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Obesity: Responding to the Global Epidemic

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Obesity has reached epidemic proportions in the United States and other developed nations. In the United States, 27% of adults are obese and an additional 34% are overweight. Research in the past decade has shown that genetic influences clearly predispose some individuals to obesity. The marked increase in prevalence, however, appears to be attributable to a toxic environment that implicitly discourages physical activity while explicitly encouraging the consumption of supersized portions of high-fat, high-sugar foods. Management of the obesity epidemic will require a two-pronged approach. First, better treatments, including behavioral, pharmacologic, and surgical interventions, are needed for individuals who are already obese. The second and potentially more promising approach is to prevent the development of obesity by tackling the toxic environment. This will require bold public policy initiatives such as regulating food advertising directed at children. The authors call not for the adoption of a specific policy initiative, but instead propose that policy research, based on viewing obesity as a public health problem, become a central focus of research.

Research on obesity has increased exponentially during the past decade and has yielded remarkable discoveries in the regulation of body weight. This same period, however, has witnessed an unparalleled increase in the prevalence of obesity, making it one of our nation's most pressing health problems. This article reviews the epidemiology and complications of obesity and the factors that have contributed to its sharp increase. Consistent with previous reviews, we examine advances in treatment including behavioral, pharmacologic, and surgical interventions (Brownell, 1982; Brownell & Wadden, 1992). Advances in treatment, however, must be complemented in the next decade by innovative public policy designed to prevent the development of obesity.

Epidemiology and Complications of Obesity

Obesity refers to an excess of body fat, which normally accounts for about 25% of weight in women and 18% in men (Bray, 1998). Body fat can be measured by a number of methods but is now usually estimated by the body mass index (BMI), which is calculated as weight in kilograms divided by height in meters squared (i.e., kg/m^2; Bray, 1998). (Figure 1 provides for this calculation using pounds and inches.) The BMI is highly correlated with measures of body fat and has replaced the more traditional measure of percentage over ideal weight (VanItallie & Lew, 1992).

The World Health Organization (WHO; 1998) has defined obesity as a BMI $\geq$ 30 kg/m^2, as has the National Institutes of Health/National Heart, Lung, and Blood Institute (NIH/NHLBI; 1998). By this criterion, in 1994 (the last period for which most complete analyses are available), 25% of U. S. women and 20% of men were obese (Flegal, Carroll, Kuczmarski, & Johnson, 1998). An additional 25% and 39%, respectively, were considered overweight, defined as a BMI of 25.0 to 29.9 kg/m^2. Thus, 55% of adult Americans were either overweight or obese; only a minority had a BMI in the desired range of 18.5–24.9 kg/m^2. As alarming are findings that the prevalence of obesity and overweight is continuing to increase precipitously. The most recent data, obtained in 1999, found that fully 61% of adult Americans were either overweight (34%) or obese (27%; National Center for Health Statistics, 1999). Similar trends have been observed in other developed nations, leading the WHO to declare obesity a global epidemic (WHO, 1998). In the United States, minority women are the most affected. In the 1994 survey, 37% of African American and 33% of Mexican American women were obese; an additional one third of each group met criteria for overweight (Flegal et al., 1998). From ages 20 to 60 years, BMI increases in both sexes, across all ethnic groups.

Obesity is associated with an enormous burden of physical, economic, and emotional suffering (NIH/NHLBI, 1998). The criterion for defining obesity (i.e., BMI of 30 kg/m^2) was selected principally on the basis of the strong relation between BMI and mortality, shown in Figure 2. At a BMI of 30 kg/m^2, the risk of

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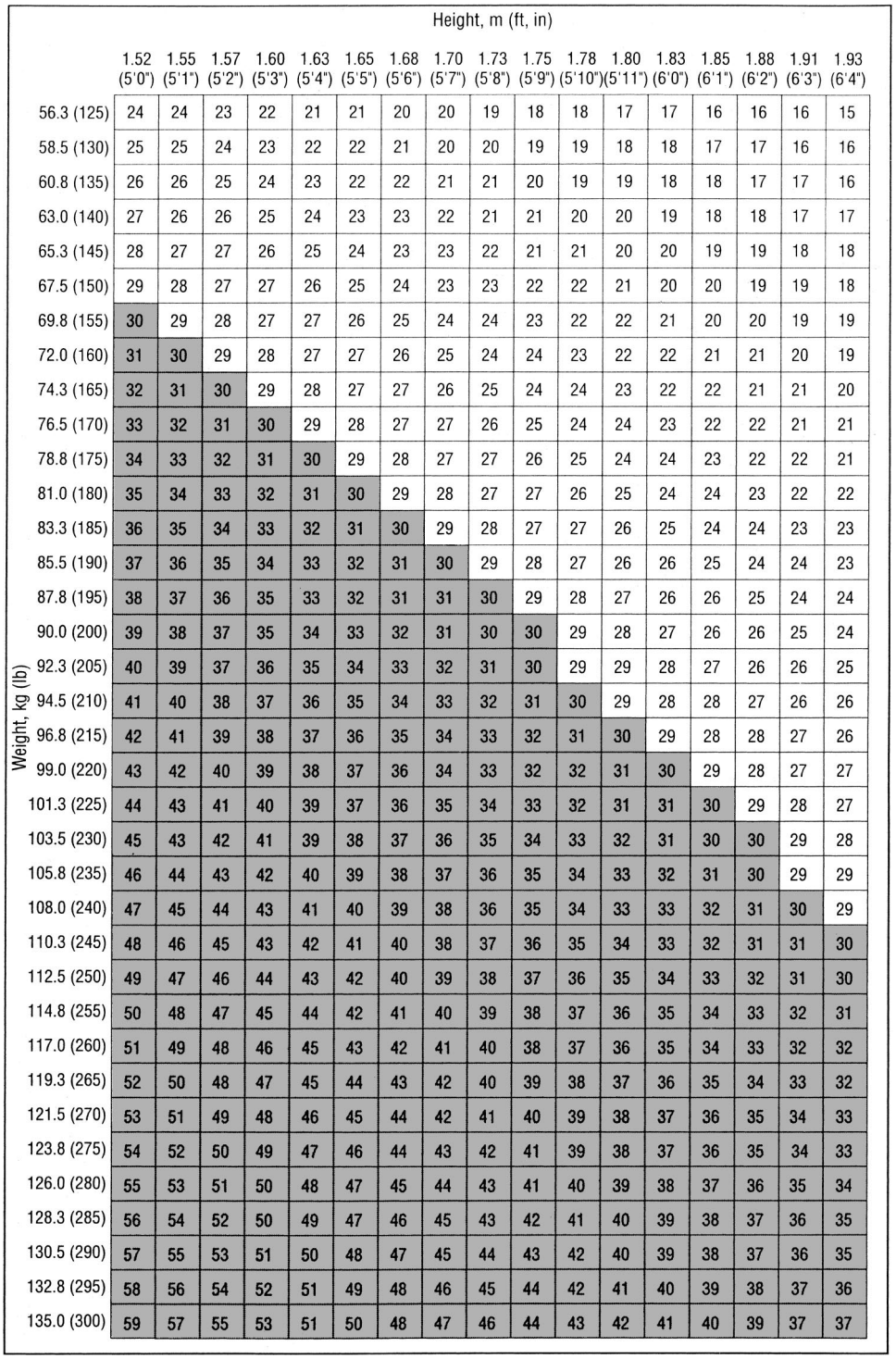

