will do so if surgery is reversed. Thus, both medication and surgery must be seen as long-term rather than short-term solutions to obesity management.

Table 1
Barriers to obesity treatment

Barrier	Intervention	Rationale
Acceptance of obesity as a chronic disease	Education of patients and health care providers.	Obesity treatment requires lifelong management to maintain weight loss.
Socioeconomic status	Address limitations. Adapt strategy to socioeconomic situation.	Long-term adherence to the obesity treatment should be facilitated.
Time constraints	Reassess motivation and readiness for change. Adapt strategy to patient's schedule. – Use of meal replacements – Flexible exercise schedule	– Offers portion control, availability, portability, ease of preparation. – Practical and sustainable.
Saboteurs	Identification of problem. Counselling, support and motivation.	Support systems are essential to long-term weight maintenance
Comorbidities	– Mental health – Sleep – Pain – CV disease – Respiratory disease – Digestive disease – Endocrine disorders	– Recognition and concomitant treatment of depression and attention deficit disorder (ADD) – Recognition and referral for specific intervention of binge eating disorder (BED) – Evaluation and treatment of obstructive sleep apnea (OSA) – Pain management – Management of symptoms of cardiovascular disease – Accurate diagnosis and treatment of the cause of dyspnea – Recognition and treatment of reflux symptoms – Recognition and treatment of insulin resistance, hypothyroidism, Cushing and hypogonadism
Medication	Choose medication with less propensity for weight gain	– Improvement of depression and impulsive behavior facilitates adherence to obesity treatment. – Cognitive behavioural therapy (CBT) focusing on binge eating and associated psychopathology facilitates adherence to lifestyle habits. Reduction of cardiovascular risk and risks of accidents. Improved sleep may positively impact adherence to lifestyle changes and physical activity. Allows patient to be more physically active. Allows patient to be more physically active. Allows patient to be more physically active.
Alcohol and substance abuse	Assessment and treatment of addictions.	Avoidance of using food to relieve reflux symptoms. Investigation of endocrine disorders is only justified when historical and clinical evidence supports the diagnosis. Prevent weight gain
		Improve adherence to obesity treatment

While bariatric surgery has been gaining popularity and acceptance as an effective way to achieve massive weight loss, it remains an option only for the extremely obese. At present bariatric surgery is the only therapeutic modality that can produce sustainable weight loss and halt or resolve comorbidities in patients with morbid obesity [16]. The generally accepted criteria for gastrointestinal surgery for weight loss include patients with BMI > 40 or BMI > 35 complicated by weight-related comorbidities [17]. These weight parameters should be viewed as a guide for the clinician, as patients with multiple weight-related comorbidities and BMI below 35 may also be considered candidates for surgery.

It is especially important for both primary care providers and subspecialists to be familiar with currently recommended approaches to the medical treatment of obesity, as it is critical that this extremely common, treatable chronic disease be recognized, intervention initiated, and therapy maintained at every appropriate clinical opportunity. Significant medical benefits can be gained even with a relatively small percentage of weight loss.

3. Socioeconomic status

Socioeconomic status can pose a significant obstacle to obesity treatment [18]. Strategies may have to be tailored to specifically address the socioeconomic situation of the patient. In Western societies, the prevalence of obesity is greater in lower socio-economic groups. This has been attributed to the greater density of fast-food restaurants in low-income neighbourhoods [19], higher cost of healthy diets [20], safety concerns that prevent walking and other outdoor activities [21] and greater social acceptance of excess body weight [18]. Affordability of membership in commercial weight-loss programs, gyms, obesity medications or surgery can likewise prove to be important obstacles. All of these factors can pose important barriers to weight management and interventions must specifically acknowledge and address these limitations.

4. Time constraints

Lack of time is one of the most frequently encountered barriers to obesity management [22]. Patients may find it difficult to find extra time to engage in physical activity or to plan a healthy diet. Implementing a strategy for obesity management that the patients can adapt to fit their own schedule is essential if they are expected to follow them in the long-term [9]. For individuals with busy schedules or travel loads, activities they can engage in on their own (e.g. walking) may be more practical and sustainable than team sports or activities involving unwieldy equipment or sporting facilities.

Where time is a major obstacle to planning and preparing a healthy diet, commercial meal replacements that provide a quick source of protein and other nutrients may be a practical alternative. The advantages of meal replacements

are portion and calorie control, availability, convenience, easy of preparation and portability [23]. As meal replacements are generally less expensive than “real” food, they are also an affordable alternative to fast food for individuals with low incomes [24].

