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Adult Obesity

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Introduction

Obesity is a multifactorial disease that is growing in epidemic proportions. It is associated with significant medical risks that may be ameliorated by modest weight loss. Exercise and behavioral modification of diet are the cornerstones of treatment; pharmacotherapy and surgery may be useful adjuncts.

Etiology

It is estimated that 40 to 70% of the variation in obesity within populations is heritable;[1] however, this predisposition can be overridden by environmental cues and behavior (physical activity and nutrient choices). Less common are secondary causes of obesity (Table 69.1).

Genetic Causes

Genetic background strongly influences the risk of obesity, as demonstrated by adoption studies, where adoptees more closely resembled their biologic parents than their adoptive parents,[2] and twin studies that showed a concordance rate in identical twins twice that of fraternal twins.[3] Both positive and negative energy balance studies in twins show greater variations between pairs than within pairs.[4] Although most forms of monogenetic obesity (Table 69.2) are rare to extremely rare, frame shift mutations of the melanocortin-4 (MC4) receptor have been described in 2 to 7% of populations.

More commonly, polygenetic influences are involved. Although extensively studied, candidate genes remain to be identified.[1] These genes may influence food intake, metabolism, energy expenditure, and hormones (Table 69.3).

TABLE 69.1

Etiology of Obesity

Primary
Genetic
Nutritional
Environmental
Secondary
Neural
hypothalamic lesions
amygdala lesions
temporal lobe lesions
Endocrine
oophorectomy
insulinoma, insulin therapy
Cushing's syndrome, corticoid therapy
Pharmacologic
Viral?

TABLE 69.2

Monogenetic Human Obesity

Prader-Willi syndrome
Laurence-Moon-Biedl syndrome
Cohen's syndrome
Kleinfelter's syndrome
Leptin deficiency
Truncated leptin receptor
Proopiomelanocortin deficiency
Melanocortin-4 receptor

Environmental Causes

Nutritional

The role of nutrient intake in promoting obesity is quantitative, qualitative, and temporal. The increasing availability of food, often in excessive serving sizes, promotes hyperphagia. The intake of dietary fat is significantly related to adiposity. Dietary fat is converted to body fat with approximately 25% greater efficiency than carbohydrate. Dietary fat may be less satiating than protein and complex carbohydrates, although foods with a high glycemic index (i.e., rapidly converted to glucose) may stimulate hunger and lead to more frequent eating. The long-chain fatty acid composition of dietary fat influences energy utilization; low ratios of polyunsaturated to saturated fat are associated with lower respiratory quotients (RQ; moles of carbon dioxide produced per mole of oxygen consumed).[5] Low protein diets are utilized less efficiently than high protein diets. The pattern of food intake may play a role in the development of obesity. Widely spaced meals are used less efficiently because of the energy cost of storage. Compared to immediate oxidation, the energy cost of converting glucose into glycogen is 5%, and into fat is 28%.

Inactivity

Physical inactivity is increasing as a result of decreased manual labor, the use of labor-saving devices, and a shift in leisure preferences (television, computers, spectator sports).

TABLE 69.3

Obesity-Related Factors Thought to be Genetically Modulated

Diet Related
Dietary fat preferences
Appetite regulation
Amount and rate of eating
Metabolism/Nutrient Partitioning
Adipose tissue distribution
Adipose tissue lipolysis
Adipose tissue and muscle lipoprotein lipase (LPL) activity
Muscle composition and oxidative potential
Free fatty acid and β-receptor activities in adipose tissue
Capacities for fat and carbohydrate oxidation
Energy Expenditure
Metabolic rate
Dietary induced thermogenesis
Nutrient partitioning
Propensity for physical activity/inactivity
Hormonal
Insulin sensitivity/resistance
Growth hormone status
Leptin action

Psychosocial Factors

A cause-and-effect relationship between low socioeconomic status and obesity has been demonstrated.[6] Societal ideals of desirable weight have various ethnic, cultural, and gender determinants. Emotional distress may promote overeating. The hormonal response to stress has been suggested to promote visceral adiposity.[7]

Secondary Obesity

Endocrine changes secondary to obesity (insulin resistance, decreased growth hormone secretion, blunted prolactin responsiveness, hyperparathyroidism, decreased serum testosterone in men) may make it difficult to determine whether obesity is primary or secondary. Secondary obesity may result from hypothyroidism, Cushing's syndrome, insulinoma, hypogonadism, Frolich syndrome, hypothalamic tumors, head injury, or drugs (Table 69.4). There are several known animal models of viral-induced obesity. Antibodies to human adenovirus AD-36 (capable of producing obesity in chickens and mice) have been observed in some obese humans.[8]

Energy Balance

Within groups, there is not a correlation between energy intake and body weight; for the individual, intake does correlate with weight. Therefore, obesity can be viewed as a

TABLE 69.4

Drugs That May Promote Weight Gain

Tricyclic antidepressants
Lithium
Sulfonylureas
Thiazolidinediones
β-adrenergic blockers
Some steroid contraceptives
Corticosteriods
Insulin
Cyproheptadine
Sodium valproate
Neuroleptics

disorder of energy homeostasis. This variability may be either innate or acquired, and may be the result of hyperphagia, energy partitioning, intermediary metabolism, the efficiency of the coupling of electron transport to ATP generation, the efficiency of ATPases, degree of physical activity, the magnitude of adaptive thermogenesis, hormonal influences on energy expenditure, and physiological demands (growth, pregnancy, lactation).