Weight, kg (lb) \ Height, m (ft, in)	1.52 (5'0")	1.55 (5'1")	1.57 (5'2")	1.60 (5'3")	1.63 (5'4")	1.65 (5'5")	1.68 (5'6")	1.70 (5'7")	1.73 (5'8")	1.75 (5'9")	1.78 (5'10")	1.80 (5'11")	1.83 (6'0")	1.85 (6'1")	1.88 (6'2")	1.91 (6'3")	1.93 (6'4")
56.3 (125)	24	24	23	22	21	21	20	20	19	18	18	17	17	16	16	16	15
58.5 (130)	25	25	24	23	22	22	21	20	20	19	19	18	18	17	17	16	16
60.8 (135)	26	26	25	24	23	22	22	21	21	20	19	19	18	18	17	17	16
63.0 (140)	27	26	26	25	24	23	23	22	21	21	20	20	19	18	18	17	17
65.3 (145)	28	27	27	26	25	24	23	23	22	21	21	20	20	19	19	18	18
67.5 (150)	29	28	27	27	26	25	24	23	23	22	22	21	20	20	19	19	18
69.8 (155)	30	29	28	27	27	26	25	24	24	23	22	22	21	20	20	19	19
72.0 (160)	31	30	29	28	27	27	26	25	24	24	23	22	22	21	21	20	19
74.3 (165)	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21	21	20
76.5 (170)	33	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21	21
78.8 (175)	34	33	32	31	30	29	28	27	27	26	25	24	24	23	22	22	21
81.0 (180)	35	34	33	32	31	30	29	28	27	27	26	25	24	24	23	22	22
83.3 (185)	36	35	34	33	32	31	30	29	28	27	27	26	25	24	24	23	23
85.5 (190)	37	36	35	34	33	32	31	30	29	28	27	26	26	25	24	24	23
87.8 (195)	38	37	36	35	33	32	31	31	30	29	28	27	26	26	25	24	24
90.0 (200)	39	38	37	35	34	33	32	31	30	30	29	28	27	26	26	25	24
92.3 (205)	40	39	37	36	35	34	33	32	31	30	29	29	28	27	26	26	25
94.5 (210)	41	40	38	37	36	35	34	33	32	31	30	29	28	28	27	26	26
96.8 (215)	42	41	39	38	37	36	35	34	33	32	31	30	29	28	28	27	26
99.0 (220)	43	42	40	39	38	37	36	34	33	32	32	31	30	29	28	27	27
101.3 (225)	44	43	41	40	39	37	36	35	34	33	32	31	31	30	29	28	27
103.5 (230)	45	43	42	41	39	38	37	36	35	34	33	32	31	30	30	29	28
105.8 (235)	46	44	43	42	40	39	38	37	36	35	34	33	32	31	30	29	29
108.0 (240)	47	45	44	43	41	40	39	38	36	35	34	33	33	32	31	30	29
110.3 (245)	48	46	45	43	42	41	40	38	37	36	35	34	33	32	31	31	30
112.5 (250)	49	47	46	44	43	42	40	39	38	37	36	35	34	33	32	31	30
114.8 (255)	50	48	47	45	44	42	41	40	39	38	37	36	35	34	33	32	31
117.0 (260)	51	49	48	46	45	43	42	41	40	38	37	36	35	34	33	32	32
119.3 (265)	52	50	48	47	45	44	43	42	40	39	38	37	36	35	34	33	32
121.5 (270)	53	51	49	48	46	45	44	42	41	40	39	38	37	36	35	34	33
123.8 (275)	54	52	50	49	47	46	44	43	42	41	39	38	37	36	35	34	33
126.0 (280)	55	53	51	50	48	47	45	44	43	41	40	39	38	37	36	35	34
128.3 (285)	56	54	52	50	49	47	46	45	43	42	41	40	39	38	37	36	35
130.5 (290)	57	55	53	51	50	48	47	45	44	43	42	40	39	38	37	36	35
132.8 (295)	58	56	54	52	51	49	48	46	45	44	42	41	40	39	38	37	36
135.0 (300)	59	57	55	53	51	50	48	47	46	44	43	42	41	40	39	37	37

Figure 1. A table for estimating body mass index (BMI) from height and weight. Overweight is defined as a BMI of 25.0 to 29.9 kg/m^2 and obese as a BMI $\geq$ 30 kg/m^2. The table is adapted from data provided by Shape Up America and The American Obesity Association, 1996. Reprinted with permission, from the Web site—www.shapeup.org—of Shape Up America!, a nonprofit organization based in Washington, DC.

mortality increases by approximately 30% and at a BMI $\geq$ 40 kg/m^2 by 100% or more (Manson et al., 1995). Death is attributable primarily to obesity's association with cardiovascular disease and type 2 diabetes, as well as with several cancers (Pi-Sunyer, 1993). Other complications include sleep apnea, gallbladder disease, and the exacerbation of osteoarthritis. In 1995, obesity-related complications were estimated to cost our nation $99 billion (Wolf & Colditz, 1998).

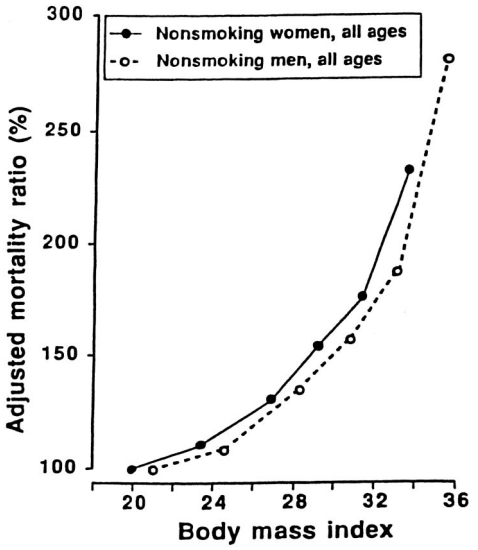

Figure 2. Mortality ratios in relation to the body mass index of nonsmoking men and women (of all ages) who participated in the American Cancer Society study (Lew & Garfinkel, 1979). From "Assessment of Morbidity and Mortality Risk in the Overweight Patient," by T. B. VanItallie and E. A. Lew in *Treatment of the Seriously Obese Patient* (p. 28), T. A. Wadden and T. B. VanItallie (Eds.), 1992, New York: Guilford Press. Reprinted with permission.

Although often overlooked, obesity disproportionately affects people of low income, particularly women. One study found it was six times more common in women of low as compared with high socioeconomic status (Moore, Stunkard, & Srole, 1962). Differences in social class contribute to the discrepancy in prevalence rates between majority and minority women, the latter of whom fall disproportionately in the lowest income levels (Kumanyika, 1987). A prospective study of female adolescents clearly revealed the social and economic perils of excess fat (Gortmaker, Must, Perrin, Sobol, & Dietz, 1993). Obese as compared with nonobese girls completed significantly fewer months of high school (despite equal grades), were significantly less likely to marry, and had lower household incomes. Other findings have confirmed that overweight individuals are subjected to prejudice and discrimination when seeking college admissions, a job, or a place to live (Wadden, Womble, Stunkard & Anderson, 2002). It is remarkable, in the face of such obstacles, that a majority of overweight individuals in the general population have essentially normal psychological functioning (Wadden et al., 2002). Those, however, who seek weight reduction are more likely to report depression, anxiety, binge eating, and other complications (Fitzgibbon, Stolley, & Kirschenbaum, 1993; Friedman & Brownell, 1995).

Etiology of the Obesity Epidemic

Bray (1998) has succinctly summarized the etiology of obesity: "Genes load the gun, the environment pulls the trigger." Genes are currently thought to explain 25%–40% of the variance in BMI (Bouchard, 1994; Price, 2002) and contribute to differences among people in resting metabolic rate, in weight gain in response to overfeeding, and in where excess fat is stored (i.e., body fat distribution; Bouchard, 1994; Bouchard et al., 1989, 1990; J. A. Levine, Eberhardt, & Jensen, 1999). Thus, some individuals appear to be born with a genetic predisposition to obesity that is readily nurtured by our nation's lifestyle, as discussed later.

