Self-Regulation of Energy Intake in the Prevention and Treatment of Obesity: Is It Feasible?

Michael R. Lowe

Abstract

LOWE, MICHAEL R. Self-regulation of energy intake in the prevention and treatment of obesity: is it feasible? *Obes Res.* 2003;11:44S-59S.

The prevalence of obesity in developed countries has been steadily increasing. Comprehensive lifestyle change programs for the treatment of obesity have garnered considerable empirical support, but most weight lost in lifestyle interventions is regained within several years. The outcome of obesity prevention programs has also been disappointing. One reason for this state of affairs may be that most weight control programs are based on an assumption of equipotentiality of their intervention components. That is, obesity prevention and treatment programs consist of a multitude of behavioral, cognitive, nutritional, physical activity, and interpersonal techniques, all of which are assumed to be of roughly equal importance in weight control. However, there is considerable evidence that our evolutionary heritage has made most humans highly sensitive to the availability and nature of food in the environment. It therefore may be unrealistic to expect that enhancing self-regulatory skills will be sufficient to overcome the combined influence of our appetitive predispositions and the obesigenic environment. However, there is growing evidence that weight control interventions that focus on the availability, structure, composition, and portion size of foods in the diet improve long-term weight control. Concerted efforts to change the availability and nature of foods at both the individual and population level may hold considerable promise for the treatment and prevention of obesity.

Key words: self-regulation, nutrition, dieting, treatment, prevention

Department of Psychology, Drexel University, Philadelphia, Pennsylvania.
Address correspondence to: Michael R. Lowe, Department of Psychology, Mail Stop 626, Drexel University, 245 N. 15th St., Philadelphia, PA 19102.
E-mail: lowe@drexel.edu

Quitting smoking is easy. I've done it a thousand times.
Mark Twain

Overview of Paper

Twain's lament about quitting smoking applies equally well to losing weight. Most weight loss diets ultimately fail. Therefore, most people prone to weight gain diet over and over again. Our obesigenic environment, combined with the great difficulty of permanently modifying energy intake and energy expenditure, means that obesity will, in the near future, probably overtake smoking as the number one cause of morbidity and premature mortality worldwide.

The topic addressed in this paper is the feasibility and suitability of using self-regulation strategies to achieve an acceptable degree of weight control (i.e., the prevention or treatment of overweight or obesity) in individuals and populations. The viewpoint developed here has been shaped by several influential perspectives on and observations about obesity that have been described over the past several decades. These perspectives include externality theory, set point theory, the development of restraint theory and the anti-dieting movement, the disappointing outcome of weight control programs, the biological "revolution" in obesity research, and the challenge of maintaining energy balance in developed countries. Therefore, these areas will be briefly reviewed to place in context the recommendations made later in the paper.

A topic of special relevance to the current paper is the controversy over dieting. Therefore, the pros and cons of restrained eating and dieting will be briefly considered. This section makes clear that the question "Is dieting good or bad?" is a gross oversimplification of a complex and multidimensional issue. Specifically, beginning to answer this question requires that one specify the meaning of "dieting," who is dieting, and why they are dieting.

The question of why it is so difficult to prevent weight gain and to sustain weight loss is then considered. The "lifestyle change" approach to treatment, which is heavily

based on cognitive-behavioral therapy, is briefly reviewed. The assumptions on which this approach to treatment is based are then evaluated in light of current psychobiological models of obesity.

Given human beings' biological predispositions and the environmental make-up of developed societies, the feasibility of achieving eating and weight control through the modification of self-regulatory skills is addressed next. Evidence is reviewed suggesting that the availability, structure, composition, and portion size of food in the environment appears to be of special significance for weight control. The concept of "personal food environments" is introduced and the potential weight control benefits of focusing intervention efforts directly on the nature of individuals' food environments are enumerated. An integrated model is developed that views the need for directly modifying individuals' food environments as a joint function of the degree to which a given environment promotes weight gain and an individual's proneness toward obesity.

It is important to emphasize that this paper addresses only the energy intake component of the energy balance equation. The creation of a chronic positive energy balance and the resulting weight gain is a function of both energy intake and energy expenditure (1). The relative importance of these two influences in the obesity epidemic is controversial (2). The present paper's focus on the energy intake half of the energy balance equation is inherent in the topic under discussion and is not meant as a statement about the relative importance of energy intake and energy expenditure in the development or maintenance of overweight.

Development of the Controversy over Dietary Restriction

In the 1960s, Stanley Schachter (3) and Richard Nisbett (4) developed the "internal-external" theory of obesity. This theory held that obese individuals, relative to those of normal weight, were over-responsive to external cues (and food cues in particular) and under-responsive to internal cues (including feelings of hunger and fullness). In food replete environments, these characteristics would make obesity-prone individuals more likely to overeat and gain weight. There were a number of studies in the late 1960s and early 1970s that supported this viewpoint. In a classic paper, Nisbett (5) reinterpreted this viewpoint by suggesting that obese individuals differed from normal weight people because they typically kept their weight below its biologically determined "setpoint," resulting in a state of chronic hunger that accounted for normal-obese differences in external and internal responsiveness. Nisbett (5) reviewed a variety of studies showing parallels between the behavior of obese individuals and hungry organisms generally. Both Schachter (3) and Nisbett (5) accepted the reality of normal-obese differences in internal and external responsiveness, but they differed in whether these characteristics were a cause (3) or a consequence (5) of obesity.

Whereas Nisbett (5) focused on the consequences of obese individuals' suppression of their weight below its biologically appropriate set point value, Herman and colleagues (6,7) extended this idea by theorizing that the chronic effort required to keep weight suppressed created the behavioral differences first documented by Schachter (3). That is, Herman and colleagues (6,7) suggested that maintaining restraint over one's food intake created exaggerated external responsiveness and impaired internal responsiveness. These investigators developed the Restraint Scale to measure this tendency and demonstrated that *normal weight* restrained eaters showed behavioral patterns similar to those previously found with overweight individuals. For instance, whereas normal weight unrestrained eaters appropriately reduced their food intake after drinking a high-calorie preload, normal weight restrained eaters showed the opposite eating pattern, increasing their food intake after the preload (6). Stress affected eating similarly [with stressed unrestrained eaters decreasing and stressed restrained eaters increasing their eating (7)]. These studies (8) supported the theory that it was restrained eating or dieting—not body weight per se—that was the critical correlate [and to Herman and Polivy (8), the primary cause] of normal-obese differences in internal and external responsiveness shown in prior research.

Four trends in the 1970s and 1980s further increased concerns about the physical and psychological effects of dieting. First, the prevalence of dieting in the general population increased, even among normal weight individuals and prepubescent children (9–12). Second, the apparently new eating disorder of bulimia nervosa was identified (13), and extreme dieting behavior was widely viewed as a likely contributor to the disorder (14–16). Third, it became apparent that the great majority of overweight individuals who lose weight fail to maintain the loss (17). Furthermore, several investigators argued that the risks of dieting for weight loss outweighed any benefits of dieting (18,19). Fourth, because women show a stronger connection between self-esteem and physical appearance than men, the societal emphasis on achieving an unrealistically thin body became a feminist issue (20). The cumulative result of these various critiques of dieting has been the development of a full-fledged "anti-dieting" movement (19,21–24).

Another development related to the topic of dietary restriction for weight control has been the "biological revolution" in the study and treatment of obesity. A variety of findings on the biology of obesity raised additional questions about the feasibility of achieving weight control through the exercise of dietary restraint. These included data supporting setpoint theory, the documentation of strong genetic influences on body weight, the movement in the medical profession to have obesity officially classified as a

disease, the demonstration of metabolic defenses against weight loss, the possibility that weight cycling has adverse effects on health, the discovery of leptin, and the growth of pharmacological treatments for obesity.

Although this paper focuses on energy intake, the level of intake required to match energy expenditure can change over time. Given a limitless supply of food in the environment, the degree to which energy intake must be restricted to maintain energy balance will depend on the level of energy expenditure required in everyday life. Nonresting energy expenditure is the variable component of total energy expenditure that determines what level of energy intake is required to maintain energy balance. In developed countries, physical activity levels have declined and are probably lower than they have ever been in human history (25). Some investigators (1,26,27) have hypothesized that there may be a lower limit of physical activity–based energy expenditure below which it becomes almost impossible to consistently keep energy intake low enough to match energy expenditure. This could be the case because the feasibility of self-regulation of body weight is based not just on the potential effectiveness of dietary restraint as a behavioral practice, but also on the degree to which dietary restraint must be exercised to achieve energy balance. To the extent that the application of dietary restraint is insufficient for people with weight problems to maintain energy balance, the problem could stem from inherent limitations of dietary restraint, from a low level of daily physical activity that creates the need for unrealistic levels of dietary restraint, or from both influences. New research will be required to determine this.

The Many Faces of Dietary Restraint

The empirical study of dietary restraint began with the work of Herman and Mack (6) and Herman and Polivy (7). These authors developed the Restraint Scale to measure the restrained eating construct. For 10 years after its introduction, nearly all research on restrained eating used this scale (28,29). Herman, Polivy, and colleagues use the terms "restraint" and "dieting" interchangeably and research using the Restraint Scale has contributed significantly to the negative reputation that dieting has developed. However, an examination of the Restraint Scale shows that it does not actually tap the construct that that most people think of when referring to dieting or "being on a diet." The dictionary defines the verb form of "diet" as "to eat and drink according to a regulated system, especially so as to lose weight" (30). The Restraint Scale, in contrast, mostly measures concerns about eating and weight, along with the size of fluctuations in weight. Denotatively, these characteristics seem to describe people with weight problems much more than they describe individuals who are on diets to lose weight. As we will see shortly, this distinction may be critical to understanding many of the negative effects associated with restraint.