When patients persistently present new time constraints their motivation and readiness for change may need to be reassessed. Strategies that employ motivational interviewing techniques to explore and address ambivalence may be beneficial in this regard [25].

5. Saboteurs

Numerous factors within a patient’s social environment can sabotage and undermine weight-loss attempts and weight-loss maintenance [26]. A person’s social and cultural milieu has an important influence on lifestyles and beliefs related to physical activity and nutrition. Going on a diet may be seen as being a “woman’s thing” and the culture of eating vast amounts of rich foods during social interactions may be difficult to overcome. A strong support network of friends, family or peers are essential to promote successful adherence to long-term lifestyle changes but getting buy in and support may pose challenging. It is also not uncommon that people closest to someone with obesity may actively or inadvertently hamper or subvert interventions [27]. A family is a dynamic system with its own language, roles, rules, beliefs, needs, and patterns. Each family member plays a part in the system and when the role of one player changes, even for the better, it can disrupt the roles of all involved. A not uncommon example is the suspicion of infidelity that may arise, when a spouse or partner suddenly attempts to lose weight. These types of dynamics may need to be addressed if they lead to what has been described as “intimate saboteurs”. It is important to anticipate such responses and pre-empt them by ensuring that the reasons and motivations for embarking on a weight management plan are clear to all.

Social and professional obligations can also sabotage the patient’s efforts as participation in activities associated with food and alcohol consumption may be linked to the way a person interacts. For example, the refusal to participate in weekend binges or festivities may lead to the loss of personal friendships and support systems. These losses need to be identified and alternative behaviors put in place. Professional counselling on “exit” strategies may be required to change long-established behavioral and social patterns without leaving a social or emotional void.

6. Comorbidities

Numerous comorbidities can promote weight gain or obstruct weight loss. While reviewing the management of co-comorbidities is beyond the scope of the present review, recognition and treatment of the following comorbidities may be essential for successful obesity treatment.

6.1. Mental health

Health care professionals should screen obese adults for eating disorders, depression and psychiatric disorders as appropriate [9]. While obese individuals in general do not report more mental health problems than normal-weight individuals [28], those seeking treatment in commercial weight-loss programs or obesity clinics have higher rates of depression, increased substance abuse and an increased prevalence in eating disorders — particularly binge eating disorder (BED) and night eating syndrome [29–31]. Eating disorders, anxiety and compulsive behaviors in obese individuals have been associated with past or ongoing physical, sexual and emotional abuse [32]. All of these behaviors in turn may be perpetuated by psychosocial stressors like bullying and stigmatization resulting in poor self esteem, personality disorders and social phobias [33].

Approximately 30% of patients seeking treatment for obesity will meet the diagnostic criteria for BED whereas in the prevalence of BED in general population is approximately 2.5% [30]. BED is characterized by recurrent episodes of eating binges associated with subjective and behavioural indicators of impaired control over, and significant distress about the binge eating without the presence of inappropriate compensatory behaviours. It is associated with more severe and earlier onset of obesity, earlier onset of dieting, more weight fluctuations and earlier psychopathology. Patients with BED may find it harder than other people to stay in obesity treatments and may be more likely to regain weight quickly. Patients with BED may require behavioural and cognitive treatment focusing on their binge eating and associated psychopathology before embarking on obesity treatments [34]. Recent studies have shown that pharmacological management of BED may promote weight loss [35,36] and that with proper counselling, patients with BED may do well following bariatric surgery [37].

Where present, treatment of depression may have to be initiated to allow patients to successfully address lifestyle changes [38]. Patients with mood disorders may present with depressed or irritable mood and lack of normal interest or pleasure in daily life, thus undermining any desire to follow a treatment regimen. The treatment of psychiatric illnesses in this patient population needs to occur with an understanding of the obesogenic potential of antidepressants prompting the preferential use of medications with lower propensity for weight gain [39].

Recent studies also suggest that attention deficit disorder (ADD) may not be uncommon among obese patients [40]. Lack of impulse control can make it difficult for patients to adhere to meal plans or medications. Thus identification and treatment of ADD and subsequent improvement in impulse control may help patients focus and adhere to weight management plans.

Patients with active eating disorders, substance abuse or personality issues that can interfere with obesity management need to be referred for specific interventions before commencing an obesity management program [34].

