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CONFRONTING THE FAILURE OF BEHAVIORAL AND DIETARY TREATMENTS FOR OBESITY

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ABSTRACT. *This review questions the appropriateness of behavioral and dietary treatments of obesity in light of overwhelming evidence that they are ineffective in producing lasting weight loss. The stigmatization of obesity, the overstatement of health risks, and the pervasive influence of the lucrative diet industry have maintained public demand for dietary treatment. However, decades of research on the biology of weight regulation make clear the unlikelihood of success with dietary treatment, information which the health professions have been slow to integrate. Recommendations are made for improving lifestyle, health risk factors, body image, and the self-esteem of the obese without requiring weight loss.*

It has been over a decade since two major reviews questioned the effectiveness and social appropriateness of behavioral treatments for obesity (Stunkard & Penick, 1979; Wooley, Wooley, & Dyrenforth, 1979b). Other papers and books have since appeared challenging the basic precepts which underlie dietary treatments for obesity (Bennett, 1984, 1987; Bennett & Gurin, 1982; Ernsberger & Haskew, 1987; Fitzgerald, 1981; Foreyt, Goodrick, & Gotto, 1981; Krieshok & Karpowitz, 1988; Wooley & Wooley, 1982, 1984; Wooley, Wooley, & Dyrenforth, 1979a). Their arguments, however, have not been embraced, accepted, or in many cases, even addressed by the mainstream of behavioral scientists and health care pro-

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fessionals who treat obesity. It is still widely held within the health care professions that obesity confers significant health risks warranting weight reduction. Weight loss as a means for achieving health and happiness is vigorously promoted by the commercial weight loss industry, now a major economic force in North America.

Behavioral or dietary treatments for mild and moderate obesity[1] continue to be advocated despite weak and often conflicting epidemiological data suggesting that these levels of obesity are linked to significant health risks (cf. Ernsberger & Haskew, 1987; Mann, 1974a, 1974b), and despite overwhelming evidence from controlled studies that weight loss programs are ineffective in producing lasting weight change (cf. Bennett, 1987; Stunkard & Penick, 1979). One can point to behavioral programs for weight control recommended in the same publications which document physiological resistances to weight change, seemingly without recognition of the contradictions or problems involved in trying to override the body's biological regulatory mechanisms. Our failure to fully confront these issues has meant that, despite new knowledge, there has been no fundamental change in our practices. Old diets with new names, such as the "set point diet," seem almost to parody the efforts of scientists to understand the causes of obesity and provide treatments consistent with this understanding.

Another expository review of obesity treatment studies is unnecessary since the topic has been comprehensively reviewed elsewhere (Bennett, 1986; Brownell, 1982; Brownell & Wadden, 1986; Foreyt, 1977; Foreyt et al., 1981; Jeffery, 1987; Stunkard & Mahoney, 1976; Wilson & Bronwell, 1980). Instead, the primary aim of this paper is to provide an evaluative and integrative appraisal of weight loss treatments within the context of what is known about (a) long-term treatment efficacy, (b) the biology of weight regulation, (c) eating patterns of the obese, (d) the genetic determinants of obesity, and (e) the health risks associated with obesity. In view of this evidence, it will be argued that mental health professionals should, under most circumstances, be advised *against* the delivery of dietary or behavioral treatments for mild or moderate obesity rather than proposing more aggressive dietary approaches (Brownell & Jeffery, 1987). When weight reduction is offered, consumers should be given complete information about risks and probable outcome. Rather than expending further resources on traditional treatments of obesity, health professionals should be encouraged to further develop alternative approaches that more adequately address the physi-

[1]There have been many attempts to define and classify obesity using either weight relative to norms, height to weight ratios, measures of body fat, or regional distribution of fat (cf. Bjorntorp, 1987; Bray, 1978; NRC, 1989). Stunkard (1984) has proposed a simple scheme of defining obesity relative to norms as mild (20%–40% overweight), moderate (41%–100% overweight), or severe (more than 100% overweight). According to this definition, about 35% of women in this country are obese and of this group, 90.5% are mildly, 9% are moderately, and 0.5% severely obese (Stunkard, 1984). These definitions of obesity are all arbitrary in the sense that they define the condition using cutoff points along a normal distribution of body weight or fat, without reference to etiology or disease. Most of the behavioral and dietary research that will be summarized in this review has been directed toward the mild and moderate groups. In the interest of avoiding tedium, the current review will not define the way that the term was used in each study cited unless it has specific relevance to the interpretation of the results.

cal, psychological, and social hazards associated with obesity without requiring dieting or weight loss.

THE SOCIAL CONTEXT OF OBESITY TREATMENT

The enormity of the efforts directed toward eradicating obesity is perhaps best illustrated in economic terms. According to a recent market analysis summarized by *Obesity and Health* ("What's Ahead?," 1989), the 1988 cost of weight loss products and services in the United States alone was estimated at over $29 billion. To put this figure in perspective, the Federal Government of the United States spent a total of $31.9 billion in 1988 on education, training, employment, and social services (Office of Management and Budget [OMB], 1990). According to the weight loss industry report in 1988, $4.5 billion were spent in 1988 in hospital based clinics and weight loss programs, another $1.5 billion in the almost 13,000 nonhospital programs, and about the same amount on residential spas offering weight loss programs. Americans additionally spent $8 billion on health spas and exercise clubs, $382 million on 54 million diet books, $10 billion on diet soft drinks, and billions more on low calorie foods and artificial sweeteners. Surgery is not included in this analysis, but there are an estimated 30,000 stomach redution and jaw wiring operations per year. Additionally, about 100,000 liposuction procedures were performed at a cost of $3,500 apiece. The weight loss industry is a major economic force in this country, projected to reach over $50 billion by 1995.

For several decades the fashion, entertainment, and publishing industries have bombarded women with role models for physical attractiveness so gaunt as to represent virtually no women in the actual population (Garner & Garfinkel, 1980; Garner, Rockert, Olmsted, Johnson, & Coscina, 1985). These representations imply that not only beauty but also success, personal happiness, and self-worth can be achieved through slenderness. There seems little doubt that cultural pressures for slimness have intensified in the past 30 years (Garner, Garfinkel, Schwartz, & Thompson, 1980; Silverstein, Perdue, Peterson, Vogel, & Fantini, 1986). Although the reasons for this are unclear, one argument holds that as women have moved into previously male-dominated activities, the "traditional" female body shape has developed negative connotations while the masculine shape has come to symbolize self-discipline and competency (Bennett & Gurin, 1982; Wooley & Wooley, 1985). Indeed, Silverstein et al. (1986) found that curvaceousness went out of style both in the 1920s and in the 1970s and 1980s, both periods of peak activity on the part of women to gain male prerogatives. Marketing geniuses, if not responsible for these trends, have certainly capitalized on them by promoting self-consciousness and personal discontent so that consumers will believe that they require the remedies the advertisers offer (Ewen, 1976; Wachtel, 1989).

The power of these influences is evidenced in studies showing that the majority of adolescent and young adult women feel fat even when their weight falls within the normal range (e.g., Dwyer, Feldman, & Mayer, 1967; Huenemann, Shapiro, Hampton, & Mitchell, 1966; Klesges, 1983; Moses, Banilivy, & Lifshitz, 1989; Nylander, 1971; Wardle & Beales, 1986). This has led Rodin, Silberstein, and Striegel-Moore (1985) to characterize the shape dissatisfaction endemic to young women as "normative discontent." Given these attitudes, it is not surpris-

ing that about two out of every three high school girls report that they are trying to lose weight (Rosen & Gross, 1987). Increasingly, there are reports indicating that substantial numbers of young women use harmful weight control behaviors, such as self-induced vomiting, with a subgroup of these manifesting serious eating disorders (Crowther, Post, & Zaynor, 1985; Killen et al., 1987; Pyle, Halvorson, Neuman, & Mitchell, 1986). Recent studies indicate that feeling fat and restricting food intake are not uncommon in schoolchildren 8–13 years of age (Davies & Furnham, 1986; Maloney, McGuire, Daniels, & Specker, 1989; Wardle & Beales, 1986). One of the more alarming manifestations of the fear of obesity is a subgroup of children with short-stature syndrome and delayed puberty caused by rigorous dieting due to fear of fatness (Puglifse, Lifshitz, Grad, Fort, & Marks-Katz, 1983). The desperation connoted by these behaviors is easily overlooked in a cultural climate which champions thinness as an indisputable virtue. Many of the behaviors used for diagnoses of anorexia nervosa and bulimia nervosa are culturally syntonic, considered neither uncommon nor abnormal by the lay public (Huon, Brown, & Morris, 1988). Even anorexia nervosa itself has developed a not altogether unfavorable connotation in our culture (Branch & Eurman, 1980; Bruch, 1985; Garner et al., 1985; Wooley & Wooley, 1982).

The intensity of the prevailing cultural pressures for thinness and the psychological risks that they impart have particularly serious implications for those who suffer from moderate to extreme obesity (Wooley et al., 1979a). Shape dissatisfaction and attempts to restrict food intake are evident in all but a small minority of women who are statistically overweight (Davies & Furnham, 1986; Wardle & Beales, 1986), and extreme body image disparagement has been recognized for years as a grim consequence of obesity (Stunkard & Burt, 1967; Stunkard & Mendelson, 1967; Wadden & Stunkard, 1985).

The social stigma against obesity is extraordinary in its magnitude and pervasiveness. As Fitzgerald (1981) notes: "Public derision and condemnation of fat people is one of the few remaining social prejudices . . . allowed against any group based solely on appearance" (p. 223). It is well documented that obese people are denied educational opportunities, jobs, promotions, and housing because of their weight (Bray, 1976; Canning & Mayer, 1966; Karris, 1977). The affront to those who are obese goes beyond the almost uniform judgment that they are unattractive (Furnham & Radley, 1989) and includes negative stereotypes that begin early in childhood. The aversion to obesity is evident by kindergarten (Lerner, 1969; Lerner & Gelbert, 1969), and grade-school children consistently associate larger body shapes with adjectives such as "stupid," "dirty," "lazy," "sloppy," "mean," "ugly," and "sad" (Maddox, Black, & Liederman, 1968; Staffieri, 1967, 1972; see Wooley et al., 1979a for a review).

Harsh attitudes toward the obese depend on the assumption that they bring their condition on themselves through lack of willpower and self-control (cf. Harris & Smith, 1982; Maddox et al., 1968). In the face of the overwhelming social rejection, obese individuals may seek professional support; however, research has shown that health professionals share in the culture's perjorative view of obesity and that this prejudice may influence their clinical judgement (Brotman, Stern, & Herzog, 1984; Maiman, Wang, Becker, Finlay, & Simonson, 1979; Richardson, Goodman, Hastorf, & Dornbusch, 1961; Young & Powell, 1985). A negative attitude toward obesity is sufficiently ingrained in our social fabric that,

in addition to social rewards for success, obesity treatment programs have at times included social shaming procedures as a treatment principle. Many authors have advocated a critical attitude toward obese patients (cf. Wooley & Wooley, 1980). Confronted with evidence of consistent failure of behavioral treatment programs — a failure seemingly understandable only as a consequence of widespread patient noncompliance — several respected researchers concluded that social pressure on obesity needed to be increased:

> The ultimate social pressure treatment would be to increase the social sanctions against obesity, so that being overweight would be a tremendously shameful thing. In this manner, obesity would be under external control, as are other social behaviors for which society has learned that internal control is not enough for some, as in the case of sexual or criminal acts. Obesity control would then be inculcated into the normal socialization process for children. (Foreyt et al., 1981, p. 170)

This statement no doubt expressed for a generation of researchers the frustration of reported failure and needs to be understood not as the position of an individual but as an illustration of the way in which ineffective treatment strategies lead, as an almost inevitable consequence, to blaming the victim.

BEHAVIORAL AND DIETARY TREATMENT OF OBESITY

The Paradox of Dietary Treatment of Obesity

There are two indisputable facts regarding dietary treatment of obesity. The first is that virtually all programs appear to be able to demonstrate moderate success in promoting at least some short-term weight loss. The second is that there is virtually no evidence that clinically significant weight loss can be maintained over the long-term by the vast majority of people. Since health professionals and professional societies recommend weight loss as the treatment of choice for the 23 million or so Americans judged to be overweight, the apparent inconsistency between the short- and long-term treatment findings needs to be illuminated.

Short-Term Follow-Up Studies

Behavioral weight control programs for obesity are often described as demonstrating promising treatment effects that are well maintained during the first year of follow-up (cf. Bennett, 1986; Brownell & Jeffery, 1987; Wadden & Stunkard, 1989). In an analysis of the results from 105 behavioral studies, Bennett (1986) reported a mean weight loss for the 6,121 participants of 5.38 kg after an average of 13.37 weeks of treatment. The modest treatment effects were maintained in the short-term with a mean weight loss of 5.11 kg recorded for 5,453 participants followed for an average of 35.5 weeks.

Although most of the behavioral treatment studies have involved only short-term evaluation of effectiveness, follow-up duration has increased in recent years. Brownell and Jeffery (1987) have reported that the average length of follow-up for a sample of studies appearing in several major psychology journals was 15.5 weeks in 1974, 30.3 weeks in 1978, 58.4 weeks in 1984, and 44 weeks in 1986. The duration of treatments provided and the amount of weight loss achieved has increased over the same period, although the number of pounds lost per week has not changed dramatically (Brownell & Jeffery, 1987).

There has been a move away from short-term, strictly "behavioral" programs to greater emphasis on multicomponent interventions that incorporate exercise, social influence, longer treatment duration, and continued therapeutic contact after the end of formal treatment. These strategies have been found to promote greater weight losses and improved maintenance during the first 18 months after treatment (Perri, McAdoo, McAllister, Lauer, & Yancey, 1986; Perri et al., 1988; Perri, Nezu, Patti, & McCann, 1989), leading some authors to the conclusion that obesity might be reconceptualized as a chronic condition "requiring some form of continuous-care program long after completing an initial period of treatment" (Perri et al., 1988, p. 533). However, over and over again the initial encouraging findings are eroded with time. Following up their initially impressive treatment results, Perri et al. (1989), for example, state "After clients conclude their active involvement in obesity treatment, they typically abandon weight loss strategies and begin to regain weight" (p. 452).