Appetite Regulation

The major loci in the central nervous system that regulate feeding behavior are the dorsomedial, paraventricular, arcuate, and lateral nuclei of the hypothalamus, the prefrontal cortex, the amygdala, the nucleus accumbens, and the nucleus of the solitary tract. These sites respond to a variety of stimuli including deprivation, intracellular glucose concentration, intracellular fat oxidation, food choice, meal timing, desire, mood, stress, metabolic rate, fidgeting, fat stores (via leptin), and ingestive behavior. These areas communicate via complex interactions of neuromodulators that either suppress or stimulate feeding (Table 69.5). Many of these peptides are found in the gut as well as the brain.

Appetite regulation can also be viewed as several interacting feedback loops. The glucostat theory involves glucose-sensitive neurons that stimulate appetite under conditions of low glucose levels, and glucoreceptors in the liver that provide the afferent signal via the vagus nerve. Leptin, a protein produced by adipocytes, appears to drive the adipostat, which is thought to measure the adequacy of fat stores. Thermogenesis in brown adipose tissue has been proposed as a thermostatic regulator of food intake.[9]

Energy Partitioning

Genetic, hormonal, nutritional, and physical activity factors influence the partitioning of energy between fat and fat-free mass, as well as preference for carbohydrate versus fat oxidation to meet energy needs. A high RQ has been shown to predict future weight gain.[10,11] Obese subjects have an attenuation of both basal- and epinephrine-stimulated rates of lipolysis[12] that may in part result from hyperinsulinemia. Dietary fat intake may also influence substrate utilization, with low ratios of polyunsaturated to saturated fats resulting in lower RQs.[13]

A propensity to excessive fat stores may result from increased LPL activity and peroxisome proliferator-activating receptor (PPAR) abnormalities. Lipoprotein lipase activity is increased in obesity, although it is unclear if this increase is a cause or result of obesity. Adipose tissue LPL activity increases during caloric restriction and may lead to rapid weight

TABLE 69.5

Neuromodulators of Appetite Regulation

Stimulatory
Norepinephrine (α-2)
Endogenous opioids
Dopamine (physiologic levels)
Neuropeptide Y
Peptide YY
Orexins
Galanin
Agouti-related protein
Melanin-concentrating hormone
Inhibitory
Norepinephrine (β)
Epinephrine (β)
Dopamine (supraphysiologic)
Serotonin
Cholecystokinin
Somatostatin
Glucagon, glucagon-like protein (GLP-1)
Urocortin
Corticotropin-releasing hormone (CRH)
Melanocortin agonists (e.g., proopiomelanocortin, melanocyte-stimulating hormone)
Cocaine/amphetamine regulated transcript
Leptin

regain when caloric restriction is abandoned. PPARs promote differentiation of preadipocytes. Mutations in gamma-2 PPAR have been described in some severely obese humans.[14]

Energy Expenditure

Basal metabolic rate (BMR) is the rate of energy expenditure upon awakening, before any physical activity and 12 to 18 hours after the last meal. More commonly assessed is resting energy expenditure (REE), which is obtained in the resting state several hours after the last meal. The difference between BMR and REE is small. This energy component is expended for maintenance of body functions and homeostasis (primarily proton pumping and protein turnover) and accounts for 60 to 75% of total energy expenditure in sedentary individuals. Subjects with low REE gain more weight than persons with normal or elevated REE.[15] The REE is increased in obesity, related to the individual's increase in lean body mass. REE increases with overfeeding. With caloric restriction, REE is decreased by both the caloric deficit and the resultant loss in lean tissue. Exercise may help minimize REE decrease. Physical training, i.e., being in the "trained state," can also increase REE independent of body composition or the residual effects of the last bout of exercise.[16]

Energy expenditure on physical activity is the most variable, and is the only component of energy balance that is under volitional control. It includes shivering and fidgeting, as well as physical work. It may range from less than 100 kcal/day in sedentary individuals to greater than 1000 kcal/day with strenuous exericise or labor. Exercise efficiency (energy expenditure/unit work) is not altered by obesity, but because of carrying excess weight, more energy will be expended during weight-bearing activities. In general, the obese are less physically active than the lean. The influence of physical activity on obesity, indepen-

dent of genotype, has been examined in twins, where discordance for obesity was associated with discordance in activity level.[17]

The thermic effect of food (TEF), also known as diet-induced thermogenesis, is the energy cost of food digestion, absorption, metabolism, and storage, as well as a component resulting from sympathetic nervous system activity. It is lowest for fat and highest for excess carbohydrate stored as fat and protein. It may account for up to 10% of daily energy expenditure and consists of both obiligatory and facultative components. The latter may be decreased in obesity. Weight loss does not normalize TEF and may contribute to weight regain.[18-19] However, longitudinal studies have demonstrated declines in glucose-induced thermogenesis with the evolution of obesity.[20] With caloric restriction, the TEF declines.