The Leptin Pathway

The specific genes that contribute to common types of human obesity have yet to be identified, and there are likely to be dozens that potentially interact (Chagnon, Perusse, Weisnagel, Rankinen, & Bouchard, 2000; Price, 2002). Investigators, however, have found at least five single-gene defects that produce obesity in laboratory animals. The past decade's most exciting discovery was that of the ob gene and its protein product leptin (Zhang et al., 1994). Leptin is secreted from adipose tissue and has been shown in the mouse to act on neural networks in the hypothalamus that regulate energy intake and expenditure (Campfield, Smith, Guisez, Devos, & Burn, 1995).

The ob/ob mouse, as a result of a genetic mutation, produces no leptin (Campfield et al., 1995). The animal's marked overeating, sedentariness, and resulting obesity are all reversed by the administration of recombinant leptin. Investigators expected to find that severely obese humans would be deficient in leptin. To date, however, only a handful of such individuals have been found worldwide; they too have lost weight when given the protein (Montague et al., 1997). Surprisingly, the vast majority of obese individuals have high leptin levels, commensurate with their increased body fat (Considine et al., 1996). This finding has led investigators to speculate that some obese individuals may be insensitive to leptin, in the same manner that persons with type 2 diabetes are insensitive to insulin (Campfield et al., 1995). Clinical trials of leptin are currently under way, and investigators are searching for abnormalities in other neuropeptides that are related to leptin (Schwartz, Woods, Porte, Seeley, & Baskin, 2000).

The Toxic Environment

Research in genetics holds promise for significantly obese individuals who have physiologic, metabolic, or related abnormalities. Such research, however, is unlikely to solve our nation's obesity epidemic. This is because changes in our eating and activity habits, not our gene pool, appear to lie at the heart of this epidemic (Hill & Peters, 1998; Price, 2002). Studies that have tracked individuals who moved from less to more modernized countries have shown increased rates of obesity. For instance, Bhatnagar et al. (1995) followed individuals who migrated to West London from Punjab, India and found significantly increased body weights in the individuals in London compared with their siblings remaining in Punjab. Another clear example is provided by Pima Indian women living in Arizona compared with their relatives remaining in Mexico (Ravussin, Valencia, Esparza, Bennett, & Schulz, 1994). Women in Arizona were found to have an average dietary fat intake of 41% of total calories, body weight of 90 kg, and BMI of 37 kg/m^2, as compared with 23% of calories from fat, body weight of 70 kg, and BMI of 25 kg/m^2 for women remaining in Mexico. Although studies of this type do not control perfectly for genetics, the staggering effects of changing environments (or cultures) cannot be dismissed. We agree with the conclusions of the Institute of Medicine (1995) that, "there has been no real change in the gene pool in this period of increasing obesity. The root of the problem, therefore, must lie in the powerful social and

cultural forces that promote an energy-rich diet and a sedentary lifestyle." (p. 152)

Brownell and colleagues (Battle & Brownell, 1997; Brownell, 1994; Horgen & Brownell, 1998, 2002) have referred to these forces as a "toxic" environment. Toxic in this case refers to unprecedented exposure to energy-dense, heavily advertised, inexpensive, and highly accessible foods. These foods have been combined with an increasingly sedentary lifestyle in which children, for example, watch an average of 28 hr of television a week; the more hours of viewing, the greater the likelihood of obesity (Gortmaker et al., 1996). Additional examples of the toxic environment include the explosion of fast food restaurants, large and ever-growing portion sizes, buffet restaurants, gasoline stations remodeled to have minimarkets, fast food franchises in school cafeterias, school districts signing contracts with soft drink companies, and powerful food advertising. What occurs in schools is especially concerning, with decreased emphasis on physical education classes, poor food available in cafeterias, vending machines, and food advertising on educational television. All signs are that the environment is worsening, so an increased prevalence of obesity, even from the high rates seen now, might well be expected (Foreyt & Goodrick, 1995b).

Genetics may help to explain why particular individuals become obese (or severely obese) when exposed to the toxic environment (Price, 2002). Such research should ultimately aid treatment but alone is unlikely to prevent the continued growth of obesity. We believe that the management of this epidemic will require better treatments for individuals who are already obese, but of more importance, it will require that greater resources and efforts be aimed at the prevention of this disorder. Such efforts must target the toxic environment that lies at the heart of the problem. In the remainder of this article, we first examine advances in the assessment and treatment of individuals who are already obese. We then discuss public health and policy initiatives designed to prevent the development of obesity.

Assessment of Obesity

Assessment of Health Risks

An expert panel convened by the NIH/NHLBI (1998) recently issued comprehensive guidelines for assessing obesity. Step 1, as shown in Table 1, is to calculate the individual's BMI. In general, the greater the increase above a value of 25 kg/m^2, the greater the risk of health complications (Bray, 1998; Manson et al., 1995). Step 2 is to determine the waist circumference, which provides an estimate of body fat distribution (NIH/NHLBI, 1998). Excess fat in the upper body (particularly in the intra-abdominal cavity) is more likely to be associated with hypertension, type 2 diabetes, dyslipidemia, and other complications than is the same amount of fat carried in the lower body (Lapidus et al., 1984; Sjöstrom, 1993). In women, a waist circumference $\geq$ 35 in. is indicative of upper body obesity, with the corresponding value in men $\geq$ 40 in. Table 1 shows that upper body obesity compounds the risk of health complications in the presence of a BMI of 25–34.9 kg/m^2. Individuals with a disease risk equal to or greater than "high," as determined by the table, should have a thorough medical examination to determine whether they have already developed hypertension or other conditions that require treatment.

Assessment of Behavioral and Psychosocial Factors

Assessment of health risks should be complemented by an evaluation of behavioral and psychosocial complications, as described previously (Wadden & Phelan, 2002). Of these, binge eating may be the most significant. Approximately 10%–30% of obese individuals who seek weight reduction suffer from binge eating disorder, in which they consume large amounts of food in a short period of time and experience loss of control during these episodes (Spitzer et al., 1993; Stunkard, 2002). They do not, however, purge after overeating. Binge eating is frequently associated with depression, negative body image, and other complications that may require treatment, independent of the individual's obesity (Marcus, 1993; Yanovski, 1993). Binge eating can be assessed by the Questionnaire on Weight and Eating Patterns (Yanovski, 1993) or by the Eating Disorder Examination–Questionnaire (Fairburn & Cooper, 1993). Options for treating this disorder are discussed later.

Methods of assessing routine eating and activity habits have been described previously (Allison, 1995; St. Jeor, 1997). Patients are likely to underestimate their calorie intake by as much as 50% when keeping food records (Lichtman et al., 1992). These records, however, are helpful clinically because they highlight patterns of

Table 1
Classification of Overweight and Obesity by BMI, Waist Circumference, and Associated Disease Risk

			Disease risk relative to normal weight and waist circumference	
	BMI (kg/m^2)	Obesity class	Men $\leq$ 102 cm ($\leq$ 40 in.) Women $\leq$ 88 cm ($\leq$ 35 in.)	Men $>$ 102 cm ($>$ 40 in.) Women $>$ 88 cm ($>$ 35 in.)
Underweight	$<$ 18.5			
Normal	18.5–24.9			
Overweight	25.0–29.9		Increased	High
Obesity	30.0–34.9	I	High	Very high
	35.0–39.9	II	Very high	Very high
Extreme obesity	$\geq$ 40	III	Extremely high	Extremely high

Note. From "Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults," by the National Institutes of Health/National Heart, Lung, and Blood Institute, 1998, *Obesity Research, 6*(Suppl.), p. 54. BMI = body mass index.

eating and track progress in treatment. Physical activity is the other behavior that must be assessed, especially considering its relationship to long-term weight reduction (Pronk & Wing, 1994). Activity can be assessed by a structured interview (Sallis et al., 1985) or by a questionnaire (Paffenbarger et al., 1993); in research studies, it is frequently evaluated by counting minutes of planned exercise. Alternatively, a new generation of pedometers provides an accurate but inexpensive method of tracking the daily number of steps walked (Bassett et al., 1996).