The problem is a subtle one that occurs repeatedly; results are presented as positive when, in fact, all indications are that the long-term projections are quite poor. This matter of interpretation is an especially questionable convention since there are consistent data on the pattern of weight gain that follows weight loss achieved through a wide range of treatment strategies. To give a fairly typical example, the findings of Craighead, Stunkard, and O'Brien (1981) are often cited as one of the more successful behavioral programs in maintaining weight loss (Figure 1). It is evident from Figure 1 that the active treatments led to statistically significant weight loss and that there was a trend for the drug and combined treatments to produce weight loss superior to the behavioral therapy alone. At one year follow-up, however, the behavior therapy group showed the least regain. In discussing these results, Stunkard (1984) states:

> This most recent study provides grounds for optimism as to the future of behavioral treatment of obesity . . . over the long run, behavior therapy clearly outperformed the most potent alternative treatment with which it has yet been compared. (p. 165)

This interpretation does not reflect an appreciation of the apparent trajectory of the posttreatment weight rebound for all treatment groups. The most parsimonious interpretation of these findings is that the participants are returning to their pretreatment weight, and the graph is simply a snapshot of this process one year after the end of treatment. It also fails to acknowledge that the trend toward weight gain, together with similar findings from other studies, probably represents a basic underlying mechanism unlikely to show a reversal or levelling off if data were to be collected at subsequent follow-up points.

Brownell and Jeffery (1987) have similarly avoided the most obvious implication of these and comparable results, arguing that the negative findings may actually be positive since those who have regained most or all of their weight at follow-up might have weighed even more had they not received treatment. To give this speculation more credence than the alternative possibilities that obese individuals would be at the same or lower weights if they had not received any treatment goes beyond data on long-term weight fluctuations in the obese (Williamson & Levy, 1988). Findings from another study by Brownell and colleagues (Brownell, Greenwood, Stellar, & Shrager, 1986), as well as others to be

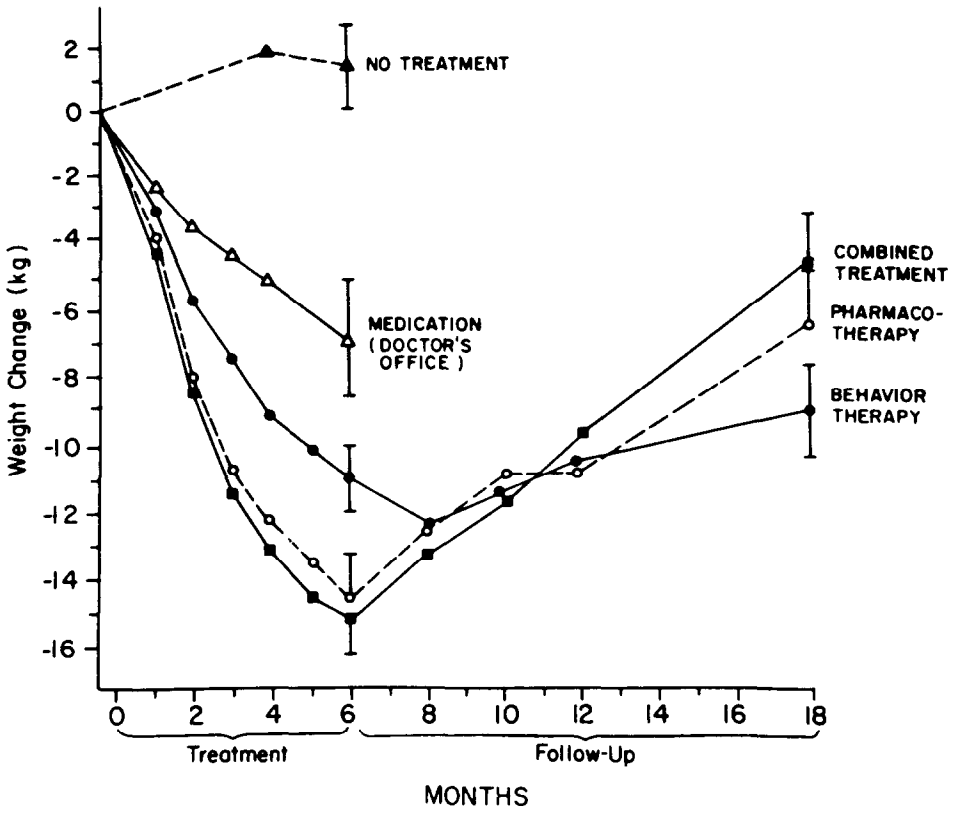

FIGURE 1. Weight Changes During 6-mo Treatment and 12-mo Follow-Up. (Vertical lines represent 1 SEM. From "Behavior Therapy and Pharmacotherapy for Obesity" by L. W. Craighead, A. J. Stunkard, and R. M. O'Brien, 1981, *Archives of General Psychiatry, 38,* pp. 763–768. Copyright 1981 by the American Medical Association. Reprinted by permission.)

discussed later, support the hypothesis that dieting itself may lead to enhanced metabolic efficiency (i.e., greater energy storage at a fixed energy inake) and a possible tendency to maintain a higher body weight.

Brownell and Jeffery (1987) also assert that the failure to demonstrate success in clinical samples does not obviate the possibility that weight loss may be a realistic goal for those who do not seek treatment. It is reasoned that treatment studies draw participants from clinical settings; these may be the most recalcitrant individuals from the potential pool of candidates for weight loss. While this is obviously a theoretical possibility and is supported by one study on a highly selected sample (Schachter, 1982), it seems to stretch the weight loss litany to its limits and leads to the curious interpretation that treatment is most appropriate for those who do not seek professional help.

Against a tradition in science which assumes that there are no treatment effects until they are demonstrated, there is an unchallenged convention by which weight loss interventions are presumed effective until there is explicit evidence

to the contrary. The reality is that we do not have effective treatment to offer, and we should be candid about this until there is reliable evidence to the contrary. To avoid confronting the failures of obesity treatment is to mislead a public desperately waiting with cash in hand for an effective remedy.

Another criticism of behavioral and certain other treatments for obesity is that the amount of weight loss achieved, while statistically significant, is generally too small to be of practical consequence (Wing & Jeffery, 1979). According to Brownell and Jeffery (1987), the mean weight loss for controlled behavioral trials reported in 1984 was 15.4 lb, with an average of just over 13 weeks of treatment; however, the initial mean weight of participants was 197 lb.

In answer to the rather modest weight losses achieved by most dietary approaches, it has been recommended that more aggressive methods be implemented in order to achieve greater initial losses (Brownell & Jeffery, 1987). These have included very low calorie diets (VLCD); longer treatments; and the addition of exercise, financial contracts, relapse prevention, and social support to behavioral regimens (Brownell & Jeffery, 1987; Brownell, Marlatt, Lichtenstein, & Wilson, 1986). These proposals all appear to presume that maintenance of weight loss is a realistic expectation and/or that failure to maintain weight loss does no harm. As will be more evident later, both of these assumptions are inconsistent with much of what we know.

Long-Term Follow-Up Studies

In contrast to the body of literature indicating that both traditional and behavioral treatments for obesity produce weight loss that is reasonably well maintained after one year, it is evident that weight is gradually regained over time, with many individuals eventually weighing more than they did at the start of treatment (Adams, Grady, Lund, Mukaida, & Wolk, 1983; Dubbert & Wilson, 1984; Kirschenbaum, Stalonas, Zastowny, & Tomarken, 1985; Murphy, Bruce, & Williamson, 1985; Rosenthal, Allen, & Winter, 1980). This effect becomes more conspicuous the longer that the follow-up is extended, and it is apparent that most individuals will regain most or all of their weight after four or five years (Bjorvell & Rossner, 1985; Graham, Taylor, Hovell, & Siegel, 1983; Jordan, Canavan, & Steer, 1985; Kramer, Jeffery, Forster, & Snell, 1989; Murphy et al., 1985; Stalonas, Perri, & Kerzner, 1984; Stunkard & Penick, 1979). For example, in a 5-year follow-up, Stalonas et al. reported that the average participant had gained 11.9 lb since the end of a behavioral program, making him or her 1.49 lb heavier than when treatment began. Murphy et al. found that the average *self-reported* weight loss after four years for several different treatment conditions was 7.12 lb, but the average *measured* weight loss was actually about one pound. Jordan et al. (1985) studied selected patients who had achieved at least a 15-lb weight loss during treatment. They found that patients showed distinct patterns of weight fluctuation, with only 12.2% remaining consistently below their post-treatment weights for 5 years. The odds against maintaining long-term weight loss are dramatically illustrated by Kramer et al. in a recent study of 114 men and 38 women who had successfully completed a 15-week behavioral weight loss program. They reported that less than 3% of the patients maintained their post-treatment weight throughout the four yearly follow-up assessments. Monotonic weight regain was the predominant pattern for the men, whereas the women tended to lose and regain weight more often during the follow-up period.

In a study that is interesting because it did not rely on a sample of patients seeking weight loss treatment, Binnie (1977) identified and treated 43 individuals in a rural general practice who were above "ideal" weight.[2] After 10 years, only one patient had achieved ideal weight, and only two had achieved 80% of their goal. These results are similar to other long-term studies indicating that only a small minority of patients are able to achieve and maintain ideal weight goals. Stunkard and Penick (1979) found that after 5 years, the majority of patients receiving both behavioral and traditional treatment regained most of the weight originally lost.

The very few long-term studies reporting somewhat less dismal findings are problematic because of possible bias in the sample of subjects studied at follow-up (Graham et al., 1983; Grinker, Most, Hirsch, Borsdorf, & Wayler, 1985). The most impressive results were those of Grinker et al., who reported that 55% of subjects followed after a residential treatment program had maintained at least a 5-kg weight loss; however, there was only a 38% response rate. Similarly, Graham et al. targeted 62 of 138 initial participants in a weight loss program on the basis of having telephone numbers and addresses available; it is unclear from the report why such a high percentage was lost to follow-up

The effects of treatment do not stop with an initial weight loss or maintenance period. Weight rebound seems to be almost as reliable a consequence of treatment as initial loss. The pattern of weight gain recorded in extended follow-up indicates that short-term results are frankly misleading indicators of long-term outcome. Our tradition of reporting and interpreting data provides a model, used in the promotion of virtually all weight loss programs and products, in

[2]The terms "ideal" or "desirable" weight are typically derived from insurance company actuarial tables to indicate weights at which insurance policy holders can expect greatest longevity. They are widely used by health professionals and the lay public to characterize the goal weight that one should try to achieve for greatest health. They have been criticized on a number of technical grounds such as sampling, variability of methodologies used to gather weight and mortality data, and the fact that they do not take into consideration individual differences in lean body mass, skeletal size, and regional fat distribution (Harrison, 1985; Knapp, 1983). "Frame sizes" reported in the 1983 Metropolitan Life Insurance height–weight tables were not derived from anthropometric measurements but were based on arbitrary groupings of the data (Weigley, 1984). Perhaps the greatest concern regarding "ideal" and "desirable" weights relates to the interpretation of the weight table figures. They fail to take into consideration that body weight, like other physical attributes, could be expected to vary naturally in the population (measures of dispersion are not included in the weight tables used to recommend desirable weights). Diverse genetic propensities within the population may mean that what is a healthy weight for a population may not have any relevance to the individual. Moreover, there is little attempt to actually inform individuals of the *magnitude of risk* associated with particular deviations from specified weights. Results from Sorlie et al. (1980) indicate that for both men and women within the middle three quintiles of the weight distribution, there is no relationship between weight and mortality. For women of average height (5 ft 3 in. to 5 ft 6 in.) the risk of death was virtually identical for those between 115 lb and 194 lb. While women in the highest and lowest 20% of the weight distribution had a higher risk of death, this was considerably lower than the mortality rate for thin and average weight men. Finally, even though certain weights may be related to longevity in the insurance company samples, as will be discussed more fully later, it is mere speculation that changing one's weight category will actually alter longevity.

which it is implied that treatments can be adequately evaluated by their immediate effects. However, if treatment studies are to be relevant, the maintenance of clinically meaningful weight loss must be the agreed-upon standard.

One would expect that existing data would lead to unanimity in the research community about the need for long-term evaluations in order to draw conclusions about treatment efficacy. It is not that failures of dietary treatment are unknown to researchers. Indeed, a number of well respected researchers have based their repeated calls for long-term follow-up upon these observations (Brownell et al., 1986; Foreyt et al., 1981; Stunkard, 1975; Wilson, 1978). Nevertheless, many others have remained undaunted by the failure to demonstrate long-term success and have argued not for the abandonment of dietary treatment but instead have called for more "agressive approaches" to treatment such as the very low calorie diet (VLCD; e.g., Brownell & Jeffery, 1987).

Very Low Calorie Diets

The very low calorie diet of 300–500 calories per day was enthusiastically received because of its ability to produce large and rapid weight loss. In a comprehensive review, Wadden, Stunkard, and Brownell (1983) described the VLCD as "the most important recent development in the medical treatment of obesity" (p. 675). More recently, Brownell and Jeffery (1987) suggested that the VLCD "may be the treatment of choice for persons who are severely obese and who have had difficulty losing sufficient weight on more moderate dietary regimens" (p. 363). Initial trials of VLCDs combined with behavior therapy seemed to support the initial fervor by achieving significant weight loss, reasonably well maintained at one year follow-up (Wadden & Stunkard, 1986; Wadden et al., 1983). However, as with most approaches, the initial optimism has been dampened by a sobering series of long-term follow-up studies indicating that the pattern with the VLCD is the same as with other dietary treatments.

In their initial report, Wadden and Stunkard (1986) found that the VLCD combined with behavior therapy produced significant weight loss (mean = 19.2 kg) and was more effective than either the behavioral or VLCD therapy alone. At the end of one year, the weight loss for the combined treatment was reasonably well maintained (12.9 kg), but after three years virtually all of the treatment effects were reversed, particularly when results were corrected for the effects of intervening weight loss efforts (Wadden, Stunkard, & Liebschutz, 1988). Failure of this approach is even more evident in a follow-up of 497 patients receiving a combination of a VLCD (Optifast) and behavior modification through a large health maintenance organization (Hovell et al., 1988). Hovell and colleagues found that 55% of the patients who started treatment dropped out before completion. While the remaining 45% lost a mean of 83.9% of their "excess weight," the four follow-up groups, illustrated in Figure 2, regained between 59% and 82% of their initial excess weight by 30 months from the start of treatment. Patients began treatment, on average, about 50% over "ideal weight" defined by weight norms.