Exercise may potentiate TEF, especially in insulin-sensitive subjects. Although study results are inconsistent, at least some obese individuals have lesser magnitudes of energy expenditure than their lean counterparts.

Adaptive thermogenesis is influenced by both genetic and environmental (ambient temperature, food intake, emotional stress) factors and usually accounts for 10 to 15% of total energy expenditure. Mechanisms include changes in the efficiency of oxidative phosphorylation, rates of protein turnover, Na pump activity and futile cycles, and activity/inactivity of brown fat (nonshivering thermogenesis). Examples of futile cycles which waste ATP are:

- Glucose to pyruvate to glucose
- Cyclic lipolysis and reesterification of triglycerides
- Glucose to lactate to glucose (Cori cycle)
- Glucose to glucose-6-phosphate to glucose
- Fructose-6-phosphate to fructose-di-phosphate to fructose-6-phosphate
- Pyruvate to phosphoenolpyruvate to pyruvate

Wastage of ATP may also result directly by phosphatases converting ATP to ADP.

Non-shivering thermogenesis as a mechanism of heat production is well established in hibernating animals and infants of various species, although its contribution to adult human obesity is controversial. This thermogenesis occurs in mitochondria of brown adipose tissue, where an uncoupling protein (uncoupling protein-1, or UCP-1) facilitates a proton leak, and thus fat oxidation is dissociated from oxidative phosphorylation. Recently, additional UCPs have been discovered (currently, five are known). Several of the UCPs have genetic linkage to human obesity, but the importance in obesity remains to be delineated. A polymorphism in the UCP-3 gene has been associated with altered REE.[21]

Prevalence of Obesity

Overweight and obesity are increasing dramatically globally. There has been a greater than 25% increase in the U.S. in the past three decades.[22] The CDC reports an astounding 49% increase in obesity in young adults from 1991 to 1998.[23] For U.S. adults, 42% of men and 28% of women are overweight (body mass index [BMI] ≥25 to 29.9); and 21% of men and 27% of women are obese (BMI ≥30).[22] African- and Hispanic-Americans have a higher prevalence of obesity than Anglo-Americans.

Assessment

Assessment of the patient should include the BMI, waist circumference (measured at the level of the iliac crest), and overall medical risk. Bioimpedance analysis is a simple noninvasive technique that measures total body water and total fat, and calculates lean body mass. Other methods of assessment (underwater weighing, doubly labeled water, calorimetry, dual-energy x-ray absorptiometry [DEXA], computerized tomography [CT] scan, and magnetic resonance imaging [MRI]), are expensive and inaccessible to most practitioners. Although skinfold measurements are inexpensive, they have poor reproducibility, especially with increasing obesity.

Body Mass Index

The BMI is highly correlated with fatness, and minimizes the effect of height. It is calculated as:

$$\text{BMI} = \text{wt (in kg)}/\text{ht}^2 \text{ (in meters) or} \qquad \text{(Eq. 69.1)}$$
$$\text{BMI} = \text{wt (in lb)} \times 703/\text{ht}^2 \text{ (in inches)}$$

As an index of mass, it does not distinguish between fat and fat-free mass. Consequently, it is possible to be overweight without having excess adiposity (very muscular individuals) as well as obese without being overweight (sarcopenic individuals). Table 69.6 presents classification for BMI.

Fat Distribution

In addition to total adiposity, distribution of body fat has medical implications (Table 69.7), with abdominal (visceral) fat presenting a greater health risk than gluteal-femoral fat.[24] A waist circumference >40 inches in men or >35 inches in women reflects excess abdominal fat. Gluteal-femoral fat, which is thought to be estrogen dependent, serves as an energy store for lactation and increases during each pregnancy. Following menopause, this fat depot decreases, while intraabdominal fat increases.

Insulin Resistance

Given the key role of hyperinsulinemia on the medical risks of obesity (see below) assessment of insulin resistance is desirable. Most sensitive is the insulin suppression test, although its performance is not practical in the usual care setting. Fasting plasma insulin levels, although less sensitive, are a useful guide.

TABLE 69.6

Classification for BMI

BMI	Weight Classification
18.5-24.9	Normal weight
25.0-29.9	Overweight
30.0-34.9	Class 1 obesity
35.0-39.9	Class 2 obesity
≥40	Class 3 obesity

TABLE 69.7

Metabolic Consequences of Upper Body Obesity

Increased insulin secretion
Decreased hepatic clearance of insulin
Insulin resistance
Increased lipolysis
Increased circulating free fatty acids
Increased free fatty acid oxidation
Increased gluconeogenesis and decreased glucose utilization
Effects of hyperinsulinemia (see Table 69.8)
Increased free testosterone and free androstenedione levels associated with decreased sex hormone-binding globulin in women
Decreased progesterone levels in women
Decreased testosterone levels in men
Increased cortisol production

Medical Risks of Obesity (Comorbidities)

The medical consequences of obesity may result from hyperinsulinemia, mechanical effects of excess weight, and alterations in sex hormones. Especially damaging is hyperinsulinemia (Table 69.8). Increased insulin secretion is related to total body fat; however, decreased hepatic insulin clearance is related specifically to the amount of abdominal fat (may be related to increased androgen effects on the liver). Exhaustion of pancreatic reserves may lead to impaired glucose tolerance. Glucose intolerance relates to increased intra-abdominal visceral fat as opposed to subcutaneous abdominal fat.[25] Other comorbidities associated with hyperinsulinemia include hypertension, hyperlipidemia, atherosclerotic disease, stroke, and polycystic ovarian syndrome.