Methods and Goals of Weight Reduction

A Treatment Algorithm

Figure 3 presents an algorithm for selecting treatment. It is similar to one developed by the NIH/NHLBI (1998) in that interventions are recommended on the basis of an individual's BMI and risk of health complications. Less intensive (and expensive) approaches are generally recommended for persons with lower BMIs. The goal for individuals with a BMI < 27 kg/m^2 may be simply to prevent weight gain by increasing physical activity and decreasing intake of high-fat, high-sugar foods. Self-directed efforts and/or advice from a primary care physician may be sufficient in this regard. Individuals with higher BMIs also are encouraged to adopt healthier eating and activity habits, but if unable to lose weight, they have the option of using more aggressive (and costly) interventions. Pharmacotherapy, for example, is an option for individuals with a BMI ≥ 30 kg/m^2 and bariatric surgery for those with a BMI ≥ 40 kg/m^2 (NIH/NHLBI, 1998). Such interventions often carry a greater risk of side effects than a traditional program of diet and exercise, but their greater efficacy in reducing excess weight and its associated health complications may warrant the risks. We believe that heavier individuals should always try less-intensive approaches before progressing to these latter interventions; many will respond satisfactorily, at least in the short term, to a comprehensive behavioral program. As shown by the algorithm's matching component, treatment selection may be modified on the basis of the individual's medical or behavioral complications. For example, pharmacotherapy may be used with a

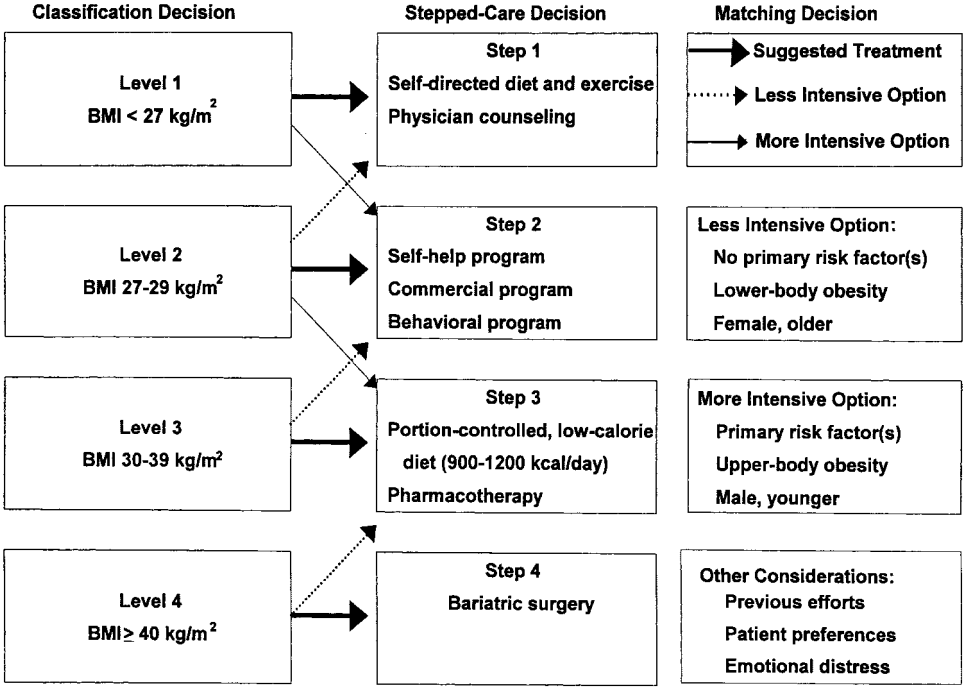

Figure 3. A conceptual scheme showing a three-stage process for selecting treatment. The first step, the classification decision, divides people into four levels based on body mass index (BMI). This level indicates which of four classes of interventions are likely to be most appropriate in the second stage, the stepped-care decision. All individuals are encouraged to control their weight by increasing their physical activity and consuming an appropriate diet. When this approach is not successful, more intensive intervention may be warranted, with the most conservative treatment (i.e., lowest cost and risks of side-effects) tried next. The thick solid arrow between two boxes shows the class of treatments that is usually most appropriate for an individual when less intensive interventions have not been successful. The third stage, the matching decision, is used to make a final treatment selection, based on the individual's prior weight loss efforts, treatment preferences, and need for weight reduction (as judged by the presence of comorbid conditions or other risk factors). The dashed lines point to treatment options for persons with a reduced need for weight reduction because of a reduced risk of health complications. The thin solid arrows show the more intensive treatment options for persons, who despite relatively low BMI levels, have increased risks of health complications. Adjunct nutritional or psychological counseling is recommended for patients who report marked problems with meal planning, depression, body image, or similar difficulties.

patient with a BMI of 27 kg/m^2 who has a significant comorbid condition (i.e., hypertension, dyslipidemia, etc.). By contrast, the absence of health complications in an older woman with lower body obesity and a BMI $>$ 30 kg/m^2 might suggest the use of diet and exercise alone, despite the patient's eligibility for pharmacotherapy. The goal in selecting treatment is to maximize improvements in weight and health while minimizing risks associated with therapy. Treatment selection also must consider the individual's prior weight loss efforts and current treatment preferences.

The proposed algorithm is based on our review of the literature and on our best clinical judgment (Clark, Guise, & Niaura, 1995). We note, however, that there have been few randomized controlled trials with long-term follow-up evaluations (i.e., $\geq$ 2 years) that have directly compared the efficacy of different interventions with persons in the same BMI class, let alone with individuals in different BMI classes (i.e., treatment matching). For example, although surgery appears to be the most successful intervention for persons with BMI $\geq$ 40 kg/m^2, no trials have directly compared it with long-term pharmacotherpy. Similarly, for persons with a BMI $\geq$ 30 kg/m^2, there are not definitive long-term data that show that a comprehensive behavioral program is more effective when combined with weight loss medication or that either of these approaches is more effective, after 2 years, than a commercial or self-help program. Ultimately, long-term data from controlled trials are needed to validate this and similar algorithms that have been proposed for the management of obesity.

New Goals of Obesity Treatment

Regardless of the treatment selected, both patient and provider must have realistic goals for weight reduction. Traditionally, all obesity treatments sought to help patients achieve an ideal weight (Foster, 1995). A greater appreciation of the biological factors that regulate body weight and the difficulty of maintaining large weight losses prompted a number of expert panels to conclude that weight losses of only 5%–15% of initial weight are a successful outcome (Institute of Medicine, 1995; NIH/NHLBI, 1998; U. S. Department of Agriculture, 1995). Losses of this size are frequently sufficient to improve health complications of obesity including hypertension, type 2 diabetes, and hypercholesterolemia (Blackburn, 1995; Goldstein, 1992). Thus, attainment of a healthier weight has replaced the ideal weight of years past (Brownell & Wadden, 1992; Foster & Kendall, 1994). A landmark randomized trial recently found that a 7 kg weight loss, combined with 150 min a week of physical activity, reduced the incidence of developing type 2 diabetes by 58% in overweight individuals with impaired glucose tolerance (Diabetes Prevention Program, 2002). A follow-up study of 5,000 overweight (or obese) individuals who already have type 2 diabetes was recently initiated to determine whether modest weight loss and increased physical activity will reduce the occurrence of heart attack, stroke, and related health complications (Yanovski, Bain, & Williamson, 1999). Observational studies have suggested that intentional weight loss is associated with reduced mortality (Williamson et al., 1995; Williamson et al., 2000).

