*THEORETICAL, PRACTICAL, AND SOCIAL
ISSUES IN BEHAVIORAL TREATMENTS
OF OBESITY*

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Although showing superior maintenance, behavioral treatments of obesity typically produce small weight losses at a decelerating rate. Rather than reflecting poor compliance with treatment, these findings are consistent with known compensatory metabolic changes that operate to slow weight loss and promote regain. Other problems associated with dieting include failure of caloric regulation, hyper-responsivity to food palatability, and hunger, which is greatest under conditions of moderate restriction and unpredictability of access to food. The inevitability of treatment failure in many instances must be faced and efforts made to prevent further worsening of the obese patient's self-esteem. Prognosis and treatment planning may be aided by consideration of the historical difficulties of weight loss, the degree of hunger experienced on diets, which may reflect important physiological differences among individuals, and the use of food to optimize arousal level. Full involvement of the patient in setting goals and planning treatment is recommended.

DESCRIPTORS: obesity, behavioral treatments, hunger, food restriction, metabolism, weight loss, physiological factors, self-esteem, cultural prejudices, humans

During the last decade, a multitude of behavioral approaches to obesity have rapidly emerged. This paper does not attempt to review the specific methods in detail, for this has been carefully done elsewhere (Bellack, 1977; Jeffery, Wing, and Stunkard, 1978; Leon, 1976; Stunkard and Mahoney, 1976; Wilson, 1978; and Yates, 1975); rather, both the achievements and the limitations of these approaches are analyzed in the context of a broader view of obesity.

Obesity treatments constituted the first and perhaps still the largest foray of psychologists into a traditionally medical field, a choice both fortuitous and calamitous, for although it speeded development of treatments it soon revealed their limitations. Psychologists had

picked one of the most perplexing and refractory of all disorders. The initial choice of problem areas probably had much to do with the objectivity and ease of measurement of body-weight. The availability of a suitable dependent variable encouraged the adoption of obesity treatment as a testing ground for behavioral techniques. The *Zeitgeist* against which the new behavioral techniques were developed was certainly favorable to new approaches. Conceptualizations of obesity in psychiatry, psychology, and psychosomatic medicine stressed the extraneous causes of eating in obesity, rooted in developmental disorders, but insight therapies based on these ideas had proven ineffective. Medical researchers had named obesity as a major health problem, but had rejected the idea that it was constitutionally determined in any but a small fraction of cases. Attempts to modify eating patterns directly seemed an obvious next step.

While these generalizations do not quite represent the true state of knowledge 10 yr ago, they do reflect what was generally known.

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Since then, sufficient data have been accumulated to disprove many well-established preconceptions and to replace them with a cacophony of findings and theories which, to one degree or another, challenge the scientific and social appropriateness of all existing modes of treatment.

The new findings make it easier to understand why behavior therapy, although showing a decided improvement over its predecessors, is still disappointing in its effectiveness, and they challenge psychologists to develop still more sophisticated approaches to treatment, taking into account complexities of the physiology and the social context of obesity.

The major assumption in behavioral treatments of obesity is that overweight is due to excess food consumption resulting from faulty eating habits. Learned behaviors hypothesized to contribute to overweight have included rapid rate of consumption, large bites, frequent feedings, ingestion of large quantities in a given meal or snack, *etc.* These behaviors are regarded as operants, reinforced by the pleasure of eating and under the partial control of environmental cues associated with eating, which serve as discriminative stimuli for ingestive behaviors. A common explanation of "overeating" is that the reinforcing consequences are immediate, whereas the aversive ones are delayed.

This formulation has led naturally to treatment strategies that (1) provide immediate reinforcement for "appropriate" eating or food abstinence (*e.g.*, money, social approval, self-reward), (2) diminish the strength of pleasure as a reinforcer for eating (*e.g.*, aversive conditioning to food tastes, covert conditioning), (3) make long-term negative consequences more salient at time of eating (*e.g.*, rehearsal of the undesirability of being fat and the value of thinness), (4) eliminate food cues that serve as discriminative stimuli (*e.g.*, ridding the house of fattening foods), or (5) narrow the range of discriminative stimuli for eating by restricting the time and place in which eating occurs.

To the extent that a simple learning model applies to eating behavior, these strategies make sense and indeed have been effective. Failures of the learning model have less to do with the ingenuity and technical adequacy of techniques than with limitations in the explanatory power of the model itself.

One problem that others have noted (Mahoney, 1975*a*; Mahoney, 1975*b*) is the difficulty in defining faulty or maladaptive eating patterns, except by fiat or tautology (as any high-frequency behavior displayed by the particular obese patient). The conditions leading to faulty learning are not defined, the relationship of particular behavior patterns to total food consumption is not known. Nor is there even good evidence to show that certain eating styles are correlated with obesity. At first glance, this seems to be a rather simple empirical problem attributable to the fact that treatment research has progressed more rapidly than basic observational research. Indeed, since Mahoney's (1975*b*) discussion of this problem in 1975, many comparative studies of eating styles in the obese and nonobese have been carried out, although they have not resulted in the clear-cut differentiation that had been desired.

However, there are more fundamental problems to be reckoned with, and these are discussed in detail in succeeding sections. As will be documented later, several decades of research have shown that on the whole, the obese eat no more than the lean, suggesting that important differences lie in energy expenditure. Energy expenditure, however, is tied to diet, showing a tendency to decrease with caloric restriction and increase with overfeeding, but to varying degrees in different individuals, so that some people's metabolic systems appear to respond in such a way as to maintain leanness while others maintain adiposity. Excess pounds may be lost readily by some, and with extreme difficulty by others, the term difficulty referring to degree of caloric restriction required, the degree of experienced hunger, and the degree of general dysphoria.

Thus, the reinforcer of eating behavior in operant theory—pleasure—(or, perhaps more descriptively in some cases “relief of discomfort”), is probably neither equivalent across people nor equivalent across degrees of deprivation or levels of bodyweight in the same person. By the same token, there is no reason to assume that the control over behavior effected by discriminative stimuli is constant, nor the relationship of eating behavior to internal states.

It seems likely that eating behavior cannot be adequately conceptualized without retaining the concept of hunger and considering the variables that influence it. In humans, hunger is neither synonymous with amount of food ingested (for voluntary and cognitive controls may greatly influence what is eaten) nor is it merely hypothetical and inaccessible to study, since several measures reliably and sensitively reflect deprivation states and drug effects in normally functioning individuals.

Finally, there is a “psychology” of obesity, which extends beyond ingestive behaviors and takes into account the cultural and social pressures on the obese to behave in certain ways, and the (ineffective) self-help strategies typically shaped by these forces. It is important to consider the impact of treatment on problems commonly associated with obesity and to define the goals of treatment more comprehensively in terms of the patient’s overall welfare.

FOOD INTAKE IN OBESITY

Total Consumption

The belief that obese people “overeat” is so widespread that one wonders if this conviction will give way to the actual data on this question. It is, of course, true that obese people overeat in the sense that their caloric intake is too great to keep them thin. But what is usually meant is that they eat abnormally large amounts of foods relative to a lean population. In Garrow’s (1974) book, the findings of 13 studies relating body weight to food intake were re-

viewed. Of these, 12 showed the intake of the heavier subjects to be less or equal to that of the thinner ones. The remaining study confirmed these results by one measurement technique while finding differences by another, and not surprisingly, concluded that the latter technique was therefore valid. An additional seven studies may be cited in support of the finding of no differences (Thomson, Billewicz, and Passmore, 1961; Hejda and Fabry, 1964; Hampton, Huenemann, Shapiro, and Mitchell, 1967; Hanley, 1969; Kissileff, Jordan, and Levitz, 1978; S. C. Wooley, Tennenbaum, and Wooley, *in press*; Coll, Meyer, and Stunkard, *in press*).

Any of these studies can be criticized on methodological grounds. In some, the range of weight is rather narrow. Others depend wholly on self-report measures. Others are based on rather small samples and are conducted in laboratories under conditions that might be expected to affect natural eating behavior. Others record only public eating. Nonetheless, the congruence of results is so striking that the burden of proof must be said to rest with those who contend that there is a difference.

This very puzzling finding is not easy to interpret. For one thing, although the same may be said of the lean subjects, the obese samples in all likelihood contain subjects in various phases of weight gain and loss as well as subjects in equilibrium. The low caloric intakes of subjects on restricted diets must therefore be balanced out by high intakes of other subjects, possibly those gaining weight. There are no data to answer the question whether during the periods in which weight is gained obese people have higher intakes than lean ones. It is tempting to assume that this is the case, but risky, as will be seen in later sections on the problem of regain after weight loss.

At the least, one may conclude from these findings that any abnormalities of overall food intake that may exist in the obese as a group probably have to do with patterning of food consumption, rather than total consumption

over long periods of time. Many overweight people maintain their weight on modest intakes and the assumption that weight loss will follow if obese patients can be made to eat "normal" amounts will prove false for many patients. In both lean and obese subjects, the range of calories required to maintain constant body-weight is very large and remains unexplained by such obvious variables as degree of physical activity. Although most authorities believe on the basis of the many animal models of obesity that there are distinct subtypes, no reliably predictive classification system for humans has yet been devised.

