1-1-1984

The effect of eating patterns in weight loss programs.

William Michael Tyson
University of Massachusetts Amherst

THE EFFECT OF EATING PATTERNS IN WEIGHT LOSS PROGRAMS

A Dissertation Presented

by

WILLIAM MICHAEL TYSON

Submitted to the Graduate School of the
University of Massachusetts in partial fulfillment
of the requirements for the degree of

DOCTOR OF PHILOSOPHY

February 1984

Psychology

THE EFFECT OF EATING PATTERNS IN WEIGHT LOSS PROGRAMS

A Dissertation Presented

by

WILLIAM MICHAEL TYSON

Approved as to style and content by:

Morton G. Harmatz, Chairperson of Committee

James I. Chumbley, Member

Arthur S. Keene, Member

Castellano B. Turner, Member

Seymour M. Berger, Chair
Psychology Department

ACKNOWLEDGEMENTS

I am indebted to Mort Harmatz, the chair of my committee, for his generous assistance and instruction and for his confidence in my ability to complete the Doctorate. I am grateful to Cass Turner, Jim Chumbley and Art Keene, the remaining members of my committee, both for their teaching and their friendship.

Jim Gronendyke, Jack Farrell, Diane Spencer, Todd Thomas, Nancy Keough and Brad Gordon contributed incredible amounts of work, companionship and inspiration. I am pleased to have known them, let alone to have taught them.

Foothills Area Programs, my current employer, has very generously allowed me administrative leave for the completion and defense of this dissertation.

I would never have been able to finish this project if it were not for Madalyn Tyson. I have been able reliably to depend on her to keep me going through the very worst. I probably wouldn't have bothered to finish without Daniel and Miriam Tyson's assistance. They make it all worthwhile.

Finally, none of this would have been possible without the cooperation of my parents, Michael and Diane Tyson. It is to them that I owe whatever I make of myself, and it is to them that this dissertation is most respectfully dedicated.

ABSTRACT

The Effect of Eating Patterns in Weight Loss Programs

February 1984

William M. Tyson, A.B., Temple University

M.S., Ph.D., University of Massachusetts

Directed by: Professor Morton G. Harmatz

A study was designed to evaluate the effect of manipulations to eating patterns on weight loss and maintenance. Twenty-eight subjects participated in a seven week diet during which time they maintained daily records of all food eaten and at what time it was eaten. In addition each subject maintained a daily record of their moods. Actual weights were obtained from each subject twice-weekly for the duration of the diet and for four weeks following termination of the diet.

The subjects were divided into four groups with each group receiving a specific set of dieting instructions. One group was given a diet which focused entirely on restricting caloric intake. A second group was given instructions focused on distributing caloric intake throughout the day. A third group was given instructions both to restrict and to distribute intake, and the fourth group was instructed to simply record their intake while following a diet of their own design.

The results of this study indicated no differential effect by diet group on weight loss, calorie intake and fluctuation,

carbohydrate intake or mood. An analysis of the subject's eating patterns indicated that the failure to obtain effects derived from a failure to obtain differences in eating pattern (the independent variable).

An internal analysis to identify differences between successful and unsuccessful dieters was conducted for the purpose of validating the method developed to pursue this line of investigation. This procedure clarified some appropriate prior questions which require investigation before questions such as those asked in this study may be productively investigated.

A sub-hypothesis regarding heightened external cue sensitivity observed among the overweight was investigated, using a word recognition and recall task keyed to food and emotion related, as well as, neutral words. This procedure yielded results which call into question the theory that this sensitivity is a trait of the overweight.

The results of this study are seen to demonstrate the development and validation of a method for the study of eating patterns, and to clarify and delineate the direction and potential productivity of further research.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

This study was designed to develop an argument that some factors associated with the development and maintenance of obesity are functionally related to the distribution of caloric intake by the overweight individual. A basic premise of the arguments to be developed here is that eating behavior is, at least, partially determined by a set of adaptive mechanisms which are part of the organism's phylogenic endowment.

This is not to say that human eating is genetically predetermined, but rather to say that the survival of the human species has, to some degree, hinged on the individual's ability to find, eat, and metabolize food. Obesity is the result of a complex interaction of biological and behavioral mechanisms, whose function is the adaptation and survival of the human organism.