The paradoxical behavior patterns shown by restrained eaters (identified by the Restraint Scale), along with concerns about the relationship between dieting and eating disorders, gave rise to an explosion of research on restrained eating and dieting that is ongoing. This research has demonstrated that, depending on what restraint-related construct is measured, the effects found with the Restraint Scale either disappear or are reversed (29,31–33). In addition, Lowe (31) and French and Jeffrey (34) noted that "dieting" and "restrained eating" can refer to a variety of practices that are associated with widely differing effects. Thus, one dieter may try to avoid eating for as long as possible each day, whereas another may try to limit portion sizes throughout the day. One dieter may avoid all high-fat products and another may be a vegetarian. Equally important as *how* someone is dieting is *who* is dieting and *why*. The consequences of dieting for a normal weight teenage girl whose self-esteem depends on becoming skinny are likely to be quite different than those experienced by the middle-aged man who is avoiding weight gain to slow the progression of heart disease. Further distinctions are also needed because many diets are based partly on changes in physical activity or lifestyle, not just in eating. It is therefore critical that any discussion of the effects of dieting or restraint specify exactly how the construct is being defined and measured (34). In the absence of such specification, asking whether dieting is desirable is akin to asking if taking drugs is desirable (when "drug-taking" can range from prescribed medications for an illness, to drinking socially, to mainlining heroin).

Is Dietary Restraint Harmful?

The central question addressed in this paper—the feasibility of successful self-regulation of eating to prevent or treat obesity—presumes that dieting is sufficiently safe and effective to even consider it as a means of preventing and treating obesity. The issue of safety—that is, whether dieting is potentially harmful, and if so, whether its benefits outweigh any risks—is beyond the scope of this paper. Brownell and Rodin (35) provide a thorough discussion of the relevant issues and studies. The stance taken in this paper is consistent with their overall conclusion: that among overweight and weight-gaining individuals, the potential benefits of dietary restriction likely outweigh the risks.

There is one dieting pattern—dieting to reach a weight lower than one's desirable weight for height—that virtually all health professionals would agree is potentially harmful. Especially among children (36), such dieting may be physically harmful and increase the risk of developing an eating disorder. Also of concern is that such efforts may be based on a misguided attempt to boost low self-esteem by reshap-

ing one's body (37). Such an outcome is not only unlikely, but may lead the dieter to neglect other, more adaptive methods for developing healthy self-esteem (38). The question of whether and how dieting may contribute to eating disorders is an important one that has been addressed elsewhere (39–42).

Before leaving this section, there is one other question worth asking. Although many health professionals and some researchers have argued that dieting is psychologically and medically harmful (18,19), almost no one asks whether *not* dieting or being restrained might be harmful [for exceptions, see Pinel et al. (43) and Peters et al. (25)]. The prevalence of obesity has grown tremendously during the past few decades in America and in many other countries around the world. No one would suggest that the millions of people who are heavier today than they were 10 or 20 years ago are better off as a result. In fact, because the majority of overweight people in the population do not join weight loss programs (34) and are presumably continuing to gain weight, it may be even more important to determine why so many people are not more restrained than it is to determine why a small percentage become so restrained that they develop disordered eating.

Why Is Weight Gain Prevention and Weight Loss Maintenance So Difficult?

In developed countries, there are several trends that illustrate the relentlessness of people's tendency to gain weight. First, despite overweight people's investments of effort, time, and money in losing weight, virtually all nonmedical approaches to weight loss are characterized by eventual weight regain, usually within several years (44). Second, programs aimed at weight gain prevention have produced disappointing results (45). Third, human populations in developed countries over the past several decades have been steadily gaining weight (46) despite significant physical, medical, social, and emotional burdens imposed by overweight.

Results from the National Weight Control Registry (47) have made it clear that, among those relatively rare individuals who are able to maintain a substantial weight loss, a major sustained effort is required. These individuals continue to eat low-calorie diets and exercise much more than most Americans (about 1 h/d of physical activity like brisk walking). Furthermore, even among members of this highly select group, 35% gained 5 lbs or more during a 1-year follow-up (47). Over 30 years of behavioral research has not materially improved the tendency of most dieters to regain weight (48). This is despite steady growth in the number of weight control procedures taught in lifestyle change programs. For instance, the popular LEARN manual (49), which has grown in length and comprehensiveness over the years, now has a "master list" of 162 techniques for helping people modify their lifestyles to lose weight and maintain the loss.

Evidence of the relentlessness of weight gain and weight regain does not mean the field should stop trying to improve people's ability to control their weight. In fact there are a number of promising leads to methods that may improve weight loss maintenance (50–52). However, it also seems appropriate to ask whether assumptions about obesity that are inherent in lifestyle change programs sufficiently reflect the actual nature of obese individuals' susceptibility to weight gain (and regain). In other words, in addition to asking how lifestyle change programs can improve overweight individuals' coping skills, social support, self-monitoring of food intake, and so on, we perhaps should also be asking if part of the reason the weight control field has made relatively little progress in developing long-lasting treatments for obesity is that the assumptions on which these treatments are based are in need of revision.

Lifestyle Change Approach to Weight Control

Comprehensive cognitive-behavior therapy programs—often also referred to as lifestyle change programs—are based on the assumption that overweight people need to acquire new lifestyle habits and skills to make permanent changes in their energy intake and energy expenditure. For instance, Brownell (53) has suggested that "lifestyle change—most notably, modification of eating behavior, physical activity, and psychological factors like attitudes, goals, and emotions—is the central determinant of whether people will lose weight and maintain the loss." The investigators who have developed and investigated these programs acknowledge that weight gain and obesity are the result of a multitude of genetic, biological, behavioral, social, and environmental factors (54–56). Whereas cognitive-behavior therapy programs assume that overweight individuals are eating too many calories and expending too little energy, there are few if any assumptions made about the relative importance of the multitudinous variables that make some people prone to weight gain. Rather, there is more of a "shotgun" approach, involving the incorporation of scores of techniques directed at a wide variety of lifestyle issues that might influence energy intake or expenditure (eating schedules, nutritional knowledge, negative beliefs about exercise, social support, coping skills, and so on). The presumption is that because there are many potential sources of overeating and sedentary behavior, the best way to control weight is to address all such sources in treatment programs. There is little or no prioritization of factors that contribute most to overweight individuals' difficulties with weight control.

Although more comprehensive cognitive-behavioral programs have produced larger weight losses than their fore-

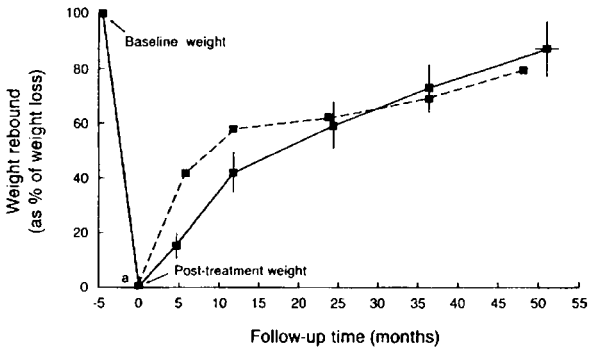

Figure 1: Weight rebound pattern of 24 obese females after being reduced to normal weight (solid line) compared with the mean results of 16 reports on diet and behavior modification programs (dotted line) (58). Reproduced with permission by the *American Journal of Clinical Nutrition.*

runners, the greater weight losses are mainly due to the longer duration of the programs (44). Furthermore, although lengthier and more comprehensive programs result in greater weight losses, there is little reason to believe that they slow the rate of weight regain when they end (57). In fact, a study by Hensrud et al. (58) suggests that there is little if any transfer of weight control behaviors learned during the weight loss period to the weight maintenance period. These investigators noted that virtually all studies on weight loss maintenance have involved a combination of weight loss and extensive instruction in weight control among individuals who actively sought out treatment. These authors wondered what would happen if a group of obese individuals were to lose weight without seeking or receiving any lifestyle intervention for weight control. Stated differently, these investigators were interested in studying the consequences of weight reduction per se, in the absence of the co-occurring influences of motivation to seek weight loss treatment and instruction in diet, exercise, and eating behavior change.

These researchers recruited a group of obese postmenopausal women, each of whom was put on an 800-kcal/d diet (until they lost at least 10 kg) so that all subjects were in the normal weight range when the study ended. The women did not join the study to lose weight and they were taught nothing about how to lose weight or maintain the weight loss they underwent. They were followed for ~5 years after the weight loss period ended. As a reference group, the authors examined 16 previously published studies that used diet and behavior modification to help overweight people lose weight and maintain the loss. They averaged the results of these 16 studies and graphed these average results along with those obtained with their group of obese women who had lost weight.

The results for the two groups are shown in Figure 1. It can be seen that the 24 obese study participants (represented by the solid line) had regained almost all of their lost weight by ~5 years after the weight loss period ended. The average rate of weight regain among participants in the 16 previous weight loss studies (represented by the dotted line) is shown for purposes of comparison. It is evident that both groups showed a rapid weight regain after weight loss, particularly in the first 2 years after weight loss ended. There was no substantial difference between the two groups, but there was a trend during the first 2 years of follow-up for the women who had received formal weight loss treatment to regain their weight faster than the women who had been given no instruction in weight control. The fact that the outcomes for the no-instruction group were no worse than those obtained by women who sought out and received a complete weight loss program strongly suggests that weight regain after a weight loss is an expected and "natural" outcome. As much as the participants in the 16 studies were motivated to lose weight permanently, and despite the fact that they were provided with the skills to do so, they were no better able to maintain their losses than were those whose weight loss was neither purposefully sought nor instructionally facilitated.