It is also important to assess patients ability to understand management strategies, as cognitive impairment (as in many syndromal forms of obesity) can pose significant barriers to obesity treatment.

6.2. Sleep

Several recent epidemiological studies suggest a relationship between disrupted or inadequate sleep and obesity [41]. This has in part been attributed to the profound influence of sleep deprivation on the neuroendocrine regulation of appetite and food intake [42]. Whether or not increased sleep can reduce the risk for obesity or play a role in obesity management has yet to be determined.

In contrast, there is little doubt that sleep disruptions due to obstructive sleep apnea is a common finding in obese individuals [43]. Prevalence of sleep apnea has been estimated to affect 40% of the adult obese population [44] and is clearly a major determinant of day-time drowsiness and irritability as well as metabolic and cardiovascular risk [45,46]. While there is little evidence that commencing treatment for obstructive sleep apnea (e.g. continuous positive airway pressure) can *per se* result in weight loss, it is not far fetched to assume that improved sleep will positively impact on physical activity levels and overall mental disposition thereby promoting motivation, initiation and adherence to lifestyle changes.

6.3. Pain

Obesity is commonly associated with musculoskeletal pain resulting in functional locomotor limitations [47]. Excess body weight is closely related to osteoarthritis [48] and lower back pain [49]. Chronic generalized pain and or chronic fatigue [50] are common findings in obese patients probably due to a combination of different factors including depression, fibromyalgia, osteoarthritis, sleep disorders and reduced cardio-pulmonary fitness. The presence of generalized pain or chronic fatigue can affect the global sense of well being, quality of life and functional capacity leading to decreased physical activity and lack of motivation to follow healthy diets. The presence of significant pain can promote immobility leading to loss of muscle mass, reduced cardio-pulmonary fitness and precipitate psychological and metabolic changes that facilitate further weight gain. Pain management may thus be an important first step to promote mobility thereby permitting a more active lifestyle.

6.4. Cardiovascular disease

Obese individuals have increased risk of cardiovascular disease [51]. The presence of angina, dyspnea, claudication or stroke sequela can limit the patients' ability to be physically active, thereby promoting sedentariness and weight gain. Thus, interventions aimed at reducing symptoms of cardiovascular disease can allow patients to be more physically active. Unless clearly indicated, the use of

medications such as beta-blockers, which can promote weight gain [52] or interfere with weight loss [53], should be avoided.

6.5. Respiratory disease

Interventions aimed at improving pulmonary function may be important to allow patients to be more physically active to promote weight loss and prevent weight regain. Dyspnea in obese patients can unmask other associated conditions such as respiratory and heart disease. There is a clear association between dyspnea and obesity in adults and children [54]. Hypercapnic respiratory failure and cor pulmonale are frequently observed in severe obesity [55]. With the exception of systemic corticosteroids, there is no evidence that modern pharmacological asthma treatment promotes weight gain [56].

6.6. Digestive disease

Assessment of dental status is particularly important in obese patients as poor dental health can limit the ability to eat food with high fiber content, such as cereals, fruits and vegetables, thus promoting the consumption of energy-dense processed foods [57].

Obesity is associated with an increased risk of gastroesophageal reflux disease symptoms, erosive esophagitis, and esophageal adenocarcinoma [58]. Symptoms of reflux can be misinterpreted as hunger and are often relieved by eating. Furthermore, elevated ghrelin levels, which may promote hunger and weight gain, were reported in patients with functional dyspepsia [59]. Thus, abnormalities of the upper gastrointestinal tract can potentially contribute to weight gain. While there is one study to suggesting that in patients with type 2 diabetes, the H₁-blocker cimetidine may promote weight loss [60], the potential mechanism and relevance of this effect remain unclear.

6.7. Endocrine disorders

While endocrine abnormalities can promote weight gain or hinder weight loss, it is important to consider the *a priori* likelihood of an endocrine disorder in a given patient before embarking upon a series of endocrine function tests. Endocrine testing cannot be justified unless there is good historical and clinical evidence to support a diagnosis other than simple obesity.

Profound insulin resistance as found in patients with type 2 diabetes or polycystic ovary syndrome has been associated with poorer response to both pharmacological and non-pharmacological weight-loss interventions [61]. Similarly, increased liver and visceral fat, common findings in patients with insulin resistance, have been linked to poorer response to lifestyle interventions [62]. This may be in part due to the strong antilipolytic effects of the hyperinsulinemia present in these patients. Recent evidence suggests that insulin resistant

individuals may respond better to low-carbohydrate or low-glycemic diets than to restriction of fat intake [63]. As discussed below, both insulin secretagogues and insulin treatment can promote weight gain and undermine weight-loss efforts in patients with type 2 diabetes.