Eating Styles

In the last few years, a number of studies have been carried out to compare obese and lean subjects with respect to various parameters of eating behavior, such as number, timing, and composition of meals and snacks, rate of bites taken, size of bites, *etc.* These studies were undertaken to uncover aspects of eating behavior associated with obesity, often with the specific goal of more clearly specifying behavioral targets for treatment interventions. Recent papers by Meyers, Stunkard, and Coll (Note 1) and Coll *et al.* (*in press*), concluded that no replicable obese-normal differences in eating styles or food choices have been found in the series of studies conducted in naturalistic settings, or in related laboratory studies. In a comparison of a large sample of obese and lean subjects who kept two-week food diaries, Kissileff *et al.* (1978) found no differences in duration, place, or frequency of meals or snacks or mood states, level of hunger, or activities associated with eating. All subjects reported behaviors often thought to typify the obese, such as eating in the absence of hunger, rapid eating, and frequent snacks. Coll *et al.* (*in press*) cited evidence from analysis of adipose tissue of lean and obese subjects which indicated that the constituents of their diets are similar. Reports from large-scale studies comparing eating pat-

terns of obese and normal subjects show the obese to eat fewer meals per day. (Fabry, Fodor, Hejl, Braun, and Zvolankova, 1964; Hejda and Fabry, 1964). A possible reason for the discrepancy between these findings and those of Kissileff *et al.* is that Fabry's subjects were not patients and were relatively old. The only difference that seems consistently to emerge from comparisons of eating behavior is that the intake of the obese is more dependent than that of the lean on food palatability (Coll *et al.*, *in press*; Rodin, 1976; O. W. Wooley and Wooley, 1975; S. C. Wooley *et al.*, *in press*).

It should be noted that the original intent of studies of eating habits was to isolate behavior patterns that led to the overeating assumed to accompany obesity. Since this critical assumption has not been upheld, the design of these studies does not, in fact, allow discovery of predictors of high food intake. Certain eating habits might indeed lead to greater overall consumption, a hypothesis that could be tested by comparing eating styles of large and small eaters, or by manipulating parameters of food intake (see Mahoney, 1975a; O. W. Wooley, Wooley, and Turner, 1975). It would also seem logical to study the effects of deprivation on parameters of eating. It may be premature to assume that differences in manner of eating reflect only differences in habits based on such factors as social learning. If they are instead indicators of hunger, their modification through behavioral interventions may have little value. Indeed, the few studies that have monitored habit change have not found these changes related to weight loss (Bellack, Rozensky, and Schwartz, 1974; Jeffery *et al.*, 1978; Brownell, cited by Wilson, 1978). However, failure may be due to the use of weight loss as a criterion. It would seem preferable to relate habit changes to changes in overall food intake, rather than to weight loss, because the latter is a considerably more remote variable that does not always vary in expected ways with changes in food intake, a point discussed in greater detail in succeeding sections.

Of course, some aspects of eating behavior are of interest apart from their relationship to total intake because of the possibility that they may directly affect the utilization of ingested calories. There is, for example, evidence that in laboratory animals, compression of eating into a few hours of the day results in a greater tendency to store the ingested calories as fat (Cohn and Joseph, 1959; Tepperman and Tepperman, 1964). Collier, Hirsch, and Hamlin (1972) found that with very high fixed-ratio schedules, rats adopted a pattern of eating only one meal per day. Their intake was only half the original maintenance value but bodyweight losses did not exceed 6%. Studies of the effect of meal frequency on rate of weight loss have produced inconsistent results (Garrow, 1974; Young, Scanlon, Topping, Simko, and Lutwak, 1971). However, the fact that infrequent meal eating leads to hyperlipogenesis does not necessarily imply that weight-loss diets composed of frequent meals should promote lipolysis and weight loss. In discussing meal spacing, Garrow (1974) suggested that the somnolence produced by large meals may reduce energy expenditure and thus reduce rate of loss.

It is clear from this discussion that further study is required to establish a clearcut rationale for modification of specific features of eating style as a treatment strategy. However, if the aim of behavioral programs is conceptualized as helping each patient to find the most comfortable way of reducing or stabilizing intake, then experimentation with modes of eating makes sense. The difference is that the rationale for any given change is that it is found to help that individual, requiring the construction of an idiographic theory of each person's eating behavior. There is no pretense that eating is being "normalized" and no standard set of instructions. In practice, the authors have found this approach quite workable, even in small groups (six or fewer). Thus, for one patient, eating very high protein meals may help to curb between meal appetite; for another, planned snacks may stabilize energy level and

reduce meal size disproportionately; for another, increasing calories during the day may prevent night eating. A technique is considered successful if it reduces daily intake or increases satisfaction. To accomplish this, therapist and patient must carefully study food records containing information on hunger, pleasure and circumstances of eating and plan and analyze the results of weekly "experiments".

Such an individualized approach is not offered as an optimal solution. It merely employs trial and error in the absence of established general principles. It is to be greatly hoped that the somewhat confusing and discouraging results obtained from the first round of studies of eating styles will not deter further efforts, posing other questions. There is, for example, some experimental as well as anecdotal evidence that slow eating (O. W. Wooley *et al.*, 1975) and ingestion of protein foods enhance satiety (Booth, Chase, and Campbell, 1970; O. W. Wooley, Wooley, and Williams, Note 2). Findings such as these may have practical value apart from any relationship to theories of obesity.

ENERGY EXPENDITURE

Energy expenditure may be somewhat arbitrarily divided into three components: basal metabolism, that is calories expended at rest and in a fasted state; the additional calories required to support sedentary activity and to metabolize food; and the calories burned in motor activity, *i.e.*, exercise. There is a current trend to replace measures of basal metabolism with measures of resting metabolism, which do not require an overnight hospitalization and fast. In most individuals, basal metabolism and sedentary activity account for by far the largest portion of calories utilized and exercise the least.

The finding that the obese eat the same amounts as the lean suggests that the storage of excess calories may be due to lesser energy

expenditure, at least for some period of time. It *cannot, however, be assumed* that the difference rests in activity level or that increasing activity levels is necessarily the most logical target of intervention, although there is indeed some empirical grounding for these statements.

Studies of activity levels in the obese have produced highly variable results. Garrow's (1974) review of studies on this topic found about equal numbers demonstrating lesser activity levels in obesity and finding no differences. These studies used such diverse measures as motion picture sampling of obese and non-obese adolescents engaged in sports (Bullen, Reed, and Mayer, 1964), self-report (Johnson, Burke, and Mayer, 1956; Stefanik, Heald, and Mayer, 1959), pedometer readings (Chirico and Stunkard, 1960), a device that discriminates sitting from standing (Bloom and Eidex, 1967), and a device that measures movement in a chair (Greenfield and Fellner, 1969). Brownell, Albaum, and Stunkard (Note 3) found that the spontaneous use of stairs in place of an escalator by obese subjects was 1.5% *versus* 6.7% in lean subjects.

Despite the tremendous methodological ingenuity in these studies, interpretation of results is quite difficult. Activity level must be converted to energy expenditure, taking account of the greater energy cost of movement for heavier persons as was done, for example, in Chirico and Stunkard's (1960) study. Generalizability is open to question in observations of activities in which the obese are typically made to feel self-conscious (*e.g.*, sports), and accuracy is uncertain in measures relying on self-report. One tends to think of distortions in self-report occurring in the direction of social desirability, but this is not always the case. Johnson *et al.* (1956) reported that their sample of obese adolescents who were found to eat less than their peers believed that they ate more. The studies based on pedometers worn for periods of days the most compelling evidence, but Stunkard (Note 4) has noted that pedometers do not always function properly when worn by

obese subjects because they do not fall vertically from the waist.

More fundamental, however, is the question of how significant, in terms of total overall caloric expenditure, the differences that have been observed may be. Although Brownell *et al.* (Note 3) found that lean subjects used stairs more than four times as often as obese subjects, neither used them much. Garrow (1974) argued that among people with primarily sedentary occupations, physical activity accounts for a relatively small proportion of total energy expenditure, insufficient to account for obesity. Thus, although it seems likely, on the basis of existing evidence, that the obese, at least obese women, are somewhat less active, the significance of this fact remains uncertain. Nor is there any prospective evidence to suggest that relative inactivity is a cause of obesity, rather than an effect. There is evidence that activity is itself a regulated variable, decreasing, for example, with caloric deprivation and varying with predictability of feeding patterns. Whether or not it would prove easier to make lasting alterations in activity level than in food intake remains an open question.

Other sources of variability in energy expenditure are of interest. It should first be noted that individual differences within the range of "normal" are quite large. Rose and Williams (1961) studied pairs of subjects matched for age, weight, height, and sex, one of whom habitually consumed twice as much as his control to maintain the same weight. The investigators were not able to attribute the differences to physical activity level. Warwick, Toft, and Garrow (1978), in a study of normal subjects under controlled conditions, found differences of as many as 400 to 500 calories per day in resting metabolism and as many as 770 per day during sedentary activity.

Effects of Caloric Restriction on Energy Expenditure

A second major source of variability in energy expenditure is diet. In studies of humans,

caloric restriction has been reported to produce decreases in basal metabolic rate of 15 to 30%, decreases in spontaneous activity and decreases in the energy cost of a particular task (Apfelbaum, Bostsarron, and Lacatis, 1971; Bray, 1969; Buskirk, Thompson, Lutwak, and Whedon, 1963; Drenick and Dennin, 1973; Garrow, 1974; Howard, Grant, Challand, Wraight, and Edward, 1978; Keys, Brozek, Henschel, Mickelson, and Taylor, 1950) Kleiber (1961) wrote that "metabolic level (in rats) decreases as an almost linear function of starvation time . . . to 60% of the level observed at the start of starvation."

This lowering of metabolic rate is generally seen as an adaptive response to food restriction, as a way to conserve sparse energy. Kollar, Slater, Palmer, Dokter, and Mandell (1964) reported an increase in the amount of time starved subjects spent in isolated "withdrawal" as a fast progressed. Subjects spent increasingly more time napping during the day. They could mobilize effort for specific tasks but found it difficult to sustain activity. The authors speculate that humans show an "inhibitory conservatory response" to diminished food supplies.

However, these changes are not restricted to lean subjects in whom caloric restriction leads to underweight (Buskirk *et al.*, 1963; Drenick and Dennin, 1973). Rather, they appear to be most marked in subjects whose initial metabolic rate is low. Martineaud and Tremoliere (see Garrow, 1974) reported that obese patients with the lowest metabolic rates had the biggest drop in rate during caloric restriction. Rixon and Stevenson (1957) also noted that in rats, low prefast metabolic rates are associated with slow rates of weight loss. These facts may help to explain why patients with the slowest initial rate of weight loss lose the least overall, (Jeffery *et al.*, 1978), an alternative to an explanation based on noncompliance with program requirements.