Perri and Corsica (57) reviewed about a dozen studies that did facilitate the maintenance of weight loss by extending treatment contacts by 1 year or longer. This review found that when the extended treatment contact finally ended, weight regain began and continued at the same rate shown by those not receiving the extended treatment. The results of both the Hensrud et al. (58) and Perri and Corsica (57) analyses indicate that the cognitive and behavioral changes taught in lifestyle programs, although effective while the program is in effect, do not produce sufficiently durable changes in obesity-prone individuals to counteract or ameliorate the pernicious effects of the obesigenic environment. A key question is whether making further refinements to lifestyle change programs might eventually succeed in equipping dieters with the motivation and self-regulatory skills required to improve the maintenance of lost weight. As Wilson (48) has suggested, the slow pace of progress in lifestyle change programs over the past several decades suggests that investing efforts to further refine such programs is unlikely to yield substantial benefits. However, there is accumulating evidence that, in terms of the energy intake side of the energy balance equation, focusing future efforts on modifying the structure and nutritional composition of the diet may hold substantial promise for improving weight loss maintenance.

Significance of the Food Environment in Long-Term Weight Control

There are three types of evidence that suggest that obese (and possibly obesity-prone) individuals have a heightened vulnerability to environments that offer unlimited quantities of a variety of foods high in caloric density (which tend to

be foods high in fat, sugar, or both). The first type of evidence involves taste preferences. Infants with overweight parents showed greater sucking avidity for a saccharin solution than infants with normal weight parents, even though the two groups of infants did not differ in body mass, body composition, or metabolic parameters (59). One year (59) and 4 years (R. Berkowitz, personal communication, 2002) later, the infants who showed the greatest sucking avidity when 3 months old also had gained the most weight. Overweight people, relative to those of normal weight, also have stronger taste preferences for food stimuli high in fat and sugar (60), passively overconsume energy after a high-fat preload (61), and select (62) and consume (63) more foods that are high in sugar and fat.

The second type of evidence involves eating regulation. Overweight individuals often, although not always, show poorer compensation for food preloads than normal weight people (64–67). The relationship between body mass and eating disregulation has been detected in children as young as 3 to 4 years old, in whom a powerful inverse relationship ($r = -0.72$) has been found between ability to regulate intake after a preload and skinfold measures of their level of body fat (68).

Normal weight restrained eaters (identified with the Restraint Scale) not only do not compensate for preloads but eat somewhat more after a preload than after no preload (8). In fact, one reason that overweight individuals have not more consistently been found to differ in eating regulation compared with normal weight people may be that many normal weight individuals are restrained eaters. The most likely reason for the similarity between eating regulation patterns in overweight individuals and normal weight restrained eaters is not that they are both restraining their intake (8) but that they share similar predispositions toward overeating and weight gain (31,41,69,70).

Understanding the source of eating regulation problems in overweight individuals is important because if it is caused by counter-regulatory responses to dieting behavior, then overweight individuals are caught in a perpetual catch-22. However, research during the past 15 years has found that none of the mechanisms that have been proposed to explain eating regulation problems in overweight and restrained normal weight individuals is capable of doing so. Neither current dieting (31,32,40,41,71,72), a history of failed dieting (69,73,74), nor weight suppression (31,47,71,75) can account for eating disturbances such as counter-regulatory eating and binge eating. [An important exception to this conclusion seems to be rapid, extensive weight loss, which is likely responsible for the emergence of binge eating in studies with both psychiatrically normal men (76) and bulimic individuals (77)]. Therefore, the difficulty that normal weight restrained eaters and many overweight individuals share with regard to eating regulation does not seem to stem from dieting behavior. Rather, the chronic dieting that is so common among both normal weight restrained eaters and overweight individuals seems to be a result of, rather than the cause of, problems with eating regulation. In line with this interpretation, various measures of dieting behavior have been shown to prospectively predict weight gain rather than weight loss (70).

The third type of evidence involves the effectiveness of nutrition change interventions in overweight individuals. An examination of both weight loss and weight regain in lifestyle change programs makes it clear that participants, on average, fall far short of reducing their caloric intake to the level prescribed by the programs (1200 kcal/d for most female participants, 1500 kcal/d for those with the highest BMIs). During weight loss, adherence to these calorie goals would produce average weight losses of roughly 4 kg/month (i.e., average prescribed intake of ~1300 kcal/d, in a group whose average energy needs would roughly be ~2500 kcal/d, would produce a 1200 kcal/d, or 36,000 kcal/mo, energy deficit). This translates into a 4- to 5-kg/mo weight loss; in an average program of 20 weeks, average weight loss in completers would be over 20 kg. However, actual average weight losses are less than one-half this amount (56). Increased metabolic efficiency might account for a small part of the discrepancy, although this will be offset to some extent by increases in physical activity shown by most participants. The point is that most participants are unable to restrict their caloric intake to the prescribed level even during the weight loss phase of lifestyle change programs, when participants are most highly motivated to lose weight. A similar conclusion was recently reached by Leslie et al. (78), who found that overweight dieters who were taught to restrict their daily intake by 600 kcal lost about the same amount of weight as those who were placed on a more restrictive 1500-kcal/d diet. The reason was that latter participants were unable to limit their intake even to a relatively lenient 1500-kcal/d diet.

Similarly, when lifestyle programs are extended to 40 to 52 weeks in length, participants lose much less weight than they would be expected to if their weight loss continued at the same pace as that achieved during the first 4 to 6 months of the program (56). Finally, it is apparent that after the weight loss phase, participants are unable to keep their food intake suppressed sufficiently to maintain their weight losses.

The evidence just reviewed suggests that the modern food-abundant environment may be particularly "toxic" (79) to individuals who are overweight or who are prone to become overweight. The modern environment also promotes weight gain and obesity because of the minimal energy expenditure required to function in everyday life. Given such an obesigenic environment, the question addressed in the following three sections is how obesity-prone people can better manage their energy intake, especially in

light of their greater than average susceptibility to overconsume energy in an environment replete with food and food cues.

Experimental Evidence of the Effects of Nutritional Interventions on Weight Maintenance

Structured Meals and Meal Replacements

If part of obese and obesity-prone individuals' susceptibility to being in positive energy balance stems from their sensitivity to the abundance of eating opportunities in the modern environment, then perhaps weight control interventions should place a much greater emphasis on *directly* controlling the food intake of such individuals. As suggested earlier, lifestyle change programs are based on an assumption of equipotentiality among their treatment components. That is, because there is approximately equal emphasis placed on modifying a wide variety of lifestyle habits, these programs assume that these habits have approximately equal influence on energy balance and, therefore, weight control. However, if interventions that focus on food itself are more likely than other interventions to actually produce long-term modifications in nutritional and caloric intake, then perhaps weight control programs should emphasize procedures that directly target what foods are consumed during weight loss and maintenance.

There is, in fact, growing evidence that interventions that specifically focus on modifying the structure, composition, and portion size of foods consumed are more effective than multipronged lifestyle change programs in promoting long-term weight control. It has been known since the 1970s that very-low-calorie diets (<800 kcal/d), which replace normal foods with preportioned liquid meals, are effective in helping people achieve large weight losses (80). There is evidence that the effectiveness of such meal replacements goes well beyond the prescription of a very low level of caloric intake and involves the minimization of contact with and preparation of foods, as well as the elimination of decisions about food choice and portion size. For instance, Foster et al. (81) found that obese individuals assigned to 420-, 660-, or 800-kcal/d diets lost equal amounts of weight. This suggests that the use of meal replacements helped participants reduce their caloric intake generally, not simply as a function of their assigned level of caloric intake. Furthermore, when meal replacements are used to make up the bulk of a low-calorie diet (~1100 kcal/d), the rate of weight loss is approximately double the rate on a prescribed 1200-kcal/d diet consisting entirely of regular foods (82,83). The apparent reason for the effectiveness of meal replacement–based weight loss diets is that they greatly facilitate dieters' adherence to the prescribed eating plan (because portion sizes are predetermined and food choices are minimized) relative to plans involving roughly the same caloric goal that require participants to purchase, prepare, and consume reduced amounts of many of the same foods that helped create the overweight state. The fact that participants in controlled trials of comprehensive lifestyle change programs—who are typically highly motivated to lose as much weight as possible (84)—cannot consistently limit themselves to the prescribed caloric intake of 1200 to 1500 kcal/d, again strongly suggests that overweight people are largely incapable of consistently restraining their food intake when choosing from the vast array of highly palatable foods that are constantly available to them.

There are two additional types of evidence that point to the powerful influence of the structure and portion size of food on weight loss and maintenance. Wing and Jeffrey (85) reviewed three pertinent studies that they and their colleagues conducted. They found that providing overweight individuals with either prepared meals and snacks for most of their food intake or with detailed meal plans and shopping lists significantly increased weight losses relative to conditions involving traditional lifestyle change programs. Metz et al. (86) also reported improved weight loss with a prepared meal plan. These studies suggest that gaining tight control over food availability and portion sizes adds substantially to the weight losses achieved with traditional lifestyle change programs that do not place such a strong emphasis on directly controlling what foods are consumed. Nonetheless, although food provision has usually [although not always (87,88)] been found helpful for facilitating weight loss, it has not facilitated weight loss maintenance (85,86).