Although there is an upward trajectory for all groups in Figure 2, it is interesting to note that the group least consistently available for follow-up assessments had the steepest slope. This may have implications for results of studies that have selected subsamples for follow-up based upon availability. These results are consistent with those of another recent study that compared VLCD with

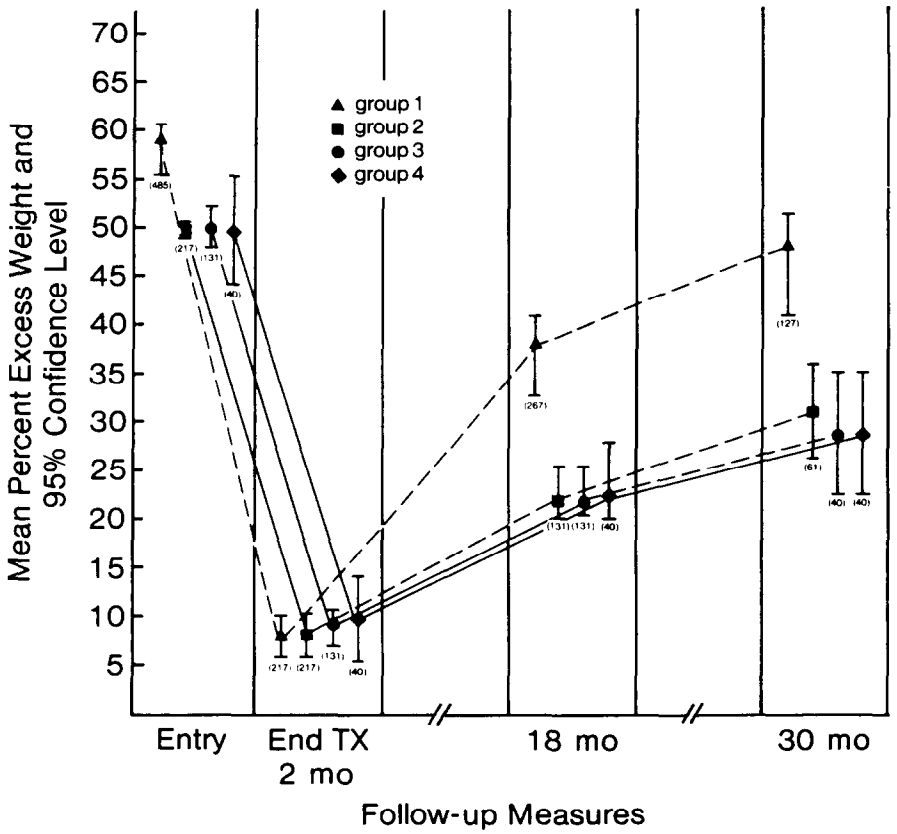

FIGURE 2. Mean Percent of Excess Weight and 95% Confidence Intervals of Patient Groups Followed After Supplemented Fasting Regimen. (Groups were defined by consistent membership across follow-up periods: Group 1 is an average of all patients available at a given follow-up, Group 2 patients were available for two, Group 3 for three, and Group 4 for all follow-up periods. From "Long-Term Weight Loss Maintenance: Assessment of a Behavioral and Supplemented Fasting Regimen" by M. F. Hovell, A. Koch, C. R. Hofstetter, C. Sipan, P. Faucher, A. Dellinger, G. Barok, A. Forsythe, and V. J. Felitti, 1988, *American Journal of Public Health, 78,* pp. 663–666. Copyright 1988 by the American Public Health Association. Reprinted by permission.)

gastroplasty and found that after five years only 3% of the VLCD group could be considered successful compared to 16% of the surgery group (Andersen, Stokholm, Backer, & Quaade, 1988). Moreover, there was no apparent leveling off of the weight regain even after 5 years.

The failure of the VLCD to lead to sustained weight loss should not have been a suprise since 15 years earlier Sohar and Sneh (1973) reported that most of the small proportion of patients who were actually able to complete a low calorie diet returned to within 10% of their pretreatment weight at a 14-year follow-up. Even those who were below their pretreatment weight at follow-up remained obese by most standards.

Perhaps of even greater concern than the negative findings is the unjustified optimism found in the initial reports of some of the more recent VLCD studies. A year prior to publication of the findings in the Optifast VLCD study mentioned earlier, a news release from the manufacturer of the protein supplement attributed the following summary of the results to one of the researchers:

> Weight loss can now be easily, safely and routinely accomplished with good probability of maintaining the loss . . .[F]ive years ago this just wasn't the case. The frontier of dieting has moved from simply accomplishing a weight loss to maintaining the loss long-term. (San Diego VLCD Study, 1989)

Although the rate and magnitude of weight loss have been the basis for recommending the VLCD, its most remarkable feature is the speed of weight regain following treatment. The association between large weight losses during treatment and rapid regain was observed by Stunkard and Penick (1979) in reference to behavioral and traditional treatments. They noted that this pattern was similar to that found following fasting treatments. In recalculating the weight losses reported by Swanson and Dinello (1970), Stunkard and Penick (1979) found that the more weight lost during treatment, the higher the follow-up weight. Accordingly, Swanson and Dinello (1970) reported that 78% of their 18 super obese patients had returned to their original weight within 3 years of ending a fasting treatment.

These findings are consistent with those of Johnson and Drenick (1977), who followed 121 moderately obese patients for an average of 7.3 years after losing an average of 28.2 kg through a supervised fast. After 2–3 years, 50% of the group had reverted to their original admission weight and 90% had done so after nine years. As Figure 3 illustrates, only 7 of the patients (5.7%) remained at reduced weights during the follow-up period. According to the authors, this outcome "required continuous conscious dieting [and] the rate of regain was quite uniform, regardless of baseline weight, degree of weight loss, length of the fast, or the duration of obesity" (p. 1382). In another report of these findings, it was stated that "a sizable number of patients who had maintained a stable obese weight for several years prior to treatment eventually ended up considerably more obese than before weight reduction" (Drenick & Johnson, 1980, p. 33). Although fasting is now regarded as carrying an unacceptable mortality risk (Wadden et al., 1983), it is important to note that the poor maintenance found with this approach is consistent with the more recent results with VLCDs.

Irrespective of the relative merits of the VLCDs and the behavioral or dietary approaches to obesity, there is remarkable consistency in the pattern of weight regain. It would seem that the correct interpretation of long-term findings is quite straightforward. Most approaches lead to weight loss during active treatment, and many individuals continue to lose in the interval directly following treatment; however, most participants ultimately regain to levels that approximate their pretreatment weight. It is only the rate of weight regain, not the fact of weight regain, that appears open to debate. While this may be discouraging to the individual intent on weight loss, it should also provide some solace to the many individuals who have failed at dieting and have attributed the failure to a personal lack of will power.

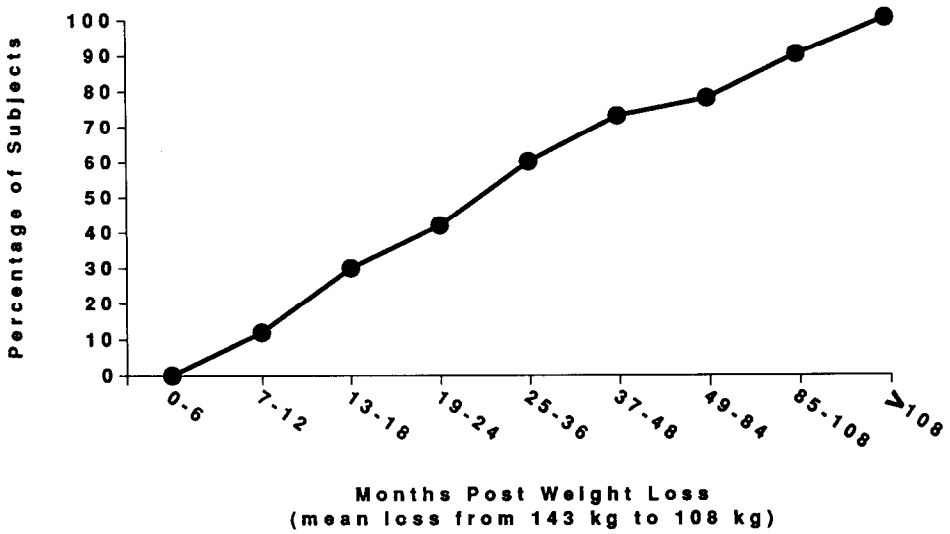

FIGURE 3. Percent of Patients Remaining at Reduced Weights at Various Time Intervals After Achieving Weight Loss. (Solid line represents mean for all patients. From "Therapeutic Fasting in Morbid Obesity: Long-Term Follow-Up" by D. Johnson and E. J. Drenick, 1977, *Archives of Internal Medicine, 137,* pp. 1381–1382. Copyright 1977 by the American Medical Association. Adapted by permission.)

What Really Accounts for the Failure to Maintain Weight Loss?

There is remarkable resemblance between the pattern of weight regain seen in humans following treatment, illustrated in Figures 1–3, and the results from experiments in which laboratory animals are exposed to caloric deprivation, made to lose weight, then returned to unrestricted eating. Figure 4 presents the results from one study in which laboratory rats lost weight in response to caloric restriction (Mitchel & Keesey, 1977). When ad lib eating was restored, the animals regained to body weight levels that were very close to their nondeprived littermates — all of this without the benefit of bathroom scales or (presumably) social pressure. As further illustrated in Figure 4, the same pattern of body weight defense was evident in a group of animals with lesions to the lateral hypothalamus, except that the absolute weight levels were lower.

The concept of "set point" has been proposed to account for these and other data from human and animal studies showing that there is remarkable stability in body weight over time. According to the set point concept, body weight is regulated by physiological mechanisms that oppose the displacement of body weight caused by either over or underfeeding (Nisbett, 1972; Powley & Keesey, 1970). There is some debate about the precise physiological mediators and the factors that influence the absolute levels at which weight regulation occurs, leading some to question the utility of the set point concept (Mrosovsky & Powley, 1977). Nevertheless, there are impressive data indicating that:

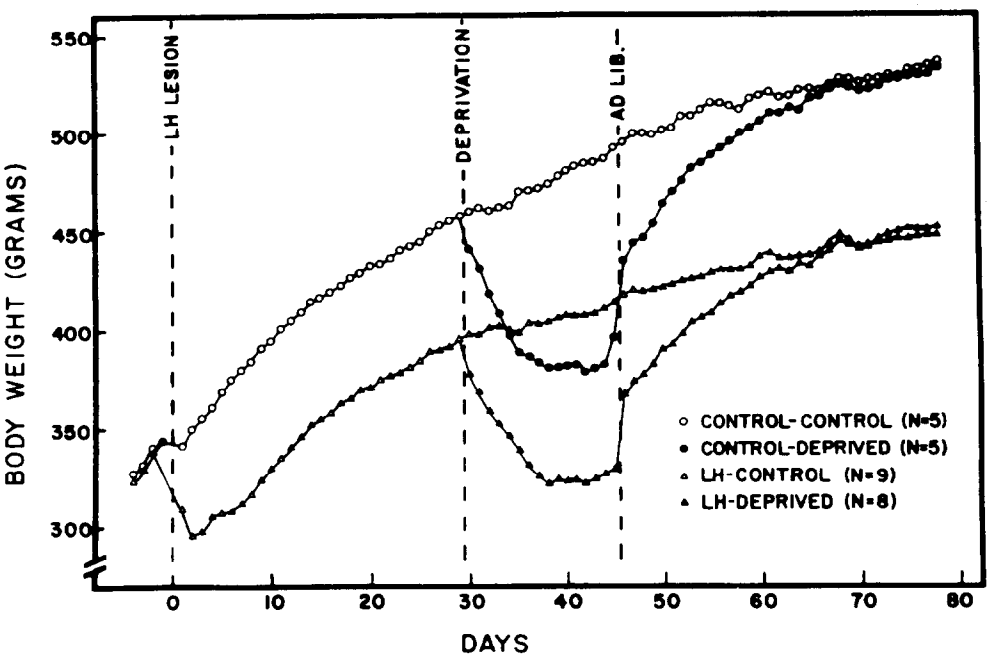

FIGURE 4. Recovery of Body Weight by Control and L-H-Lesioned Rats Following Food Restriction and return to Ad Libitum Feeding Schedule. (From "Defense of a Lowered Weight Maintenance Level by Lateral Hypothalamically Lesioned Rats: Evidence From a Restriction-Refeeding Regimen" by J. S. Mitchel and R. E. Keesey, 1977, *Physiology and Behavior, 18,* p. 1123. Copyright 1977 by Pergamon Press plc. Reprinted by permission.)

1. Displacement of body weight usually results in "homeostatic" metabolic adjustments designed to return the organism to the body weight normally maintained.
2. This body weight "defense" occurs in obese as well as nonobese animals.
3. Genetic factors influence both regulated weight and the propensity toward upward and downward regulation of body weight under certain circumstances in response to environmental challenges.
4. Certain environmental factors, such as diet palatability, exercise, smoking, climate, and certain drugs, appear to influence the absolute levels (within certain limits) at which body weight is regulated (Keesey, 1986), and thus the term "set point" is probably less satisfactory than "regulated weight."

Metabolic Adaptation to Changes in Body Weight

It is so commonly assumed that obesity reflects a failure of body weight regulation that the idea it may represent an adaptive or "normal" state seems almost inconceivable. However, there is consistent evidence from both human and animal studies that weight loss in the obese as well as nonobese leads to reductions of 15–30% in energy requirements (Barrows & Snook, 1987; Boyle, Storlien,

Harper, & Keesey, 1981; Bray, 1969; Donahoe, Lin, Kirschenbaum, & Keesey, 1984; Dulloo & Girardier, 1990; Elliot, Goldberg, Kuehl & Bennett, 1989; Finer, Swan, & Mitchell, 1986; Geissler, Miller, & Shah, 1987; Keesey, 1988: Leibel & Hirsch, 1984; Ravussin, Burnand, Schutz, & Jequier, 1982). Corbett, Stern, and Keesey (1986) reported that reducing the body weight of rats by 14.9% through caloric restriction led to a 24.6% decline in resting metabolic rate. Comparable declines in metabolic rate have been demonstrated in humans exposed to semi-starvation conditions (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). Obese patients have been shown to make the same or greater metabolic adjustments as their lean controls during reduced caloric intake and weight loss (Barrows & Snook, 1987; Elliot et al., 1989; Finer et al., 1986; Leibel & Hirsch, 1984). Leibel and Hirsch reported that obese humans who had lost a significant amount of weight (52 kg) and were still judged to be 60% overweight had caloric requirements 28% below pre-weight-loss levels, which were themselves 25% lower than would be predicted from body size alone. Even short-term restrictions involving small decrements in weight are accompanied by rather dramatic metabolic adjustments. For example, Bray (1969) has reported a 17% decline in resting metabolic rate in obese patients with only a 3% weight reduction! This observation parallels Keesey's (1988) finding that, while the resting metabolism of genetically obese rats was initially 26% higher than that of lean littermates, following only a 4.4% diet-induced weight loss, energy requirements were reduced to that of the lean controls weighing less than half as much. Studies of metabolic changes during weight loss indicate that there is tremendous individual variability in the degree of resistance to displacement resulting from caloric restriction. Absolute weight alone provides little information about caloric needs and the speed of weight loss given a set caloric intake (Miller & Parsonage, 1975).

Importantly, when food intake is normalized after a period of food restriction, there is a tendency for energy to be redeposited preferentially as body fat (Dulloo & Girardier, 1990; Ozelci, Romsos, & Leveille, 1978). In animal studies, Dulloo and Girardier (1990) have shown that this is true even if the fat content of the diet is negligible during regain (i.e., 3%). Their research has also shown that when the extra energy cost of fat synthesis during refeeding is considered it accounts for even further metabolic efficiency.