Mechanical consequences of obesity include congestive heart failure, sleep apnea, restrictive lung disease, surgery risks (pneumonia, wound infection), high risk pregnancy, cellulitis, degenerative arthritis, and steatohepatitis. Alterations in sex hormones contribute to decreased fertility, menorrhagia, oligomenorrhea, and certain cancers (breast, uterus).

Comorbidities are present in approximately two-thirds of subjects with a BMI >27. Hypertension is the most common obesity-related health risk and its prevalence increases markedly with increasing levels of obesity, as does the incidence of type 2 diabetes, gallbladder disease, and osteoarthritis. Hypercholesterolemia, while more common in the overweight and obese than in normal weight individuals, does not show an increase with increasing levels of obesity. However, the risk of coronary heart disease in women does increase with increasing level of obesity.[22,26] The risk for death from cardiovascular disease, cancer, or other diseases increases with increasing degrees of obesity.[27,28] For more extensive detail, the reader is referred to the NIH Clinical Guidelines[29] or the WHO Consultation on Obesity Report.[30] A modest (5 to 15% of initial weight) sustained weight loss will improve many of the health complications of obesity.[31-36]

Treatment

The most effective weight-loss programs combine diet, exercise, behavior modification, and social support. The patient's motivation can be assessed using the Diet Readiness

TABLE 69.8

Effects of Hyperinsulinemia

Renal
Uric acid — increased production, decreased clearance
Decreased potassium and sodium excretion
Lipid Metabolism
Increased VLDL
Decreased HDL
Decreased clearance of chylomicron remnants and IDL
Increased postprandial lipemia
Increased small, dense LDL
Increased "oxidizabilty" of LDL
Decreased lipolytic activity
Glucose Metabolism
Glucose intolerance
Nervous System
Increased sympathetic activity
Cardiovascular
Increased heart rate
Hypertension
Increased plaque formation
Decreased plaque regression
Smooth muscle and connective tissue proliferation
Enhanced LDL receptor activity
Hemostasis
Increased PAI-1
Increased fibrinogen
Increased von Willebrand factor
Increased factor X
Increased adhesion of mononuclear cells to endothelium
Gonadal
Polycystic ovaries

Questionnaire.[37] For descriptions of popular weight loss programs, see *Weighing the Options*,[38] pg 66-80.

Goals

Practitioners should help patients to set realistic goals to help prevent the patient from being overwhelmed or relapsing. Without guidance, most patients choose goals based on cosmetic criteria, that are usually unachievable. A reasonable or healthy weight loss goal needs to take into account health risks, genetic predisposition to obesity, and whether the patient has hyperplastic (excess fat cell number) versus hypertrophic (excess fat cell size) obesity. When fat cells hypertrophy, they develop insulin resistance, resulting in increased medical risk. When adipocytes reach some maximum size (approximately double optimal

TABLE 69.9

NIH Guidelines for Choosing a Weight-Loss Program

The diet should be safe and include all the Recommended Dietary Allowances for vitamins, minerals, and protein
The program should be directed towards a slow, steady weight loss unless a more rapid weight loss is medically indicated
A doctor should evaluate health status if the client's weight-loss goal is greater than 15 to 20 lb, if the client has any health problems, or if the client takes medication on a regular basis
The program should include plans for weight maintenance
The program should give the prospective client a detailed list of fees and costs of additional items

size), there is a stimulus to proliferate. Hyperplastic obesity is common in patients with childhood/adolescent-onset obesity, morbid obesity, and in some cases of excessive weight gain during pregnancy. Because it is impossible to reduce the number of adipocytes, an appropriate goal for the patient with hyperplastic obesity is to reduce excess fat by approximately one-half.

For patients with a BMI >30, a 10% weight loss over six months (i.e., 1 to 2 lb or 0.5 to 1.0 kg/wk) is a reasonable goal. Slower weight loss is appropriate for those with lesser degrees of obesity. For overweight subjects who are not motivated to lose weight, the goal should be prevention of further weight gain.

Criteria for Choosing a Weight Loss Program

Guidelines for choosing a weight loss program have been developed by the NIH (Table 69.9) and The Institute of Medicine.[38]

Diet

Diets should be individually planned to help create a deficit of 500 to 1000 kcal/day. Succcessful weight reduction is more likely to occur when consideration is given to a patient's food preferences in tailoring a particular diet. The dietitian should ensure that all of the recommended dietary allowances are met; this may necessitate the use of a dietary or vitamin supplement. The diet should also be realistic, i.e., based on dietary modification and practical changes in eating habits. The nutritional recommendations should be based on the patient's current eating habits, lifestyle, ethnicity and culture, other coexisting medical conditions, and potential nutrient-drug interactions.