Thyroid hormones are generally within normal levels [64]. Routine testing of thyroid hormones should be therefore discouraged and limited to patients with symptoms suggesting hypothyroidism (such as dry hair and skin, cold intolerance, hair loss, difficulty concentrating, poor memory, constipation, muscle cramping, menorrhagia, goiter). Thyroid hormone should not be used as an obesity treatment in euthyreote individuals.

Although obesity is one of the cardinal features of Cushing syndrome [65], routine investigation for corticosteroid excess in unselected obese patients is not warranted. Only the presence of other features characteristic of Cushing syndrome (central obesity without affecting extremities, easy bruising, purple striae — different from the stretch marks seen in obesity —, skin atrophy, proximal myopathy, impaired glucose tolerance, hypokalemia, etc.) should prompt further investigations [66].

Testosterone and sex hormone binding globulin (SHBG) blood concentrations progressively decrease in obese men, but free testosterone levels are generally normal [64]. In a patient with hypogonadism, testosterone treatment may increase libido and restore body fat distribution [67].

7. Medications

Iatrogenic weight gain as a consequence of pharmacotherapy occurs with many commonly used drugs, leading to exacerbation of comorbidities related to obesity and to non-compliance with therapy (Table 2) [68]. When these medications are necessary and cannot be replaced by medications with less propensity for weight gain, patients should be counselled regarding the possibility of weight gain and preventive measurements should be emphasized.

Improvement in glycemic control is often associated with some degree of weight gain, a collateral effect that is common to many antidiabetic treatments including insulin, thiazolidinediones, and sulfonylureas [69]. Thiazolidinediones are associated with a redistribution of fat from visceral to subcutaneous depots which is associated with improvements in metabolic control [70]. Metformin [71] and acarbose [72] are not

Table 2
Medications that may be associated with weight gain

-
- Antidiabetic medication (insulin, sulphonylureas, thiazolidinediones)
 - Antipsychotics (olanzapine, clozapine, risperidone)
 - Antidepressants (tricyclic antidepressants)
 - Mood stabilizers/antiepileptic drugs (valproate, carbamazepine)
 - Lithium
 - Steroid hormones (glucocorticoids)
 - Antiretroviral therapy
 - Beta-blockers
-

associated with weight gain and in some cases may produce modest weight loss. Newer antidiabetic agents targeting the incretin system like glucagon like peptide 1 (GLP-1) receptor agonists (e.g. exenatide), amylin analogues (e.g. pramlintide) or dipeptidylpeptidase (DPP) IV inhibitors (e.g. vildagliptin) [73] as well as long-acting insulin analogues (e.g. glargine, detemir) [74], may limit or promote weight loss.

Weight gain is a well-documented side effect of many psychotropic medications including antipsychotic drugs, antidepressants and mood stabilizers and may have serious long-term health and psychosocial consequences [39,75]. Atypical antipsychotics, tricyclic antidepressants, mood stabilizers and lithium are all associated with weight gain and while newer antidepressants, like SSRIs, were initially reported to be associated with an early minimal weight loss, recent evidence indicates that their use can also be followed by long-term weight gain. Bupropion may be the only antidepressant routinely shown to cause weight loss [76]. In one small study, sibutramine and topiramate were shown to prevent weight gain in patients with bipolar disorder [77]. Topiramate and zonisamide are anticonvulsants with possible mood stabilizing properties and may induce substantial weight loss [78].

Weight gain is also seen in long-term pharmacological treatment with systemic glucocorticoids [79], antiretroviral therapy [80] and non-selective beta-blockers [81].

8. Substance abuse

Moderate alcohol consumption is sometimes associated with higher BMI [82]. Although alcohol can be an important source of calories (7 kcal/g), it has no effect on satiety [83]. Alcohol consumption can lead to positive fat balance through the sparing effect of alcohol on fat oxidation, leading to increased fat storage [84].

Substance abuse or the use of diuretics and laxatives for purging purposes should be investigated as these disorders need to be treated before embarking on a weight management plan.