The conditions that appear to maximize the potential of energy-conserving responses to food restrictions are discussed in later sections,

but defensive conservatory responses have been observed in the obese when food is unavailable or when control over food supplies is reduced. O. W. Wooley, Wooley, and Dunham (1976) found that obese (but not lean subjects) showed decreased salivary responses under conditions of deprivation and unavailability of food. Decreased salivation has been reported by Bogdanoff, Bogdanoff, and Wolf (1961) to characterize a defensive energy-conserving response as opposed to an aggressive response to threat, and has been found in the 20 studies in which it has been measured, to accompany depression, or "learned helplessness". O. W. Wooley and Wooley (Note 5) found that during a week at a fitness camp in which food intake was limited to 900 calories per day, subjects showed changes in resting unstimulated salivation comparable to those seen in depression.

The effect of changes in metabolic rate on the course of weight loss during a diet is not trivial. According to Apfelbaum and Bostsarron (1971), an individual can expect to lose about 40 g per day if calories are cut from 2000 to 1500 per day. However, "the weight loss will soon be stopped by adaptive changes in energy expenditure induced by the restricted diet itself" (p. 426), dropping to 20 g per day the second month, and 10 g per day the third month, then stopping altogether. An approximate halving of the weight lost each month on a fixed reducing diet corresponds well to figures reported by Jeffery *et al.* (1978) for average rates of weight loss in successive four-week intervals of a comprehensive behavioral treatment program: 6.3 pounds, 3.6 pounds, 2.1 pounds, and 1.7 pounds.

Adaptive changes in energy expenditure may become more pronounced with each diet. Garrow (1974) described evidence that with longer periods of underfeeding, recovery of metabolic rate to prerestriction levels takes longer and that metabolic rate falls more rapidly with return to underfeeding than it did initially. Thus, with progressive attempts at weight reduction, weight may be lost more slowly and regained

more readily, one of many possible explanations for the finding of some investigators that behavioral treatment of older or more chronically obese patients is less effective than is treatment of younger patients.

The fact that metabolic changes appear to persist after termination of fasts so as to enhance weight regain poses a problem even more perplexing than the slowing of rate of loss. Studies with animals suggest that, even though increased length of starvation leads to lessened intake with refeeding, the weight gain is the same as seen with lesser deprivation and higher refeeding intakes (Hamilton, 1969). In a dramatic demonstration of metabolic compensation, Boyle, Storlien, and Keesey (1978) found that rats starved to 20% below normal weight gained 29.6 g during a period of refeeding in which they ate less than baseline amounts (the calories adjusted downward in proportion to the loss of metabolically active tissue), while *ad lib* controls actually receiving more food gained only 1.6 g. This represents an 18-fold increase in efficiency. Metabolic changes associated with dieting not only predispose to rapid gain, but bias the system toward excessive storage of adipose tissue.

During refeeding, the starved normal volunteers of Keys *et al.* (1950) gained to 110% of initial weight (with eventual return to baseline) and to 140% of initial amount of adipose tissue. The percentage of regained weight that was fat varied from 33% to 67%, depending on the calories eaten during the refeeding period. Tepperman and Tepperman (1964) found that "the tissues of animals which have been starved for 2 or more days and then refed a high carbohydrate diet show an extremely high capacity for converting nonfat precursors into fatty acids", a phenomenon they call "adaptive hyperlipogenesis". Szepesi (1978) found that following deprivation and refeeding to initial weight, rats had 30 to 50% more adipose tissue than at baseline. Stepwise increases in the refeeding diet did not prevent this, although it was lessened by certain psychoactive drugs

(haloperidol, spiroperidol, and pimozide), a surprising finding that they attribute to the action of these drugs in reducing the effects of stress associated with deprivation. As discussed earlier, hyperlipogenesis also appears to be associated with infrequent meals.

The import of greater fat stores at equivalent body weights is not clear. One might speculate that this change in body composition could affect the course of later attempts at weight loss or have implications for health. At present there is no way to know, but the clearest implication of this research is that caloric deprivation produces marked changes in the body's utilization of ingested food, changes in the direction of increasing stores of energy as though to fortify against future food shortages.

Effects of Overfeeding on Energy Expenditure

A final source of variation in energy expenditures is that produced by overfeeding, one of the most interesting and controversial topics in obesity research. Two kinds of metabolic responses to food ingestion have been studied, responses to individual loads of food and responses to prolonged overfeeding. The former, known as the "thermic effect", is the increase in metabolic rate and heat loss (as opposed to storage of calories) that immediately follows food ingestion. The thermic effect (formerly believed to be specific to protein foods and known as "specific dynamic action") is universal, but there is some evidence that it is greater in lean subjects. Pittet, Chappuis, Acheson, DeTechtermann, and Jequier (1976) found that obese subjects showed a smaller increase in metabolic rate, smaller heat loss, and lower body temperature after a glucose load than controls. Kaplan and Leveille (1974) reported that lean subjects showed metabolic increases of 19.6% and 25.3% at 1 and 2 hr after ingesting 800 calories of protein, compared to increases of 3% and 9% by obese subjects. These findings are in disagreement

with negative findings on obese-normal differences by Strang and McCluggage (1931).

More germane to the question of long-term regulation of bodyweight are studies of prolonged overfeeding. A number of studies have demonstrated that when normal weight subjects overeat for relatively long periods of time (*i.e.*, a minimum of two weeks) the amounts of weight gained fall far short of those predicted if excess calories were stored as fat (Ashworth, Creedy, Hunt, Mahon, and Newland, 1962; Mann, Teel, Hayes, McNally, and Bruno, 1955; Miller and Mumford, 1967; Miller, Mumford, and Stock, 1967; Sims and Horton, 1968). For example, as noted by Garrow (1974), the first four subjects studied by Sims and Horton ate two or three times their normal intake for three to five months but increased their weight only 10 to 12%. During the second half of a 20-day period of overfeeding, subjects of Mann *et al.* (1955) gained only a quarter of the expected weight. Miller, Mumford, and Stock (1967) reported that the thermogenic effect (disposal of excess calories) was dependent on moderate levels of exercise; however, after recalculation of their data, Garrow (1974) concluded that increases in resting metabolic rate (31 to 58%) were sufficient to account for half the "missing" calories; the addition of exercise and its effects on metabolism following mealtimes appeared to account for the remainder.

Alterations in metabolic rate sufficient to dispose of a portion of excess calories do not necessarily require enormous increases in food intake, although the effects seem to be most pronounced under those conditions. In experiments conducted upon himself in 1922, Gulick (see Garrow, 1974) found that during a time period (41 days) in which he ate an average of 2744 calories per day, his expenditure averaged 2800 calories per day and he lost one pound. During a later period (73 days), in which he ate 3545 calories per day, and reported that his activity level was virtually identical to that in the former period, he expended

3300 calories per day, gaining only six pounds. In other words an increase of 800 calories in daily intake was met by an increase in expenditure of 500 calories, nearly offsetting the effects of the extra food.

When one considers these findings in conjunction with the fact that lean subjects made experimentally obese (with great difficulty in most cases) return rapidly and spontaneously to normal weight at the cessation of the forced feeding, it becomes clear that overeating alone is not sufficient to cause most people to become or to remain obese.

The few overfeeding experiments that have used obese subjects suggest that they do not dispose as readily of excess calories. Obese women studied by Passmore, Strong, Swindells, and El Din (1963) gained about twice as much weight as thin men (Passmore, Meiklejohn, Dewar, and Thow, 1955) for a given calorie excess consumed over nine days. Mahler (1972) found that during three weeks on 150% of baseline intake, obese subjects gained 12 pounds, while lean ones gained only four.

These results could indicate that lesser ability to dispose of excess calories was a cause of overweight in the obese sample. However, recalling that metabolic rate is depressed during dieting, it may be a consequence of recent eating histories. An adaptive mechanism, which acts to conserve calories in the face of shortages, might also operate to reduce waste of energy once refeeding is begun, *i.e.*, to encourage energy storage. Possibly this bias could persist. It would be adaptive, for example, under conditions of uncertain food supplies to expend energy freely in the service of other needs so long as food was abundant, but to reduce expenditure when food-searching behaviors were unsuccessful, and to store fat upon refeeding. The problem of obesity could be due to irreversibility of such mechanisms. Thus, overfeeding may not lead to rapid weight gain *except* in individuals who have experienced semistarvation, calling into play a survival mechanism unsuited to modern society.

In any event, these findings begin to make it clear how people may be overweight without eating more than their lean peers and quite apart from differences in activity level. The implications for behavioral treatment include the need to attend to individual differences; to understand that treatment failures may not reflect noncompliance or cheating; consideration of the pattern of dieting least likely to produce unwanted metabolic effects; and attention to the need for special support during periods of diet rebound in which patients may become understandably demoralized. Specific suggestions might include increases in activity during "re-feeding" to promote thermogenesis, and the use of only brief periods of caloric restriction with intermittent return to normal eating. It would be gratifying to see serial measurements of metabolic rate included in treatment studies, as an additional parameter of treatment outcome. Although Bellack (1977) argued that weight loss of less than one pound per week is not clinically significant, it may be preferable in the long run.

Unless therapist and patient anticipate the problems that can occur with dieting, many negative consequences may ensue (dropout, triggering of overeating, depression). Even with support and understanding, the process of continuing weight loss may be so difficult for some patients as to be effectively impossible, a conclusion that may seem pessimistic but which is certainly borne out by the data.

The Effects of Prolonged Caloric Restriction on Hunger and Eating Behavior

Four generalizations can be made about the effect of prolonged caloric restriction on hunger and feeding behavior:

1. Hunger is a direct function of the number of calories eaten per unit of time during the period of caloric restriction.
2. Hunger is an inverse function of the predictability or regularity of the periods of access to food during the period of caloric restriction.