Skinner (1953) has argued that the emergence of ontogeny, the ability to acquire behavior during the lifetime of the individual, is an important development in the evolution of organisms. Organisms which were able to adapt to rapidly changing features of the environment were more likely to survive. Species evolved which could, through ontogenic mechanisms, efficiently modify behavior to meet environmental demands which varied from generation to generation, or from individual to in

dividual. He further proposes that in the progress of evolution, those organisms which are considered the most evolved are those for whom the largest proportion of behavior is acquired through the experience of the individual member, rather than being predetermined by genetic endowment. The limiting case, of course, being imposed by phylogenic constraints. That is, the best behavioral programming to the contrary, a rat cannot learn to fly. In the case of eating behavior, the phylogenic limits are likely to be encountered by virtue of the fact that biological factors mediate much of that behavior.

Some of the theories of obesity which will be reviewed here would seem to suggest that obesity is due to constitutional factors, either present at birth or occurring early in development. The approach taken here is to assume that the biological and behavioral patterns which have been identified as characteristic of the obese are modifiable within the context of the current environment. This approach seems to be more parsimonious, more consistent with evolutionary patterns (Curtis, 1975), and finally, less pessimistic.

The Literature on Eating

Conventional (Dwyer, Feldman & Mayer, 1973) and Medical (Stunkard, 1976; Bruch, 1973) wisdom historically has attributed obesity to a failure of the regulatory mechanisms involved in feeding. Regulation of body weight is seen to be a function of caloric intake balanced with energy output. It is assumed that some malfunction of this mechanism

causes obesity. Simply put, the fat person either eats too much or does not exercise enough, or both. Research focuses on an attempt to identify the causes of this imbalance, whether behavioral or biological, and treatment focuses on an attempt to get the individual to either eat less, exercise more, or both. There are at this time three major lines of research into the causes of obesity. These are the (a) psychosomatic, (b) environmental, and (c) physiological.

The psychosomatic hypothesis

Psychosomatic theories and research assume that disorders of eating are essentially psychological in nature. They further specify that overeating is a function of unresolved emotional conflicts for which eating serves as an anxiolytic. Similarly, the inactivity--lack of exercise--of the obese is due to depression which is also the result of unresolved emotional conflict (Bruch, 1973). Bruch suggests that obese patients do not seem to know when they are physically hungry and suggests that, during childhood, these patients were not taught to discriminate between hunger and such states as fear, anger, and anxiety. The obese may label any arousal state as hunger, or may label no internal state as hunger (Bruch, 1961).

Experimental support for the psychosomatic hypothesis has been at the very least, equivocal (Schachter, 1971; Stunkard, 1976; Rodin, 1977). Schachter, Goldman, and Gordon (1968) manipulated both food deprivation and fear (fear = anticipation of electric shock) and measured subsequent eating. Contrary to the expectations of the psycho-

somatic hypothesis, fear reduced the amount eaten by normals as did preloading, whereas neither had a differential effect on the amount eaten by overweight subjects. The overweight subjects ate approximately the same amount whether they were fearful or not fearful, and whether they were deprived or not deprived. Schachter suggests two possible interpretations of the finding that the overweight subjects ate more than normal weight subjects regardless of internal state: (a) the psychosomatic position that the obese label different physiological states as hunger, or (b) the alternative position that the obese do not respond at all to internal states and eating is unrelated to internal state.

Schachter (1971; 1973) reports an elaborate series of experiments by a number of researchers, which were designed to investigate this hypothesis. The findings of these experiments do not support the psychosomatic hypothesis. Overweight subjects do not eat more when they are anxious or otherwise aroused and eating does not have an anxiolytic effect on those internal or psychic states. Schachter reports that originally he had speculated that the obese label all arousal states as hunger, but his data does not bear this out. Instead the data overwhelmingly suggest the alternative hypothesis that, for the obese, eating is not controlled by internal stimuli, but rather occurs under the control of external food-related stimuli.

Stunkard (1971) and Stunkard and Mendelsohn (1973) suggest that, while it is true that some people with emotional problems are also obese, the hypothesis that obesity is the result of emotional disorders is a

function of the restricted sample seen in a psychiatric clinic. That is, the people who tend to come to a psychiatric clinic for difficulties with obesity might tend to be those for whom obesity coexists with emotional disorder. They cite data which suggest that this generalization to the general population of the overweight is unwarranted. The majority of overweight people are emotionally normal in their adjustment. They suggest only two emotional disorders can be seen to be related to obesity, these are (a) disorders of body image, and (b) pathological overeating. By the latter, Stunkard (1971) refers to such phenomena as compulsive binge eating and the night eating pattern (these will be described in more detail in a later section). Overeating itself is not by definition pathological; it may only be classified as such when accompanied by emotional disorder and/or bizarre behavior. The frequency of obesity-related emotional disorders in the general population of the overweight is seen to be quite small.