9. Barriers to pharmacological and surgical treatment of obesity

Unlike medical treatments for other chronic conditions, the use of anti-obesity treatments is generally not reimbursed by health care systems. This puts the whole burden of cost for these treatments on patients, often making them unaffordable. This may in part explain the poor long-term adherence with these compounds [85], although modest efficacy and lack of recognition of the need for long-term treatment to sustain weight-loss maintenance may likewise play an important role. It is reasonable to assume that counselling patients on the potential benefits of long-term weight-loss maintenance may improve long-term adherence to pharmacotherapy.

Although recent studies document the significant reduction in morbidity and mortality associated with bariatric surgery [86,87], access is still not widely available. A recent French survey found that although primary care physicians and sub-

specialists perceive its effectiveness as high, rate of referrals for obesity surgery are remarkably low [88]. Limited coverage by public and private payers poses another important barrier to accessing this treatment. Educating health professionals, policy makers, patients and the general public on the substantial benefits to be derived from modern bariatric surgery should serve to improve access to this treatment in the coming years.

10. Conclusions

In this review we emphasize the importance of identifying barriers that could interfere with obesity treatment. Identifying and addressing these barriers may be essential before patients can make the necessary lifestyle changes and adhere to therapy. Failure to identify and address these barriers may perpetuate a sense of failure and further undermine the often low sense of self esteem and self efficacy of obese patients. On the other hand, addressing these barriers and adapting obesity management strategies to the specific situation of each patient is likely to save resources and increase success rates of obesity treatments.

11. Learning points

- Obesity is a chronic and progressive disease.
- Obesity requires lifelong treatment.
- Early identification of treatment barriers can save resources and increase the prospect of long-term success.

References

- [1] World Health Organization. Obesity: preventing and managing the global epidemic (who technical report series no. 894). Geneva: World Health Organization; 2000.
- [2] Kopelman PG. Obesity as a medical problem. *Nature* 2000;404: 635–43.
- [3] Katzmarzyk PT, Janseen I. The economic costs associated with physical inactivity and obesity in Canada: an update. *Can J Appl Physiol* 2004;29:90–115.
- [4] Visscher TL, Rissanen A, Seidell JC, Heliövaara M, Knekt P, Reunanen A, et al. Obesity and unhealthy life-years in adult Finns: an empirical approach. *Arch Intern Med* 2004;164:1413–20.
- [5] Puhl R, Brownell KD. Bias, discrimination, and obesity. *Obes Res* 2001;9:788–805.
- [6] Pi-Sunyer FX. A review of long term studies evaluating the efficacy of weight loss in ameliorating disorders associated with obesity. *Clin Ther* 1996;18:1006–35.
- [7] Tsai AG, Wadden TA. Systematic review: an evaluation of major commercial weight loss programs in the United States. *Ann Intern Med* Jan 4 2005;142:56–66.
- [8] Wing RR, Phelan S. Long-term weight loss maintenance. *Am J Clin Nutr* 2005;82:222S–5S.
- [9] Lau DC, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E, et al. Canadian clinical practice guidelines on management and prevention of obesity in adults and children. *CMAJ* 2006;176: S1–13.
- [10] Foster GD, Sarwer DB, Wadden TA. Psychological effects of weight cycling in obese persons: a review and research agenda. *Obes Res* 1997;5: 474–88.
- [11] Forman-Hoffman V, Little A, Wahls T. Barriers to obesity management: a pilot study of primary care clinicians. *BMC Fam Pract* Jun 6 2006;7:35.