3. The ability to compensate for change in caloric density of food is impaired by very restrictive regimes.
4. The ability to overcome the effects of innate palatability factors on regulatory eating is impaired and the tendency to acquire aversions to and preferences for discriminable sensory qualities of food is enhanced by very restrictive regimes.

Hunger and Level of Caloric Restriction

Generalization 1 above is paradoxical and runs counter to common sense, but less so when one considers that it is *prolonged* periods of caloric restriction that are being discussed, *i.e.*, fasts, diets, "deprivation schedules" that are in effect for many days or weeks. A comparison of studies by Collier and his associates (Collier *et al.*, 1972; Collier, Hirsch, and Kanarek, 1977), and Hamilton (1969) demonstrates well this relationship between hunger and level of caloric restriction. Collier and his associates have shown that rats at bodyweights no more than 6% below free-feeding values will work harder to overcome "constraints" on food availability than will animals 20% or 30% below their free-feeding body weights. Hamilton reported an inverse relationship ($r = -0.91$) between the length of a total fast (2 to 10 days) and the amount of food eaten on the first day of free access to food. Lindner and Blackburn (1976) reported that among their fasting obese patients, hunger reactions "invariably" subsided after a few days. Other studies of fasting obese people (Bloom, 1959; Drenick, Swendseid, Bland, and Tuttle, 1964), reported the same phenomenon. It may be that these patients "denied" their hunger for fear that they would not be allowed to continue fasting; however, Kollar *et al.* (1964) found the same absence of hunger with fasting "healthy, normal weight men" who "all agreed that they were most aware of hunger during the first 24 hours of fasting and seldom noticed it after 48 hours" (p. 115).

The phenomenon of decreased hunger and activity level while fasting is paradoxical and would be *behaviorally* unadaptive in most cases, in the sense that a starving animal might find food somewhere else if hunger "drove" it to search elsewhere. Studies of "semi-" starving human subjects have not found an absence of hunger. Keys *et al.*'s (1950) subjects—conscientious objectors who had volunteered to lose 25% of their bodyweight—were given an average of 1700 calories per day and yet complained bitterly about hunger and thought about, dreamed of, and became totally obsessed with food. Apfelbaum (1976) reported that in dieting obese women, hunger was much less prominent on 220 calories a day than on 1000 to 2000 calories a day. One way of understanding this phenomenon is to consider the stimulating effect of the presence of food on appetite. Powley (1977) provided a compelling documentation of the many metabolic changes that constitute the anticipatory or cephalic phase of digestion and which tend to drive appetite. He argued that it is the exaggeration of these responses in VMH lesioned animals that accounts for hyperphagia and hyperlipogenesis mediated by excess calories.

The greater suppression of hunger on more restrictive regimens may additionally be related to a general activational system (Stricker, 1978; Wolgin and Teitelbaum, 1978) which is closely tied to feeding; both feeding and changes in general activation levels depend on an intact lateral hypothalamus, and each represents a mechanism for adapting to the existing food supply. When food is relatively plentiful, feeding predominates; as food becomes more scarce, a lowering of general activation represents an increasingly important means of protecting endangered fuel supplies.

Stricker (1978) described this activation system as "a central excitatory neural system that is not restricted to feeding but participates in its control by mediating components of arousal and motor function that are common to all voluntary behaviors" (p. 1012). Wolgin and

Teitelbaum (1978) wrote that "feeding (and perhaps all motivated behavior) depends not only upon internally generated error signals but also upon the ability of sensory stimuli to energize appropriate behavior reactions" (p. 496).

The increased incidence of depression sometimes reported during dieting (Stunkard and Rush, 1974) would appear related to this excitatory system; two of the primary symptoms of depression are loss of appetite (weight loss) and "retardation" (Noble and Lader, 1972) of motor activity. Feeding may be an adaptive counterresponse to depression: "the principal effect on the brain of feeding (and of any other motivated behavior) may be to restore central catecholaminergic activity to some optimal level" (p. 1012, Stricker, 1978). Polivy and Herman (1976) showed that when subjects high on restraint become depressed, they *gained* weight. They speculated that "emotions will disrupt the chronic self-control of the restrained eater, increasing consumption" (p. 330).

A purely behavioral analysis of greater hunger on less restrictive regimens was provided by Staddon and Simmelhag (1971). They observed the frequency of 16 activities or responses by pigeons at 80% of free-feeding bodyweights during the interval between food reinforcements delivered on response-dependent and response-independent schedules. They noted that on both kinds of schedules "the presentation of food at a predictable time resulted, after training, in the regular occurrence of a food-related behavior in anticipation of food delivery, quite independently of the demand characteristic of the situation (*i.e.*, the reinforcement schedule)" (p. 15). The three most common of these food-related behaviors involved orienting the head and body toward the food magazine, pecking movements directed toward "some point on the magazine wall", and pecking movements directed toward the key. It stands to reason that the more food per unit of time that is delivered during the period of caloric restriction, the more often food-related behavior will be displayed. If "food-related behavior"

can be equated with "hunger", then the more frequent the food presentations, the greater the hunger.

An implication of these findings is that, on the average, behavioral programs that involve moderate restriction of calories will lead to greater levels of hunger than more extreme diets. Also, as noted earlier, weight loss is apt to slow nearly to a stop after several months. More restrictive diets, such as the protein-sparing fast, may produce less discomfort while sustaining much higher rates of weight loss, but the metabolic depression will be considerably more severe, refeeding will present a difficult problem, and there is a greater risk to health.

Lindner and Blackburn (1976) reported that the combination of a protein-sparing fast with subsequent behavioral training led to large losses, which were well maintained by those who completed the behavior therapy portion of the treatment. The good maintenance of weight loss in this program is in some ways surprising in view of the low energy expenditures and hyperlipogenesis induced by fasts. This may be attributable to the inclusion of a vigorous exercise program during refeeding. On the other hand, there may be a selective bias operating to produce the results; that is, patients who experienced extreme hunger and rapid weight gain on refeeding may have been the most likely to drop out of the behavioral treatment component that followed the fast.

Obviously, despite the generalizations that can be drawn from research on feeding, there are pronounced individual differences in response to dieting. A predictive schema for these differences is discussed in a later section.

Hunger and Predictability or Regularity of Food

Studies by Baker (1955), Lawrence and Mason (1955), Mandler (1957), Gross (1968), and Weinstock (1972) all support the generalization that rats on irregular schedules behave as if they were operating on a higher level of drive than rats regularly fed and kept at

comparable body weights. The more predictable food presentations are, the more time the animal has to engage in other activities; the most adaptive, *i.e.*, energy-saving, activity would be sleep, because it is during sleep that energy expenditure is lowest. Increases in sleeping in fasting subjects have been described (Kollar *et al.*, 1964).

Konorski (1967) postulated a "hunger anti-drive center" in the limbic system that "suppresses hunger on the basis of conditionally acquired information to the effect that in a given situation food is unavailable" (p. 326-327).

Staddon and Simmelhag (1971) spoke of "animals' need to budget time effectively . . . (A) time or stimulus, reliably associated with the absence of a given reinforcer provides information just as useful as a time perfectly correlated with the delivery of that reinforcer, since it permits the animal to attend to present and future needs other than the one associated with the absent reinforcers".

When there is a certainty that food is temporarily unavailable, hunger is suppressed or suppressible. This effect is illustrated by Konorski's (1967) discussion of "negative conditioned stimuli", stimuli which signal that no food will be available. When a negative conditioned stimulus is paired with a positive conditioned stimulus (*i.e.*, a signal that food will be available), the effect of the latter on conditioned salivary responses is decreased. Suppression of hunger "is manifested by the striking fact that a voracious animal who displays a strong motor excitement during the intertrial intervals due to the hunger drive . . . , momentarily calms down when the . . . no-food (stimulus) is presented" (p. 325).

The stabilization of eating patterns has been among the most prominent features of behavioral programs. Indeed, the establishment of temporal control was set forth by Ferster, Nurnberger, and Levitt (1962) as a central goal in their paper, which launched research on behavior therapy for eating disorders. The advantages of predictability of feeding times

in the "management" of hunger appears to have been supported by subsequent research and may well be among the most important accomplishments of treatment, one that almost surely enhances weight maintenance, if not rapid loss.

Compensation for Changes in Caloric Density during Periods of Caloric Restriction

Jacobs and Sharma (1969) noted that studies that show animals capable of making appropriate volumetric adjustments in food intake when diet is diluted or enriched, used subjects on *ad lib* schedules, while studies with other results used subjects on deprivation schedules. Booth (1972) showed that as the time of access to food is increased, the ability to make appropriate adjustments increases. He said that rats on more restrictive schedules might have refused to eat food of low-calorie density because "the caloric deficit has been so extreme for so long that behavioral depression arising from debilitation had set in or at least that the capacity to adapt to high bulk diet had been reduced by loss of time" (p. 878).

This "behavioral depression" may be the result of decreased catecholaminergic activity in the brainstem activational system (Stricker, 1978). The loss of capacity to handle high bulk diets suggests that what Collier *et al.* (1977) called the "cost of use"—i.e., the energy required to ingest, digest, and absorb the caloric components of the diet—are too high for an animal whose energy supplies are depleted.

Herman, Polivy, and their associates have investigated the effects of dietary restraint, i.e., chronic dieting, in humans. Their general finding was that dieters, whether obese or not, did not respond to "internal cues" such as anxiety (Herman and Polivy, 1975) and calories ingested as "preloads" (Herman and Mack, 1975; Hibscher and Herman, 1977). Instead, consumption of a high caloric preload markedly increased food ingestion (Schachter, 1968).

Overall, the pattern and significance levels of results in this study accorded well with the comparable previous research (i.e., by Schachter and associates) categorizing subjects on the basis of degree of overweight. The notion that relative deprivation rather than obesity per se may be the most viable conceptualization underlying individual differences in eating behavior appears to have considerable merit. Furthermore, the restrained-unrestrained distinction becomes more powerful when obese subjects are eliminated from the data. (p. 671, Hibscher and Herman, 1977).