The diet should be prescribed by the physician and implemented by the dietitian. The active involvement of the physician in such cases is essential, while the role of the dietitian can be invaluable, since caloric intake should be evaluated monthly. Food records should be completed by the patient to assess the relationship of caloric intake to weight loss. However, changes in body weight may not reflect changes in body fat if the patient has edema or has been adding muscle tissue due to an aggressive exercise program. The rate of weight loss can be expected to decline as the patient's energy requirements decline.

Low Calorie Diets (LCD)

Caloric restriction is an integral component of weight loss regimens. The restriction can be moderate to severe; however, compliance decreases with unrealistic restrictions. In general, a 500 to 1000 kcal reduction from maintenance caloric requirements is recommended. Maintenance requirements may be determined by the resting energy equation (REE) recommended by Mifflin et al.[39]

$$\text{REE} = (9.99 \times \text{wt in kg}) + (6.25 \times \text{ht in cm}) - (4.92 \times \text{age in yr}) + (166 \times \text{sex [male = 1; female = 0]}) - 161 \quad \text{(Eq. 69.2)}$$

Multiply the REE by an activity factor (1.5 for women; 1.6 for men) to determinine maintenance requirements.[40]

The Harris-Benedict equation can also be used to calculate REE or basal metabolic rate (BMR); however, this equation overpredicts REE by 5 to 24%.[39] The Harris-Benedict equations are:[40]

$$\text{BMR for males} = 66 + 13.8\ (\text{wt in kg}) + 5\ (\text{ht in cm}) - 6.8\ (\text{age in yr}) \quad \text{(Eq. 69.3)}$$

$$\text{BMR for females} = 655 + 9.6\ (\text{wt in kg}) + 1.8\ (\text{ht in cm}) - 4.7\ (\text{age in yr}) \quad \text{(Eq. 69.4)}$$

Subtract 500 to 1000 kcal to determine the caloric intake needed to achieve a weight loss of approximately 1 to 2 pounds per week.[38]

Implementation of Diet

The dietitian usually uses the exchange system, or the Food Guide Pyramid, to prescribe a specified number of exchanges (or servings) of foods from each food group, and a defined portion size for each food. Thus, weighing and measuring foods is an important requirement in terms of patient compliance. The subject can choose a variety of foods within each food group, and some higher calorie foods can be built in, occasionally. Once caloric needs have been determined, Table 69.10 can be used as a guide for various caloric levels. A sample meal plan for a 1500-calorie exchange diet is shown in Table 69.11.

Because overweight individuals need to lose weight over a period of time, it is imperative that the diet be acceptable. The diet must fit the taste preferences and habits of the individual and be flexible enough to allow eating outside the home as well. Dietary education is necessary to assist in the adaptation to an LCD and should address the topics[29] listed in Table 69.12.

Low Calorie, High Fiber Diets

Reducing dietary fat, along with an increase in dietary fiber and a decrease in refined sugars, is a sound program for weight loss as well as weight maintenance, especially when consuming the recommended number of servings from the Food Guide Pyramid. Consuming ample fruits, vegetables, and whole grains can aid in weight loss because increased fiber intake can increase satiety. The National Research Council and the American Heart Association recommend consuming 25 to 35 grams of fiber each day. A fat intake of 20 to 30% of total kcalories is appropriate, as long as total kcalories from refined sugars are not

TABLE 69.10

Food Group Exchanges for Various Caloric Levels

Calorie Level, kcal	1200	1500	1800
Starch group exchanges	5	6	8
Fruit group exchanges	2	3	4
Vegetable group exchanges	3	4	5
Milk group exchanges	2	2	2
Meat group exchanges	5 oz	6 oz	7 oz
Fat group exchanges	≤3	≤4	≤5
Sweets	use sparingly	use sparingly	use sparingly

TABLE 69.11

Sample Meal Plan for 1500 Calories

Food	Calories (kcal)
Breakfast	
Whole wheat toast, 2 slices	140
Banana, 1 small	60
Milk, fat-free, 8 fl. oz	90
Margarine, 2 tsp	90
Coffee/tea	0
Lunch	
Turkey sandwich	
Turkey breast, 2 oz	70
Bread, 2 slices	140
Mayonnaise, fat-free, 1 Tb	10
Lettuce, 1 leaf	0
Mini carrots, raw, 1 cup	25
Yogurt, fat-free, vanilla, 1 cup	200
Coffee/tea	0
Supper	
Fish, baked, 4 oz	140
Potato, baked, 1 med (6 oz)	160
Sour cream, light, 2 Tb	40
Broccoli, steamed, 1 cup	50
Margarine, 1 tsp	45
Strawberries, fresh, 1 1/4 cup	60
Whipped topping, fat-free, 2 tb	15
Coffee/tea	0
Snack	
Popcorn, air-popped, 6 cups	180
Total calories	1515