- [12] Merchant A, Yusuf S, Sharma AM. A cardiologist's guide to waist management. *Heart* 2006;92:865–6.
- [13] Bray GA, Ryan DH. Drug treatment of the overweight patient. *Gastroenterology* 2007;132:2239–52.
- [14] Padwal R, Kezouh A, Levine M, Etmann M. Long-term persistence with orlistat and sibutramine in a population-based cohort. *Int J Obes (Lond)* Apr 10 2007 [Electronic publication ahead of print].
- [15] Kral JG, Naslund E. Surgical treatment of obesity. *Nat Clin Pract Endocrinol Metab* 2007;3:574–83.
- [16] Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 2004;292:1724–37.
- [17] Gastrointestinal surgery for severe obesity. National institutes of health consensus development conference statement. March 1991. *Am J Clin Nutr* 1992;55:615S–9S.
- [18] Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 1989;105:260–75.
- [19] Block JP, Scribner RA, DeSalvo KB. Fast food, race/ethnicity, and income: a geographic analysis. *Am J Prev Med* 2004;27:211–7.
- [20] Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* 2004;79:6–16.
- [21] Miles R, Panton L. The influence of the perceived quality of community environments on low-income women's efforts to walk more. *J Commun Health* 2006;31:379–92.
- [22] Ruelaz AR, Diefenbach P, Simon B, Lanto A, Arterburn D, Shekelle PG. Perceived barriers to weight management in primary care—perspectives of patients and providers. *J Gen Intern Med* 2007;22:518–22.
- [23] Heymsfield SB, van Mierlo CA, van der Knaap HC, Heo M, Frier HL. Weight management using a meal replacement strategy: meta and pooling analysis from six studies. *Int J Obes Relat Metab Disord* 2003;27: 537–49.
- [24] Huerta S, Li Z, Li HC, Hu MS, Yu CA, Heber D. Feasibility of a partial meal replacement plan for weight loss in low-income patients. *Int J Obes Relat Metab Disord* 2004;28:1575–9.
- [25] Rollnick S. Behavior change in practice: targeting individuals. *Int J Obes Relat Metab Disord* 1996;20:S22–6.
- [26] Verheijden MW, Bakx JC, van Weel C, Koelen MA, van Staveren WA. Role of social support in lifestyle-focused weight management interventions. *Eur J Clin Nutr* 2005;59:S179–86.
- [27] Andrews G. Intimate saboteurs. *Obes Surg* 1997;7:445–8.
- [28] Mustajoki P. Psychosocial factors in obesity. *Ann Clin Res* 1987;19: 143–6.
- [29] van Hout G, van Oudheusden I, van Heck G. Psychological profile of the morbidly obese. *Obes Surg* 2004;14:579–88.
- [30] Spitzer RL, Yanovski S, Wadden T, Wing R, Marcus MD, Stunkard A, et al. Binge eating disorder: its further validation in a multisite study. *Intl J of Eat Disord* 1993;13: 137–53.
- [31] Wadden TA, Sarwer DB, Womble LG, Foster GD, McGuckin BG, Schimmel A. Psychosocial aspects of obesity and obesity surgery. *Surg Clin North Am* 2001;81:1001–24.
- [32] Grilo CM, White MA, Masheb RM, Rothschild BS, Burke-Martindale CH. Relation of childhood sexual abuse and other forms of maltreatment to 12-month postoperative outcomes in extremely obese gastric bypass surgery. *Obes Surg* 2006;16: 454–60.
- [33] Janssen I, Craig WM, Boyce WF, Pickett W. Associations between overweight and obesity with bullying behaviors in school-aged children. *Pediatrics* 2004;113:1187–94.
- [34] Dingemans AE, Bruha MJ, Furth EF. Binge eating disorder: a review. *Int J Obes* 2002;26:299–307.
- [35] McElroy SL, Hudson JI, Capece JA, Beyers K, Fisher AC, Rosenthal NR. Topiramate Binge Eating Disorder Research Group. Topiramate for the treatment of binge eating disorder associated with obesity: a placebo-controlled study. *Biol Psychiatry* 2007;61:1039–48.
- [36] McElroy SL, Kotwal R, Guerdjikova AI, Welge JA, Nelson EB, Lake KA, et al. Zonisamide in the treatment of binge eating disorder with obesity: a randomized controlled trial. *J Clin Psychiatry* 2006;67: 1897–906.