Additional studies have evaluated the use of meal replacements (liquid meals and snack bars) during weight loss maintenance (82,89,90). Meal replacements (MRs)[1] are used to replace one meal, and in some studies, one snack per day to facilitate control of caloric intake and weight loss maintenance. A recent review of six meal replacement studies concluded that MRs produced significantly greater weight loss than all-food reduced calorie plans 3 and 12 months after the initiation of treatment (91). A particularly impressive outcome was reported by Ditschuneit et al. (82), who found that virtually all of a clinically significant weight loss was maintained after a 4-year period during which MRs were used to replace one meal and one snack per day. The fact that such a simple addition to weight maintenance programs is capable of producing substantial improvements in weight loss maintenance suggests that that these interventions may be effectively addressing a specific vulnerability (to the food-laden environment) that has contributed to relapse in many past studies. However, unresolved questions about specific studies need to be answered before definitive conclusions can be drawn. For instance, in the

[1] Nonstandard abbreviations: MR, meal replacement; REDE, reduced energy density eating.

Ditschuneit et al. (82) study, none of the 100 subjects dropped out during the 12-week weight loss phase, suggesting that the sample studied may not have been representative of participants in most weight control studies. Also, because both conditions in the Ditschuneit et al. (82) study received MRs during maintenance, the superior weight loss maintenance shown by both groups cannot be unequivocally attributed to the use of MRs. In the Ashley et al. (89) study, participants in all three conditions maintained their weight losses during the year-long treatments (albeit at different levels of body mass), making it difficult to know what role MRs played in the results obtained.

Role of Energy Density

All of the interventions described in the previous section depended on reducing or eliminating exposure to and preparation of a variety of foods. A completely different nutritional approach to eating and weight control is to systematically reduce the energy density of the diet consumed. There is increasing evidence that a major influence on people's short-term eating regulation is the volume of food they consume. The rationale for reduced-energy density eating is based on research showing that, when palatability is held constant, people eat a constant volume (or, in some studies, weight) of food regardless of the macronutrient composition or energy density of the food (92,93). Furthermore, reducing the energy density of a diet reduces energy intake without increasing hunger or producing short-term caloric compensation (92). Longer-term studies have found that when diets are systematically modified to lower their energy density, subjects do not completely compensate for the reduction in energy intake and gradually lose a small amount of weight (93).

Rolls and Bell (92) argue that when there is an abundant supply of palatable, high-calorie foods in the environment, it will be very difficult for people prone to weight gain to permanently control their food intake and avoid weight gain. They also note that if people try to avoid weight gain by limiting themselves to smaller portions of foods that are relatively high in energy density, they will have to restrict their food intake and cope with resulting feelings of hunger and deprivation. In addition, feelings of hunger and deprivation can have as much or more to do with "wanting" to eat food high in palatability as "needing" to eat food for energy repletion (94). The virtue of a low-energy-dense diet is that it allows people to eat a satisfying amount of food while limiting energy intake.

Weight control programs based on reduced energy density eating (REDE) are sometimes assumed to be indistinguishable from low-fat diets. Fat is the most energy dense macronutrient, so reductions in fat intake are, of course, emphasized in REDE programs (95,96). However, the concept behind REDE goes well beyond recommendations to reduce fat intake. Other approaches to reducing the energy density of the diet include eating water-rich and fiber-rich foods (96) and using sugar (97,98) and fat (99) substitutes to reduce calories and enhance palatability. Whether programs using REDE principles will differ from existing low-fat diets in terms of changes in nutritional intake or long-term weight control remains to be determined. However, there is preliminary evidence that a specific focus on reducing the energy density of the diet is more successful in promoting weight loss maintenance than interventions that attempt to restrict overall caloric intake (95,100).

Potential Role of High-Protein Diets in Weight Control

There is some empirical support for one other way in which the nutritional composition of the diet might facilitate weight control. There is evidence that protein enhances satiety more than equicaloric amounts of carbohydrate or fat (101,102) and that increased protein intake may facilitate weight loss (103,104). It is possible that both MRs and the Atkins diet facilitate weight control, at least in part, because they are higher in protein than the typical American diet. Research that manipulates protein composition of the diet during weight loss and weight maintenance will be needed to investigate this possibility.

Asymmetry in Humans' Body Weight Regulation

A major reason for the present emphasis on nutritional interventions for weight control is that it has become quite evident that body weight regulation in humans is asymmetrical. That is, the human body defends against long-term energy deficits much more vigorously than it does long-term energy surfeits (26). This means that 1) weight gain can occur effortlessly, and the accumulation of adipose tissue proceeds with little physiological resistance, and 2) weight loss occurs with difficulty, and the loss of adipose tissue is resisted tenaciously. It seems that the reason for this is that, throughout humans' evolutionary history, "overconsumption" typically enhanced survival, whereas inadequate energy intake threatened it (25). In fact, the adaptive value of eating when food is available (even when short- and long-term energy stores are adequate) is reflected in the presence of two different brain systems that underlie eating motivation. One system is activated by a negative energy balance and the other by the presence of food—and highly palatable food in particular (94). Thus, the mere presence of food is often sufficient to elicit a drive to eat it. In summary, the radical differences between the environment in developed countries and the environment that shaped our evolution facilitates both excessive energy intake and inadequate energy expenditure, producing the current obesity epidemic (25).

Self-Regulation of Energy Intake and Body Weight in Obesity-Prone Individuals: Is It Feasible?

We are now in a position to address the question posed in the title of this paper. Scientific research on energy balance regulation is providing the outlines of a model of obesity that is at odds with assumptions about obesity commonly held by both laymen and by many health care professionals. Weight problems are often viewed by laymen as a consequence of defects in character (e.g., a weak will, sloth) and by many professionals as a consequence of bad habits (e.g., eating in front of the TV, having negative weight-related thoughts, eating for emotional reasons). The model of weight regulation emerging from research on weight regulation (25,26,93,101), however, is one that views weight gain and obesity as an inevitable outcome of the mismatch between our evolutionary endowment and modern lifestyles in developed countries.

Given this evolutionary endowment, the key question is whether treatment and prevention programs that focus on improving self-regulation skills have the potential to sufficiently modify energy intake and energy expenditure to ameliorate or prevent obesity. On the energy intake side, it seems that in the present environment the answer is probably "no." [Interestingly, an apparent exception to this conclusion is the treatment of childhood obesity through programs that include parents as agents of change (105). The long-term success of these programs may be because of the fact that parents exert significant long-term control of their children's food intake and physical activity. Thus, these successful programs may teach us more about the wisdom of environmental (in this case, familial) change than they do about the effectiveness of self-regulation.]

Given the current obesigenic environments in developed countries, the modification of obese or obese-prone individuals' cognitive and behavioral skills does not seem to be sufficient to produce the type of permanent lifestyle change that will foster the maintenance of weight loss or the avoidance of weight gain. As Blundell and Gillett (26) put it, "attempted self-control of behavior is frequently unreliable because it tends to oppose biological tendencies and environmental pressures." Although long-term maintenance of substantial weight loss is sometimes achieved (47,106), this occurs in a very small proportion of those who attempt to lose weight. Similarly, educational and motivational programs aimed at the prevention of weight gain have had disappointing results (45).

Stated differently, it seems that the battle between our self-regulatory abilities and the obesigenic environment is not a fair fight (25); in the long run, most of those with a predisposition toward weight gain will, despite their best efforts, eventually gain weight. A promising approach to improving weight control is to focus preventive and treatment efforts on the nature of the foods to which vulnerable individuals are exposed. To the extent that such modifications are made, there will be less need to depend on self-regulation to achieve eating and weight control. As Skinner (107) has argued, "self-control" may best be achieved by arranging the environment in a way that maximizes the likelihood that the sought-after behavior patterns will occur. In the present case, this means modifying people's food environments, whether through individual-level changes such as the provision of meal replacements, or through society-wide changes in the food supply.

Changing the Environment vs. Changing Ourselves

Up to this point in the paper, most of the variables considered relevant to obesity have focused on the individual (e.g., dietary restraint, food self-monitoring, slowing eating rate, modifying weight-related beliefs). This focus reflects where most treatment and prevention interventions have been directed—toward the individual. The emphasis on changing individuals overlooks the fact that, of the factors affecting body mass that are potentially controllable, the most powerful ones are in the environment (food variety, energy density, portion size, etc.), not within individuals (personality, belief systems, attitudes). The reason that that the qualifier "potentially controllable" is used here is to differentiate between outcomes associated with *individual differences* in self-control and outcomes associated with attempts to *modify* self-control. In the former case, there are obviously tremendous individual differences in ability to exercise self-control, whether of eating or other health-related behaviors. For example, the participants in the National Weight Control Registry (47), who have demonstrated an impressive (and rare) ability to maintain a large weight loss, presumably are at the upper end of the distribution of obesity-prone individuals' ability to make long-term changes in their food intake and activity level. However, the fact that some people demonstrate such tight and prolonged control over their food intake, physical activity, and body weight does not mean that weight control professionals are able to instill this ability among those who do not naturally possess it. In fact, it is likely that a major determinant of why certain obese individuals choose to attend weight control programs in universities and medical centers—which have produced the vast majority of outcome studies on weight loss and maintenance—is that they feel so little control over their eating and weight.