Unfortunately, a significant disparity exists between the 10% weight losses recommended by professionals and the 20%–35% reduction in initial weight desired by overweight patients (Foster, Wadden, Vogt, & Brewer, 1997; Jeffery, Wing, & Randall, 1998; O'Neil, Smith, Foster, & Anderson, 2000). This discrepancy can lead to unrealistic and negative evaluations of treatment outcomes. For example, before treatment, obese women characterized a loss of 25% of initial weight as "one that I would not be happy with" and a 17% weight loss as "one that I could not view as successful in any way" (Foster, Wadden, Vogt, & Brewer, 1997). Thus, the average 10% weight loss produced by the best nonsurgical approaches may be very disappointing to patients and lead them to abandon the diet and exercise regimen needed to maintain weight loss. This belief, that dissatisfaction with outcome adversely affects the maintenance of weight loss, is based on the broader literature of goal setting (Carver & Scheier, 1982; Cervone, Jiwani, & Wood, 1991), as well as our clinical experience. One study (Jeffery, Wing, & Randall, 1998), however, found no relationship between reaching goal weight and maintenance of weight loss, but no ratings of satisfaction were obtained and very few patients (i.e., 17%), mostly men, reached their goal. Further studies of this issue are needed.

Advances in the Treatment of Obesity

The next several sections review advances in behavioral, pharmacologic, and surgical interventions. Lack of adequate data precludes us from making evidence-based recommendations concerning commercial or self-help programs (shown in the algorithm). This situation may improve in the next decade with the Federal Trade Commission's (1999) call for commercial weight loss programs to provide information on the costs of their programs, as well as their results of treatment. Weight Watchers recently showed that its group program, which offers social support and sensible diet and exercise recommendations at a cost of approximately $12 a week, induced a loss of 5 kg in 6 months (Heshka et al., 2000). Overeaters Anonymous, a group self-help program, is free of charge and frequently appeals to obese individuals who report binge eating and/or emotional distress (Malenbaum, Herzog, Eisenthal, & Wyshak, 1988). The LEARN Program for Weight Management offers another self-help option (Brownell, 2000). Unfortunately, little if anything is known about the effectiveness of diet books, such as the ever-popular *Dr. Atkins' Diet Revolution* (Atkins, 1999), which are purchased by millions of persons each year (Freedman, King, & Kennedy, 2001). Data are similarly needed to help consumers (and practitioners) make informed decisions about a host of off-the-shelf products, most notably herbal supplements (Allison, Fontaine, Heshka, Mentore, & Heymsfield, 2001).

Group Behavioral Treatment

The results of group behavioral treatment for obesity are well known and have not changed substantially over the past 15 years. A 20-week program induces an average loss of 8.5–9.0 kg (i.e., 9% of initial weight; Foreyt & Goodrick, 1993; Wadden & Foster, 2000; Wilson, 1994; Wing, 2002). Without further treatment, patients regain one third of lost weight in the year following treatment, with increasing regain over time. Recent efforts have focused on inducing larger weight losses and improving the maintenance of weight loss.

Inducing larger weight losses. The most reliable method of increasing short-term weight loss is restricting calorie intake more severely. Numerous randomized trials have shown that very-low-calorie diets (VLCDs), providing 400–800 kilocalories per day (kcal/day), produce short-term weight losses nearly double those

resulting from a standard 1,200–1,500 kcal/day diet of conventional foods (used in traditional behavioral programs). VLCDs, however, are associated with rapid weight regain, such that 1 year after treatment there are not statistically significant differences in weight loss between the two dietary approaches (Ryttig, Flaten, & Rossner, 1997; Torgerson, Lissner, Lindross, Kruijer, & Sjöstrom, 1997; Wadden, Foster, & Letizia, 1994; Wing, Blair, Marcus, Epstein, & Harvey, 1994). This has led investigators to use less calorically restricted diets but to retain the use of portion-controlled servings, a prominent feature of liquid VLCDs (Wadden et al., 1997). Jeffery et al. (1993), for example, found that patients who were prescribed a 1,200-kcal/day diet, and were provided much of the food they were to eat, lost significantly more weight after 6 and 18 months of treatment than did patients prescribed the same calorie intake but who consumed a self-selected diet of conventional foods. A follow-up study showed that simply giving patients detailed menu plans was as effective in increasing weight loss as was providing the prescribed foods (Wing et al., 1996). Two studies of a popular meal-replacement plan (i.e., SlimFast) showed similar benefits of using a portion-controlled diet (Ditschuneit, Flechtner-Mors, Johnson, & Adler, 1999; Rothacker, 2000). In the most successful study, conducted in Germany, patients who replaced one meal and one snack a day with portion-controlled servings had a mean loss of 8% of initial weight at the end of 4 years (Flechtner-Mors, Ditschuneit, Johnson, Suchard, & Adler, 2000). These very favorable findings await replication in the United States and other nations.

Increasing the length of treatment also increases weight loss, but not by as much as expected. Three studies found that weekly behavioral treatment of 40 to 52 weeks produced losses of 12%–14% of initial weight, whereas losses of approximately 18%–25% were anticipated on the basis of results obtained during the first 4–6 months (Perri, Nezu, Patti, & McCann, 1989; Wadden et al., 1994; Wing et al., 1994). The repeated finding that most patients cannot lose more than 10%–15% of initial weight has led to the more modest weight loss goals discussed previously.

Improving long-term weight loss. Improving the maintenance of weight loss remains the critical challenge for all obesity treatments (Foreyt & Goodrick, 1993). Two developments have facilitated progress. The first is the recognition that obesity, for most individuals, is a chronic disorder that requires long-term care. Perri and colleagues (Perri et al., 1988; Perri, Nezu, & Viegner, 1992) showed in a series of studies in the 1980s that continuing behavioral care, whether provided by phone, by mail, or in clinic, improved the maintenance of a 10%–15% weight loss, achieved with a 1,200–1,500 kcal/day diet. The shortcoming of this approach is that participation in maintenance sessions declines over time (Wadden et al., 1997; Wing, Venditti, Jakicic, Polley, & Lang, 1998). Innovative approaches, including the use of the Internet and television, are needed to facilitate patients' participation in long-term care (Meyers, Graves, Whelan, & Barclay, 1996; Tate, Wing, & Winett, 2001).

The second development concerns new methods to facilitate patients' increased physical activity, which is the best predictor of weight loss maintenance (Pronk & Wing, 1994). Two studies found that patients who exercised at home, as compared with on-site (i.e., health club or clinic), adhered better to their activity program and/or lost more weight (King, Haskell, Young, Oka, & Stefanick, 1995; Perri, Martin, Leermakers, Sears, & Notelovitz, 1997). Home exercise appears to reduce barriers to adherence. Similarly, multiple short bouts of activity (i.e., 10 min) have been found to be as effective in facilitating exercise adherence and weight loss, as a single long bout (i.e., 40 min; Jakicic, Wing, Butler, & Robertson, 1995; Jakicic, Winters, Lang, & Wing, 1999). Short bouts provide more opportunity to fit exercise into a busy day, as does increasing lifestyle activity, which involves burning more calories throughout the day as a result of small changes, such as using stairs rather than escalators or walking rather than riding (Epstein, Wing, Koeske, & Valoski, 1985). Two recent studies showed the benefits of lifestyle activity, as compared with traditional structured exercise, for improving cardiorespiratory fitness and weight control (Andersen et al., 1999; Dunn et al., 1999). In both cases, the lifestyle intervention was as effective as the traditional exercise intervention. Lifestyle activity would appear to be the perfect alternative for obese individuals who hate to exercise. Moreover, modest improvements in physical fitness, consistent with those that can be achieved with lifestyle activity, may be sufficient to decrease cardiovascular mortality (Blair et al., 1989; Lee, Blair, & Jackson, 1999).