Herman and Polivy attributed the failure of restrained eaters to respond appropriately to manipulation of internal state to voluntary constraints on eating, which were disrupted, (so that eating behavior was "disinhibited") under specific conditions such as when unscheduled eating occurred. Studies of restrained eaters using conditioned salivation as a measure of appetite suggest that the phenomena described extend beyond voluntary or cognitive processes.

In a series of studies, Wooley and associates found that lean subjects displayed sensitive differentiation of salivary response to food as a function of the caloric value of preloads, discriminating differences as small as 150 calories (O. W. Wooley, Wooley, and Williams, 1978). By contrast, obese male inmates of a correctional institute (probably unrestrained eaters) showed equal and *large* salivary responses after preloads differing by as much as 450 calories (O. W. Wooley, Wooley, and Woods, 1975). In these subjects, there was an evident failure of satiety. Study of restrained eaters again showed failure to differentiate levels of caloric intake but appetitive responses were consistently *low*. These data imply that inhibition of appetite is accompanied by dysfunction of caloric sensing mechanisms.

Note that failure to respond to internal signals in obesity is of two types. In one, there is a failure of satiety, in the other an unstable

inhibition of appetite. Both may be responses to sub-optimal food intakes. In the former case, some factor such as rapid removal of nutrients from the system may prevent normal satiety and produce overeating until a weight sufficient to slow hyperlipogenesis is achieved (Booth, 1976; Powley, 1977; Stricker, 1978). In the latter case, the failure of regulation appears to be a direct effect of caloric restriction that may correct itself on refeeding unless, as seems likely, this brings with it a failure of satiety. Study of these forms of abnormal responses to calories might prove to have some value in predicting outcome of treatment. Apart from eating, no behavioral "cure" for either is readily apparent.

Sensory Qualities of Food and Caloric Restriction

The effects of the sensory qualities of food on eating behavior are of two types, innate and acquired. Peck (1978) has shown that the body weight an animal will "defend" is a direct function of diet palatability. Feeding in cats recovering from hypothalamic lesions that impair endogenous activation appears earlier in the recovery period if innately palatable foods are offered (Wolgin and Teitelbaum, 1978).

Booth (1972) showed that the ability of hungry rats to make appropriate volumetric adjustments to caloric dilution and enrichments is also a direct function of innate palatability factors. Of greater interest here is his finding that, if foods of high and low caloric density are masked with discriminably different odors, rats at high levels of caloric restriction will learn to eat more of the high calorie food than of the low calorie food. When the foods are offered repeatedly—one one day, one the next—the rats eat 83 Kcal per day of the high calorie food, 30 Kcal per day of the low calorie food. When the odors were offered simultaneously (for 1 hr) incorporated in a pair of samples of medium caloric diet, the intake of the high-density-odor-sample was 10.9 g; of

the low-density-odor sample, 6.1 g. Thus, aversions and preferences can develop under high levels of caloric restriction. These acquired palatability factors probably work in the same way that innate ones work—*i.e.*, by differentially energizing the appropriate behavior reaction, which in this case, is eating. Rozin and Kalat (1971) and Revusky and Garcia (1970) have given learning theory explanations of how these aversions and preferences might be learned.

The heightening of the influence of innate and acquired reactions to the sensory qualities of food by caloric restriction probably accounts for whatever evidence there is for "externality" (Rodin, 1976) or "finickiness" in the obese. Booth's results suggest that these characteristics may be related to dieting history, rather than being a primary defect.

These findings may support the general utility of stimulus control procedures that limit availability of palatable foods, at least during periods of weight loss. On the other hand, they suggest that perhaps these procedures should be phased out during refeeding, so that high palatability does not continue to be paired with eating at the high levels of hunger that typically lead to diet breaking, thereby perpetuating learned preferences for rich foods.

A SCHEMA FOR INDIVIDUAL DIFFERENCES IN RESPONSE TO TREATMENT

In contrast to earlier views positing satiety receptors sensitive to specific nutrients, recent formulations have stressed the total availability of metabolic fuels in determining hunger (see Booth, 1976). Availability of lipids appears to shift as a function of bodyweight (possibly as a result of changes in fat cell size), and hyperlipogenesis has been observed in animals with VMH lesions, experimentally produced hyperinsulinism, genetic obesity, and following starvation (Booth, 1976; Friedman and Stricker, 1976; Powley, 1977; Tepperman and

Tepperman, 1964). Stricker (1978) proposed that the obese be divided into two groups—those with *primary obesity*, in which hyperlipogenesis leads to rapid removal of nutrients from the bloodstream with consequent failure of satiety, and those with *primary hyperphagia*, in which overeating unrelated to metabolic abnormalities is a cause of obesity. Although in both instances, Stricker implied that overeating (of whatever origin) was the sole cause of obesity, it is important to recall that obesity may occur even in the absence of intake in excess of population norms.

Stricker (1978) speculated that in humans, genetic factors and postnatal feeding may be the causes of hyperlipogenesis. On the basis of existing evidence, long-term stringent dieting may be added to this list as an additional possible cause. Note that hyperlipogenic disorders could have the effect of a "set point" mechanism, if hunger is curbed only by weight gain and a repletion of adipose cells that offsets the tendency to hyperlipogenesis. Although the mechanism for failure of satiety is controversial, the fact of its existence in some individuals is less so. Some patients complain of intense hunger while dieting, while others rarely report having sensations they label as hunger, and categorization on this dimension would appear to be useful.

Primary hyperphagia Stricker hypothesizes to be due to attempts to optimize general activation level by taking advantage of the stimulating properties of the sensory qualities of food and the sedative effects of food ingestion. He suggests that in the case where food is used to decrease arousal, sedative anorexigenic drugs such as fenfluramine may be of benefit.

Assuming that the dimensions of primary hyperphagia and level of arousal are orthogonal, one arrives at four categories of obese patients, as shown in Table 1. This system represents an undoubtedly oversimplified schema, if only because the dimensions involved are probably not truly dichotomous. However, it leads to testable hypotheses. The patient with

primary obesity and low arousal will be motivated to eat both to quiet hunger and to increase arousal level. This is likely to lead to a pattern of both large and frequent feedings and probably to a considerable degree of overweight. Although both moderate dieting and fasting will increase hunger, the energization produced by hunger will not be experienced adversely, so that the net effect will be chronic moderate discomfort with the motivation for diet breaking present almost equally at all times. In contrast, the patient with metabolically determined hunger and high general arousal level will probably tend toward a pattern of extremely large meals, eaten for sedative effect to facilitate relaxation. Although one might argue that arousal would drive such patients to frequent eating, it seems more likely that the relationship between hunger and arousal is curvilinear and that at extremely high levels of arousal associated with external pressures, appetite would be suppressed. Instead, eating might be initiated to speed decreases in arousal level attendant on removal from stressful situations. For this type of patient, diets and fasts will produce not only hunger but irritability approaching frenzy, with a great likelihood of diet-breaking by the end of the day.

Patients with primary hyperphagia will not report particularly high levels of hunger, either at maintenance intake or while dieting. Those with low arousal who eat for stimulation will show a pattern of frequent snacking, especially during the parts of the day when external demands are greatest, typically morning and afternoon. Moderate diets will produce lethargy, boredom, and possibly depression. However, these patients may be the ones who experience the ketosis accompanying fasting as euphoria. Again, a tendency toward high arousal is apt to result in larger meals sufficient to have the desired sedative effect, and dieting and fasting will be likely to produce irritability, which may be tolerated until late evening at which point fear of or actual insomnia may become the impetus for night eating.

By this schema, prognosis would of course be worst for patients with primary obesity, especially those in whom it is coupled with high arousal. It is difficult to say which form of primary hyperphagia would be most intractable. However, because the stimulating potential of food can be somewhat diminished by controlling its availability and palatability, and because suitable behavioral alternatives to eating can be found for those with some freedom to do as they choose, the patients with low arousal seem most likely to profit from existing behavioral treatment modalities. An exception to this rule might be patients trapped in crippling economic and social situations. More appropriate techniques emphasizing stress management and relaxation training might be devised for the high arousal group in which eating has become an anxiety relief response.

Location of patients on the arousal dimension could thus help determine choice of treatment techniques. Whether or not the patient has "primary obesity" is likely to be the major determinant of therapeutic outcome, but may be difficult to ascertain. Detailed history of past weight losses and gains and the eating patterns associated with these, along with careful monitoring of hunger, may help to generate testable predictions. Although speculative, this classificatory system could prove heuristic and has the appealing feature of simultaneously considering physiologic and emotional determinants of eating.

Self-Esteem and Obesity

Although poor self-esteem undoubtedly constitutes a problem for many people, in the case of obesity it deserves explicit and special consideration. Obese people, like the physically handicapped, wear their "problem" for all to see at all times, and yet unlike those groups are held responsible for their condition. They can scarcely avoid interactions with others in which weight and eating behavior are an explicit topic of discussion, concern, and criticism,

or a covert determinant of others' evaluations. An appreciation of the intensity of cultural prejudice against overweight is found in reviews of studies of the attitudes of children toward overweight peers, which show that from age 5 yr onward, endomorphic children are judged negatively on nearly every dimension studied (Lerner, 1969; Learner and Gelbert, 1969; Lerner and Korn, 1972; Staffieri, 1967; Staffieri, 1972). Chubby children are, for example, judged to be ugly, stupid, mean, sloppy, lazy, dishonest, and frequently teased. Children indicate a desire to maintain greater personal distance from overweight than from average or thin children (Lerner, 1973). And samples of adults as well as children, including groups of health professionals, rate obese children as less likeable than children with a variety of handicaps, disfigurements, and deformities (Goodman, Dornbusch, Richardson, and Hastorf, 1963; Maddox, Beck, and Liederman, 1968; Mathews and Westie, 1966; Richardson, Goodman, Hastorf, and Dornbusch, 1961). In a recent study of preschoolers, ranging in age from 2 to 5 yr, Dyrenforth, Freeman, and Wooley (Note 6) found that only three of 56 subjects expressed preference for a fat *versus* a thin rag doll. Three of the five obese children studied judged themselves to be "more like" the fat than the thin doll, but all three liked the thin doll better. These results show a surprising evaluative bias at a very early age.