Rodin (1977) has reviewed this literature in some depth, and concludes that the evidence for emotional causes of obesity is quite weak, but that there is some evidence that emotional hyperresponsiveness is at least a collateral if not a consequence of obesity. Stunkard (1971) reports findings which suggest that there are some emotional and psychological sequelae to weight loss dieting which are quite serious. He refers to this as the "dieting depression syndrome" and describes it as including: weakness, nervousness manifested in anxiety and restlessness, irritability, fatigue, difficulty in concentration and general apathy.

Dieting depression has been attributed to disturbances in body image which occur when the subject loses weight and to underlying emotional disorders which are revealed during weight loss. As evidence to the contrary, Stunkard cites an experiment by Keys, Brozek, Henschel, Mickelson and Taylor (1950) in which 32 healthy young men of normal weight lost 25 per cent of their body weight in six months. These subjects evidenced psychological symptoms similar to those described in the dieting depression syndrome.

Environmental Perspectives

Environmental approaches to obesity will be divided here into two categories. These are (a) the simple behavioral approach, which characterizes most clinical interventions, and is limited to applying principles of behavior change derived from laboratory investigation to the behavior of eating or exercising, and (b) experimental and theoretical analyses of the behavior of eating.

Behavior control and weight

The former category of investigation accepts as its premise the conventional wisdom that the obese eat too frequently and exercise too infrequently (Abramson, 1977a). The interventions then are designed to decrease the frequency of the former and/or increase the frequency of the latter. This is accomplished by manipulations of stimuli and consequences which are involved in eating behavior. Techniques employed

include positive reinforcement, covert conditioning, aversive control, self control, bibliotherapy, various techniques of antecedent stimulus control, education, and social and emotional support. Excellent reviews of this literature, detailed descriptions of the various techniques and their effectiveness are available in Foreyt (1977), Abramson (1977b) and Stuart and Davis (1972), and will not be reviewed here.

These techniques of behavior control are all effective to some degree. Some subjects in the various treatment programs have lost some weight. None, however, have been effective for all subjects, and in follow-up studies many of the subjects were seen to have regained at least some of their weight (Rimm & Masters, 1979). Many explanations for this have been offered, but at this point none has emerged as particularly compelling. The most parsimonious explanation of the limited success of weight loss programs is that obesity and weight loss are more complex than our currently available technology. If this simple conclusion is accepted by the reader, then it stands to reason that we should further investigate both obesity and weight-loss dieting.

It should be added that even though no procedure for weight control has emerged as perfectly or completely effective, behavior control procedures are the most effective technology available (Stunkard, 1971; Stuart & Davis, 1972). Where, and for whom, each of these techniques produce weight loss, they do so effectively.

Successful weight loss programs generally consist in the development and maintenance of what are considered to be good eating habits (Stuart & Davis, 1972). These include reduced caloric intake, regular

meals, reduced snacking between meals, and increased awareness of eating behavior as well as regular activity and exercise. Two of these components are particularly interesting, these are (a) increased awareness of eating behavior, and (b) regular meals.

Monitoring daily caloric intake and the circumstances of eating is likely to result in weight loss for at least some subjects (Stunkard, 1971; Stuart, 1967). Schachter (1973) claims that the overweight are likely to under-report daily eating and exercise, whether through lack of awareness or deliberate minimization, and cites Stunkard's finding that when chronically obese patients were fed everything that they admitted to eating in interviews, they all steadily lost weight. There is then a likelihood that subjects are unaware of how much they eat, and that by monitoring their own intake, and presumably increasing awareness, they are able to lose weight.

While this is an interesting finding in light of the understanding of obesity, it also has methodological implications. This is that any study of eating behavior or weight loss dieting must take into account the effect of monitoring on eating. This is not much of a problem in studies which employ within-subject designs, where the subject serves as its own control, because monitoring takes place during a baseline phase and the effects of experimental manipulations are compared to effects obtained during baseline (Sidman, 1960). It becomes more of a problem in group studies where the control group may or may not have its eating as closely monitored as the treatment group.

The second component of these programs which is of direct interest

to this discussion is the injunction to eat regularly. Most effective diet plans involve instructions to eat at least three regular meals each day and to eliminate snacking between meals. The focus, however, is usually on eliminating between meal snacks, and little or no attention has been given to the relative effects of regular eating on weight maintenance or loss. Again, conventional wisdom has it that eating three regular, nutritious meals is a good thing to do, and the behavioral interventions designed to inculcate skills based on conventional wisdom have simply incorporated meal regularity. It is the relative effects of distributed and regular caloric intake which constitutes the focus of this study.