Wadden, Foster, Letizia, and Mullen (1990) have recently challenged the interpretation that Resting Metabolic Rate (RMR) is adversely affected by severe caloric restriction by showing that the dramatic initial decline in RMR following a VLCD is largely reversed when the diet is stopped. In this study, 18 obese women were assigned to either a Balanced Deficit Diet (BDD), in which 1200 calories were prescribed for the 48 weeks of the study, or a VLCD in which 420 calories were consumed for 16 of the first 17 weeks and a conventional reducing diet was followed for the remainder of 48 weeks of treatment. Resting Metabolic Rate and body weight were assessed nine times over the course of the 48 weeks (see Table 1). Although Table 1 illustrates that the dramatic short-term decline in RMR following the VLCD is partially reversed when participants increased their intake, several findings are particularly noteworthy. First, although the authors concluded that "neither dietary regimen, combined with modest physical activity, was associated with long-term reductions in metabolic rate that exceeded decreases anticipated with the achievement of a lower body weight" (p. 707), the caloric intake for both groups indicates that they were still actively restricting

TABLE 1. Changes in Weight and Resting Metabolic Rate Over 48 Weeks in Patients in the BDD and VLCD Conditions

	Changes in Weight, kg[a]		Changes in Resting Metabolic Rate, kJ/d[a]	
Week	BDD	VLCD	BDD	VLCD
5	−4.1 ± 0.7	−7.5 ± 0.6*	−808 ± 396	−1622 ± 397**
9	−7.0 ± 1.0	−14.2 ± 0.7*	−895 ± 267	−1629 ± 388**
13	−8.7 ± 1.8	−18.7 ± 1.0*	−1114 ± 295	−1677 ± 338***
17	−11.0 ± 2.0	−23.1 ± 1.1*	−1053 ± 356	−1865 ± 552***
19	−12.9 ± 2.1	−25.2 ± 1.6*	−1035 ± 234	−1237 ± 321
21	−12.6 ± 2.5	−26.0 ± 1.6*	−936 ± 264	−1095 ± 397
25	−14.3 ± 3.0	−24.7 ± 2.5*	−786 ± 341	− 948 ± 330
48	−18.2 ± 3.5	−21.6 ± 2.9	−851 ± 358	− 697 ± 200

Note. BDD indicates balanced-deficit diet; VLCD, very-low-calorie diet. Values are mean ± SEM. Through Week 21, $n = 9$ for both conditions; $n = 8$ and $n = 7$ in both conditions at weeks 25 and 48, respectively. From "Long-Term Effects of Dieting on Resting Metabolic Rate in Obese Outpatients" by T. A. Wadden, G. D. Foster, K. A. Letizia, and J. L. Mullen, 1990, *Journal of the American Medical Association, 264,* pp. 707–711. Copyright 1990 by the American Medical Association. Reprinted by permission.

[a]Changes in weight and resting metabolic rate were significantly different from baseline for both conditions at all assessment periods ($p<.001$).

*Difference between conditions was significant at $p<.001$. **Difference between conditions was significant at $p<.01$. ***Difference between conditions was significant at $p<.05$.

their food intake. The VLCD and the BDD subjects reported a mean caloric intake at Week 25 of 1118 calories and 1198 calories, respectively, and at Week 48 of 1392 and 1285 calories, respectively. Although RMR for both conditions was less than 10% below baseline (adjusted for changes in weight), it should be pointed out that levels of caloric intake at the end of treatment were approximately 500 calories a day *below* the average caloric requirements for the average woman of the same age (National Research Council [NRC], 1968). Secondly, by Week 25 it is evident that the weight of the women in the VLCD was beginning to rebound, indicating that metabolic homeostasis had not yet been achieved despite continued caloric restriction. Finally, the obese condition was not cured in these women. The women began treatment at an average of more than 235 pounds (at 5 ft 5 in. tall), and at the end of the 48 weeks, women in both treatment conditions still weighed more than 190 pounds. While it is true that the acute changes in metabolic rate initially observed with highly restrictive dieting were partially reversed, it is important not to underestimate the chronic dietary restriction that is apparently necessary to maintain the losses achieved through dieting which, in turn, may account for the ultimate weight rebound that can be anticipated for these women.

The persistence of the metabolic changes that accompany weight loss are particularly relevant to the small minority of obese individuals who actually do manage to maintain a lowered body weight for long periods of time. Geissler et al. (1987) found that previously obese women who had maintained their target weights for an average of 2.5 years had a metabolic rate about 15% less and ate

significantly less (1298 vs. 1945 calories) than lean controls. Leibel and Hirsch (1984) have reported that the reduced metabolic requirements endure in obese patients who have maintained a reduced body weight for 4–6 years. *Thus, successful weight loss and maintenance is not accomplished by "normalizing eating patterns" as has been implied in many treatment programs but rather by sustained caloric restriction.* This raises questions about the few individuals who are able to sustain their weight loss over years. In some instances, their eating patterns are much more like those of individuals who would earn a diagnosis of anorexia nervosa than like those with truly "normal" eating patterns.

Moreover, reducing body weight in the obese may make them superficially resemble those who are naturally lean, but basic differences in body fat stores persist. Trembley, Despres, and Bouchard (1984) studied a group of men who, through a program of long distance running, had lost an average of almost 40 kg and maintained a stable weight for at least one year. They found that the ex-obese runners were similar to sedentary controls on measures of body fat and fat metabolism, but that both groups differed significantly from elite runners. The tendency to preferentially defend body fat at the expense of lean body mass is illustrated by studies showing that both food restriction and jejunoileal bypass surgery are successful in keeping genetically obese rats at reduced body weight levels; however, *they preserve elevated body fat levels at the expense of muscle mass and vital organs such as brain, heart, kidney, and liver* (Cleary & Vesselli, 1984; Greenwood, Maggio, Koopmans, & Sclafani, 1982).

"Yo-yo" or intermittent dieting may make weight reduction and maintenance even harder over time. Metabolic efficiency may be enhanced by repeated cycles of weight loss followed by weight gain; returning to the initial weight often does not completely reverse this ability to store more energy with less energy intake (Brownell, Greenwood, Stellar, & Shrager, 1986; Reed, Contreras, Maggio, Greenwood, & Rodin, 1988). Brownell, Greenwood, Stellar, & Schrager found that rats became more efficient in their utilization of energy with repeated periods of weight loss and regain. Reed et al. (1988) reported that repeated weight cycling not only produced a four-fold increase in metabolic efficiency but also led to increased dietary fat selection, larger adipose deposits, and greater plasma insulin values in female rats. Thus, there is evidence that dieting, if it is followed by return to initial weight levels, results in a lower metabolic rate making subsequent weight loss more difficult. Moreover, independent of whether typical resting metabolic rate is in fact influenced by repeated bouts of weight change, weight cycling may lead to greater accumulation of body fat (Dulloo & Girardier, 1990; Reed et al., 1988) and increased cardiovascular risk factors (Reed et al., 1988). The long-term effects of the possible hypometabolic adjustment and the generalizability of these findings to humans is not clear at this time.

There are conflicting findings from the animal literature on whether or not weight cycling leads to a higher weight. Several studies have indicated that one or more cycles of weight loss and regain lead to higher weights than are evident in noncycled controls (Brownell et al., 1986; Coscina & Dixon, 1983; Szepesi, 1978; Szepesi & Epstein, 1977; Szepesi, Vegors, Michaelis, & DeMouy, 1975). Other studies indicate that cycling leads to increased metabolic efficiency, not to elevated weights (Cleary, 1986; Reed et al., 1988). The divergence may relate to such factors as the age, species, genetic strain, and sex of the animal as well as the length, frequency, and magnitude of food restriction and the palatability of diet.

There are also conflicting data on cycling and body weight for humans, but there is some evidence for increased metabolic efficiency with weight fluctuations. Steen, Oppliger, and Brownell (1988) reported that wrestlers who experienced repeated cycles of weight loss and regain had a 14% lower resting metabolic rate than those who did not display this pattern of weight change. This is in contrast to findings by Melby, Schmidt, and Corrigan (1990), who compared the resting metabolic rate of 12 weight-cycling wrestlers and 13 weight-stable nonwrestler controls. They found that when the wrestlers lost weight for competition, their RMR was significantly lower compared to preseason levels, but it was not lower than that of the weight-stable controls.

The weight history of women defined as "restrained eaters" (i.e., chronic dieters) indicates that they have been on weight reducing diets where they have lost more than 4 kg of body weight more often than "unrestrained eaters" (Laessle, Tuschl, Kotthaus, & Pirke, 1980; Tuschl, Platte, Laessle, Stichler, Pirke, 1990). Although restrained eaters generally weigh more and have higher maximum weight since childhood, they report eating less (Wardle & Beales, 1987) and expending about 620 kcal per day less than those with the unrestrained eating style (cf. Tuschl, 1990). Tuschl concludes that the restrained eating style probably leads to regulation of body weight below that which would be maintained without dietary restrictions, and this leads to compensatory changes in energy expenditure which are decreased even further by frequent weight fluctuations.

A recent study has shown a significantly slower velocity of weight loss the second time obese patients participated in a VLCD, suggesting greater metabolic efficiency after their first cycle of weight loss (Blackburn et al., 1989). Keys et al. (1950) found that during refeeding, semi-starved men initially overshot their control weight by 10%, eventually returning to baseline. There is some evidence that children show this same pattern of weight overcompensation following certain forms of malnutrition (Ashworth, 1969). There are many clinical reports of obese patients who, following treatment, gain to a level well above their previous maximum weight (cf. Stalonas et al., 1984). Commenting on their follow-up study, Drenick and Johnson (1980) indicate that "a sizeable number of patients who had maintained a stable weight for several years prior to treatment eventually ended up considerably more obese than before weight reduction" (p. 33). It is a moot point whether or not these individuals would have gained to the same degree or more in the absence of treatment, but clinically, patients often indicate that the dramatic overeating associated with posttreatment weight gain persisted well beyond their previous weight plateau. Thus, one of the most surprising consequences of treatment is that it could lead to *increased* obesity in some individuals.

Just as the body resists weight loss by making metabolic adjustments, it also resists gain. In a classic experiment, prisoners volunteered to gain between 20% and 25% of their original body weight by eating about twice their usual caloric intake for about 6 months (Sims et al., 1968). Most of the men gained the initial few pounds with ease but quickly became hypermetabolic and resisted further weight gain despite continued overfeeding (Sims, 1976). One prisoner stopped gaining weight even though he was consuming close to 10,000 calories per day. With return to normal amounts of food, most of the men returned to the weight levels that they had maintained prior to the experiment. Again, there were individual differences in the response to overfeeding since a few of the men with a

family history of obesity did not immediately return to normal weight levels with the reduction in food intake. Other studies of experimental overfeeding indicate that there is a variable increase in resting metabolic rate (Levin, Triscari, & Sullivan, 1986; Welle, Seaton, & Campbell, 1986) and that these metabolic adjustments result in much less weight gain than would be expected with a given caloric intake (Ashworth, Creedy, Hunt, Mahon, & Newland, 1962; Miller & Mumford, 1966; Miller, Mumford, & Stock, 1967; Neumann, 1902; Welle et al., 1986). Of interest, the metabolic resistance to weight gain is reduced in genetically obese animals (Himms-Hagen, 1981; James & Trayhurn, 1981; Romsos, 1983) and in obese humans (Kaplan & Leveille, 1974; Pittet, Chappuis, Acheson, De Techtermann, & Jequier, 1976).

The evolutionary significance of the metabolic adaptations to weight loss and the preference to replenish and enlarge fat stores when food becomes available is not difficult to understand. Throughout history one of the most profound threats to organism reproductive capability and survival has been food shortage (Brown & Konner, 1987). Animal studies have shown that duration of survival is directly related to body fat reserves and metabolic efficiency (Rixon & Stevenson, 1957). Thus, organisms with the abilities to increase efficiency of caloric utilization when food is in short supply, and to selectively increase fat stores when food becomes available to cope with future food shortages, preferentially contribute to the gene pool. This may seem implausible at first within the context of our current social preferences for thinness. However, whereas fatness is inversely related to socioeconomic status in developed countries, it has a strong positive association in developing countries where food supplies are, and have long been, scarce (Sobal & Stunkard, 1989). Probably because of its relationship to survival, our ancestors and the peoples of developing societies have viewed fatness as a symbol of social prominence and sexual desirability.

Genetic Contributions to Obesity

Although the mechanisms are probably varied and complex, there is increasing evidence that genetic factors play an important role in the predisposition to obesity (Borjeson, 1976; Bouchard, 1986; Bouchard et al., 1990; Garn, 1986; Poehlman et al., 1986; Price, Cadoret, Stunkard, & Troughton, 1987; Ravussin et al., 1988; Roberts, Savage, Coward, Chew, & Lucas, 1988; Stunkard, Foch, & Hrubec, 1986; Stunkard, Harris, Pedersen, & McClearn, 1990; Stunkard, et al., 1986). In a study of same-sex, monozygotic and dizygotic twin pairs, Borjeson (1976) estimated heritability of obesity to be 88%. Concordance rates for different degrees of obesity are twice as high for monozygotic as for dizygotic twins (Stunkard, Foch, & Hrubec, 1986). In a large scale study, a strong positive relationship was found between the Body Mass Index (BMI) of adoptees and their biological parents, whereas no relationship was found in the BMI of adoptees and adoptive parents (Price et al., 1987; Stunkard, Sorenson, Hanis, Teasdale, Chakraborty, Schull, & Schulsinger, 1986). In a reinterpretation of these data, Costanzo and Schiffman (1989) have suggested that it may be thinness rather than obesity that is inherited. Stunkard et al. (1990) assessed the relative importance of genetic and environmental effects on BMI in another recent study of identical and fraternal twin pairs reared together or reared apart. The intrapair correlations for BMI for twins reared apart (r = .70 for males, r = .66 for fe-

males) were only slightly lower than for twins reared together. Morever, sharing the same childhood environment did not influence BMI in later life (i.e., only potential environmental influences unique to the individual rather than those shared by family members were important contributors to the remaining variance). The authors concluded that genetic influences on BMI are substantial and childhood environment has "little or no influence." Nevertheless, environment does exert an influence on body weight, as indicated by another recent study of 250 white male identical twin pairs studied both at military induction and again between 20 and 34 years later (Newman et al., 1990). At follow-up, there was a mean difference of over 12 pounds (5.5 kg) between the heavier and lighter twin.