The best maintenance of weight loss is likely to be achieved by combining lifestyle and structured exercise to raise activity-related energy expenditure to approximately 1,500–2,500 kcal per week, as compared with the 1,000 kcal/week traditionally targeted in behavioral programs. Post hoc analyses of several studies revealed the benefits of this higher goal (Andersen et al., 1999; Jakicic et al., 1999; Jeffery, Wing, Thorson, & Burton, 1998). Similarly, participants in the National Weight Control Registry, who had lost at least 14 kg and kept it off for 1 year, reported that they expended approximately 2,800 kcal/week (Klem, Wing, McGuire, Seagle, & Hill, 1997). It is important to note that, although this may be a desired level, any increase in physical activity is better than none. Obese individuals must initially set modest activity goals that they can achieve rather than such ambitious goals that they are destined to fail. Individuals of all weights should be encouraged to increase their physical activity to improve their physical health and emotional well-being, regardless of the effects on body weight (Blair, 1991).

Generalization of treatment effects. It should be noted that nearly all of what we know about the behavioral treatment of obesity comes from studies conducted in academic research centers. It could be argued that persons who enroll in such studies have more refractory obesity and, therefore, are more difficult to treat than those in community samples. Alternatively, such individuals may be more motivated, as indicated by their willingness to commit to long-term participation in research trials. In either case, such differences highlight the need to conduct effectiveness studies to determine whether the findings obtained in tertiary care centers extend to the greater numbers of persons who are treated in community settings (Brownell & Wadden, 1991; Womble, Wang, & Wadden, 2002). The efficacy–effectiveness distinction is important in all areas of clinical research, but particularly with obesity, given its widespread prevalence.

We also note that most treatment studies have been of predominantly Caucasian samples. This is troubling given the significantly higher rates of obesity among African Americans and Hispanic Americans (Flegal et al., 1998), particularly in women. With most weight loss methods, including balanced-deficit diets, VLCDs, and bariatric surgery, African Americans lose significantly less weight than do Caucasians (Kumanyika, 2002). Reasons for the smaller losses are not well understood. Cultural and social factors are

likely to influence attitudes and behaviors concerning eating, activity, and weight reduction. Lack of attention to these factors when designing and evaluating interventions is a serious gap in our current knowledge. The few programs to date that have been designed specifically for African Americans have yielded modest weight losses (Kumanyika, 2002).

Pharmacologic Treatment of Obesity

Pharmacotherapy is likely to play an increasingly important role in the treatment of obese individuals, specifically those with a BMI $\geq$ 30 kg/m^2 (or 27 kg/m^2 in the presence of health complications; 1996; Yanovski & Yanovski, 2002). In particular, medications could help facilitate the maintenance of weight loss if patients took them long-term, in the same manner that antihypertensive or antidiabetic agents are taken (Hill et al., 1999; Stunkard, 1982). Investigators no longer expect the short-term use of weight loss medications to cure obesity any more than they expect a 3-month trial of medication to cure diabetes. Long-term pharmacotherapy is needed in both cases (Bray & Greenway, 2000).

To be prescribed long-term, weight loss medications must be both safe and effective. Two agents, sibutramine (Meridia) and orlistat (Xenical), are approved by the Food and Drug Administration "for weight loss and the maintenance of weight loss." Sibutramine is a combined serotonin-norepinephrine re-uptake inhibitor that appears to act on receptors in the hypothalamus that control satiation (i.e., feelings of fullness). In randomized trials, sibutramine plus diet produced a 7% reduction in initial weight at 1 year, as compared with a 2% loss for patients treated by placebo plus diet (Lean, 1997). Losses of 10%–15% were achieved when sibutramine was combined with a more intensive program of diet and exercise modification, suggesting that behavior therapy may improve the effects of weight loss medication (James et al., 2000; Wadden, Berkowitz, Sarwer, Prus-Wisniewski, & Steinberg, 2001). Sibutramine is associated with small increases in heart rate and blood pressure and, thus, should not be used in patients with uncontrolled hypertension or cardiovascular disease. Sibutramine also is not recommended for use in combination with several medications, including selective serotonin re-uptake inhibitors (SSRIs) for depression (Abbott Laboratories, 2001).

Orlistat is a gastric lipase inhibitor that blocks the absorption of about one third of the fat contained in a meal (Sjöstrom et al., 1998). The undigested fat (oil) is passed in stool, leading to the loss of about 150–180 kcal/day. In addition, orlistat requires the consumption of a low-fat diet. If patients consume more than 20 g of fat per meal, or a total of 70 g/day, they increase the risk of adverse gastrointestinal events that include oily stools, flatus with discharge, and fecal urgency. Thus, patients are negatively reinforced to eat a low-fat diet, which further reduces their caloric intake. In randomized trials, orlistat plus diet produced a 10% reduction in initial weight at 1 year as compared with a 6% loss for placebo plus diet (Davidson et al., 1999; Sjöstrom et al., 1998). Patients who remained on the drug for an additional year maintained a loss of 8% of weight at the end of this time. Because orlistat does not affect the central nervous system, it may be used with patients who take SSRIs or other centrally acting medications.

The next decade will witness an intensified search for medications that decrease energy intake and/or increase energy expenditure (Bray & Greenway, 2000). The results of initial leptin trials have been disappointing; the highest doses of the protein induced losses of only about 8% of initial weight (Heymsfield et al., 1999). Further research, however, on the genetics of body weight regulation is likely to identify additional candidates for intervention. We make two points in this regard. First, trials of 2 years or more are needed to compare pharmacologic treatment with strong behavioral interventions. Current medications do not appear to be more effective than group behavior therapy in inducing weight loss but may maintain weight loss more effectively and efficiently. Second, more research is needed on methods of combining pharmacologic and behavioral interventions. An early study suggested that these two interventions together were more effective than either used alone (Craighead, Stunkard, & O'Brien, 1981). This may be because the two approaches have different but complementary mechanisms of action (Craighead & Agras, 1991). Behavior therapy appears to help obese individuals control the external environment, whereas pharmacotherapy modifies the internal environment by enhancing satiety (as with sibutramine) or by causing malabsorption (as with orlistat; Phelan & Wadden, in press; Wadden, Berkowitz, et al., 2001).

Surgical Treatment of Obesity

The significant health hazards of extreme obesity (BMI $\geq$ 40) and the modest weight losses produced by conservative treatments led to the development of surgical interventions. The first widely used procedure was an intestinal (jejunoileal) bypass that produced drastic weight losses (45 kg in 80% of patients) but was abandoned because of the high complication rate, including liver failure, protein-calorie malnutrition, and vitamin and mineral deficiencies (Albrecht & Pories, 1999). In search of a safer procedure, Mason and Ito (1969) developed a gastric bypass (GB) procedure that remains widely used today. The GB creates a small (50 ml) gastric pouch at the base of the esophagus to limit intake. In addition, the stomach and part of the intestine (duodenum) are bypassed by attaching the small pouch and the jejunum. Various modifications, including a smaller gastric pouch of 20–30 ml, have produced average weight losses of approximately 30% of initial weight during the first 18 months, with maintenance of a 25% loss up to 14 years later (Albrecht & Pories, 1999).

A second frequently used procedure is vertical banded gastroplasty (VBG). VBG also creates a small pouch to limit intake, but no alterations are made in the gastrointestinal tract. Weight loss appears to be greater for GB than for VBG (30% vs. 25%, respectively), probably because of the slight malabsorption associated with the former procedure (Sugerman, Londrey, & Kellum, 1989). GB may be particularly effective for sweet eaters because it induces an unpleasant "dumping syndrome" following the ingestion of refined carbohydrates. Patients experience nausea and other adverse gastrointestinal events that discourage future consumption of sweets. Both procedures and their resultant weight loss are associated with significant improvements in medical conditions (Kral, 1998) and psychosocial functioning (Powers, Rosemurgy, Boyd, & Perez, 1997; Stunkard, Stinnet, & Smoller, 1986), although some studies found regression in mood over time, even with maintenance of weight loss (Karlsson, Sjöstrom, & Sullivan, 1998; Waters et al., 1991).