The analysis of eating behavior

While there is a large literature on obesity in the field of Behavior Analysis and Behavior Therapy, these publications have usually focused on treatment procedures and have not focused on analyses of the behavior itself.

A notable exception is the work of Ferster, Nurnberger and Levitt (1962) who provided an analysis of eating behavior within the framework of the Experimental Analysis of Behavior and proposed a treatment strategy with which to validate the account. This treatment program produced results no better than the existing treatments.

Stuart (1967) adopted the strategy reported by Ferster and his colleagues and with some modifications to the procedures was able to produce dramatic results in weight loss among his subjects. Subsequent

research has focused on examining the effectiveness of various components of the diet programs and the validation of new wrinkles in behavioral procedures.

Research on food related behavior has been minimal in the field of Experimental Behavior Analysis and except for some studies of shock-induced (Hutchinson, 1977) and schedule-induced (Staddon, 1977) overeating, little attention has been paid to obesity.

Perhaps the most interesting analysis of food-related behavior among overweight and normal weight populations comes from a series of studies by Schachter and his colleagues (Schachter, 1971; 1973; Rodin, 1977). Schachter had been studying situational determinants of emotional responses, and had become interested in obesity as an extension of this research. Schachter (1973) gives an account of differences in eating patterns and the circumstances of eating among the overweight, underweight, and normal weight and integrates research into human eating patterns with research in the fields of biology and physiology on the experimental manipulation of eating behavior in animals. This is one of the few thorough investigations of food-related behavior in the literature to date. The major findings of this line of inquiry will be briefly described here.

Stimulus Bound Eating

As previously described, Schachter, Goldman, and Gordon (1968) manipulated both food deprivation and fear, and compared the amount

eaten by normal and overweight subjects. Fear reduced the amount eaten by normals as did preloading, whereas it had no differential effect on the obese. On the basis of this Schachter suggested that overweight subjects might not eat in response to internal hunger cues, b^{2}t rather eat when food is present regardless of internal state. He extended this argument by proposing that eating in the obese occurred in response to external food-related cues.

Nisbett (1968) performed two experiments which manipulated both taste and quantity of food. Nisbett also included underweight subjects in his sample. He hypothesized that the degree of susceptibility to external food cues is a dimension directly related to degree of overweight. He examined the amount of food eaten by underweight, normal and overweight subjects when the food tasted good or bad, and when subjects were food deprived or not food deprived. Overweight subjects ate more ice cream than normal and underweight subjects, when the ice cream tasted good.

The data are more equivocal with regard to bad taste as overweight, normal and underweight subjects all ate approximately the same, very small amount of the bad tasting ice cream. Schachter attributes this to the fact that the ice cream used by Nisbett was really awful (containing 2.5g of quinine per quart). Schachter (1971) cites an unpublished study by Decke which suggested that when the ice cream was less awful (only 0.4g of quinine per quart) the obese did indeed eat less. Similarly, Hashim and Van Itallie (1965) found that obese subjects drastically reduced food intake when limited to a dull bland diet, whereas normal

subjects maintained about the same intake.

Nisbett did an internal analysis of his data to test the hypothesis that the quantity of ice cream eaten by the obese subjects was proportional to their ràting of the taste. This was not supported by the data, instead it was found that overweight subjects who rated the ice cream as only fairly good overate as much as subjects who rated the ice cream as very good. Among normals, quantity eaten was directly related to ratings of quality.

Schachter and Gross (1968) manipulated the amount of time that a subject thought had elapsed by means of a rigged clock and found that obese subjects ate more when they thought that more time had elapsed. They found no such relationship between perceived time and eating for normal weight subjects.

Nisbett (1968b) compared thin, normal and overweight individuals on amount eaten where quantity was the relevant independent variable. He presented the subjects with either one or three sandwiches and informed them that more were in the refrigerator across the room. Overweight subjects ate more than normals when the food was immediately present (three sandwiches), and ate less than normals when the sandwiches were not immediately present (one sandwich).

Ross (1969) demonstrated that overweight subjects ate more shelled nuts when a table lamp illuminating the bowl containing the nuts was bright than they did when it was dim, providing further support for the notion that quantity eaten is controlled by the salience of the external stimuli.

On the basis of these, and similar, findings Schachter concludes that the eating behavior of the obese is largely controlled by external food cues rather than by internal cues. Thus, the food related behavior of the obese could be seen to be of a different type than that of individuals of normal weight.