TABLE 69.12

Educational Topics for Weight Loss Counseling

Energy value of different foods
Food composition — fats, carbohydrates (including dietary fiber), and proteins
Evaluation of nutrition labels to determine caloric content and food composition
New habits of purchasing — give preference to low-calorie foods
Food preparation — avoid adding high-calorie ingredients during cooking (e.g., fats and oils)
Avoid overconsumption of high-calorie foods (both high-fat and high-carbohydrate foods)
Maintain adequate water intake
Reduction of portion sizes
Limiting alcohol consumption

excessive. Reducing the percentage of dietary fat alone will not produce weight loss unless total kcalories are also reduced.[29] Although lower-fat diets without targeted caloric reduction help promote weight loss by producing a reduced kcalorie intake, lower-fat diets with targeted caloric restriction promote greater weight loss than lower-fat diets alone.[29]

TABLE 69.13

Sample Meal Plan for Low-Fat, High-Fiber Diet

Food	Calories (kcal)	Fat (g)	Fiber (g)
Breakfast			
Orange juice, 1/2 cup	56	0	0.5
Fiber One cereal, 1/4 cup	30	0.5	6.5
Fruit 'n Fiber cereal, 1/2 cup	105	1.5	3
Banana, 1/2 med	52	0.2	1.3
Milk, fat-free, 8 fl oz	86	0.4	0
Whole wheat toast, 1 slice	65	2	2
Margarine, light, 1 tsp	17	2.7	0
Coffee/tea	0	0	0
Lunch			
Split pea soup, 1 cup	133	1.6	3.7
Triscuit crackers, reduced fat, 8	130	3	4
Chicken breast, grilled, skin removed, 2 oz	95	2.1	0
Hamburger bun, 1	123	2.2	1.2
Honey mustard, 1 Tb	25	0	0
Sliced tomato, 1/2 med	13	0.2	0.7
Lettuce, 1 leaf	2	0	0.3
Pear, 1 med	98	0.7	4
Coffee/tea	0	0	0
Supper			
Spinach salad, 1 cup	12	0.2	1.6
Salad dressing, light, 2 Tb	100	8	0
Grouper, baked, 4 oz	133	1.4	0
Brown rice, 1/2 cup	108	0.9	1.8
Asparagus, steamed, 6 spears	22	0.3	1.4
Whole wheat roll, 1	75	1.3	2.1
Margarine, light, 2 tsp	34	5.4	0
Yogurt, fat-free, vanilla, 1 cup	200	0	0
Strawberries, 1 cup	45	0.6	3.4
Coffee/tea	0	0	0
Totals	1759	35.2[a]	37.5

[a] Fat calories provide 18% of total kcalories.

Implementation of Diet

The dietitian should prescribe a caloric level appropriate for a weight loss of one-half to one pound per week. The dietitian should also recommend the fat intake (20 to 30%) and the fiber goal (25 to 35 g). Advising the patient to gradually increase fiber in the diet will help avoid gastrointestinal side effects such as gas, cramps, and bloating. Increasing fluid intake while increasing fiber intake will prevent constipation. Educating the patient on high-fiber cereals, eating the peels on apples and potatoes, and eating the whole fruit rather than just drinking the juice will help fulfill the requirement for fiber. Beans are also an excellent low-fat source of fiber. Patients should be encouraged to keep food records of total kcalories, fat, and fiber to monitor compliance. Recommendations of references for counting calories, fat, and fiber should be given to the patient along with sample meal plans. A sample meal plan is shown in Table 69.13.

TABLE 69.14

Contraindications to Very Low Calorie Diets (VLCDs)

Recent myocardial infarction
Cerebrovascular disease
Chronic renal failure
Hepatic disease
Type I diabetes mellitus
Severe psychiatric disorders
Alcoholism
Cancer
Infection
Acute substance abuse
Human immunodeficiency virus infection

Very Low Calorie Diets (VLCDs)

A very low calorie diet (VLCD) is defined as one that provides <800 kcalories per day. Such diets may severely restrict carbohydrates and induce ketosis (ketogenic diets) or may simply restrict all macronutrients, and can be either a liquid formulation or a food diet.[41] The following discussion will refer to ketogenic VLCDs. Ketosis produces anorexia and thus improves dietary compliance. This diet is appropriate only when a patient has a major health risk(s) and the physician has determined that the diet can be used safely. Indications for patients for VLCDs are a BMI ≥35, or a BMI ≥30, in association with comorbid conditions. The natriuresis associated with ketosis and the rapid reduction in insulin resistance make the diet especially useful in patients with fluid overload and diabetes, respectively. Candidates for VLCDs should have failed prior weight-loss attempts and should demonstrate motivation to adhere to the VLCD. Additionally, the patient should understand that this is a temporary method for weight loss, and that transitioning to a more balanced eating pattern will be necessary for further weight loss and weight maintenance. Patients should not follow this diet for more than 12 to 16 weeks. Contraindications for use of VLCDs are listed in Table 69.14.