Negative attitudes toward obesity are, if anything, intensified during adolescence, especially for females. Few adolescent girls feel they are thin enough and very high proportions attempt to diet (Clifford, 1971; Crisp, 1977; Douty, Moore, and Hartford, 1974; Halmi, Goldberg, and Cunningham, 1977; Jourard and Secord, 1955). Not surprisingly, obese adolescent girls show personality test responses comparable to those shown by ethnic and racial minorities in the 1960s (Monello and Mayer, 1963).

The effects of prejudice against fat are clearly seen in the modern obsession with attaining a degree of slenderness well below that required

for good health. Many applicants to weight clinics are minimally overweight or even underweight. Boskind-Lodahl's discovery of over 100 undergraduates at Cornell University manifesting a syndrome of binge-eating followed by vomiting called attention to the epidemic proportions of this disorder and many authorities believe *anorexia nervosa* to be steeply on the rise (Boskind-Lodahl, 1976; Boskind-Lodahl and Sirlin, 1977).

The dread of fatness is clearly expressed by these patients, but those who have failed, who are fat, may not be so open about feared or actual contempt of others. Usually they believe that the only cure for their problems would be to reduce; to discuss these problems is to complain about troubles they have brought on themselves, a behavior they may feel will not be tolerated. Identification with cultural values is so strong that overweight women frequently defend their critics and consider themselves lucky, for example, to have husbands, families, or friends at all. Such a patient who had engaged in weight-reduction attempts for nearly 20 yr never hinted at any feeling other than optimism, gratitude to her therapists and her family for their patience, and enthusiasm for the new opportunity provided by a behavioral treatment program. Some years later she showed the authors a journal kept during this same period of time. It chronicled her struggle with self-hate, anguish, and rage at the responses of others toward her. After her first behavioral session she wrote:

"A mountain of fat, great volumes of larded flab, covered with over-stretched and sagging skin. Buried deep within is a bit of Joy slowly drowning in the emollient folds. Hope, too, is suffering the death-agony, going down for the third time in the enormous body of fat. There is only one cure for the indignities. . . . Marking time, counting calories, recording activities, avoiding people, hiding misery, extravagantly wasting my life."

The point to be made is that whether or not they are expressed, such feelings typically run very deep in overweight people and, when one considers the fact that treatments rather rarely succeed in helping the obese to achieve or maintain normal weights, to omit this aspect of their problem from plans for treatment is in an important sense to ignore their real needs.

In the recent past, some feminist writers have addressed the problems surrounding obesity and its treatment. (See O. W. Wooley, Wooley and Dyrenforth, *in press* for a review of these writings.) Aldebaran (1975) stated succinctly: "The secret rage and humiliation of fat women could burn this country to the ground." Treatments that do not address this problem are criticized. "You see your role as therapist to help . . . us stay on reducing diets. Reducing programs are . . . ineffective at producing weight loss and this . . . failure is well-known in the medical literature. . . . The . . . failure of reducing diets is fat people's collective experience, and therapy tells us to ignore it." (Aldebaran, 1977).

A point made by these writers is that given the difficulty, perhaps in some cases the impossibility, of achieving slenderness, the obese should carefully consider the choice to persist in attempts to diet.

Finally, fat people are beginning to say we have a right to eat as much as we want . . . and to be fat. This is a powerful assertion for a fat person to make in light of the persecution of fat. However, the right to be fat is only a theoretical right for, rightful or not, we are fat and our efforts to change ourselves almost always fail. . . . One problem with the "right" to be fat is that it requires fat people to "choose" to remain fat in order to justify no longer dieting. Because of our conditioning we have to develop a way to move from self-hatred to self-love. (Aldebaran, 1975, p. 6)

It would seem that there is a great deal to be gained, and little to be lost, in therapists help-

ing obese patients to achieve greater self-acceptance and to make a reasoned choice in which their own needs and feelings are given at least as much importance as the attitudes of others. Sometimes behavioral treatment programs have included elements designed to increase shame over obesity, as by teaching patients to rehearse the ideas that fat is ugly and slenderness attractive. It seems doubtful that lack of shame over eating or appearance can be a cause of obesity; on the contrary, shame frequently leads to perpetuation of such self-destructive attempts at self-help as solitary eating, avoidance of public gatherings, and intermittent self-starvation. There is no evidence that the techniques that foster shame show any advantage over other techniques and even if weight losses were found to be greater, it could still be argued that the overall benefit to the patient is diminished.

Instead, it would seem to be consistent with the philosophy and tradition of behavior therapies to acquaint patients with the facts about obesity, including the lack of evidence that the obese eat more than others, the effects of dieting on metabolism, and the modest outcomes of most treatment, and to engage them in a process of goal-setting based on these facts. Although therapists tend to be reluctant to discourage patients, the *facts* of their own experience will have discouraged them or given them hope. If the experience of dieting has been one of constant failure, despite enormous effort, they will be relieved to have their experience confirmed and understood, and some may be better able to withstand the difficulties knowing that they are not exclusively attributable to their own failings. Some may choose to give up dieting and work on minimizing the negative consequences of being obese. Others will recognize that weight loss is, in fact, relatively easy for them and worth the effort. It has been our experience that few patients embrace these pieces of information as "rationalizations"; the concept of rationalization, of course, implies a self-serving denial of the

"truth", which in the case of obesity is not known. Put otherwise, patients typically become interested in discovering what holds true for them. What is their weight maintenance calorie level? How rapidly and for how long will they lose at a given level? How unpleasant is it? What, exactly, are the benefits to be expected from weight loss?

Replacing mythology with fact and counter-acting the diverse effects of social prejudice should be among the goals of comprehensive treatment for the obese. This means that efficacy of procedures in promoting weight loss must be balanced against the extent to which they further erode dignity and self-esteem. With the lifting of the stigma of emotional illness formerly associated with obesity and constructive involvement in the great practical difficulties involved in dieting, behavior therapy may be said to have improved the treatment climate for obese patients. However, it is appropriate now to reevaluate certain treatment strategies, for example those that call negative attention to obese patients or to their eating behavior and to question further treatments that infantilize patients through the application of exclusively external controls and contingencies.

Viewed positively, there are many previously ignored areas ripe for creative applications of learning theory in obesity treatment. These include development of techniques to improve body image, increasing socialization, and training in interactional skills aimed at discouraging others from behaving in belittling ways. Sexual dysfunction secondary to poor body image should be a matter of concern, as should helping patients to recognize and combat educational or vocational discrimination by weight. As in other forms of behavioral treatment, involvement of the patient in selecting treatment goals would seem desirable.

SUMMARY

Behavioral treatments of obesity have produced weight losses as good or better than

most previous forms of outpatient treatment, with clearly superior maintenance of results. However, despite the ingenuity and technical competence applied to this problem, permanent losses still tend to be small. Reasons for this include the difficulties in reducing the essentially normal food intakes of many patients, the marked decreases in energy expenditure associated with dieting, and hyperlipogenesis during the refeeding period.

Hunger during caloric restriction is inversely proportional to the degree of restriction, and inversely proportional to the predictability of food availability. Hunger can be suppressed, but at the cost of suppression of activity. During deprivation, caloric sensing mechanisms are impaired and there is a heightened responsiveness to diet palatability, which seems partially dependent on innate mechanisms and partly due to preference/aversion learning.

It appears that for some patients, failure of satiety has a physiological basis related to constitutional factors and possibly to the long-term effects of stringent dieting on metabolism. Classification of patients into those with and without excessive hunger and characterized by high and low general arousal levels allows predictions of response to treatment and is consistent with new formulations emphasizing the interrelationship of activation of feeding and other behaviors.

In view of the intractability of many cases of obesity, it is advantageous to define more broadly the goals of treatment so as to deal with the severe problems in self-esteem stemming from social prejudice and repeated failure. Behavior therapy has made an important contribution, but it needs to go the extra mile.

REFERENCE NOTES

1. Meyer, J., Stunkard, A. J., and Coll, M. Eating in public places: *There is no "obese eating style," it's where you eat that matters.* Paper presented at the meeting of the Association for the Advancement of Behavior Therapy, Atlanta, December, 1977.
2. Wooley, O. W., Wooley, S. C., and Williams, B. S. *Effect of prior protein intake on appetite for palatable food.* Paper presented to the meeting of the American Psychological Association, San Francisco, September, 1977.
3. Brownell, K. D., Albaum, J. M., and Stunkard, A. J. *Evaluation and modification of activity patterns in the natural environment.* Unpublished manuscript, Department of Psychiatry, University of Pennsylvania, 1978.
4. Stunkard, A. J. Personal communication, February, 1975.
5. Wooley, O. W. and Wooley, S. C. *Out of touch with the body: Internal signals, awareness, and obesity.* Paper presented at the meeting of the American Psychological Association, Washington, D.C., September, 1976.
6. Dyrenforth, S. R., Freeman, D. G., and Wooley, S. C. *Self-esteem, body type preference, and sociometric ratings of peers in pre-school children.* Unpublished manuscript, Department of Psychiatry, University of Cincinnati College of Medicine, 1978.