There is growing recognition that the mechanism leading to the expression of obesity in vulnerable individuals relates to metabolic factors. Research has shown that metabolic rate varies among individuals and that it tends to have a pattern of familial association. Using heart rate to estimate metabolic rate, Griffiths and Payne (1976) concluded that the metabolic rate of the 4-year-old children of obese parents was 10% lower than that of 4-year-old children of nonobese parents. Roberts and colleagues (1988) studied the rate of energy production in infants of lean and obese mothers. They found that at three months, the babies of lean and obese mothers were indistinguishable in terms of BMI and skin-fold thickness; however, the energy expenditure was more than 20% lower in the infants who later became overweight. Similar observations were made by Ravussin et al. (1988) in a very different experimental situation. Southwestern American Indians with low 24-hour energy expenditure at an initial visit were four times more likely to gain in excess of 7.5 kg over the next two years. This study also indicated that there was a significant correlation in the metabolic rates of siblings, suggesting that low energy expenditure leads to the aggregation of obesity in families. These findings account for the later development of obesity in the children of obese parents in the absence of differences in caloric intake among the obese and the lean. In another recent study, Bouchard et al. (1990) overfed 12 pairs of identical twins by 1000 kcal per day, 6 days per week for over three months, and then measured changes in body weight, body composition, and topography of fat distribution. Although there was considerable variability in the amount of weight gained, there was much more similarity within pairs than between pairs in body weight gain, percentage of fat, fat mass, and estimated subcutaneous fat. There was about six times as much variance between as within pairs for regional distribution of fat. These results strongly support the hypothesis that genetic factors play an important role in determining individual differences in the tendency toward obesity and the distribution of body fat.

Dietary Factors and Obesity

The metabolic findings just described are congruent with the tremendous body of research employing a great variety of methodologies that has failed to yield any meaningful or replicable differences in the caloric intake or eating patterns of the obese compared to the nonobese (Rolland-Cachera & Bellisle, 1986; Striegel-Moore & Rodin, 1986; Wooley et al., 1979b). Although it strains credibility to imagine such a concordance of incorrect results, these findings must be reconciled with other findings that tend to keep alive the question of whether the obese eat more than the nonobese and therefore can lose weight by eating "nor-

mally." The most important of these is the consistent observation of an inverse relationship between weight and social class, among women but not among men in developed societies (cf. Sobal & Stunkard, 1989). Sobal and Stunkard (1989) attribute these findings to the mediating influence of certain social attitudes toward obesity and thinness in developed societies that selectively influence members of different social classes through the mechanisms of dietary restraint, physical activity, social mobility, and inheritance. Accordingly, upper class women may be more committed to the view that slimness is desirable and have greater resourses to suppress their weight through dietary restraint. They may also engage in more recreational activities because of greater leisure time, access to resources, and social pressures to exercise. These two factors may allow upper class women to exert some control over their weight, but as Sobal and Stunkard (1989) point out, the extreme orientation toward weight control may have alarming social and personal consequences, as reflected in the high rates of eating disorders and shape dissatisfaction in this socioeconomic group. The downward mobility of obese women, the upward mobility of thin women, and the interaction between these patterns and inheritance may account for the genetic transmission of weight propensities along socioeconomic status lines (Sobal & Stunkard, 1989). Other factors that could contribute to the higher levels of obesity in women of lower socioeconomic class include greater number and earlier onset of pregnancies, higher levels of chronic stress, and ethnic differences in female adiposity.

The interaction of genetic and environmental factors in the modulation of body weight is supported by two recent studies of schoolchildren (Rolland-Cachera & Bellisle, 1986; Rolland-Cachera et al., 1988). Rolland-Cachera and Bellisle found that food intake was about 500 calories greater and obesity about four times more common in the lowest versus the highest socioeconomic groups studied; however, within each socioeconomic group, there were comparable levels of caloric intake among lean, average weight, and obese children. These apparently contradictory findings are explained by suggesting that a given caloric intake leads to the expression of obesity in those who are metabolically predisposed and that the higher calorie diet found in lower socioeconomic classes simply challenges the adaptive threshold for obesity in larger numbers of individuals.

The argument that overeating is the major cause of obesity has also been fortified by the observation that diet palatability can lead to obesity (Peck, 1978; Schemmel, Mickelsen, & Gill, 1970; Sclafani, 1980; Winn & Herberg, 1985). The potentiation of obesity by a palatable diet is particularly marked for certain genetic strains of rats and humans whose natural habitat has historically provided rather bland or meager foodstuffs (Coleman, 1979). When exposed to a palatable diet, animals not only ingest more calories but also convert a larger proportion of ingested food to body fat (Sclafani, 1980).

Some experiments with laboratory animals have shown that body weight increases in response to a palatable diet and reverts to normal levels once the animals are returned to chow (Gale, Van Itallie, & Faust, 1981; Peck, 1978; Rothwell & Stock, 1979; Sclafani & Gorman, 1977). Other studies indicate that the ingestion of palatable foods, particularly over a prolonged period, may lead to elevated body weight that resists subsequent downregulation even when the animal is returned to a normal diet or exposed to dietary restriction (Corbett et al., 1986; Levin et al., 1986; Rolls, Rowe, & Turner, 1980; Winn & Herberg, 1985). These animals make the initial metabolic adjustments designed to defend

the lower weight, but the process attenuates with prolonged exposure to the palatable diet. The animals appear to have attained an elevated set-point, in that they regulate or defend their new higher weight level against further gain or loss (Keesey, 1986).

This gradual recalibration or "sliding" in the homeostatic mechanism for body weight maintenance is similar to the lowering of body temperature during hibernation (Mrosovsky & Barnes, 1974). Both changes provide a means of maintaining internal equilibrium in the face of environmental challenge. With regard to body weight, the precise mechanisms responsible for this transformation are unclear. They may relate to the proliferation of adipose cells and other physiological changes that are irreversible (Bjorntorp, 1986; Faust, 1981; Keesey, 1986; Mandenoff, Lenoir, & Apfelbaum, 1982). On the other hand, they may simply reflect the influence of another motivated behavior system of weight regulation. The idea that a distasteful diet would cause animals to maintain a lower than normal body weight seems understandable without positing a changed physiology or rejecting altogether the concept of weight regulation.

Although genetic factors play a role in the expression of body weight, there are conflicting findings regarding stability in individual body weight over time. Noppa and Hallstom (1981) found adult body weight to be relatively stable, reporting a mean weight gain over six years for 1,302 Swedish women of 1.4 kg (SD = 5.1 kg). Only 28 women (2.2%) had lost more than 10 kg, and 59 women (4.5%) had gained more than 10 kgs. Similarly, Borjeson (1962) found that more than 80% of obese adolescents remain obese as adults, and this tendency is most evident for those who are at the upper end of the weight continuum. On the other hand, other studies have shown that there is considerable redistribution of relative weights in children assessed over time (Bradden, Rodgers, Wadsworth, & Davies, 1986; Zack, Wiens, & Kennedy, 1984).

It may be concluded that nature and nurture both exert influences on body weight and that the eventual expression of obesity is a complicated matter. To the degree that social and economic factors determine the palatability or type of the diet, they might act in a permissive manner by challenging the adaptive capacity of particular gene pools. Van Itallie (1986) has emphasized that "cafeteria diets" in laboratory animals and certain changes in the Western diet in humans leads to general increases in the prevalence of obesity. Obesity attained through either dietary or genetic routes may in turn lead to certain metabolic adaptations that cause weight to be maintained at elevated levels.

Regardless of these factors, the myth of overeating by the obese is sustained for the casual observer by selective attention. Each time that a fat person is observed to have a "healthy appetite" or an affinity for sweets or other high calorie foods, a stereotypic leap into causality is made. The same behaviors in a thin person attract little or no attention. Even in instances in which food intake is large, it may not represent "overeating," since it may relate to large metabolic demands. Thus, the major premise of dietary treatments of obesity, that the obese overeat with respect to population norms, must be regarded as unproven, casting doubt on the appropriateness of dietary interventions from yet another vantage point.

HEALTH RISKS ASSOCIATED WITH OBESITY

Perhaps the most ardent justification for treatment of obesity stems from the contention that even moderate obesity poses serious health risks. However, re-

views of the health risks associated with obesity have profoundly different interpretations or points of emphasis with respect to the same epidemiological data. Examination of the actual mortality data from many epidemiological studies makes it clear that, across studies, there is often no reliable pattern of association between premature death and relative weight (cf. Ernsberger & Haskew, 1987). It is not our intention to review in detail the immense literature on mortality, morbidity, and obesity, but rather to point to data that raises questions about the necessity and even the desirability of weight loss, especially in view of the likelihood of regain. Several topics are particularly relevant, including the importance of regional distribution of body fat, the conflicting findings related to mortality risk and morbidity, the effects of weight loss on mortality, and the health risks associated with weight cycling.

Regional Distribution of Body Fat

Recent research has indicated that body build and regional distribution of body fat are more important than overall obesity in predicting mortality. It is much less healthy to have a large concentration of fat around the abdomen (apple shape) than to have body fat more evenly distributed or centered below the waist (pear shape) (Donahue, Abbott, Bloom, Reed, & Yano, 1987; Ducimetiere, Richard, & Cambien, 1986; Hartz et al., 1990; Lapidus, Andersson, Bengtsson, & Bosaeus, 1986; Larsson et al., 1984; Larsson et al., 1989). These and other studies show that mortality rates and many cardiovascular changes previously attributed to obesity are more closely related to regional distribution of body fat, body build, or lean body mass than to adiposity or relative body weight (cf. Ernsberger & Haskew, 1987; NRC, 1989). Some have observed that subjects with high waist to hip circumference ratio (WHR) but low BMI have the highest risk for cardiovascular disease (Larsson et al., 1984; Lapidus et al., 1984). A recent study has shown that WHR, independent of BMI was positively associated with blood pressure, cholesterol, triglycerides, fibrinogen, smoking, sick leave, frequent use of health facilities, and other measures of illness (Larsson et al., 1989). In contrast, BMI, independent of WHR, was not related to physical health variables measured, and thus, generalized obesity was associated with good health. Moreover, the Swedish study by Larsson et al. (1984) indicated that, *within each of the body build groups,* obese men had a *lower* death rate than those from the medium and low body mass tertiles (cf. Ernsberger & Haskew, 1987). Even with the added predictive precision derived from examining body build, Seidell et al. (1989) have cautioned that fat distribution and body mass index "do not explain more than approximately 9–13% of the variance in cardiovascular risk factors." These important data are often ignored in making recommendations about the health risks of obesity. It is important to point out that the "apple" pattern is relatively uncommon in women who are nevertheless urged to reduce as much or more than men.

Conflicting Views on Mortality Risks and Morbidity

There are conflicting opinions on the mortality and morbidity risks associated with obesity; the conclusion that obesity is dangerous represents a selective review of the data. In 1985, the NIH convened a consensus panel of distinguished obesity researchers to consider the data on obesity and health and to prepare a published statement on

the "Health Implications of Obesity" (National Institutes of Health Consensus Development and Conference Statement, 1985; Simopoulos & Van Itallie, 1984). The conclusions from the conference received wide publicity and echoed the prevailing view that even mild obesity is associated with elevated health risks. Weight reduction was recommended for everyone exceeding population norms. Manson, Stampfer, Hennekens, and Willett (1987) reviewed the epidemiological literature on mortality risk and obesity and arrived at a similar conclusion, stating that minimum mortality occurs at relative weights at least 10% below the U.S. average. The recent NRC (1989) review of obesity and eating disorders has a much more moderate tone but also emphasizes the health risks of obesity.

Since these reports appeared, an additional study has found increased mortality with obesity. In a prospective study of nurses, Manson et al. (1990) reported that obesity and weight gain were associated with an increased incidence in fatal and nonfatal coronary heart disease. Although the authors concluded that 40% of all coronary events were attributable to adiposity and therefore potentially preventable, it should be noted that too few women in the survey lost weight over the follow-up period to assess the influence of weight reduction on coronary risk. This is in contrast to Wilcosky, Hyde, Anderson, Bangdiwala, and Duncan (1990), who reported that BMI was positively related to all-causes mortality among men but not women, after an average 8.4-year follow-up. However, neither coronary heart disease nor cancer, the two leading causes of death, was significantly associated with BMI. Moreover, even the all-causes mortality data for men in the Wilcosky et al. study is questioned by a reviewer (Rimm, 1990) who recommended not publishing the study since the authors were unwilling to reveal actual mortality rate data that may have contradicted key conclusions.

In all of the summary reports described above, the studies chosen for emphasis are those that support the conclusion that obesity is an independent risk factor. However, there is much conflicting evidence on this point. Challenging the view that there is a clear association between body weight and mortality, a number of epidemiological studies and reviews have concluded either that obesity does not confer elevated health risks or that such risks have been greatly exaggerated (Andres, 1980; Barrett-Connor, 1985; Ernsberger & Haskew, 1987; Fitzgerald, 1981; Jarrett, 1986; Keys, 1980; Keys et al., 1984; Larsson, Bjorntorp, & Tibblin, 1981; Mann, 1974a). In an examination of 16 prospective studies of body weight and mortality, Keys and colleagues (Keys, 1980; Keys et al., 1984) found that obesity was not a major risk factor for death. It should be noted that the conclusions prevailed, even when controlling for the effects of smoking which may reduce the average weight while increasing mortality. In a detailed review of epidemiological studies, Ernsberger and Haskew mount a persuasive argument that the preponderance of evidence fails to support the contention that obesity is associated with an elevated mortality risk.

Many studies indicate that the *group most at risk for early death is the underweight rather than average weight or moderately obese groups* (Blair et al., 1989; Jarrett, 1986; Keys, 1980; Keys et al., 1984; McGee & Gordon, 1976; Paffenbarger, Hyde, Wing, & Hsieh, 1986; Simopoulos & Van Itallie, 1984; Sorlie, Gordon, & Kannel, 1980; Wannamethee & Shaper, 1989; Wilcosky et al., 1990). Although the elevated mortality in thinner groups has been attributed to the overrepresentation of "hidden illnesses" or smoking in the lower weight groups, this interpretation fails to account for the findings in a significant number of studies (cf.

Ernsberger, 1987; Ernsberger & Haskew, 1987; NRC, 1989). Andres (1990) has reported that restricting analyses to nonsmokers does not eliminate the excess mortality among those who are underweight nor does their inclusion shift the weight at which lowest mortality occurs downward. Studies that have eliminated deaths for the first five years of follow-up (i.e., individuals who may be losing weight due to cancer) still find the strong association between mortality and thinness (Waaler, 1983; Wilcosky et al., 1990). That there have been no major health campaigns designed to encourage those below the national weight norms to embark on a program of weight gain in order to improve their health stands in stark contrast to the urgent pleas for weight loss directed to those above the norms.