Two Fundamentally Different Ways in Which Environmental Change Can Affect Weight Control

A major distinction can be drawn between two ways that the environment affects weight control. One is that the nature of the existing environment affects the behavior of

those living in it. This can be seen, for example, in the worsening of the obesity epidemic during that past 20 years or in the tendency for émigrés from nonwestern to western countries to gain weight. Once overweight or obesity develops, it is very difficult to permanently reverse the condition (56,108). This paper argues that programs aimed at achieving and maintaining medically significant (5% to 10% of starting body weight) weight losses should shift their emphasis from the modification of a diverse set of self-regulatory behaviors and cognitions to the direct modification of individuals' food environments. However, from a population perspective, where the goal should be the primary prevention—rather than the treatment—of overweight, even this approach is insufficient.

The primary prevention of obesity will require widespread structural changes in the availability, nature, and cost of foods, as well as major changes in parental feeding practices. This latter goal represents the second major way in which the environment affects weight control: through the development of appropriate eating-related knowledge and habits early in life. The significance of cultural and familial transmission of healthy eating habits is reflected in marked differences in prevalence of overweight and obesity in countries whose environments would seem to be similar in their obesity-promoting influences (e.g., the United States and France). Some of the apparent reasons for the reduced level of obesity in France—which have to do with cultural and familial norms and practices concerning food and eating—are starting to be documented (109).

Because there are large individual differences in the ability to regulate eating, which begin early in life (68,110), it is equally important for parents to learn how to recognize and respond to signs that their children are prone to excessive weight gain. Research on these topics is in its infancy, and it is by no means clear how parents can help prevent, or respond to, excessive weight gain in their children. Children as young as 3 to 4 years of age, like many older children and adults, do not compensate well for caloric loads (68). Fisher and Birch (110) found in young girls that amount eaten in the absence of hunger at age 7 was independently predicted by the girls' relative weight, eating in the absence of hunger, and level of parental restriction of their child's intake (all measured at age 5). Fisher and Birch (110) concluded that "parents' use of restrictive feeding practices is not effective in limiting children's food intake and can actually promote children's consumption of the restricted foods, even in the absence of hunger," although they also note that it is likely that "the relation between parents' use of restriction and child weight status is bidirectional." Similarly, Johnson (68) examined the relationship between parents' dieting and disinhibitory eating, on one hand, and their children's eating regulation, on the other. Levels of both dieting and disinhibited eating in mothers were related to problems with self-regulation of intake in their children. Johnson suggested that "mothers may be serving as role models for maladaptive eating strategies and behaviors."

Both Fisher and Birch (110) and Johnson (68) note that it is impossible to determine from their studies how much of parents' potential influence on their children's eating and weight is transmitted behaviorally and how much genetically. However, work by de Castro (111) suggests that genes may be more important than the environment in determining both body mass and the many aspects of energy intake that contribute to it. He concluded a review of such influences by noting that they "demonstrated that the genes independently affect body weight, height, overall daily intake, and meal intakes" (111). In a study analyzing genetic and common environmental influences on such variables (111), he found that "there was no discernible effect of familial (common) environment on any of these factors. Hence, these results agree with those of others. . . that the parental contribution to the intake regulation of their offspring results from their genetic contribution and not from their influence on the environment of their children." The contrasting viewpoints presented here are similar to the controversy, reviewed above, over whether restrained eating represents a cause or a consequence of eating and weight regulation problems. Obviously, resolving this fundamental issue will be key to determining the best strategies for preventing and treating obesity.

Concept of Personal Food Environments

Returning to the "environment vs. individual" comparison made above, it is important to note that environments are both chosen for us and chosen by us (112). In particular, our exposure to relevant food and physical activity environments is partially predetermined by the culture, community, and family we live in, but degree of exposure to certain contexts within those environments (e.g., going to fast food restaurants, regularly having dessert after dinner) is based on individuals' decision-making.

Efforts to modify the obesigenic environment are only beginning. On a society-wide basis, the current level of effort being devoted to educate the public and modify the food and physical activity environment to enhance weight control is comparable to the level of effort devoted to addressing the problem of smoking in the 1950s and 1960s. Furthermore, the complexity of the issues related to energy intake and energy expenditure and the challenge of modifying relevant environments make addressing the obesity epidemic even more challenging than addressing cigarette smoking. Therefore, it is likely that it will take many years to implement environmental changes that might have a meaningful impact on the still-worsening (46) epidemic of obesity. Based on these considerations, it would be helpful to distinguish what might be called the "personal food environment" from the "population food environment." In relation to energy intake, the personal food environment is

the sum total of all the food-related situations individuals encounter, create, or seek out in the course of their daily lives. Obviously, the degree to which individuals are exposed to food-related situations will normally depend heavily on the nature of food availability in the population as a whole. However, whereas modifying the food environment at the population level will be an enormous, long-term undertaking, helping people modify their "personal food environment" is far more feasible. Therefore, it seems that, from an obesity treatment and prevention perspective, the field should be working on two separate, although complementary, tracks. First, given the great difficulty of producing structural modifications in the food environments of populations, we should continue to develop ways to facilitate individuals' selection and consumption of foods that will enhance their personal weight control. Second, given the fact that evolutionary adaptations have made human beings highly vulnerable to weight gain in modern environments, a variety of population-level changes in the food supply will be needed to reduce the phenotypic expression of our genotypic vulnerability.

Rationale for a Nutrition-Focused Approach

The primary goals of lifestyle change programs for weight control are to modify factors that promote energy intake and limit physical activity. On the energy intake side, relevant lifestyle factors are manifold, complex, and often distal from the act of choosing, preparing, and consuming food. Furthermore, based on the literature reviewed in the previous several pages, it seems that food and food-related stimuli are more powerful determinants of food intake than are most of the other influences addressed in lifestyle programs. Thus, there are two separate rationales underlying the current emphasis on nutritional change for improving eating and weight control. First, lifestyle change programs are aimed at too many lifestyle factors, the lifestyle factors are too complex to realistically expect that they can be permanently modified, and most of the lifestyle factors are too distal from the primary target of interest (food intake) to expect that their modification, even if possible, would be sufficient for long-term weight control. Second, whereas lifestyle programs recognize the influence of environmental factors generally, they do not sufficiently take into consideration the power of food over obese individuals and, therefore, do not sufficiently emphasize the need to gain direct control over food and food stimuli.

Obesigenic Environments, Obesity-Proneness, and Personal Food Environments: An Integrated Approach

The recommendations made above concerning the importance to weight control of modifying the availability, structure, composition, and portion size of foods were made without much reference to the two major factors that should determine their appropriateness. The first factor is how a given environmental context affects energy balance at the population level—that is, how much the environment promotes sustained positive energy balance among the individuals living in it. As many commentators have observed (1,2,25,56), the environment in modern industrialized societies is highly obesigenic. The second factor is obesity-proneness. Because of individual differences in responsiveness to an obesigenic environment [much of it genetically based (113,114)], some individuals will be much more prone to gain weight over time, even if the nature of the environment is held constant.

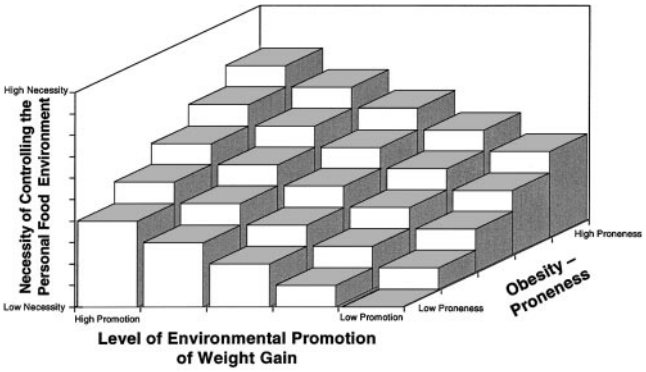

Figure 2: Schematic illustration of the hypothesized relationship between extent to which the environment promotes weight gain (*x* axis), proneness toward obesity (*z* axis), and the degree to which it may be necessary to control the personal food environment to achieve long-term weight control (*y* axis).

The argument advanced in this paper is that the more obesigenic the environment and the greater the level of obesity-proneness in a given individual, the more that an affected individual will need to develop direct control over his or her "personal food environment" to avoid weight gain (for those prone to weight gain) or maintain a weight loss (for those already overweight). These hypothesized relationships are illustrated in Figure 2, which is a schematic illustration of the presumed additive effects of obesigenic environments and obesity-proneness on the need to control one's personal food environment. (This figure is not based on data or particular studies but is meant only to illustrate the proposed relationships.)

Although the *x* and *z* axes represents continua, they are arbitrarily divided into five levels for illustrative purposes. If the *x* axis (environmental "obesigenicity") was used to characterize countries, then countries where the absolute level of obesity, and the extent of increase in obesity prevalence in recent years, is relatively low (e.g., many countries in Asia), would be at the lower end of this continuum. At the upper end would be countries where the absolute level of obesity, and the rate of increase in obesity, is high (e.g., the United States and some south Pacific islands). This dimen-

sion could capture other environmental continua such as socioeconomic status in developed countries, where obesity prevalence usually shows an inverse relation with socioeconomic status (115).

The *z* axis represents an individual's obesity-proneness, whether or not it is currently being manifested. This term is meant to encompass both degree of susceptibility to weight gain (for individuals in the normal weight range) and obesity severity (for those already overweight or obese). Obesity-proneness is assumed to interact with environmental variables (factors affecting energy intake and energy expenditure) to determine whether, and how much, weight is gained over time in a particular environment (as well as the feasibility of sustaining a weight loss). For those who are already overweight or obese, this dimension reflects the severity of the condition. Severity could be defined according to a number of overlapping criteria, including BMI, age of onset, length of time in an overweight state, binge eating frequency, and weight cycling history. For the sake of simplicity, all of these distinctions are subsumed under the term "obesity proneness" in Figure 2. This continuous dimension is also arbitrarily divided into five categories for illustrative purposes.