Table 1

	Primary Obesity		Primary Hyperphagia	
	Low Arousal	High Arousal	Low Arousal	High Arousal
<i>Maintenance Eating Patterns</i>				
Meal size	large	very large	small	large
Meal frequencies	high	low	very high	average
<i>Response to Moderate Restriction</i>				
Hunger level	high	high	low	low
Affective change	well-being	irritability	depression, boredom	irritability, insomnia
Type of diet breaking	unpatterned	evening eating	snacking early in day	night, weekend eating
<i>Response to Fasting</i>				
Hunger level	very high	very high	low	low
Affective change	energetic	extreme agitation	euphoric	nervous

REFERENCES

- Aldebaran. Uptight and hungry: The contradiction in the psychology of fat. *RT: A Journal of Radical Therapy*, October, 1975, 5-6.
- Alderbaran. Fat liberation—A luxury? An open letter to radical (and other) therapists. (Quotes taken from an unpublished manuscript supplied by the author; this article subsequently appeared in *State and Mind*, 1977, 5, 35.)
- Apfelbaum, M. The effects of very restrictive high protein diets. *Clinics in Endocrinology and Metabolism*, 1976, 5, 417-430.
- Apfelbaum, M., Bostsarron, J., and Lacatis, D. Effect of caloric restriction and excessive caloric intake on energy expenditure. *American Journal of Clinical Nutrition*, 1971, 24, 1405-1409.
- Ashworth, N., Creedy, S., Hunt, J., Mahon, S., and Newland, P. Effect of nightly supplements on food intake in man. *Lancet*, 1962, 2, 685.
- Baker, R. A. The effects of repeated deprivation experience on feeding behavior. *Journal of Comparative and Physiological Psychology*, 1955, 48, 37.
- Bellack, A. S. Behavioral treatment for obesity: Appraisal and recommendations. In M. Hersen, R. M. Eisler, and P. M. Miller (Eds), *Progress in behavior modification*. New York: Academic Press, 1977. Pp. 1-38.
- Bellack, A. S., Rozensky, R. H., and Schwartz, J. A comparison of two forms of self-monitoring in a behavior weight reduction program. *Behavior Therapy*, 1974, 5, 523.
- Bloom, W. L. Fasting as an introduction to the treatment of obesity. *Metabolism*, 1959, 8, 214.
- Bloom, W. L. and Eidex, M. F. Inactivity as a major factor in adult obesity. *Metabolism*, 1967, 16, 679.
- Bogdonoff, M. D., Bogdonoff, M. M., and Wolf, S. Q. Studies on salivary function in man: Variation in secretory rates as part of the individual's adaptive pattern. *Journal of Psychosomatic Research*, 1961, 5, 170.
- Booth, D. A. Approaches to feeding control. In T. Silverstone (Ed), *Appetite and food intake*. Berlin: Dahlem Konferenzen, 1976. Pp. 417.
- Booth, D. A. Caloric compensation in rats with continuous or intermittent access to food. *Physiology and Behavior*, 1972, 8, 891-899.
- Booth, D., Chase, A., and Campbell, A. Relative effectiveness of protein in the late stages of appetite suppression in man. *Physiology and Behavior*, 1970, 5, 1299.
- Boskind-Lodahl, M. Cinderella's stepsisters: A feminist perspective on anorexia nervosa and bulimia. *Signs: Journal of Women in Culture and Society*, 1976, 2, 341-356.
- Boskind-Lodahl, M. and Sirlin, J. The gorging purging syndrome. *Psychology Today*, 1977, 10, 50.
- Boyle, P. C., Storlien, H., and Keesey, R. E. Increased efficiency of food utilization following weight loss. *Physiology and Behavior*, 1978, 21, 261.
- Bray, G. Effect of caloric restriction on energy expenditure in obese patients. *Lancet*, 1969, 2, 397.
- Bullen, B. A., Reed, R. B., and Mayer, J. Physical activity of obese and non-obese adolescent girls, appraised by motion picture sampling. *American Journal of Clinical Nutrition*, 1964, 14, 211-223.
- Buskirk, E. R., Thompson, R. H., Lutwak, L., and Whedon, G. D. Energy balance of obese patients during weight reduction: Influence of diet restriction and exercise. *Annals of the New York Academy of Sciences*, 1963, 110, 918-940.
- Chiroco, A. M. and Stunkard, A. J. Physical activity and human obesity. *New England Journal of Medicine*, 1960, 263, 935-940.
- Clifford, E. Body satisfaction in adolescence. *Perceptual and Motor Skills*, 1971, 33, 119-125.
- Cohn, C. and Joseph, D. Changes in body composition attendant on force feeding. *American Journal of Physiology*, 1959, 196, 965.
- Coll, M., Meyer, A., and Stunkard, A. J. Obesity and food choices in public places. *Archives of General Psychiatry*, (in press).
- Collier, G., Hirsch, E., and Hamlin, P. H. The ecological determinants of reinforcement in the rat. *Physiology and Behavior*, 1972, 9, 705.
- Collier, G., Hirsch, E., and Kanarek, R. The operant revisited. In W. K. Honig and J. E. R. Staddon (Eds), *Handbook of operant behavior*. Englewood Cliffs, New Jersey: Prentice-Hall, 1977. Pp. 28-52.
- Crisp, A. H. Some psychobiological aspects of adolescent growth and their relevance for the fat/thin syndrome (anorexia nervosa). *International Journal of Obesity*, 1977, 1, 231.
- Douty, H. I., Moore, J. B., and Hartford, D. Body characteristics in relation to life adjustment, body-image and attitudes of college females. *Perceptual and Motor Skills*, 1974, 39, 499-521.
- Drenick, E. J. and Dennin, H. F. Energy expenditure in fasting men. *Journal of Laboratory and Clinical Medicine*, 1973, 81, 421-430.
- Drenick, E. J., Swendseid, M. E., Bland, M. E., and Tuttle, S. G. Prolonged starvation as treatment for severe obesity. *Journal of the American Medical Association*, 1964, 187, 100.
- Fabry, P., Fodor, J., Hejl, Z., Braun, T., and Zvolankova, K. The frequency of meals: Its relation to overweight hypercholesterolaemia and decreased glucose tolerance. *Lancet*, 1964, 2, 614-615.
- Ferster, C. B., Nurnbeger, J. I., and Levitt, E. B. The control of eating. *Journal of Mathematics*, 1962, 1, 87-109.
- Friedman, M. I. and Stricker, E. M. The physiological psychology of hunger: A physiological perspective. *Psychological Review*, 1976, 83, 409.
- Garrow, J. *Energy balance and obesity in man*. New York: American Elsevier, 1974.

- Goodman, N., Dornbusch, S. M., Richardson, S. A., and Hastorf, A. H. Variant reactions to physical disabilities. *American Sociological Review*, 1963, 28, 429-435.
- Greenfield, N. S. and Fellner, C. H. Resting level of physical activity in obese females (Guest Editorial). *American Journal of Clinical Nutrition*, 1969, 22, 1418-1419.
- Gross, L. P. *Scarcity, unpredictability and eating in rats*. Unpublished doctoral dissertation, Columbia University, 1968. (Cited by Jacobs and Sharma, 1969).
- Halmi, K. A., Goldberg, S. C., and Cunningham, S. Perceptual distortion of body image in adolescent girls: Distortion of body image in adolescence. *Psychological Medicine*, 1977, 7, 253-257.
- Hamilton, C. L. Problems of refeeding after starvation in the rat. *Annals of the New York Academy of Sciences*, 1969, 157, 1004.
- Hampton, M., Huenemann, R., Shapiro, L., and Mitchell, B. Calorie and nutrient intakes of teen-agers. *Journal of American Dietetic Association*, 1967, 50, 385.
- Hanley, T. The etiology of obesity. In I. M. Baird and A. N. Howard (Eds), *Obesity: medical and scientific aspects*. Edinburgh, 1969.
- Hejda, S. and Fabry, P. Frequency of food intake in relation to some parameters of the nutritional status. *Nutrition Dieta*, 1964, 6, 216.
- Herman, C. P. and Mack, D. Restrained and unrestrained eating. *Journal of Personality*, 1975, 43, 646-660.
- Herman, C. P. and Polivy, J. Anxiety, restraint, and eating behavior. *Journal of Abnormal Psychology*, 1975, 84, 666-672.
- Hibschler, J. A. and Herman, C. P. Obesity, dieting and the expression of 'obese' characteristics. *Journal of Comparative and Physiological Psychology*, 1977, 91, 374.
- Howard, A., Grant, A., Challand, G., Wraight, E., and Edwards, O. Thyroid metabolism in obese subjects after a very low calorie diet. In G. A. Bray (Ed), *Recent advances in obesity research*, Vol. 2. London: Newman, Ltd., 1978.
- Jacobs, H. L. and Sharma, K. N. Taste versus calories: Sensory and metabolic signals in the control of food intake. *Annals of the New York Academy of Sciences*, 1969, 157, 1084-1125.
- Jeffery, R. W., Wing, R. R., and Stunkard, A. J. Behavioral treatment of obesity: The state of the art 1976. *Behavior Therapy*, 1978, 9, 189-199.
- Johnson, M. L., Burke, B. S., and Mayer, J. Relative importance of inactivity and over-eating in the energy balance of obese high school girls. *American Journal of Clinical Nutrition*, 1956, 4, 37-44.
- Jourard, S. M. and Secord, P. R. Body-cathexis and the ideal female figure. *Journal of Abnormal and Social Psychology*, 1955, 50, 243-246.
- Kaplan, M. L. and Leveille, G. A. Calorigenic effect of a high protein meal in obese and nonobese subjects. *Federation Proceedings*, 1974, 33, 701.
- Keys, A., Brozek, J., Henschel, A., Mickelson, O., and Taylor, H. *The biology of human starvation*. Minneapolis: University of Minnesota Press, 1950.
- Kissileff, K., Jordan, H., and Levitz, L. Eating habits of obese and normal weight humans. In G. A. Bray (Ed), *Recent advances in obesity research*, Vol. 2. London: Newman, Ltd., 1978.
- Kleiber, M. *The fire of life: An introduction to animal energetics*. New York: Wiley & Sons, 1961.
- Kollar, E. J., Slater, G. R., Palmer, J. O., Docter, R. F., and Mandell, A. J. Measurement of stress in fasting man. *Archives of General Psychiatry*, 1964, 11, 113-125.
- Konorski, J. *Integrative activity of the brain*. Chicago: University of Chicago Press, 1967.
- Lawrence, D. H. and Mason, W. A. Food intake in the rat as a function of deprivation intervals and feeding rhythms. *Journal of Comparative and Physiological Psychology*, 1955, 48, 267.
- Leon, G. R. Current directions in the treatment of obesity. *Psychological Bulletin*, 1976, 83, 557-578.
- Lerner, R. M. The development of stereotyped expectancies of body build-behavior relations. *Child Development*, 1969, 40, 137-141.
- Lerner, R. M. The development of personal space schemata toward body build. *The Journal of Psychology*, 1973, 84, 229-235.
- Lerner, R. M. and Gelbert, E. Body build identification, preference, and aversion in children. *Developmental Psychology*, 1969, 5, 256-462.
- Lerner, R. M. and Korn, S. J. The development of body-build stereotypes in males. *Child Development*, 1972, 43, 908-920.
- Lindner, P. C. and Blackburn, G. L. Multidisciplinary approach to obesity utilizing fasting modified by protein-sparing therapy. *Obesity/Bariatric Medicine*, 1976, 5, 198.
- Maddox, G. L., Beck, K. W., and Liederman, V. R. Overweight as social deviance and disability. *Journal of Health and Social Behavior*, 1968, 9, 287-298.
- Mahler, R. The relationship between eating and obesity. *Acta Diabetologica Latina* (Suppl. 1), 1972, 449.
- Mahoney, M. J. The obese eating style: bites, beliefs, and behavior modification. *Addictive behaviors*, 1975, 1, 47-53. (a)
- Mahoney, M. J. Fat fiction. *Behavior Therapy*, 1975, 6, 416-418 (b)
- Mandler, J. M. Irregular maintenance schedules and drive. *Science*, 1957, 126, 505-507.
- Mann, G. V., Teel, K., Hayes, O., McNally, A., and Bruno, D. Exercise in the disposition of dietary calories: Regulation of serum lipoprotein and cholesterol in human subjects. *New England Journal of Medicine*, 1955, 253, 349.