Like the literature on mortality, the research on morbidity is beset with complicated and often conflicting findings. There are undoubtedly certain diseases that are more common in the obese; however, most reviews emphasize increases in diabetes, in cardiovascular risk factors, or in cancer without placing these within the context of what is known about the overall health risks and benefits of obesity (cf. Bradley, 1982; Ernsberger & Haskew, 1987; Fitzgerald, 1981; Wooley et al., 1979b). For example, while it is widely accepted that obesity is an independent risk factor for cardiovascular disease, it is important to note that Barrett-Connor (1985) concluded from a review of almost 100 studies that adiposity is not a risk factor for atherosclerosis or coronary heart disease. Moreover, it is rarely emphasized that cardiovascular health risks, when they do exist, may not necessarily translate into higher mortality rates (cf. Blair et al., 1989; Bloom, Yano, Curb, Reed, & MacLean, 1987; Ernsberger & Haskew, 1987).

Research on the relationship between obesity and cancer provides another good example of the problem of interpretation. Certain types of cancer appear to be more common with obesity (e.g., obese women have higher rates of gallbladder, biliary duct, endometrial, postmenopausal breast and cervical cancer; obese men have higher rates of colon and prostate cancer). But obesity appears to protect against overall cancer death and against death from specific cancer types that are the leading causes of cancer death (e.g., in women, premenopausal breast, lung, stomach, and colon, and in men, lung and stomach; cf. Ernsberger & Haskew, 1987; NRC, 1989).

In addition, there is evidence that obesity is associated with a lowered incidence of respiratory disease, infectious disease, osteoporosis, and several other ailments. Obesity also confers a more favorable prognosis in certain other diseases (Ernsberger & Haskew, 1987; Fitzgerald, 1981; Wooley et al., 1979b). The findings on mortality, morbidity, and obesity have led Andres (1980) to conclude that:

> the major population studies of obesity and mortality fail to show that overall obesity leads to greater risk. It is suggested that not only does advice on the subject of obesity need reappraisal but research on possible associated benefits of moderate obesity would be worthwhile. (p. 382)

Weight Loss and Mortality

There are studies indicating that cardiovascular risk factors decline with weight loss (e.g., Bloom et al., 1987) and that they increase with weight gain (Ashley & Kannel, 1974; Borkan, Sparrow, Wisniewski, & Vokonas, 1986). *However, there are few studies in the medical literature that indicate that mortality risk is actually reduced*

by weight loss, and there are some that suggest that weight loss increases the risk of death. In an American Cancer Society prospective survey of over 1 million people, individuals indicating that they had lost weight in the past 5 years *were more likely* to die from cardiovascular disease than those whose weight was stable (Hammond & Garfinkel, 1969). In a 10-year follow-up of men who were asked their weight at age 25, Rhoads and Kagan (1983) reported that heavy respondents who had later lost weight had almost twice as high a death rate as those who maintained a high but stable weight. Moreover, those with a high but stable weight had the same or lower death rate as thinner men. A study by Avons, Ducimetiere, and Rakoto (1983) indicated that weight loss in a sample of civil servants in Paris was associated with heightened mortality from all causes. These findings are consistent with recent findings that weight loss during adulthood is associated with increased mortality among men participating in the Lipid Research Clinics Program (Wilcosky et al., 1990). Although weight change was unrelated to mortality for women in the Wilcosky et al., study, the odds ratio extrapolated from the regression coefficients for men indicated that each 10% loss of weight was associated with a 14% increase in all-causes mortality and a 27% increase in cancer mortality.

Finally, in a study of mortality risks among 16,936 Harvard alumni, Paffenbarger et al. (1986) not only found that the highest mortality occurred in those with the *lowest body mass index* (below 32), but also that those who had gained weight since college had a *significantly lower* mortality risk compared to those who had minimal weight gain since college. According to the authors, "alumni with the lowest net gain since college had a 29% higher risk of death than their classmates that had gained the most" (p. 607). Thus, even if one accepted the premise that obesity is a dangerous condition and weight reduction a realistic goal, it is an unproven hypothesis that weight reduction actually translates into increased longevity.

Health Risks Associated With Weight Cycling

Health risks associated with obesity are not separated from those which may be independently attributed to weight cycling or oscillations in weight due to dieting and weight regain (i.e., treatment). As discussed earlier, those who lose weight almost always regain it, and the health risks associated with weight loss and gain are rarely considered in studies of mortality associated with obesity. And yet, the yo-yo syndrome has a number of dangerous effects. As mentioned earlier, weight cycling may lead to increased metabolic efficiency, an adaptation response that further thwarts attempts to achieve and maintain a lower weight. Weight cycling can not only cause health problems that were temporarily ameliorated by weight loss to reemerge but can also create new ones. For example, Ernsberger and colleagues have provided compelling theoretical arguments (Ernsberger & Haskew, 1987) and complementing data (Ernsberger & Nelson, 1988; Nelson & Ernsberger, 1984) showing that repeatedly depriving laboratory animals until they lose weight and then allowing them to regain it leads to hypertension and other cardiovascular anomalies similar to those observed in obese humans. Thus, not only is weight loss of limited benefit in the treatment of hypertension, often delaying the implementation of more effective intervention (Haynes, 1986), but also repeated failures at weight loss may actually aggravate hypertension.

Although actual dieting was not directly examined, a recent study by Hamm, Shekelle, and Stamler (1989) has shown that men who have had at least one cy-

cle of rather large weight loss and gain are at significantly greater risk for death from cardiovascular disease than men who progressively gain weight or than men who have a stable weight over a 25-year period, the condition associated with the lowest mortality. The risk of death due to cardiovascular disease was twice as high for the gain and loss group compared to the weight gain only group, even after adjusting for age, serum cholesterol, blood pressure, cigarette smoking, alcohol intake, and body mass index. The authors suggest, following Keys (1979), that this effect may stem from acceleration of atherogenesis caused by the high levels of serum cholesterol during weight regain.

Two additional data sets have confirmed that weight fluctuations are associated with increases in cardiovascular disease deaths. In an analysis of the 32-year follow-up data from the Framingham population, Lissner et al. (1991) reported that variability in body weight was associated with increased coronary heart disease and total mortality in men and women but was not related to cancer incidence. In a multiple risk factor intervention trial with men (Lissner, Collins, Blair, & Brownell, 1989), a positive association was found between cardiovascular disease death and weight fluctuation for smokers who received special lifestyle intervention (as well as those who reported quitting smoking prior to their last assessment). No such association was found for nonsmokers or for the control group of smokers.

Another case in point is a study by Drenick, Gurunanjappa, Seltzer, and Johnson (1980) entitled "Excessive Mortality and Causes of Death in Morbidly Obese Men." This paper is often cited as evidence of serious health risks associated with obesity (Bray, 1986; Brownell, 1982; Stunkard, 1984; Stunkard, Stinnett, & Smoller, 1986; Wadden & Stunkard, 1989). Indeed, the results from this follow-up of 200 obese men are dramatic, with 50 of the men dying during the course of the study. There was a 12-fold increase in the death rate in men 25–34 years of age, and 54% of the deaths were attributed to cardiovascular disease. Although the authors state that "it appears that no unusual factors other than obesity could have caused such extraordinary mortality" (p. 445), they note that their subject pool was drawn from those who had lost and then regained considerable weight through participating in the "therapeutic fast" referred to earlier (Johnson & Drenick, 1977) and illustrated in Figure 3. Whereas it is apparent from studies reviewed earlier that relatively stable obesity has modest or no mortality risk, results from the Drenick et al. (1980) study suggest that "treatment" followed by the weight rebound was associated with a profound vulnerability to premature mortality, particularly from cardiovascular disease. Similarly, in the 5-year follow-up of 27 patients treated for obesity, Stunkard and Penick (1979) reported that "four patients, all of whom had lost large amounts of weight at the first follow-up, died before the second follow-up" (p. 802).

Discussion of the results from the Drenick et al. (1980) study is consistent with the generally shortsighted appraisal of the risk factors associated with weight loss in obesity. Improvements in risk factors accompanying initial weight loss have been well documented and include reductions in cholesterol, free fatty acid, triglycerides, and blood pressure, as well as improvements in glucose tolerance and insulin secretion (cf. Bray, 1986; Brownell & Stunkard, 1981; NRC, 1989; Wood et al., 1988). However, it is rarely pointed out that all of these indices quickly rebound and often surpass the previously unhealthy levels during the inevitable weight regain (cf. Ernsberger & Haskew, 1987). Thus, the almost certain consequence of treatment (weight regain) results in a greater threat to health than

would occur if the obese patient were simply assisted in maintaining a higher but stable weight. This prompted Stunkard and Penick (1979) to conclude over a decade ago that "the medical consequences (of weight regain) may be unfortunate enough that if people cannot maintain weight loss, they would be better off not trying to lose weight!" (p. 806).

In summary, the area of health risk and obesity is distinguished by controversy and conflicting findings making future research in this area of vital importance. Obesity may predispose to certain health risks and protect against others. Evidence that it is more dangerous to be thin than fat is either ignored or minimized in analyses that shape public policy toward weight loss. It is often suggested that risks may be permanently modified by weight loss without attention to the probable health risks associated with weight loss itself or with repeated diet failures. What evidence exists for an association between obesity and mortality or morbidity is usually found not to apply to those with mild to moderate obesity. Nevertheless, mildly to moderately obese women are the major consumers of weight loss products and services. Zealous warnings that obesity is a major threat to human health are of grave concern because of their tremendous impact on professionals and the lay public already poised to view obesity as malignant. Even if health risks were clearly linked to obesity, it is important to caution against the view that this would further justify the types of dietary treatments currently available. In the absence of long-term maintenance of weight loss, we may simply be engaging in what Hirsch (1978) has characterized as the "modern day equivalent of beating the insane to keep them quiet" (p. 2).

The discussion of physical and psychological risks associated with obesity treatment has not included some of the well documented negative consequences of such radical interventions as bypass surgery, gastric stapling, jaw wiring, and the gastric balloon. In addition are the risks associated with the staggering number of fad diets, diet supplements, over-the-counter "appetite suppressants," laxatives, "cellulite solutions," dietetic food replacements, and countless other diet products that are relentlessly thrust upon the diet-hungry consumer (cf. Bennett & Gurin, 1982; Ernsberger & Haskew, 1987). Dangerous or obviously ineffective fad diets have not been reviewed because the objections voiced throughout this paper are more fundamental, in that they also apply to supposedly "sensible" or conservative weight loss methods.

PSYCHOLOGICAL RISKS ASSOCIATED WITH OBESITY TREATMENT

Psychological Symptoms Associated With Dieting and Weight Loss

In many studies it is difficult to distinguish between psychological symptoms that motivate dieting and those which result from dieting. Perhaps the best data on psychological effects of uncomplicated dieting and weight loss is the classic semistarvation study conducted by Keys and associates (1950). The experiment involved restricting the caloric intake of 36 young men who volunteered to participate as an alternative to military service. Although referred to as a study of "semistarvation," it is noteworthy that during the 6 months in which the men lost approximately 25% of their former body weight, they were restricted to about half of their former caloric intake, precisely the caloric deficit that defines "conservative" treatments for obesity (Stunkard, 1987).

Although the participants were psychologically healthy prior to the experi-

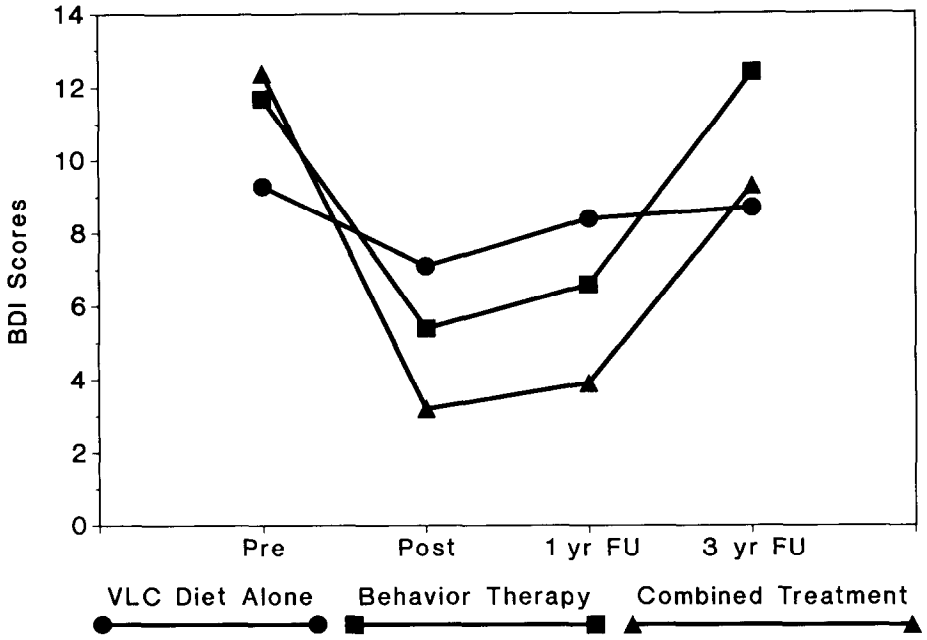

FIGURE 5. Mean Beck Depression Inventory Scores for Subjects Participating in Very Low Calorie Diet Alone (N = 15), Behavior Therapy (N = 16), and Combined Treatment (N = 19) for Treatment and Follow-Up Periods. (From "Three-Year Follow-Up of the Treatment of Obesity by Very Low Calorie Diet, Behavior Therapy, and Their Combination" by T. A. Wadden, A. J. Stunkard, and J. Liebschutz, 1988, *Journal of Consulting and Clinical Psychology, 56,* pp. 925–928. Copyright 1988 by the American Psychological Association. Adapted by permission.)

ment, most experienced dramatic attitudinal, behavioral, and emotional changes during weight loss (see Garner et al., 1985, for a summary). Most experienced periods of depression, anxiety, irritability, anger, and mood swings, transitory for some but protracted for others. Almost 20% experienced extreme emotional deterioration, and some had to be hospitalized as a result of debilitating psychological distress. One man chopped off three fingers of one hand. Personality testing confirmed clinical impressions, with some of the men showing dramatic changes in their personality profile after losing as little as 10 lb. These emotional aberrations did not immediately vanish with nutritional rehabilitation but persisted for weeks, with some of the men actually becoming more depressed, irritable, argumentative, and negativistic than they had been during semistarvation. More recent studies of normal weight individuals engaged in dieting or weight loss have confirmed earlier research and documented further psychological and physical complications (Fichter, 1985; Laessle et al., 1989; Rosen, Tacy, & Howell, 1990; Schweiger, Laessle, Fichter, & Pirke, 1988; Thompson, Palmer, & Petersen, 1988). Thus, emotional problems may motivate some to diet and lose weight, but there is convincing evidence that psychological symptoms are a common result of dieting and weight loss in those who are not obese.