- [37] Bocchieri-Ricciardi LE, Chen EY, Munoz D, Fischer S, Dymek-Valentine M, Alverdy JC, et al. Pre-surgery binge eating status: effect on eating behavior and weight outcome after gastric bypass. *Obes Surg* 2006;16:1198–204.
- [38] DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med* 2000;160:2101–7.
- [39] Fava M. Weight gain and antidepressants. *J Clin Psychiatry* 2000;61: 37–41.
- [40] Altfas JR. Prevalence of attention deficit/hyperactivity disorder among adults in obesity treatment. *BMC Psychiatry* 2002;2:9–18.
- [41] Vorona RD, Winn MP, Babineau TW, Eng BP, Feldman HR, Ware JC. Overweight and obese patients in a primary care population report less sleep than patients with a normal body mass index. *Arch Intern Med* 2005 Jan 10;165(1):25–30.
- [42] Copinschi G. Metabolic and endocrine effects of sleep deprivation. *Essent Psychopharmacol* 2005;6(6):341–7.
- [43] Gami AS, Caples SM, Somers VK. Obesity and obstructive sleep apnea. *Endocrinol Metab Clin N Am* 2003;32:869–94.
- [44] Resta O, Foschino-Barbaro MP, Legari G, Talamo S, Bonfitto P, Palumbo A, et al. Sleep-related breathing disorders, loud snoring and excessive daytime sleepiness in obese subjects. *Int J Obes Relat Metab Disord* 2001;25:669–75.
- [45] Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med* 2005;353:2034–41.
- [46] Wolk R, Kara T, Somers VK. Sleep-disordered breathing and cardiovascular disease. *Circulation* 2003;108:9–12.
- [47] Larsson UE, Mattsson E. Perceived disability and observed functional limitations in obese women. *Int J Obes Relat Metab Disord* 2001;25: 1705–12.
- [48] Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis. The Framingham Study. *Ann Intern Med* 1988;109: 18–24.
- [49] Flamme CH. Obesity and low back pain: biology, biomechanics and epidemiology. *Orthopade* 2005;34:652–7.
- [50] Patucchi E, Fatati G, Puxeddu A, Coaccioli S. Prevalence of fibromyalgia in diabetes mellitus and obesity. *Recenti Prog Med* 2003;94:163–5.
- [51] Sowers JR. Obesity as a cardiovascular risk factor. *Am J Med* 2003;115(S8):37S–41S.
- [52] Pischon T, Sharma AM. Use of beta-blockers in obesity hypertension: potential role of weight gain. *Obes Rev* 2001;2:275–80.
- [53] Scholze J, Grimm E, Herrmann D, Unger T, Kintscher U. Optimal treatment of obesity-related hypertension: the Hypertension–Obesity–Sibutramine (HOS) study. *Circulation* 2007;115:1991–8.
- [54] Sin DD, Jones RL, Man SF. Obesity is a risk factor for dyspnea but not for airflow obstruction. *Arch Inter Med* 2002;162:1477–81.
- [55] Poulain M, Doucet M, Major GC, Drapeau V, Sériès F, Boulet LP, et al. The effect of obesity on chronic respiratory diseases: pathophysiology and therapeutic strategies. *CMAJ* 2006;174:1297–9.
- [56] Hedberg A, Rossner S. Body weight characteristics of subjects on asthma medication. *Int J Obes Relat Metab Disord* 2000;24:1217–25.
- [57] Forslund HB, Lindroos AK, Blomkvist K, Hakeberg M, Berggren U, Jontell M, et al. Number of teeth, body mass index, and dental anxiety in middle-aged Swedish women. *Acta Odontol Scand* 2002;60:346–52.
- [58] Hampel H. Meta-analysis: obesity and the risk for gastroesophageal reflux disease and its complications. *Ann Intern Med* 2005;143:199–211.
- [59] Nishizawa T, Suzuki H, Nomoto Y, Masaoka T, Hosoda H, Mori M, et al. Enhanced plasma ghrelin levels in patients with functional dyspepsia. *Aliment Pharmacol Ther* 2006;24(Suppl 4):104–10.
- [60] Stoa-Birketvedt G, Paus PN, Ganss R, Ingebretsen OC, Florholmen J. Cimetidine reduces weight and improves metabolic control in overweight patients with type 2 diabetes. *Int J Obes Relat Metab Disord* 1998;22:1041–5.