The *y* axis represents the extent to which gaining control over an individual's "personal food environment" may be necessary for that person to avoid weight gain or to maintain a weight loss. [Some of these considerations are also relevant to losing weight, but the purpose of the figure is to capture variables related to long-term weight control (avoidance of weight gain or maintenance of lost weight), not short-term weight loss.]

As can be seen in the pattern of bars in the figure, it is assumed that the more obesigenic the environment and the more obesity-prone the individual, the more that gaining direct control over the personal food environment will be necessary to manage the weight problem. The "food environment" encompasses a wide variety of food- and nutrition-related variables, many of which were reviewed earlier in this paper. These include use of structured meals (detailed menu plans, provision of premeasured prepared foods, meal replacements), controlling portion size, limiting food variety within meals, limiting exposure to foods high in energy density, ensuring continual availability of a variety of low-energy dense foods in the immediate environment (e.g., choosing appropriate restaurants, stocking one's home with appropriate ingredients and foods), increased use of sugar and fat substitutes, and increased intake of lean protein and fiber-rich foods (to facilitate satiety).

The inclusion of "personal" in the term "personal food environment" refers to the fact that the omnipresence of energy dense foods in the general environment makes it essential that obesity-prone individuals purposefully engineer the eating-related situations they regularly encounter to ensure that their food selections will be based more on their weight control needs than on the lure of foods that happen to be available in a given environment. The reason for the emphasis on directly controlling what foods people expose themselves to is that foods are biologically powerful stimuli, and through learning, can become psychologically powerful as well. As such, the appeal of food can easily overpower what people "know" is good for them.

This perspective may help clarify why lifestyle change programs have been largely unable to help people achieve long-lasting reductions in their weight. Most components of lifestyle change programs are aimed at information, thought patterns, and behaviors that are rather distal to the critical objective of influencing the type and amount of foods people eat. This approach is limited for at least two reasons. One is that the number of factors putatively related to food intake is so great (television watching, speed of eating, social support, negative emotions, self-defeating beliefs, fear of success, etc.). The other is the great difficulty of changing many of these lifestyle factors, especially on a long-term basis. The point is that if the primary objective of trying to change these influences is ultimately to modify food intake, why not first attempt to modify food intake directly by teaching people how to gain control over the nature of their personal food environments?

Returning to Figure 2, the "necessity of controlling personal food environment" axis reflects the hypothesis that this need increases with increases in the other two dimensions (shown on the *x* and *z* axes). (Because the graph is schematic, it shows equal increases in both dimensions as one moves from "low" to "high" on each. This implies that the two dimensions do not interact, which is probably incorrect.) In environments that are not highly conducive to weight gain and in people that have only a modest problem with (or predisposition toward) overweight, the need to base weight control on direct modifications of an individual's personal food environment is assumed to be low or nonexistent. In such cases, the provision of motivation, support, knowledge, and skills that comprise lifestyle change programs may be sufficient to avoid weight gain or produce a long-lasting reduction in weight. On the other hand, in environments or individuals at the other end of these respective dimensions, the potential for achieving long-term weight control is assumed to depend heavily on directly modifying an individual's personal food environment. Of note is the fact that most obesity treatment research has been aimed at individuals living in highly obesigenic environments (in Western countries) who suffer from relatively serious weight problems (e.g., with BMIs in the 30 to 40 range, a significant degree of binge eating problems, and many previous dieting failures). In this regard, it is also interesting that the most successful example to date of the use of a structured meal intervention (meal replacements) for long-term weight control used participants who became eligible for the meal replacement study only after they had

failed to lose significant weight after at least 3 months of an energy-restricted diet prescribed by the referring practitioner (82). However, because Figure 2 is not based on data, its purpose at this point is purely heuristic—that is, as a possible guide for designing weight control interventions based on the level of obesity-promoting influences in a given environment and the degree of susceptibility of the participants to weight gain or (in weight loss studies) weight regain.

It should also be noted that the relevance of creating personal food environments depends on how obesigenic a given environment is. To the extent that incipient efforts to modify social policies affecting the food supply (56,116) eventually succeed, the need for the personal food environment concept will decline.

To date, controlling the "food environment" has mostly taken the form of providing structured meals (82,85) or teaching people how to systematically reduce the energy density of their diet (93,95,96,100). However, although the steps that need to be taken to create personal food environments that would facilitate weight control among the general public are well-known, the proportion of people who are taking such steps is relatively small. For instance, the benefits of consuming more fruits and vegetables—both to enhance weight control and for better health generally—have been widely publicized. However, adults' consumption of fruits and vegetables remains far below recommended levels (117). The reasons for this are manifold, but the current potential for most adults to choose and consume a reduced energy-dense diet far outstrips actual practice. The point is that much work needs to be done among both individual consumers and food providers to modify the nutritional composition of the habitual diet. To the extent that such steps are taken, the need for highly structured eating plans and meal replacements—and lengthy, expensive clinical intervention programs—will be reduced.

The most effective approach to controlling the obesity problem at the population level would be to apply the principles of structured eating (e.g., smaller serving sizes) and reduced energy density eating not to individuals but to the food supply (food manufacturers, supermarkets, convenience stores, cafeterias, restaurants) so that many more foods are made available that could facilitate, rather than undermine, weight control in whole populations. At the same time, a massive effort to educate the public—and children in particular—about healthy eating and weight control will be needed to help ensure that there will be a market for healthier food choices when they are developed (25).

In conclusion, it is clear that solving the obesity epidemic will depend on making fundamental changes to the food environment, on one hand, and educating the population about healthy nutrition and eating habits, on the other. As Peters et al. (25) recently stated:

> "We can begin with the recognition that obesity is not a problem of defective physiological regulation, but is an environmental and societal problem and therefore, must be approached through environmental and social solutions. . . . We must recognize individual differences in susceptibility to obesity and realize that cognitive regulation of body weight will be harder for some than for others. Some may succeed by being given only minimal knowledge and skills and others may need considerable help, including substantial environmental change, to succeed at cognitive control of body weight."

From the evidence reviewed in this paper, it seems likely that focusing environmental efforts on modifying the availability, structure, and nutritional composition of foods may have a particularly important role to play in facilitating weight control. These efforts might include taking steps such as financial incentives for the purchase of healthier food items, the removal of vending machines from schools, the manufacture of a greater variety of low-energy-dense foods, and in commercial food establishments, the provision of nutrition information on all menu items and the inclusion of more reduced energy dense menu choices. Such a focus on changing the food environment is far more likely to succeed than is the alternative of attempting to better educate and motivate people to make healthier food choices while living in the midst of an obesigenic environment.

Acknowledgments

I thank Rachel Annunziato, Meghan Butryn, Jason Halford, Julie Ello Martin, Barbara Rolls, Tom Wadden, and Margriet Westerterp-Plantenga for comments on an earlier version of this paper and Meghan Butryn and Chris Ochner for assistance with references.

References

1. **Hill JO, Melanson EL.** Overview of the determinants of overweight and obesity: current evidence and research issues. *Med Sci Sports Exerc*. 1999;31:515–21.
2. **Jeffery RW, Utter J.** The changing environment and population of obesity. *Obes Res.* 2003;11(suppl):12S–22S.
3. **Schachter S.** Obesity and eating. *Science*. 1968;161:751–6.
4. **Nisbett RE.** Determinants of food intake in human obesity. *Science.* 1968;159;1254–5.
5. **Nisbett RE.** Hunger, obesity, and the ventromedial hypothalamus. *Psychol Rev*. 1972;79:433–53.
6. **Herman CP, Mack D.** Restrained and unrestrained eating. *J Personality*. 1975;43:647–60.
7. **Herman CP, Polivy J.** Anxiety, restraint and eating behavior. *J Abnormal Psychol*. 1975;84:666–72.
8. **Herman CP, Polivy J.** Restrained eating. In: Stunkard AJ, ed. *Obesity.* Philadelphia, PA: Saunders; 1980, pp. 208–25.
9. **Edmunds H, Hill AJ.** Dieting and the family context of eating in young adolescent children. *Int J Eat Disord*. 1999; 25:435–40.