For obese individuals undergoing weight loss, there are conflicting findings among research studies of the psychological consequences (cf. Smoller, Wadden, & Stunkard, 1987). Some studies report adverse reactions (cf. Glucksman, Hirsch, & Levin, 1973; Robinson & Winnik, 1973; Stunkard & Rush, 1974), and others find beneficial effects or no change (cf. Wing, Epstein, Marcus, & Kupfer, 1984). Wadden, Stunkard, and Smoller (1986) have provided data suggesting that method of assessment may explain the discrepant findings from earlier studies. They found high rates of adverse reactions when assessment was frequent during treatment, retrospective, and open-ended. In a survey of treatment studies, Smollar et al. (1987) found a strong positive association between amount of weight loss and adverse mood change (r = .77). Thus, achieving clinically significant weight loss greatly increases the risk of untoward emotional reactions.

Further illumination of the psychological consequences of dieting and weight loss come from the 3-year follow-up by Wadden et al. (1988). They reported that subjects who had received behavior therapy reported significant reductions in depression at the end of treatment and at the 1-year follow-up, but at the three year follow-up there was a reemergence of the initial symptoms (Figure 5). Moreover, subjects reported that the weight regain had untoward consequences on their self-esteem, self-confidence, happiness, and on other areas of physical and psychological health.

It is likely that two competing forces are operating in determining the psychological effects of dieting in the obese. On the one hand, many individuals begin their diet feeling depressed or upset about their weight and its effects on their life. Initial success may lead some to feel better psychologically. On the other hand, the "semistarvation neurosis" described by Keys et al. (1950) may predominate. At best, weight loss can only promise temporary psychological benefits, and at worst, it sets the occasion for yet another failure experience. Sadly, obese individuals attribute their failure to character weaknesses, such as poor self-discipline, low self-image, and emotional problems.

We are not aware of systematic research that has attempted to determine the psychological effects of repeated failures in weight loss attempts; however, it has been our clinical impression that the effects can be profoundly devastating even for those who are psychologically robust (Wooley & Garner, 1991). Rather than helping obese individuals to accept themselves at their current weight, treatment programs may unwittingly or deliberately sabotage healthy self-regard, conveying the message that only weight loss justifies self-esteem. The sobering reality is that the newfound anchor in self-acceptance is ephemeral. Although the evidence that dieting has untoward effects is striking, it is important to point out that it is incomplete and it remains as a significant area for future research.

Binge-Eating and Eating Disorders

Concerns about weight and shape lead to dieting and stringent weight control efforts that may precipitate anorexia nervosa and bulimia nervosa (Garner & Garfinkel, 1980). It has been increasingly recognized that the frenzied attack on obesity and body fat in general may have had the grave side effect of leading to an increased incidence of eating disorders (Foreyt, 1987; Garner et al., 1980; Garner et al., 1985; Keys, 1986; Wadden & Stunkard, 1985; Wooley & Wooley, 1982, 1984).

Binge eating is the defining symptom of bulimia nervosa and occurs in approximately one-half of patients with anorexia nervosa (Garfinkel, Moldofsky, &

Garner, 1980); both of these eating disorders are characterized by severe dieting behavior. It is well established that habitual dieters display marked overcompensation in eating behavior that is similar to the binge eating observed in eating disorders (Garner et al., 1985; Polivy & Herman, 1985; Wardle, 1980; Wardle & Beinart, 1981; Wooley et al., 1979b). Admittedly there are limitations to the parallels drawn between laboratory demonstrations of "disinhibited eating" and clinical eating disorders (Charnock, 1989a, 1989b); however, it has been observed that dieting almost invariably precedes binge eating in patients with bulimia nervosa (Abraham & Beumont, 1982). Nevertheless, it is important to point out that binge eating does not occur with all those who diet (cf. Tuschl, 1990). Future research should be directed toward determining factors that lead to its expression in vulnerable individuals. Moreover, for some, binge-eating is a transitory phenomenon, while for others it progresses to a chronic condition. Research suggests that binge-eating comes to serve psychological functions for certain people (cf. Abraham & Beumont, 1982; Steinberg, Tobin & Johnson, 1990), which could compound the physiological pressures that have already been mentioned.

Of further interest is the finding that both obese and lean dieters eat more when depressed, a pattern which is reversed in nondieters (Baucom & Aiken, 1981; Polivy & Herman, 1976). Therefore, dieting may cause depression which in turn makes the individual more vulnerable to bouts of overeating. Again, the Keys et al. (1950) study of semistarvation is instructive in this regard. Some of the volunteers were unable to tolerate the weight loss and reported episodes of extreme overeating followed by self-reproach. This tendency became more pronounced during the refeeding stage of the experiment, during which some of the men reported greater hunger following meals and uncontrollable bouts of binge-eating that persisted for many months after they were permitted free access to food. Also, as noted earlier, the men initially gained to approximately 10% above their control weight before gradually returning to normal.

Since binge eating is characteristic of dieters and patients with eating disorders (who rigidly restrict their intake) and may be "experimentally" reproduced among normal men who lose weight, it is not at all surprising that it is a common problem among the obese (Gormally, Black, Daston, & Rardin, 1982; Hawkins & Clement, 1980; Hudson et al., 1988; Kolotkin, Revis, Kirkley, & Janick, 1987; Loro & Orleans, 1981; Marcus, Wing, & Lamparski, 1985; Stunkard, 1958). Although binge eating may not be of clinical significance in some individuals, for others it is a serious complication of treatment. Drenick et al. (1980) reported that two of the deaths in their study resulted from choking on food during binge episodes. Marcus and Wing (1987) found that between 20% and 46% of obese individuals participating in a behavioral weight control program reported binge eating. The quantity of food consumed and the duration of episodes were similar to patients with bulimia nervosa. Recent research indicates that binge eating is more frequent among the heaviest subjects presenting for weight loss treatment (Telch, Agras, & Rossiter, 1988). These researchers suggest that excessive dieting associated with being overweight may lead to binge eating, which in turn leads to increased caloric intake and greater adiposity. Whether or not binge eating among obese individuals actually leads to an escalation in weight, it is a prevalent and serious complication of the treatment most often recommended for obesity.

These observations have implications for the unfortunate but increasingly popular tendency to label obese binge-eaters as "compulsive overeaters" and to offer

treatment based upon principles derived from the Alcoholics Anonymous or abstinence model of treatment used in addictive disorders (Goldner, 1984; Malenbaum, Herzog, Eisenthal, & Wyshak, 1988). The persistent preoccupation, craving and apparent "compulsion" to consume prodigious amounts of food may be the direct result of self-imposed or formal weight loss programs. Whereas other factors may, in some cases, be implicated, "compulsive overeating" is most parsimoniously understood as a result of the conflict between cultural pressures for thinness and the biological adaptations to suboptimal weight. The use of an addiction model of treatment may have iatrogenic effects since it depends upon intensifying restraint rather than normalizing food intake (Bemis, 1985; Garner, 1985; Vandereycken, 1990; Wardle, 1987). If binge eating is best understood as a natural response to dieting and weight suppression, then psychiatric labels may lead to inappropriate treatment by deflecting attention away from crucial underlying social and biological factors.

When Is Weight Loss Justified in the Treatment of Obesity?

The discouraging outcome of obesity treatment occasionally leads to an acknowledgement that weight loss may be an inappropriate goal for the mildly to moderately obese without specific health risks. But what about the extremely overweight or those who already have illnesses, such as hypertension, diabetes, or heart disease? It would seem to take an extraordinary leap of faith to assume that treatment that has been ineffective for patients *without* medical problems would be more effective or appropriate in patients with these problems. In fact, the information presented earlier on the health risks associated with treatment suggests that the opposite might hold true.

Nevertheless, there may be some for whom the benefits of immediate weight loss outweigh its potential long term risks. Temporary weight loss in some instances may reduce the risks of surgery enough to offset the risks of dieting. The quality of life for some may be so poor that the temporary benefits of weight loss, even with the risk of eventually becoming even heavier, may be justified. This is a problematic criterion since it can be applied to anyone, and in our weight-concerned society, most overweight people will willingly accept inappropriate risks in the hope of becoming thin.

A small minority of patients, able to endure the hunger and emotional hardship of treatment and to sustain their undernutrition for years of maintenance, feel that the sacrifices have been worth the effort. Indeed, Stunkard (1984) has stated that "much of the doctor's job is to help patients to exercise cognitive controls over powerful biological systems, in effect, learning to live in a semistarvation manner" (p. 157). Although we have concerns about the appropriateness of encouraging obese patients to make such a difficult adaptation, it may be unfair to deny help to those who choose this path with entirely informed consent or to cause them to seek even more dangerous treatment outside the medical establishment.

It may be that health risks for some obese individuals can be mitigated by relatively small weight losses or qualitative changes in diet. In these cases, more weight loss may not necessarily be better, and the individual should be encouraged to have health risk factors reassessed periodically.

Finally, there may be some who respond to caloric restriction without great physical or emotional distress and are able to lose and maintain the losses rela-

tively easily (Wooley et al., 1979b). It would, therefore, seem inappropriate to refuse obese individuals the option of weight loss. But it is the contention of this review that health professionals should disclose completely the very poor likelihood of success, while offering "treatments of choice" that do not involve weight loss.

It could be argued that weight loss is justified despite fleeting results, since with many disorders the success rates of certain psychological and behavioral treatments are imperfect or transient. We believe that these situations are not parallel for several reasons:

1. Most psychotherapy patients can be promised better than 5% to 10% success rates.
2. There are physical risks associated with the highly likely failure of obesity treatment.
3. There are underlying physiological mechanisms that are responsible for resistance to downregulation of body weight.
4. Traditional treatment of obesity reinforces the prevailing cultural message that weight loss is the preferred solution to poor self-esteem.

Some have argued that since obesity is a "chronic disorder," patients should be managed under a rehabilitative model involving sustained treatment over many years (Brownell & Jeffery, 1987; Perri et al., 1988). Indeed, the best results have been obtained in treatments offered for the entire duration of follow-up (Bjorvell & Rossner, 1985). Not only is this model expensive, but it again assumes that obesity is inherently more deleterious than treatment. The risks associated with subnormal intake must come under even greater scrutiny if such restriction is offered not as a short-term but as a long-term or even permanent solution.

We want to emphasize that we do not believe that the poor long-term treatment findings should necessarily discourage all research into obesity treatment, but we believe that it is legitimate to question when further dietary treatment of adult obesity should be put to rest both as a subject of investigation and as a clinical technique. At the very least, future research should demonstrate a clear appreciation of the strength of the biological and genetic factors that determine body weight given our current environmental context. Moreover, it should be demonstrated that new mechanisms are being explored rather than a simple reorganization or intensification of methods that have a high probability of failure.

ALTERNATIVES TO WEIGHT LOSS TREATMENT

It may be that health risks associated with some forms of obesity are more clearly related to lifestyle factors, such as excessive consumption of alcohol, dietary fats, and sugar, as well as inadequate exercise, than with obesity per se. If indeed the lifestyle factors are responsible for health risks, then it may be more prudent to focus directly on lifestyle rather than obesity, where treatment compliance and long-term weight reduction results have been so poor. There is some evidence that, in the absence of weight loss, improved physical and psychological health may be achieved by nondieting interventions designed to increase exercise, normalize eating and reduce weight oscillations, and bring about qualitative changes in diet composition. These and other psychological treatments designed to di-

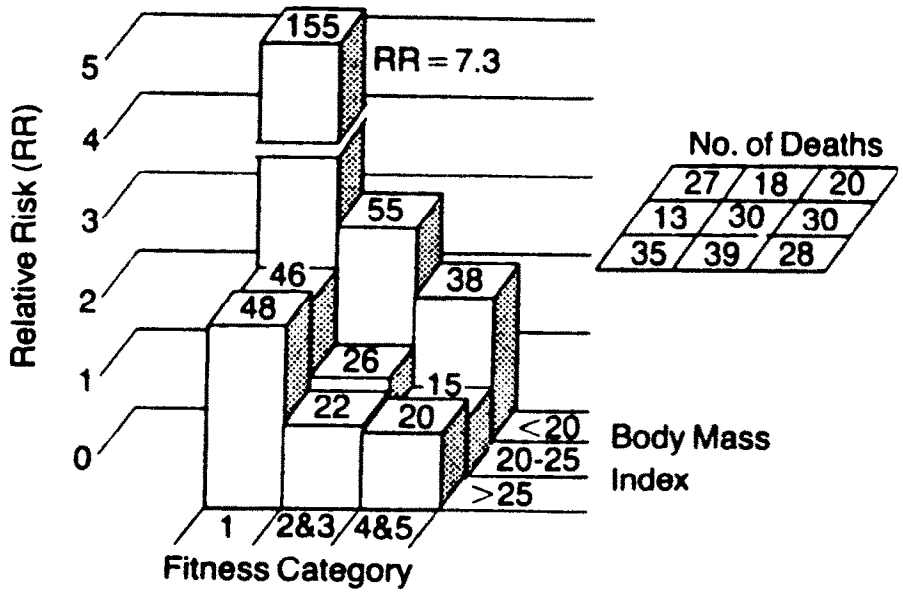

FIGURE 6. Relative Risks of All-Cause Mortality in 10,224 Men by Physical Fitness Category and Body Mass Index. (Each bar represents the relative risk [1 = least fit, 5 = most fit] based on age-adjusted all-cause death rates per 10,000 person-years of follow-up, with the relative risk of the front-right cell set at 1.0. The numbers of actual deaths are shown in the parallelogram. From "Physical Fitness and All-Cause Mortality. A Prospective Study of Healthy Men and Women" by S. N. Blair, H. W. Kohl, III, R. S. Paffenbarger, D. G. Clark, K. H. Cooper, and L. W. Gibbons, 1989, *Journal of the American Medical Association, 262,* pp. 2395–2401. Copyright 1989 by the American Medical Association. Reprinted by permission.)

rectly address self-esteem and body image disparagement may be useful alternatives to traditional treatments for obesity; however, their long-term effects on behavior are not known and their value must be assessed empirically.

Increases in Exercise

Exercise has been recommended as an important component of weight loss programs (cf. Bennett, 1986; Foreyt, 1987; Thompson, Jarvie, Lahey, & Cureton, 1982) and has been associated with better weight loss maintenance (Bennett, 1986; Graham et al., 1983; Perri et al., 1986). However, controlled trials indicate that exercise levels are rarely well maintained at follow-up (Bray, 1990; Perri et al., 1988). Moreover, Pacy, Webster, and Garrow (1986) have concluded "exercise alone appears largely ineffective regarding weight loss and almost certainly has to be coupled with caloric restriction" (p. 89).

Nevertheless, exercise may have a powerful effect on health and quality of life, and if it is not coupled with the discouraging enterprise of dieting it may be

more easily sustained. The Harvard alumni study mentioned earlier (Paffenbarger et al., 1986) illustrates that, in contrast to the debatable health risks associated with obesity, exercise can have a tremendously positive effect on longevity. These results have been impressively confirmed recently by Blair et al. (1989), who found that mortality associated with a range of health risk factors such as elevated cholesterol levels, hypertension, family history of cardiovascular death, and smoking was dramatically reduced with even moderate exercise. Of interest, thinness, not obesity, was associated with increased mortality risk and, as illustrated in Figure 6, exercise had a moderating effect in the untoward effects of thinness!