- [61] Norris SL, Zhang X, Avenell A, Gregg E, Schmid CH, Lau J. Pharmacotherapy for weight loss in adults with type 2 diabetes mellitus. *Cochrane Database Syst Rev* 2005;1:CD004096.
- [62] Thamer C, Machann J, Stefan N, Haap M, Schafer S, Brenner S, et al. High visceral fat mass and high liver fat are associated with resistance to lifestyle intervention. *Obesity* 2007;15:531–8.
- [63] Pittas AG, Roberts SB. Dietary composition and weight loss: can we individualize dietary prescriptions according to insulin sensitivity or secretion status? *Nutr Rev* 2006;64:435–48.
- [64] Kokkoris P, Pi-Sunyer FX. Obesity and endocrine disease. *Endocrinol Metab Clin North Am* 2003;32:895–914.
- [65] Plotz CM, Knowlton AI, Ragan C. The natural history of Cushing syndrome. *Am J Med* 1952;13:597–614.
- [66] Orth DN. Cushing syndrome. *N Engl J Med* 1995;332:791–803.
- [67] Lovejoy JC, Bray GA, Greeson CS, Klempere M, Morris J, Partington C, et al. Oral anabolic steroid treatment, but not parenteral androgen treatment, decreases abdominal fat in obese, older men. *Int J Obes Relat Metab Disord* 1995;19:614–24.
- [68] Ness-Abramof R, Apovian CM. Drug-induced weight gain. *Drugs Today* 2005;41:547–55.
- [69] United Kingdom Prospective Diabetes Study Group. United Kingdom Prospective Diabetes Study 24: a 6-year, randomized, controlled trial comparing sulfonylurea, insulin, and metformin therapy in patients with newly diagnosed type 2 diabetes that could not be controlled with diet therapy. *Ann Intern Med* 1998;128:165–75.
- [70] Fonseca V. Effect of thiazolidinediones on body weight in patients with diabetes mellitus. *Am J Med* 2003;115:42S–8S.
- [71] Davidson MB, Peters AL. An overview of metformin in the treatment of type 2 diabetes mellitus. *Am J Med* 1997;102:99–110.
- [72] Van de Laar FA, Lucassen PL, Akkermans RP, Van de Lisdonk EH, Rutten GE, Van Weel C. Alpha-glucosidase inhibitors for type 2 diabetes mellitus. *Cochrane Database Syst Rev* 2005;2:CD003639.
- [73] Amori RE, Lau J, Pittas AG. Efficacy and safety of incretin therapy in type 2 diabetes: systematic review and meta-analysis. *JAMA* 2007;298:194–206.
- [74] Gough SC. A review of human and analogue insulin trials. *Diabetes Res Clin Pract* 2007;77:1–15.
- [75] Zimmermann U, Kraus T, Himmerich H, Schuld A, Pollmächer T. Epidemiology, implications and mechanisms underlying drug-induced weight gain in psychiatric patients. *J Psychiatr Res* 2003;37:193–220.
- [76] Raeder MB, Bjelland I, Emil Vollset S, Steen VM. Obesity, dyslipidemia, and diabetes with selective serotonin reuptake inhibitors: the Hordaland Health Study. *J Clin Psychiatry* 2006;67:1974–82.
- [77] McElroy SL, Frye MA, Altshuler LL, Suppes T, Helleman G, Black D, et al. A 24-week, randomized, controlled trial of adjunctive sibutramine versus topiramate in the treatment of weight gain in overweight or obese patients with bipolar disorders. *Bipolar Disord* 2007;9:426–34.
- [78] Schwartz TL, Nihalani N, Virk S, Jindal S, Chilton M. Psychiatric medication-induced obesity: a review. *Obes Rev* 2004;5:115–21.
- [79] Hopkins RL, Leinung MC. Exogenous Cushing's syndrome and glucocorticoid withdrawal. *Endocrinol Metab Clin North Am* 2005;34:371–84.
- [80] Hirsch MS, Klibanski A. What price progress? Pseudo-Cushing's syndrome associated with antiretroviral therapy in patients with human immunodeficiency virus infection. *Clin Infect Dis* 1998;27:73–5.
- [81] Pischon T, Sharma AM. Use of beta-blockers in obesity hypertension: potential role of weight gain. *Obes Rev* 2001;2:275–80.
- [82] Prentice AM. Alcohol and obesity. *Int J Obes* 1995;19(S5):S44–50.
- [83] Yeomans MR. Effects of alcohol on food and energy intake in human subjects: evidence for passive and active over-consumption of energy. *Br J Nutr* 2004;92:S31–4.
- [84] Suter PM. Alcohol, lipid metabolism and body weight. *Ther Umsch* 2000;57:205–11.
- [85] Risser JA, Vash PD, Nieto L. Does prior authorization of sibutramine improve medication compliance or weight loss? *Obes Res* 2005;13:86–92.
- [86] Adams TD, Gress RE, Smith SC, Halverson RC, Simper SC, Rosamond WD, et al. Long-term mortality after gastric bypass surgery. *N Engl J Med* 2007;357:753–61.
- [87] Sjöström L, Narbro K, Sjöström CD, Karason K, Larsson B, Wedel H, et al. Swedish Obese Subjects Study. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med* 2007;357:741–52.
- [88] Avidor Y, Still CD, Brunner M, Buchwald JN, Buchwald H. Primary care and subspecialty management of morbid obesity: referral patterns for bariatric surgery. *Surg Obes Relat Dis* 2007;3:392–407.