- Mathews, V. and Westie, C. A preferred method for obtaining rankings: Reactions to physical handicaps. *American Sociological Review*, 1966, **31**, 851-854.
- Miller, D. S. and Mumford, P. Gluttony: An experimental study of overeating on low or high protein diets. *American Journal of Clinical Nutrition*, 1967, **20**, 1212.
- Miller, D., Mumford, P., and Stock, M. Gluttony: Thermogenesis in overeating man. *American Journal of Clinical Nutrition*, 1967, **20**, 1223-1229.
- Monello, L. F. and Mayer, J. Obese adolescent girls: An unrecognized "minority" group? *American Journal of Clinical Nutrition*, 1963, **13**, 35-39.
- Noble, P. and Lader, M. A physiological comparison of 'endogenous' and 'reactive' depression. *British Journal of Psychiatry*, 1972, **120**, 541-542.
- Passmore, R., Meiklejohn, A. P., Dewar, A. D., and Thow, R. K. An analysis of the gain in weight of overfed thin young men. *British Journal of Nutrition*, 1955, **9**, 27.
- Passmore, R., Strong, J., Swindells, Y. E., and El Din, N. The effect of overfeeding on two fat young women. *British Journal of Nutrition*, 1963, **17**, 373.
- Peck, J. W. Rats defend different body weights depending on palatability and accessibility of their food. *Journal of Comparative and Physiological Psychology*, 1978, **92**, 555-570.
- Pittet, P., Chappuis, P., Acheson, K., De Techtermann, F., and Jequier, E. Thermic effect of glucose in obese subjects studied by direct and indirect calorimetry. *British Journal of Nutrition*, 1976, **35**, 281.
- Polivy, J. and Herman, C. P. Clinical depression and weight change: A complex relation. *Journal of Abnormal Psychology*, 1976, **85**, 338-340.
- Powley, T. The ventromedial hypothalamic syndrome, satiety, and a cephalic phase hypothesis. *Psychological Review*, 1977, **84**, 89.
- Richardson, S. A., Goodman, N., Hastorf, A. H., and Dornbusch, S. M. Cultural uniformity in reaction to physical disabilities. *American Sociological Review*, 1961, **26**, 241-247.
- Revusky, S. H. and Garcia, J. Learned association over long delays. In C. H. Bower and J. T. Spence (Eds), *The psychology of learning and motivation. Advances in research and theory*, Vol. 4. New York: Academic Press, 1970. Pp. 1-84.
- Rixon, R. H. and Stevenson, J. A. F. Factors influencing survival of rats in fasting. Metabolic rate and body weight loss. *American Journal of Physiology*, 1957, **188**, 332-336.
- Rodin, J. The role of perception of internal and external signals on the regulation of feeding in overweight and normal individuals. In T. Silverstone (Ed), *Appetite and Food Intake*. Berlin: Dahlem Konferenzen, 1976. Pp. 265-283.
- Rose, G. A. and Williams, R. T. Metabolic studies on large and small eaters. *British Journal of Nutrition*, 1961, **15**, 1.
- Rozin, P. and Kalat, J. W. Specific hunger and poison avoidance or adaptive specialization of learning. *Psychological Review*, 1971, **78**, 459-486.
- Schachter, S. Obesity and eating. *Science*, 1968, **161**, 751.
- Sims, E. A. H. and Horton, E. S. Endocrine and metabolic adaptation to obesity and starvation. *American Journal of Clinical Nutrition*, 1968, **21**, 1945.
- Staddon, J. E. R. and Simmelhag, V. L. The "superstition" experiment: A re-examination of its implications for the principles of adaptive behavior. *Psychological Review*, 1971, **78**, 3-43.
- Staffieri, J. R. A study of social stereotype of body image in children. *Journal of Personality and Social Psychology*, 1967, **7**, 101-104.
- Staffieri, J. R. Body build and behavior expectancies in young females. *Developmental Psychology*, 1972, **6**, 125-127.
- Stefanik, P. A., Heald, F. P., and Mayer, J. Caloric intake in relation to energy output of obese and non-obese adolescent boys. *American Journal of Clinical Nutrition*, 1959, **7**, 55-62.
- Strang, J. M. and McCluggage, H. B. The specific dynamic action of food in abnormal states of nutrition. *American Journal of Medical Science*, 1931, **182**, 49.
- Stricker, E. M. Hyperphagia. *The New England Journal of Medicine*, 1978, **298**, 1010-1013.
- Stunkard, A. J. and Mahoney, M. J. Behavioral treatment of the eating disorders. In H. Leitenberg (Ed), *Handbook of behavior modification and behavior therapy*. Englewood Cliffs, New Jersey: Prentice-Hall, 1976. Pp. 45-73.
- Stunkard, A. J. and Rush, J. Dieting and depression re-examined. A critical review of reports of untoward responses during weight reduction for obesity. *Annals Internal Medicine*, 1974, **81**, 526.
- Szepesi, B. A model of nutritionally induced overweight: Weight "rebound" following caloric restriction. In G. A. Bray (Ed), *Recent advances in obesity research*, Vol. 2. London: Newman, Ltd., 1978.
- Tepperman, H. and Tepperman, J. Adaptive hyperlipogenesis. *Federation Proceedings*, 1964, **23**, 73.
- Thomson, A. M., Billewicz, W. Z., and Passmore, R. The relation between calorie intake and body-weight in man. *Lancet*, 1961, **1**, 1027.
- Warwick, P., Toft, R., and Garrow, J. Individual differences in energy expenditure. In G. A. Bray (Ed), *Recent advances in obesity research*, Vol. 2. London: Newman, Ltd., 1978.
- Weinstock, R. B. Maintenance schedules and hunger drive: An examination of the rat literature. *Psychological Bulletin*, 1972, **78**, 311.
- Wilson, G. T. Methodological considerations in treatment outcome research on obesity. *Journal*

- of Consulting and Clinical Psychology*, 1978, **46**, 687-702.
- Wolgin, D. L. and Teitelbaum, P. Role of activation and sensory stimuli in recovery from lateral hypothalamic damage in the cat. *Journal of Comparative and Physiological Psychology*, 1978, **92**, 474-500.
- Wooley, O. W. and Wooley, S. C. The experimental psychology of obesity. In T. Silverstone and J. Fincham (Eds), *Obesity: Pathogenesis and management*. Lancaster: Medical and Technical Publishing Company, 1975. Pp. ???
- Wooley, O. W., Wooley, S. C., and Dunham, R. B. Deprivation, expectation and threat: Effects on salivation in the obese and nonobese. *Physiology and Behavior*, 1976, **17**, 187.
- Wooley, O. W., Wooley, S. C., and Dyrenforth, S. R. Obesity and women II: A neglected feminist topic. *Women's Studies International Quarterly*. In press.
- Wooley, O. W., Wooley, S. C., and Turner, K. The effect of rate of consumption on appetite in the obese and nonobese. In A. Howard (Ed), *Recent advances in obesity research*. London: Newman, Ltd., 1975. Pp. 212-215.
- Wooley, O. W., Wooley, S. C., and Williams, B. S. Restraint of appetite and sensitivity to calories. In G. A. Bray (Ed), *Recent advances in obesity research*, Vol. 2. London: Newman, Ltd., 1978.
- Wooley, O. W., Wooley, S. C., and Woods, W. Effect of calories on appetite for palatable food in obese and nonobese humans. *Journal of Comparative and Physiological Psychology*, 1975, **89**, 619.
- Wooley, S. C., Tennenbaum, D., and Wooley, O. W. A naturalistic observation of the influence of palatability on food choices of the obese and non-obese. *International Journal of Obesity*, (in press).
- Yates, A. J. *Theory and practice in behavior therapy*. New York: Wiley & Sons, 1975.
- Young, C. M., Scanlan, S. S., Topping, C. M., Simko, V., and Lutwak, L. Frequency of feeding, weight reduction and body composition. *Journal of the American Dietetic Association*, 1971, **59**, 466-472.

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