Surgical treatment is only appropriate for patients with a BMI $\geq$ 40 kg/m^2 or $\geq$ 35 kg/m^2 in the presence of significant health complications (Kral, 1998). In addition, patients should undergo a comprehensive assessment by a multidisciplinary team (i.e., psy-

chologist, nutritionist, and internist) to identify any contraindications and, as important, to give patients realistic expectations about the procedure and its outcome. A comprehensive behavioral evaluation for surgical patients has been described elsewhere (Wadden, Sarwer, et al., 2001).

Binge Eating, Obesity, and Nondieting Approaches

Binge Eating

As noted previously, approximately 10%–30% of obese persons who seek weight reduction suffer from binge eating (Spitzer et al., 1993; Stunkard, 2002). A central question is whether these individuals, in view of their disordered eating and psychopathology, need different treatment than obese nonbingers. Some investigators, for example, believe that binge eaters should not diet because of a possible link between food restriction and binge eating (Polivy & Herman, 1985). (The term *dieting*, as used here, refers to the intentional and sustained restriction of food intake to induce negative energy balance and, thus, weight loss.) Several studies, however, found that consumption of a 1,200–1,500 kcal/day diet, as prescribed in behavioral programs, was associated with decreases, not increases, in binge eating symptoms (National Task Force on the Prevention and Treatment of Obesity, 2000). Other investigations, summarized by Gladis et al. (1998), showed that binge eaters and nonbinge eaters responded equally well to weight loss interventions, although there have been exceptions (Marcus, Wing, & Hopkins, 1988; Yanovski, Gormally, Lesser, Gwirtsman, & Yanovski, 1994).

Both cognitive–behavioral therapy (CBT), designed specifically to control disordered eating, and interpersonal therapy, originally intended for depression, significantly reduce binge episodes (Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990; Wilfley et al., 1993), as do exercise (M. D. Levine, Marcus, & Moulton, 1996), self-help (Carter & Fairburn, 1998), and nondieting approaches (Goodrick, Kimball, Reeves, Poston, & Foreyt, 1998). The few controlled studies of pharmacotherapy have shown little significant effect compared with placebo (McCann & Agras, 1990, Stunkard, Berkowitz, Tanrikut, Resiss, & Young, 1996; Wilfley & Cohen, 1997). Despite reducing binge eating, none of these treatments, including CBT, reliably results in weight loss. It is possible that patients reduce the number of binge episodes but compensate by increasing food intake during nonbinge episodes. Agras, Telch, Arnow, Eldredge, and Marnell (1997) found that abstinence from binge eating was associated with greater weight loss than was only partial remission from bingeing.

As a whole, the literature suggests that moderate caloric restriction, in the context of a standard behavioral weight loss program, not only improves binge eating but also induces weight loss (National Task Force on the Prevention and Treatment of Obesity, 2000). CBT and nondieting approaches improve binge eating but without inducing significant weight loss; thus, the adverse medical consequences of excess weight remain. Identification of the optimal treatment for obese binge eaters will require long-term comparisons of weight-reducing and non-weight-reducing approaches. Patients should be assessed on changes in eating behavior, weight, and physical and psychosocial health.

Nondieting Approaches for Obesity

Extending beyond binge eating disorder, some investigators have proposed that dieting should not be used in the treatment of obesity itself (Garner & Wooley, 1991; Polivy & Herman, 1992). This argument is based on the generally poor long-term results of weight loss efforts, as well as concerns that dieting may be associated with adverse physical or psychological effects, in addition to binge eating (Polivy & Herman, 1985). Few studies of obese dieters support the latter concerns (National Task Force on the Prevention and Treatment of Obesity, 2000). Nonetheless, several nondieting or undieting approaches have been proposed that, although differing in their specific methods, generally seek to (a) increase awareness about dieting's ill effects, (b) provide education about the biological basis of body weight, (c) help patients stop restricting their caloric intake and avoiding prohibited foods, (d) encourage the use of internal cues such as hunger and fullness to guide eating rather than external cues such as calories or fat grams, (e) improve self-esteem and body image through self-acceptance rather than through weight loss, and (f) increase physical activity (Foreyt & Goodrick, 1995a; Foster & Johnson, 1998; Foster & McGuckin, 2002; Hirschmann & Munter, 1988; Polivy & Herman, 1992).

Two uncontrolled trials of this approach showed significant improvements in mood and self-esteem (Polivy & Herman, 1992; Roughan, Seddon, & Vernon-Roberts, 1990). Participants in one study gained 5.9 kg over 10 weeks (Polivy & Herman, 1992), whereas those in a second lost 1.8 kg in 8 weeks (Roughan et al., 1990). In a controlled evaluation, Tanco, Linden, and Earle (1998) showed that an 8-week nondieting approach, compared with standard weight loss treatment and a wait-list control, showed greater improvements in mood and some measures of eating-related psychopathology. Both the nondieting and weight loss groups lost small amounts of weight (2.6 and 1.8 kg, respectively). A more recent study found that a program that combined elements of nondieting with a moderately restricted diet of 1,800 kcal/day produced better weight loss at 1 year than did a traditional behavioral weight loss program that included a 1,200 kcal/day diet (Sbrocco, Nedegaard, Stone, & Lewis, 1999).

Nondieting approaches merit further investigation in randomized controlled trials. Such trials will need to clearly define what is meant by "dieting" and "nondieting," as the nondieting approaches and standard behavioral interventions for weight loss have more in common than might be thought (e.g., eating a variety of foods in moderation, using stimulus control, increasing physical activity). Moreover, it may be possible to blend these approaches, as Sbrocco et al. (1999) apparently did. Optimal interventions are likely to be at least 6–12 months in duration, to include follow-up evaluations of 2 years or more, and to compare participants on changes in weight, health complications, and psychosocial status. Measures of body image dissatisfaction (Sarwer, Wadden, & Foster, 1998) should be included, given that this complication improves with weight loss (Cash, 1994; Foster, Wadden, & Vogt, 1997) but also with CBT interventions that do not produce weight reduction (Rosen, Orosan, & Reiter, 1995).

Child and Adolescent Obesity

This review has focused principally on the treatment of obese adults. Obesity, however, is also on the rise in America's youth. In children ages 6 to 11 years, the prevalence of overweight nearly doubled from 1980 to 1994 (i.e., from 6.5% to 11.4% in boys and from 5.5% to 9.9% in girls; Troiano & Flegal, 1998). Similar increases were observed in adolescents (i.e., from 4.7% to 11.4%

in boys and from 4.9% to 9.9% in girls). Although the medical consequences of childhood obesity are less severe than in adults, excess weight in childhood and adolescence is associated with hyperlipidemia, especially in youth with upper body fat distribution (Dietz, 1998). The increase in type 2 diabetes observed among adolescents (Pinhas-Hamiel et al., 1996) is thought to be largely attributable to the corresponding increase in obesity (Dietz, 1998). Independent of any deleterious effects during childhood or adolescence, obesity persists. Approximately 25%–50% of obese adolescents become obese adults (Charney, 1998; Guo, Roche, Chumlea, Gardner, & Siervogel, 1994; Must & Strauss, 1999). Moreover, the medical consequences of excess weight among adults are greater in those who were overweight as adolescents (DiPietro, Mossberg, & Stunkard, 1994; Must, Jacques, Dallal, Bajema, & Dietz, 1992).

In contrast to the treatment of adult obesity, family-based behavioral treatments for obese children produce significant long-term reductions in percentage overweight. Pooling results from four studies, Epstein (1994) reported that 10 years after treatment children had experienced an average reduction in percentage overweight of 8%, and nearly 30% were no longer obese. Studies are now needed to determine whether methods used in state-of-the-art university-based programs can be successfully implemented in community settings.