10. **Hill AJ.** Prevalence and demographics of dieting. In: Fairburn CG, Brownell KD, eds. *Eating Disorders and Obesity: A Comprehensive Handbook*, 2nd ed. New York: Guilford; 2002, pp. 80–3.
11. **Huon GF, Godden VM, Brown LB.** Reports of dieting among children: effects of interview style and approach to information collection. *Br J Clin Psychol*. 1997;36:33–40.
12. **Rodin J, Silberstein L, Striegel-Moore R.** Women and weight: a normative discontent. *Nebr Symp Motivation*. 1984;32:267–307.
13. **Russell GFM.** Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychol Med*. 1979;9:429–48.
14. **Garner DM, Fairburn CG.** Relationship between anorexia nervosa and bulimia nervosa: diagnostic implications. In: Garner DM, Garfinkel PE, eds. *Diagnostic Issues in Anorexia Nervosa and Bulimia Nervosa.* New York: Brunner/Mazel; 1988, pp. 56–79.
15. **Patton CC, Johnson-Sabine E, Wood K, Mann AH, Wakeling A.** Abnormal eating attitudes in London schoolgirls—a prospective epidemiological study: outcome at twelve month follow-up. *Psychol Med*. 1990;20:383–94.
16. **Polivy J, Herman CP.** Dieting and binging: a casual analysis. *Am Psychol*. 1985;40:193–201.
17. **Wadden TA, Bell ST.** Understanding and treating obesity. In: Bellack AS, Hersen M, Kazdin AE, eds. *International Handbook of Behavior Modification and Therapy*, 2nd ed. New York: Plenum; 1990, pp. 449–73.
18. **Garner DM, Wooley SC.** Confronting the failure of behavioral and dietary treatments for obesity. *Clin Psychol Rev*. 1991;11:729–80.
19. **Polivy J, Herman CP.** *Breaking the Diet Habit.* New York: Basic; 1983.
20. **Orbach S.** *Fat Is a Feminist Issue: A Self-Help Guide for Compulsive Eaters.* New York: Berkley; 1985.
21. **Ciliska D.** *Beyond Dieting: Psychoeducational Interventions for Chronically Obese Women: A Non-Dieting Approach.* New York: Brunner/Mazel; 1990.
22. **Cogan JC, Ernsberger P.** Dying to be thin in the name of health: shifting the paradigm. *J Social Issues*. 1999;55:187–205.
23. **Foreyt JP, Goodrick GK.** *Living Without Dieting.* Houston, TX: Harrison: 1992.
24. **Roth G.** *Why Weight? A Guide to Ending Compulsive Eating.* New York: Plume; 1989.
25. **Peters JC, Wyatt HR, Donahoo WT, Hill JO.** From instinct to intellect: the challenge of maintaining healthy weight in the modern world. *Obes Rev*. 2002;3:69–74.
26. **Blundell JE, Gillett A.** Control of food intake in the obese. *Obes Res*. 2001;9:263–70.
27. **Mayer J, Roy P, Mitra KP.** Relation between caloric intake, body weight and physical work: studies in an industrial male population in West Bengal. *Am J Clin Nutr*. 1956;4:169–75.
28. **Herman CP, Polivy J.** A boundary model for the regulation of eating. In: Stunkard AJ, Stellar E, eds. *Eating and Its Disorders.* New York: Raven; 1984, pp. 141–56.
29. **Ruderman AJ.** Dietary restraint: a theoretical and empirical review. *Psychol Bull*. 1986;99:247–62.
30. **Costello RB, ed.** *American Heritage College Dictionary*, 3rd ed. New York: Houghton Mifflin; 1997.
31. **Lowe MR.** The effects of dieting on eating behavior: a three-factor model. *Psychol Bull*. 1993;114:100–21.
32. **Presnell K, Stice E.** An experimental test of the effect of weight-loss dieting on bulimic pathology: tipping the scales in a different direction. *J Abnorm Psychol.* 2003;112L:166–70.
33. **Van Strien T, Cleven A, Schippers G.** Restraint, tendency toward overeating and ice cream consumption. *Int J Eat Disord.* 2000;28:333–8.
34. **French SA, Jeffery RW.** Consequences of dieting to lose weight: effects on physical and mental health. *Health Psychol.* 1997;13:195–212.
35. **Brownell KD, Rodin J.** The dieting maelstrom: is it possible and advisable to lose weight? *Am Psychologist*. 1994;49: 781–91.
36. **Davison KK, Markey CN, Birch LL.** Etiology of body dissatisfaction and weight concerns among 5-year-old girls. *Appetite*. 2000;35:143–51.
37. **Brownell KD.** Dieting and the search for the perfect body: where physiology and culture collide. *Behav Ther*. 1991;22: 1–12.
38. **Kearney-Cook A, Striegel-Moore R.** The etiology and treatment of body image disturbance. In: Garner DM, Garfinkel PE, eds. *Handbook of Treatment for Eating Disorders*, 2nd ed. New York: Guilford; 1997, pp. 295–306.
39. **Heatherton TF, Polivy J.** Chronic dieting and eating disorders: a spiral model. In: Crowther J, Hobfall SE, Stephans MAP, Tennenbaum DL, eds. *The Etiology of Bulimia Nervosa: The Individual and Familial Context.* Washington, DC: Hemisphere; 1992, pp. 133–55.
40. **Lowe MR, Gleaves DH, DiSimone-Weiss RT, et al.** Restraint, dieting, and the continuum model of bulimia nervosa. *J Abnorm Psychol*. 1996;105:508–17.
41. **Lowe MR.** Dietary restraint and overeating. In: Fairburn CG, Brownell KD, eds. *Eating Disorders and Obesity: A Comprehensive Handbook*, 2nd ed. New York: Guilford; 1993, pp. 88–92.
42. **Wilson GT.** The controversy over dieting. In: Fairburn CG, Brownell KD, eds. *Eating Disorders and Obesity: A Comprehensive Handbook*, 2nd ed. New York: Guilford; 2002, pp. 93–7.
43. **Pinel JP, Assanand S, Lehman DR.** Hunger, eating, and ill health. *Am Psychol*. 2000;55:1105–16.
44. **Sarwer DB, Wadden TA.** The treatment of obesity: what's new, what's recommended. *J Women's Health Gender-Based Med*. 1999;8:483–93.
45. **Jeffery RW, French SA.** Preventing weight gain in adults: the Pound of Prevention study. *Am J Public Health*. 1999; 89:747–51.
46. **Flegal KM, Carroll MD, Ogden CL, Johnson CL.** Prevalence and trends in obesity among US adults, 1999–2000. *J Am Med Assoc*. 2002;288:1723–7.
47. **Wing RR, Hill JO.** Successful weight loss maintenance. *Annu Rev Nutr*. 2001;21:323–41.
48. **Wilson GT.** Behavioral treatment of obesity: thirty years and counting. *Adv Behav Res Ther*. 1994;16:31–75.
49. **Brownell KD.** *The LEARN Program for Weight Control*, 8th ed. Dallas, TX: American Health Publishing Co.; 1999.

50. **Perri MG, Nezu AM, McKelvey WF, Schein RL, Renjilian DA.** Effects of relapse prevention training and problem solving therapy in the long-term management of obesity. *J Consult Clin Psychol*. 2001;69:722–6.
51. **Wadden TA, Berkowitz RI, Sarwer DB, Prus-Wisniewski R, Steinberg C.** Benefits of lifestyle modification in the pharmacologic treatment of obesity: a randomized trial. *Arch Intern Med*. 2001;161:218–27.
52. **Wing RR, Jeffery RW.** Benefits of recruiting participants with friends and increasing social support for weight loss and maintenance. *J Consult Clin Psychol*. 1999;67:132–8.
53. **Brownell KD.** The central role of lifestyle change in long-term weight management. *Clin Cornerstone*. 1999;2:43–51.
54. **Brownell KD, Wadden TA.** Etiology and treatment of obesity: understanding a serious, prevalent, and refractory disorder. *J Consult Clin Psychol*. 1992;60:505–17.
55. **Perri MG, Nezu AM, Viegener BJ.** *Improving the Long-Term Management of Obesity: Theory, Research Clinical Guidelines.* New York: Wiley; 1992.
56. **Wadden TA, Brownell KD, Foster GD.** Obesity: responding to the global epidemic. *J Consult Clin Psychol*. 2002;70: 510–25.
57. **Perri MG, Corsica JA.** Improving the maintenance of weight lost in behavioral treatment of obesity. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment.* New York: Guilford; 2002, pp. 357–79.
58. **Hensrud DD, Weinsier RL, Darnell BE, Hunter GR.** A prospective study of weight maintenance in obese subjects reduced to normal body weight without weight-loss training. *Am J Clin Nutr*. 1994;60:688–94.
59. **Stunkard AJ, Berkowitz RI, Stallings VA, Schoeller DA.** Energy intake, not energy output, is a determinant of body size in infants. *Am J Clin Nutr*. 1999;69:524–30.
60. **Drewnowski A.** The behavioral phenotype in human obesity. In: Capaldi ED, ed. *Why We Eat What We Eat: The Psychology of Eating.* Washington, DC: American Psychological Association; 1996, pp. 291–308.
61. **Speechly DP, Buffenstein R.** Appetite dysfunction in obese males: evidence for role of hyperinsulinaemia in passive overconsumption with a high fat diet. *Eur J Clin Nutr*. 2000;54:225–33.
62. **Westerterp-Plantenga MS, Ijederma MJW, Wijckmans-Duijsens NEG.** The role of macronutrient selection in determining patterns of food intake in obese and non-obese women. *Eur J Clin Nutr*. 1996;50:580–91.
63. **Macdiarmid JI, Cade JE, Blundell JE.** The sugar-fat relationship revisited: differences in consumption between men and women of varying BMI. *Int J Obes Relat Metab Disord*. 1998;22:1053–61.
64. **Rodin J.** The current status of the internal-external obesity hypothesis: what went wrong? *Am Psychol*. 1981;36:361–72.
65. **Schachter S, Rodin J.** *Obese Humans and Rats.* Washington, DC: Erlbaum/Halsted; 1974.
66. **Roe LS, Thorwart ML, Pelkman CL, Rolls BJ.** A meta-analysis of factors predicting energy compensation in preloading studies. *FASEB J*. 1999;A871.
67. **Rolls BJ, Kim-Harris S, Fischman MW, et al.** Satiety after preloads with different amounts of fat and carbohydrate: implications for obesity. *Am J Clin Nutr*. 1994;60:476–87.
68. **Johnson SL.** Improving preschoolers' self-regulation of energy intake. *Pediatrics*. 2000;106:1429–35.
69. **Lowe MR, Timko A.** Toward an understanding of behavioral differences between restrained dieters and restrained nondieters. 2003, submitted for publication.
70. **Stice E, Cameron R, Killen JD, Hayward C, Taylor CB.** Naturalistic weight reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *J Consult Clin Psychol*. 1999;67:967–74.
71. **Lowe MR, Kleifield E.** Cognitive restraint, weight suppression, and the regulation of eating. *Appetite*. 1988;10:159–68.
72. **National Task Force on the Prevention and Treatment of Obesity.** Dieting and the development of eating disorders in overweight and obese adults. *Arch Intern Med.* 2000;160: 2581–9.
73. **Foster GD, Wadden TA, Kendall PC, Stunkard AJ, Vogt RA.** Psychological effects of weight loss and regain: a prospective evaluation. *J Consult Clin Psychol*. 1996;64:752–7.
74. **Wadden TA, Bartlett S, Letizia KA, Foster GD, Stunkard AJ, Conhill A.** Relationship of dieting history to resting metabolic rate, body composition, eating behavior, and subsequent weight loss. *Am J Clin Nutr*. 1992;56:203S–8S.
75. **Kleifield E, Lowe MR.** Weight loss and sweetness preferences: the effects of recent versus past weight loss. *Physiol Behav*. 1991;49:1037–42.
76. **Franklin JC, Schiele BC, Brozek J, Keys A.** Observations on human behavior in experimental semistarvation and rehabilitation. *J Clin Psychol.* 1948;4:28–45.
77. **Garner DM, Fairburn CG.** Relationship between anorexia nervosa and bulimia nervosa: diagnostic implications. In: Garner DM, Garfinkel PE, eds. *Diagnostic Issues in Anorexia Nervosa and Bulimia Nervosa.* New York: Brunner/ Mazel; 1988, pp. 56–79.
78. **Leslie WS, Lean MEJ, Baillie HM, Hankey CR.** Weight management: a comparison of existing dietary approaches in a work-site setting. *Int J Obes Relat Metab Disord*. 2002;26: 1469–75.
79. **Brownell KD.** The environment and obesity. In: Fairburn CG, Brownell KD, eds. *Eating Disorders and Obesity: A Comprehensive Handbook*, 2nd ed. New York: Guilford; 2002, pp. 433–8.
80. **Wadden TA, Bartlett SJ.** Very low calorie diet: an overview and appraisal. In: Wadden FA, Vanitallie TB, eds. *Treatment of the Seriously Obese Patient.* New York: Guilford; 1992, pp. 44–79.
81. **Foster GD, Wadden TA, Peterson FJ, Letizia KA, Bartlett SJ, Conill AM.** A controlled comparison of three very-low-calorie diets: effects on weight, body composition and symptoms. *Am J Clin Nutr*. 1992;55:811–7.
82. **Ditschuneit HH, Fletchtner-Mors M, Johnson TD, Adler G.** Metabolic and weight-loss effects of a long-term dietary intervention in obese patients. *Am J Clin Nutr*. 1999;69:198–204.
83. **Lowe MR, Foster GD, Kerzhnerman I, Swain RM, Wadden TA.** Restrictive dieting versus "undieting": effects on eating regulation in obese clinic attenders. *Addict Behav*. 2001;26:253–66.