VLCDs are not recommended for weight-loss therapy for most patients because they require special monitoring and supplementation.[42] Specialized practitioners experienced in the use of VLCDs are preferable for screening and supervising patients for this diet. Medical monitoring will help the physician detect any patients who may react adversely to the VLCD. Potential complications include excessive loss of lean body mass, orthostatic hypotension, constipation (inadequate fiber in diet), gout (ketones compete with uric acid for excretion), and a likelihood for recidivism. Diuretics should be discontinued to minimize the increased risk of electrolyte imbalance,[43] and diabetic drugs will need to be reduced or discontinued. Clinical trials show that LCDs are as effective as VLCDs in producing sustained weight loss after one year.[44] As with any type of weight loss program, including behavioral therapy and physical activity along with the VLCD seems to improve maintenance of weight loss.[42,45]

Implementation of Diet

The VLCD should provide 1.2 to 1.5 g protein/kg of desirable body weight per day. This protein must be of high biologic value in order to maximize preservation of lean body mass. Lean meat, fish, poultry, and egg whites are recommended. The dietitian determines the protein needs of the patient and converts grams of protein to ounces of meat (7 g protein = 1 oz meat). The meat is divided into three meals per day, and the patient is encouraged not to skip meals. The patient is given the following directions:

TABLE 69.15

VLCD Sample Meal Plan

Breakfast

Lean ham, 4 oz
Coffee, 8 fluid oz

Lunch

Lean ground beef, 5 oz
Carrots, raw, 2 whole
Soda, diet, 12 fluid oz

Supper

Baked chicken breast, skinless, boneless, 6 oz
Green beans, 1/2 cup cooked
Tea, sugar substitute, 12 fl oz

Drink other fluids throughout the day

- Choose only lean meats
- Prepare meats without adding fats, breading, or sauces
- Weigh meat after cooking to comply with prescribed amount
- Include two servings non-starchy vegetables per day
- Drink at least two quarts of non-caloric fluids per day
- Take one multiple vitamin-mineral supplement per day
- Take a calcium supplement providing 1000 to 1500 mg elemental calcium per day
- Test urine for ketones one time per day with KetoStix (available over the counter)

A sample meal plan is shown in Table 69.15 for a VLCD providing 15 oz meat per day.

In the case of liquid diets, the protein should be from dairy sources, soy, or albumin. Most liquid formulations provide between 0.8 and 1.5 g protein/kg of desirable body weight, up to 100 g carbohydrate, the minimum of essential fatty acids, and the recommended allowances of vitamins and minerals.

Refeeding, the process of gradual weaning from the VLCD back to a balanced diet, generally takes three to six weeks. The dietitian should follow the patient closely at this time and gradually increase the daily caloric intake, because the decline in resting metabolic rate usually continues for about three months after the VLCD has been discontinued.[43] Patients should be informed that some water weight will most likely be regained when they return to a balanced diet (reversal of the natriuresis associated with ketosis).

High-Protein, Low-Carbohydrate Diets

Diet books such as *Dr. Atkins New Diet Revolution, Protein Power, The Carbohydrate Addict's LifeSpan Program, Sugar Busters,* and *The Zone* all emphasize protein and/or limit carbohydrates or sugar. These diets are similar in some respects to the VLCDs, but typically are not medically supervised. Many of these diets allow/encourage excessive fat and protein, and the authors often suggest that these diets can be followed indefinitely. The diets are based on the idea that carbohydrates are bad, and that many people are insulin-

resistant and therefore gain weight when they eat carbohydrates. Proponents espouse severely limiting carbohydrates to force the body to use the fat it already has in storage for energy instead of adding to those fat stores. The authors of these books usually conflict with most mainstream nutritional professionals who recommend ample servings of carbohydrates, especially complex carbohydrates in the form of whole grains, vegetables, and fruit. The authors of these diet books are quick to point out that because of excessive carbohydrate consumption, people are heavier than ever before. However, they don't address the real reason people are overweight — that they are eating more total kcalories and are more sedentary.

Consequences of Dieting

Caloric restriction produces a natriuresis and diuresis that is reversed with resumption of higher kcalorie levels. This may lead to discouragement and abandonment of dietary efforts. With severe caloric restriction, adaptation to starvation produces hypometabolism. Loss of lean tissue will also reduce the REE. Weight loss also produces a reduced capacity for fat oxidation.[46] However, the evidence thus far does not support adverse effects of weight cycling on REE, body composition, or body fat distribution.[47]

Temporary consequences of weight loss include secondary amenorrhea and hair loss. Gallstones may develop during weight loss; a large percentage of these dissolve spontaneously.

Exercise

Adding exercise to a calorie-restricted diet marginally increases weight loss but minimizes the decline in the REE due to the caloric deficit. The major benefits of exercise are its effects on health, mood, and maintenance of weight loss (Table 69.16). The slightly greater proportion of fat used with low-intensity aerobic exercise is offset by the greater total energy

TABLE 69.16

Proposed Mechanisms Linking Exercise with Successful Weight Maintenance

Increased energy expenditure
Improved body composition
Fat loss
Preservation of lean body mass
Reduction of visceral fat depot
Increased capacity for fat mobilization and oxidation
Control of food intake
Short-term reduction of appetite
Reduction of fat intake
Stimulation of thermogenic response
RMR
Diet-induced thermogenesis
Change in muscle morphology and biochemical capacity
Increased insulin sensitivity
Improved plasma lipid and lipoprotein profile
Reduced blood pressure
Better aerobic fitness
Positive psychological effects
Improved mood
Improved self-esteem
Increased adherence to diet

TABLE 69.17

Behavior Modification Techniques

Self monitoring — using food and exercise diaries
Stimulus control — keeping food out of sight
Stress management
Social support
Eating management — eating slower
Behavior substitution — exercising instead of eating
Rewards
Relapse prevention
Cognitive restructuring — positive self-talk
Environmental engineering — eating only at the table
Covert sensitization — imagining unpleasant consequences

expended by high-intensity aerobic exercise. Physical training increases oxidation of fatty acids. By increasing muscle mass, resistance exercise is also beneficial.