Thus, to emphasize the impact of exercise on weight loss may be to overlook its highly beneficial effects for the obese in the absence of weight change. Krotkiewski et al. (1979) found that 6 months of physical training in a group of 27 moderately obese women did not lead to overall changes in body weight or body fat but did lead to significant improvements in blood pressure. Although fasting plasma insulin levels did not change for the group as a whole, those with higher pretreatment insulin values showed a significant improvement with physical training. Zelasko, Rosen, and Heusner (1988) examined the effect of moderate progressive exercise without any planned dietary intervention on a group of moderately obese individuals. The results after 6 months indicated that while body weight and body fat remained unchanged, fasting plasma insulin, total triglycerides, and low- and high-density lipoproteins all showed significant shifts in the desired direction. These findings are particularly important when taken together with the studies that show that, in contrast to the dubious benefits of weight reduction on longevity, there is convincing evidence that physical fitness reduces mortality risk (Blair et al., 1989; Ekelund et al., 1988; Paffenbarger et al., 1986).

In addition to beneficial effects on health risk factors, exercise may have a positive influence on self-concept and mood. Ossip-Klein et al. (1989) found that both weight lifting and running improved the self-concept and the mood of clinically depressed women. Holloway, Beuter, and Duda (1988) reported that, in adolescent women, 12 weeks of strength training led to significant improvements on several measures of self-esteem.

Thus, rather than continuing to coerce obese people into reducing their body weight, perhaps health promotion programs could be reformulated into physical fitness regimens to meet the special needs of the obese. Procedural adaptations to accommodate a large body mass or poor initial fitness have been described in two manuals for exercise training in the obese (Kingsbury, 1988; Lyons & Burgard, 1988). Many obese people are deterred from active lives out of shame and fear of ridicule. Others believe that exercise in the absence of weight loss is pointless or even dangerous. It may be important to emphasize that it is possible to be heavy and still strong and fit. Nevertheless, adherence to exercise programs is generally poor, and even with improved programming, the obese, like the nonobese, may fail to take advantage of this means of improving health.

"Normalizing" Food Intake

Many obese individuals have spent much of their lives either restricting their intake or overeating when cognitive controls are relaxed or break down in response to a variety of emotional, interpersonal, or situational factors (Polivy &

Herman, 1985; Wardle, 1987). A fundamental premise of "normalizing" food intake is that the extraordinary food cravings experienced by many obese individuals are the result of dietary restriction and are similar to those seen in dieters of all weights.

Chronic dieting often involves an elaborate "morality" surrounding eating in which certain foods are "good" (i.e., calorie sparing) and others "bad" (i.e., fattening). Dieters report feeling "virtuous" when they restrict their intake and guilt ridden when they "blow their diet" (Garner et al., 1985; Polivy & Herman, 1985). After years of this "dichotomous" approach to eating, some obese individuals report short circuiting the process, overeating because they know that they are going to fail, so that there is no reason to restrict their intake in the first place. The basic approach to normalizing eating involves the genuine recognition that dieting is self-defeating and that virtually every type of food may be consumed in moderation. The quantity, quality, and spacing of meals to approximate a normal eating pattern may have to be completely relearned (Garner et al., 1985).

Ciliska (1989) reported on the effectiveness of a nondieting approach to the treatment of obesity that emphasized the elimination of caloric restriction based on principles developed for the treatment of anorexia nervosa and bulimia nervosa (Garner et al., 1985). The nondieting approach was delivered in a group format that involved either education alone or education plus experiential exercises (Ciliska, 1989). The experiential component involved an adaptation of certain body image exercises recommended for bulimia nervosa (Wooley & Kearney-Cook, 1986; Wooley & Wooley, 1985). Groups met weekly for 12 weeks and were assessed on a range of psychological parameters before treatment, posttreatment, and at six month follow-up. Results indicated that subjects in the experiential/educational group showed improvements on measures of self-esteem and dietary restraint, which were maintained at follow-up. Additional improvements on measures of body dissatisfaction, depression, and social adjustment were not maintained at follow-up testing. Efforts to persuade participants to suspend dietary restriction not only led to reduced scores on measures of dietary restraint, but also dramatic declines on the Bulimia and Drive for Thinness subscales of the Eating Disorder Inventory (cf. Garner, 1991), without a significant increase in body weight at the end of treatment or at follow-up.

A somewhat different approach was applied by Roughan, Seddon, and Vernon-Roberts (1990) to a group of 87 obese women selected on the basis of eating and weight preoccupation. A 10-session group focused on eating in response to hunger while abandoning restrictive dieting, increasing acceptance of the body regardless of size, and coping strategies to deal with emotional distress. Results on the 80 women who completed the group indicated significant improvements in eating attitudes, body image, self-image, depression, and self-esteem, as well as a small but significant weight loss (1.8 kg, $p<.004$). A 2-year follow-up of 57 of the women revealed that improvements on psychological measures were maintained or enhanced along with a mean weight loss of 3.1 kg compared to baseline. These are encouraging results, but efforts need to be made to determine the status of those not followed as well as to extend the duration of follow-up.

Qualitative Changes in Dietary Intake

There have been a number of studies that indicate that qualitative changes in diet, rather than weight loss, may have a positive effect on health risk factors. In

an early report, Dahl, Silver, and Christie (1958) found that salt restriction, rather than weight loss, was responsible for blood pressure improvements in obese hypertensives. In fact, the patients who were restricted to 600 to 800 calories per day experienced significant weight loss but generally remained hypertensive. Of those who only had drastic sodium restriction, most had a significant improvement in both systolic and diastolic blood pressure. Dahl et al. (1958) speculated that the improvements in blood pressure observed with weight loss in many studies may be due to inadvertent salt restriction. In two studies involving 87 mildly to moderately obese hypertensive men, Gillum et al. (1983) found that even modest weight reduction (an average of 3.9 kg in one sample) and salt restriction had independent effects in lowering blood pressure. More recent studies have confirmed earlier findings (Australian National Health and Medical Research Council Dietary Salt Study, 1989; MacGregor, Markandu, Sagnella, Singer, & Cappuccio, 1989; Stamler et al., 1989).

Of particular interest is the 5-year trial by Stamler et al. (1989), in which 201 men and women with mild hypertension at baseline were randomly assigned to either an intervention group receiving intensive individualized guidance from physicians and nutritionists to achieve lifestyle changes or to a control group that was monitored every 6 months. The intervention group received advice aimed at weight reduction, change in diet composition (including salt reduction), reduction in alcohol intake, and increase in exercise. Dropout rates were small for both groups, with 87% participating for more than 4 years. The intervention group achieved a 25% reduction in sodium intake and a 30% decline in alcohol consumption, with 75% of the participants reporting an increase in moderate exercise. The mean weight loss for the intervention group was small but significant (2 kg) compared to a slight gain for the control group (0.8 kg). Intervention led to significant improvements in blood pressure, with one in five of the participants on the control group moving from hypertension-prone to hypertensive during the 5-year trial versus 1 in 11 in the treatment group. Whether similar results would have been obtained by simply achieving a reduction in sodium intake is not clear. In this regard, the double blind study by MacGregor et al. (1989) is of interest, since it demonstrated a progressive decline in blood pressure over one year with salt restriction alone that was well maintained for 16 of 20 participants.

Several epidemiological studies have suggested blood pressure lowering effects of vegetarian diets (Armstrong et al., 1979; Armstrong, Van Merwyk, & Coats, 1977; Rouse, Armstrong, & Beilin, 1982; Sacks, Rosner, & Kass, 1974). Rouse, Beilin, Armstrong, and Vandongen (1983) conducted a controlled study of 59 healthy omnivorous subjects (30 women and 29 men) who were randomly allocated to a control group which ate an omnivorous diet for 14 weeks, or one of two experimental groups whose members ate an omnivorous diet for the first two weeks and then a lacto-ovo-vegetarian diet for one of two 6-week blocks of time. Results indicated that mean systolic and diastolic blood pressure did not change in the control group, but it fell significantly in the two experimental groups during the vegetarian diet and then rose significantly when returned to the omnivorous diet.

Many studies have shown that large amounts of polyunsaturated fats in the diet have a marked hypolipidemic effect (cf. Connor & Connor, 1982). Phillipson, Rothrock, Connor, Harris, and Illingworth (1985) reported that a diet high in dietary fish oil had a profoundly beneficial effect in each of 20 patients with

hypertriglyceredemia. Total plasma cholesterol, very-low-density lipoprotein, and triglyceride levels fell in each patient although body weights remained constant. Very-low-density lipoproteins were also reduced dramatically.

Modifying Negative Body Image

Studies of the nonobese have shown an association of negative body image with poor self-esteem, social anxiety, vulnerability to depression, and general psychological distress (cf. Archer & Cash, 1985; Cash & Brown, 1987; Garner, Garfinkel, & Bonato, 1987; Noles, Cash, & Winstead, 1985). Cognitive-behavioral interventions have been designed to help those with eating disorders overcome reliance on weight or shape as the sole or predominant referent for inferring self-worth (Garner & Bemis, 1982, 1985; Garner, Garfinkel, & Bemis, 1982); these may be useful in addressing the self-loathing of some obese clients. A number of recent controlled studies have indicated that body dissatisfaction can be significantly improved using cognitive-behavioral methods (Butters & Cash, 1987; Dworkin & Kerr, 1987; Rosen, Saltzberg, & Srebnik, 1989). Of importance, improvements in body image in these studies were associated with positive changes on a range of measures of other areas of self-concept and psychological functioning. Self-loathing related to shape among the obese is more pervasive and more extreme but perhaps would be amenable to change with some of the new treatments that have been offered for body disparagement.

Experiential body image therapy has been shown to be effective for bulimia nervosa patients and has also been used in the treatment of obese clients who report extreme body disparagement (Wooley & Kearney-Cooke, 1986; Wooley & Wooley, 1985). These treatments typically involve exploration of the history of body image development with opportunities to reexperience and master negative influences. Accordingly, patients are encouraged to reframe their negative body image as having a social derivation rather than reflecting intrinsic personal deficits (cf. Wooley & Wooley, 1980; Wooley et al., 1979a) and then helped to adopt strategies to address real obstacles presented by friends, family, and employers.

As indicated earlier, the pressures to be thin in order to meet the stringent cultural standards for physical attractiveness are aimed almost exclusively at women and have particularly devastating consequences for those who are much heavier than the norm. There may be value in addressing body image disparagement through the exploration of the social stigmatization of obesity and the questioning of the acquiescence to social prejudice implied in a life of chronic hunger and the pursuit of thinness. Wooley and Wooley (1980) and Wooley et al. (1979a) have reviewed social attitudes toward obesity from a feminist perspective with three themes: (a) Prevailing attitudes toward fatness have specific feminist implications; (b) fatness is an ethical, political problem more than a medical, psychiatric, or behavioral problem; and (c) there is a need for drastically revised attitudes toward obesity and its treatment. Treatments informed by feminism have emphasized helping women to question rather than conform to destructive cultural norms for body image, have attended to past sexual and physical abuse which has left a residue of bodily shame, and have had as a major focus the revaluing of women's strengths including revaluing of the normal female body.

Examination of alternative treatments should be undertaken with caution since there is little empirical evidence to recommend their superiority to traditional

treatments. It seems evident that their implementation should avoid simply "redefining the problem" of obesity in a manner that is disrespectful of the emotional and financial investment that the obese client has made in weight loss. It is important to remain cognizant of the social context within which obese people have lived, receiving weight loss advice from the health care establishment and encouragement to diet from family, friends, and media, and the weight loss industry. Most important, they have long believed that in order to feel good about themselves, they must lose weight. Giving up weight loss as a goal may be viewed as a surrender to self-loathing and relinquishment of hope for a degree of self-acceptance that most thin people take for granted. Thus, a non-weight-loss alternative would need to be explored in a gradual and sensitive manner without the expectation that it will be readily accepted. This involves beginning "where the client is," usually with the belief that weight loss is necessary and helping him or her to evaluate the costs and benefits of further dieting and other approaches.

SUMMARY AND CAVEATS

It is difficult to find any scientific justification for the continued use of dietary treatments of obesity. Regardless of the specific techniques used, most participants regain the weight lost. The inevitability of this result is often obscured by the use of follow-up periods insufficient to capture the later phases of weight regain. No longer can we reasonably expect improved results with greater sophistication of techniques as was appropriate during the early period of development and refinement of behavioral technology. Indeed, failure of dietary treatments seems rooted in the biology of weight regulation. As Bennett (1984) aptly put it, the effect of sustained caloric deprivation is "to pit the individual's 'will' against an untiring opponent, the set point mechanism" (p. 331).

If inefficacy were not reason enough to abandon dietary treatments, additional arguments can be put forward. The most common justification for treating obesity is its apparent adverse effects on health and longevity. As has been shown, these effects are in fact the subject of considerable controversy, and there is much evidence to suggest that maintenance of high but stable weight is safer than weight fluctuation, perhaps safer even than weight reduction. Dieting can lead to binge eating in individuals who have not experienced the symptom prior to attempting weight loss; it also may precipitate serious eating disorders such as anorexia nervosa and bulimia nervosa. Dieting and weight loss may have untoward psychological effects including depression, anxiety, social withdrawal, and personality changes. Finally, dieting, leading to repeated cycles of weight loss and regain, may actually contribute to obesity by increasing metabolic efficiency.

We suggest that at the least, if weight loss is offered, it should be done with full disclosure of the lack of long-term efficacy and of the possible health risks. It is further recommended that alternative nondieting approaches aimed at improving the physical and psychological well-being of the obese individual be given priority over dietary treatments as a subject of research and that such treatments be offered on an experimental basis. The collection of data on the efficacy of such treatments on health risk factors and social adjustment should allow evaluation of their appropriateness for widespread use.

It could be argued that we have not included all of the scientific literature related to each point of emphasis. This is true since each area surveyed has an enormous literature summarized in numerous books, monographs, and reviews.

While our review does not present all of the contradictory evidence on every point, we believe that it represents a fair integration and synthesis of behavioral biologic or social factors that must be considered in evaluating obesity treatment. Considering what is currently known about obesity and its treatment, we believe it remarkable that there have been so few calls of reexamination of the fundamental premises that form basic health care policy regarding weight loss. It could also be suggested that we have not adequately addressed the multiple pathways to obesity considering the different genetic, neuroendocrine, nutritional, seasonal, and pharmacological contributing factors that have been described (Sclafani, 1984). We fully recognize the complex and multidetermined nature of obesity and further recognize that discoveries in any number of areas might result in effective and appropriate treatment of some or all obese persons. However, at this time we can enhance the possibilities of meaningful scientific progress in other areas by reallocating resources currently invested in developing, applying, and studying dietary treatment that have little rational hope of success.

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