84. **Foster GD, Wadden TA, Vogt RA, Brewer G.** What is a reasonable weight loss? Patients' expectations and evaluations of obesity treatment outcomes. *J Consult Clin Psychol*. 1997;65:79–85.
85. **Wing RR, Jeffery RW.** Food provision as a strategy to promote weight loss. *Obes Res.*, 2001;9:271S–5S.
86. **Metz JA, Stern JS, Kris-Etherton P, et al.** A randomized trial of improved weight loss with a prepared meal plan in overweight and obese patients: impact on cardiovascular risk reduction. *Arch Intern Med.* 2000;160:2150–8.
87. **Ash S, Reeves M, Yeo S, Morrison G, Carey D, Capra S.** Effect of intensive dietetic interventions on weight and glycaemic control in overweight men with Type II diabetes: a randomized trial. *Int J Obes Relat Metab Disord*. 2003;27: 797–802.
88. **Pi-Sunyer FX, Maggio CA, McCarron DA, et al.** Multicenter randomized trial of a comprehensive prepared meal program in type 2 diabetes. *Diabetes Care*. 1999;22:191–7.
89. **Ashley JM, St. Jeor ST, Schrage JP, et al.** Weight control in the physician's office. *Arch Intern Med*. 2001;161:1599–604.
90. **Rothacker DQ.** Five-year self-management of weight using meal replacements: comparison with matched controls in rural Wisconsin. *Nutrition*. 2000;16:344–8.
91. **Heymsfield SB, van Mierlo CA, van der Knaap HC, Heo M, Frier HI.** Weight management using a meal replacement strategy: meta and pooling analysis from six studies. *Int J Obes Relat Metab Disord*. 2003;27:537–49.
92. **Rolls BJ, Bell EA.** Dietary approaches to the treatment of obesity. *Med Clin North Am.* 2000;84:401–18.
93. **Yao M, Roberts SB.** Dietary energy density and weight regulation. *Nutr Rev*. 2001;59:247–58.
94. **Levine AS, Billington CJ.** Why do we eat? A neural systems approach. *Annu Rev Nutr*. 1997;17:597–619.
95. **Lowe MR, Annunziato R, Riddell L, et al.** Controlled trial of a nutrition-focused treatment for weight loss maintenance. Oral presentation given at the International Congress on Obesity, Sao Paulo, Brazil, August 2002.
96. **Rolls BJ, Barnett R.** *Volumetrics. Feel Full on Fewer Calories.* New York: HarperCollins Publishers; 2000.
97. **Blackburn GL, Kanders BS, Lavin PT, et al.** The effect of aspartame as part of a multidisciplinary weight-control program on short- and long-term control of body weight. *Am J Clin Nutr*. 1997;65:409–18.
98. **Raben A, Vasilaras TH, Moller AC, Astrup A.** Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *Am J Clin Nutr*. 2002;76; 721–9.
99. **Eldridge AL, Cooper DA, Peters JC.** A role for olestra in body weight management. *Obes Rev*. 2002;3:17–23.
100. **Toubro S, Astrup A.** Randomized comparison of diets for maintaining obese subjects' weight after major weight loss: ad lib, low fat, high carbohydrate diet versus fixed energy intake. *Br Med J*. 1997;314:29–42.
101. **Bell EA, Rolls BJ.** Regulation of energy intake: factors contributing to obesity. In: Bowman BA, Russell RM, eds. *Present Knowledge in Nutrition*, 8th ed. Washington, DC: ILSI Press; 2001; pp. 31–40.
102. **Stubbs J, Ferres S, Horgan G.** Energy density of foods: effects on energy intake. *Crit Rev Food Sci Nutr*. 2000;40: 481–515.
103. **Eisenstein J, Roberts SB, Dallal G, Saltzman E.** High-protein weight-loss diets: are they safe and do they work? A review of the experimental and epidemiologic data. *Nutr Rev*. 2002;60:189–200.
104. **Johnston CS, Day CS, Swan PD.** Postprandial thermogenesis is increased 100% on a high-protein, low-fat diet versus a high-carbohydrate, low-fat diet in healthy, young women. *J Am Coll Nutr*. 2002;21:55–61.
105. **Goldfield GS, Raynor HA, Epstein LH.** Treatment of pediatric obesity. In: Wadden TA, Stunkard AJ, eds. *Handbook of Obesity Treatment.* New York: Guilford; 2002, pp. 532–55.
106. **Lowe MR, Miller-Kovach K, Phelan S.** Weight-loss maintenance one to five years following successful completion of a commercial weight loss program. *Int J Obes Relat Metab Disord*. 2001;25:325–31.
107. **Skinner BF.** *Science and Human Behavior*. New York: The Free Press; 1953.
108. **Levin BE.** Metabolic imprinting on genetically predisposed neural circuits perpetuates obesity. *Nutrition.* 2000;16:909–15.
109. **Rozin P, Kabnick K, Pete E, Fischler C, Shields C.** The ecology of eating: part of the French paradox results from lower food intake in French than Americans, because of smaller portion sizes. *Psychol Sci.* (in press).
110. **Fisher JO, Birch LL.** Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. *Am J Clin Nutr*. 2002;76:226–31.
111. **de Castro J.** Inheritance of premeal stomach content influences on food intake in free living humans. *Physiol Behav*. 1999;66:223–32.
112. **Bandura A.** *Social Foundations of Thought and Action: A Social Cognitive Theory*. Englewood Cliffs, NJ: Prentice-Hall; 1986.
113. **Bouchard C, Tremblay A, Despres JP, et al.** The response to long-term overfeeding in identical twins. *N Engl J Med*. 1990;322:1477–82.
114. **Stunkard AJ, Harris JR, Pedersen NL, McClearn GE.** The body mass index of twins who have been reared apart. *N Engl J Med*. 1990;322:1483–7.
115. **Sobal J, Stunkard AJ.** Socioeconomic status and obesity: a review of the literature. *Psychol Bull*. 1989;105:260–75.
116. **Nestle M, Jacobson MF.** Halting the obesity epidemic: a public health policy approach. *Public Health Rep*. 2000;15: 12–24.
117. **U. S. Department of Agriculture and U. S. Department of Health and Human Services.** *Nutrition and Your Health: Dietary Guidelines for Americans*, 5th ed. Home and Garden Bulletin 232. Washington, DC: U.S. Government Printing Office; 2000.