Behavior Modification

Behavior modification refers to tools or skills used to improve compliance with diet and exercise regimens. Table 69.17 outlines several approaches. Behavioral therapy is an essential component of any adequate obesity treatment program.

Surgery

Surgical options for the treatment of obesity have been reviewed by Kral.[48] The most common are gastroplasty (creation of a small gastric pouch with restricted outlet along the lesser curvature of the stomach) and gastric bypass (construction of a proximal gastric pouch whose outlet is a Roux-en-Y limb of small intestine). Both procedures produce a >50% reduction of excess weight in the majority of patients, with the bypass having superior results. Additional procedures include gastric banding (adjustable band creating a proximal gastric pouch) and biliopancreatic diversion. Jejunoileal bypasses, which have produced severe complications, are not recommended. Candidates for surgical treatment are those with severe obesity (BMI >35 with comorbidities or >40 without comorbidity) who are well informed and highly motivated, but have failed prior dietary attempts.

Drugs

To be considered for pharmacotherapy, subjects should have a BMI ≥30 without risk factors, or a BMI of ≥27 with obesity-related comorbidities. Centrally acting noradrenergic agents are approved for short-term use, whereas both sibutramine and orlistat are approved for long-term use (Table 69.18). Common side effects of noradrenergic agents are headache, insomnia, nervousness, irritability, and increased blood pressure and pulse. In addition to these effects, sibutramine may cause dry mouth. Side effects of lipase inhibitors are a consequence fat malabsorption and may include abdominal pain, diarrhea, oily stools, fecal incontinence, and malabsorption of fat-soluble vitamins.

The likelihood of long-term effectiveness can be predicted by weight loss during the first month of therapy. Weight loss drugs are not a substitute for a healthy diet and regular exercise, nor are they a cure for obesity. They can, however, promote modest weight loss sufficient to improve health risks.

TABLE 69.18

Weight Loss Agents

Drug	Usual Dose per Day
Noradrenergic Agents	
Phendimetrazine	105 mg
Phentermine	15-37.5 mg
Mazindol	1-3 mg
Diethylproprion	75 mg
Adrenergic/Serotonergic Reuptake Inhibitors	
Sibutramine	5-15 mg
Gastrointestinal lipase inhibitors	
Orlistat	120 mg tid

Outcomes

Treatment outcomes have been reviewed by Brownell and Wadden.[49] Predictors of weight loss and weight maintenance are listed in Tables 69.19 and 69.20, respectively.

TABLE 69.19

Predictors of Weight Loss

Positive Predictors
Personal Factors
High initial body weight or BMI
High REE
High self-management skills
Process Factors
Attendance at program
Weight loss early in program
Treatment Factors
Increased length of treatment
Having social support
Engaging in physical activity
Incorporation of behavior modification techniques
Self-monitoring
Goal setting
Slowing rate of eating
Negative Predictors
Repeated attempts at weight loss
Experiencing perceived stress
(Others include the opposites of the positive indicators)
Nonpredictors
Total body fat, fat distribution, and body composition
Personality/psychopathology test results
Dietary restraint
Binge eating

TABLE 69.20

Predictors of Maintenance of Weight Loss

Positive Predictors
Physical activity
Self-monitoring
Positive coping style
Continued contact
Normalization of eating
Reduction of comorbidities
Negative Predictors
Negative life events
Family dysfunction

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Aggregate Database for Investigations on Publications in Obesity and Sequelae (ADIPOS), www.adipos.com

American Council on Exercise, 5820 Oberlin Dr., Suite 102, San Diego, CA 92121, (800) 825-3636, www.acefitness.org

American Dietetic Association, 216 West Jackson Blvd., Chicago, IL 60606, (800) 887-1600, www.eatright.org

American Heart Association, 7272 Greenville Ave., Dallas, TX 75231, (800) AHA-USA1, www.americanheart.org

American Obesity Association, 1250 24th St. NW, Suite 300, Washington, DC 20037, (800) 98-OBESE, www.obesity.org

Center for Nutrition Policy and Promotion, 1120 20th St. NW, Washington, DC 20036, (202) 418-2312, www.usda.gov/cnpp

International Food Information Council, 1100 Connecticut Ave. NW, Suite 430, Washington, DC 20036, (202) 296-6540, www.ific.health.org

Shape Up America, 901 31st St. NW, Washington, DC 20007, (202) 333-7400, www.shapeupamerica.org

Weight-Control Information Network (WIN), 1 Win Way, Bethesda, MD 20892-3665. 1-800-WIN-8098. www.niddk.nih.gov/health/nutrit/win.htm