The results of behavioral treatment programs for obese adolescents are usually less successful than those for children (Brownell, Kelman, & Stunkard, 1983; Epstein, Myers, Raynor, & Saelens, 1998; Mellin, Slinkard, & Irwin, 1987). This finding suggests the potential advantage of addressing obesity in childhood, while parental influence is still strong and before obesity-promoting behaviors have become well ingrained (Epstein et al., 1998). Increased focus on physical activity may prove particularly beneficial. It is well known that sedentary behavior (e.g., watching television, playing video/computer games) is associated, both cross-sectionally and longitudinally, with obesity in children and adolescents (Andersen, Crespo, Bartlett, Cheskin, & Pratt, 1998; Dietz & Gortmaker, 1985; Gortmaker et al., 1996). Epstein, Saelens, and O'Brien (1995) have evaluated the reinforcement value of various types of sedentary and active behaviors within the context of behavioral choice theory. This emerging body of research suggests that sedentary behavior is intrinsically more reinforcing than various forms of physical activity (Epstein, Saelens, Myers, & Vito, 1997; Epstein et al., 1995). Notably, making highly desired and frequently occurring sedentary behavior contingent on increasing physical activity appears to increase physical activity and decrease sedentary behavior (Saelens & Epstein, 1998). Future research on the reinforcing value of sedentary and active behaviors may lead to theory-driven and empirically validated methods to decrease sedentary behavior among children and adolescents.

Tackling the Obesity Crisis: Prevention and Policy

There is always the hope that the obesity epidemic will be reversed with advances in treatment. Given, however, that obesity is multiply determined and driven by such a powerful environment, we doubt that behavioral and pharmacologic interventions alone will halt, let alone reverse, the current epidemic. If the environment is the primary cause of the obesity epidemic, it is reasonable to search for environmental solutions, and because obesity is so difficult to treat, prevention may be more reasonable than remedy. At this point, relatively few researchers are working on prevention, and relatively little funding is directed at this problem. Far greater effort is needed.

Prevention of Obesity

One approach is to adopt a clinical model but apply it to the prevention of weight gain rather than to weight loss. Jeffery and French (1999) did this with the Pound of Prevention Study. Participants given an educational program on diet and activity aimed at weight gain prevention were compared with those who received no program. At the end of 3 years, no advantage was observed for the intensive educational–behavioral program, as compared with the control group. Klem, Viteri, and Wing (2000) have reported more promising results with the use of an intensive, small-group intervention, and thus, such approaches deserve further study. The costs, however, of such interventions must be carefully assessed to determine whether they could be reasonably implemented with the millions of Americans at risk of becoming obese.

Large-scale lifestyle intervention programs may also play a role in prevention. Although there are examples of successful programs for adults, effects are typically small and tend to be transitory, as shown in programs in both communities and work sites (Howard-Pitney, Winkleby, Albright, Bruce, & Fortmann, 1997; Luepker et al., 1994; Patterson et al., 1998). Schools are also a likely venue for prevention. Several studies have shown positive effects of school-based interventions that focused on diet (Manios & Kafatos, 1999) and physical activity (Sallis et al., 1997). For the most part, however, effects have been modest and short-lived. Small effects spread across large numbers of people could potentially have a public health impact, but the question arises whether such school programs could ever be implemented on a broad scale. Teachers and administrators, who find it difficult to teach even basic subjects, are now beset by demands to provide instruction in diversity, AIDS, sexuality, alcohol, drugs, and social development, to name but a few issues. It will be a challenge to raise obesity prevention to the top of the priority list.

This brief synopsis suggests that traditional approaches to prevention, which emphasize increasing education in schools and extending clinical interventions to the general population, may be helpful for some individuals and should be developed further. In their current form, however, they alone are unlikely to reverse the obesity epidemic.

Policy as Means for Prevention

One approach to prevention is to examine public policy as a means for changing diet and physical activity (Battle & Brownell, 1997; Brownell, 1994; Horgen & Brownell, 1998, 2002; Jacobson & Brownell, 2000; Nestle & Jacobson, 2000; Schmitz & Jeffery, 2000; Sherwood & Jeffery, 2000). Proposals in this area are in their infancy and are largely untested, but they are worthy of further discussion and testing (Brownell, 1994; Nestle & Jacobson, 2000; Sallis, Bauman, & Pratt, 1998). Those that follow are drawn from Horgen and Brownell (2002).

1. *Regulate food advertising aimed at children.* The average American child sees 10,000 food advertisements on television each year; 90%–95% of these are for sugared cereals, fast food, soft drinks, and candy (Horgen, Choate, & Brownell, 2001). There is clear evidence that exposure relates to food preferences and that

the content of food ads aimed at children overwhelmingly favors foods of poor nutritional quality (Horgen et al., 2001). Direct regulation might be possible, but perhaps more feasible would be offering equal time for pronutrition messages. This issue is of particular concern in schools, where commercial television now couples food advertisements with education and news programming. There is evidence that early intervention can increase preferences for healthy foods (Birch, 1999).

2. *Prohibit fast foods and soft drinks from schools*. Increasingly, school systems are importing fast food franchises into cafeterias and signing contracts with soft drink companies that increase exposure to foods low in nutrition. Encouraging consumption of healthy foods by enhancing education is only part of the picture. Education by modeling, by having healthy foods predominate in cafeterias and vending machines, is also necessary.

3. *Subsidize the sale of healthy foods*. Decreasing the price of healthy foods, at least in small, controlled environments, increases sales of these foods. Several well-controlled studies of vending machine purchases, for example, have shown that reducing prices of healthy options leads to a significant increase in sales of these items (French, Jeffery, Story, Hannan, & Snyder, 1997; Jeffery, French, Raether, & Baxter, 1994). More of such research is needed to develop policy-level proposals. Broad subsidies of healthy foods at the national level, thus increasing availability and perhaps desirability of foods high in nutrient density, should be explored.

4. *Tax unhealthy foods*. A tax on unhealthy food, if done in the service of raising money for food subsidies, might have the dual effect of enabling subsidies and decreasing consumption of food poor in nutrition. Jacobson and Brownell (2000) have speculated that small taxes placed on snack foods and soft drinks, a number of which are now in effect, are acceptable to consumers and could raise considerable funds to be earmarked for nutrition education or perhaps even subsidies. It is not known how high the taxes must be to affect consumption.

5. *Provide resources for physical activity*. Given the strong contribution to obesity of physical inactivity, easy access to opportunities to be physically active, additional physical activity requirements and opportunities in the schools, and other initiatives must be considered (Sallis et al., 1998). Whether additional facilities and opportunities to be active increase activity in sedentary individuals must be studied. Simple low-cost interventions, such as posting signs encouraging use of stairs, should be explored on a broad level (Andersen, Franckowiak, Snyder, Bartlett, & Fontaine, 1998; Brownell, Stunkard, & Albaum, 1980).

It is not known whether any or all of these policy initiatives would be effective, would be acceptable to the public, even if effective, or would have unanticipated negative consequences. We call here not for the adoption of specific policy initiatives, but we propose in the strongest terms that policy research, based on viewing obesity as a public health issue, be funded and become a central area of study in the obesity field. This approach can be dismissed as unrealistic and politically naive. Similar reactions, however, initially greeted calls to equip cars with seat belts, to regulate cigarette advertising, and to get tough on drunk driving.

Summary and Conclusions

Obesity is a problem out of control. Despite the best efforts of health officials, low-calorie products galore, and ubiquitous pressure to be fit and thin, the problem grows in magnitude and does so in country after country. There is no sign of the problem abating. In the absence of bold prevention and policy initiatives, we expect that prevalence will increase still more in years to come. The cost to the health and well-being of tens of millions of individuals worldwide, and to the health care systems supporting them, will be enormous.

We applaud advances in genetics and biology and hope that treatments will continue to improve. These offer our best hope of helping obese individuals; these individuals deserve safe and effective treatments. These advances, however, are not likely to help an obese nation. At best, they subtract a small number of persons from the obese pool, while many multiples of this number are being recruited into the pool by a toxic environment. A focus on prevention, with innovative policy initiatives, may be our most promising approach.

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