Goldman, Jaffa and Schachter (1968; described in detail in Schachter, 1971) have replicated these findings and theoretical concerns in a variety of field settings. The data obtained in these studies have reliably supported the predictions derived from the basic thesis that the eating among the obese is more likely to be under the control of external food related cues than internal physiological sensations as is the case with normal and underweight individuals.

Schachter (1971) reports that subjects who had once been obese but who were now of normal weight--presumably by some form of self-imposed control--still manifested behavior characteristic of external control. Schachter also points out that given these findings, it is also likely that externally controlled persons could become emaciated if food were not immediately available or were by some means rendered unpalatable. He cites data from Bliss and Branch (1960) which suggests that out of 21 cases of Anorexia Nervosa eleven were previously obese. In contrast, attacks of anxiety or nervousness, states which produced reduced eating in normal subjects (Schachter, Goldman & Gordon, 1968), seemed to characterize the development of Anorexia in the cases which were originally normal size.

Physiological Factors

Studies conducted in the fields of biology and physiology have focused on attempting to isolate mechanisms regulating the intake of food and expenditure of energy. These include the isolation of neural, endocrinological and neurochemical mechanisms in food regulation. Most of this literature will not be reviewed here as it is largely irrelevant to the thesis of this paper (the reader is referred to Rodin, 1977, for an overview of this literature).

The premise for much of the research to be reported here is the assumption of some irregularity or malfunction of the regulatory mechanisms involved in eating. Research then progressed into the isolation and understanding of these regulatory metabolic processes or into the nature and determinants of hunger itself. On the whole, this line of research has failed to find any systematic differences between the overweight and normal weight in metabolism or regulatory hunger processes which might explain the development of obesity in some individuals. Where differences have been found, they are better considered as collateral of obesity. Some of these findings are of interest and will be briefly described here.

Hunger mechanisms

Stunkard (1959) studied Gastric Motility and found no differences in Gastric Motility in 37 Obese and 37 Normal weight subjects. He found that obese and normals did not differ in their reporting of hunger when

their stomachs were not contracting, but that they differed greatly in their reports of hunger when their stomachs were contracting (71.0% for normals, 47.6% for obese), indicating that normal weight subjects were more likely to label stomach contractions as hunger.

Griggs and Stunkard (1964) demonstrated that the obese can be trained to recognize gastric contractions, suggesting that there were no defects in the visceral sensorium, although trained subjects did not lose any weight. Stunkard (1976) reports that the obese showed no difference in the suppression of gastric motility when injected with glucagon (Stunkard, Van Itallie & Reiss, 1955). This finding suggests that the obese show no impairment of the feedback mechanisms which might be expected to modulate food intake.

Metabolic functions

Various metabolic functions have been implicated in obesity. Generally, the obese are suspected of reduced basal metabolism as compared to normals. The obese display increases in blood levels of insulin and free fatty acids. The former finding has been proposed to explain the inability of the obese to burn excess fat. The latter finding has been proposed to account for increased storage of adipose (fat) tissue. Research suggests that these phenomena are correlates of obesity rather than causes, and treatment based on these hypotheses has not been successful (Galton, 1971; Rodin, 1977).

Another difference between obese and normal weight individuals occurs in the development of adipose tissue itself. Increases in weight

can be accomplished by either an increase in the total number of fat (adipose) cells, or by an increase in the size of the adipose cells, or both (Galton, 1971). The total number of fat cells is determined early in the life of the individual, after which it seems to be impossible to reduce that number. Weight loss then must proceed by decreasing the size of the existing fat cells. This has led some to suggest that, for the obese, reduction in weight is accomplished only by the starving of these adipose cells and that, as such, the obese person who does lose weight is in a constant state of "starvation" (Nisbett, 1972).

Nisbett (1972) proposed that weight was regulated by a "set-point", that is, that different people had different normal weights (presumably determined by total number of adipose cells) and that obese people simply had higher set-points. He supported this supposition by appealing to studies of food deprivation and experimentally produced obesity. While this theory is high on speculation and low on experimental verification, there are some points of comparison which are of interest.

Studies have shown that both increased blood levels of free fatty acids and insulin are collateral with increases in food deprivation. Additionally, free fatty acid levels decrease with weight loss, and increase with weight gain among the obese, whereas artificially induced obesity in normal weight subjects does not produce increases in free fatty acid levels (Schonitzer, 1979). While it is possible that the obese are in a state of "deprivation", it is not clear that this owes to increased numbers of adipose cells. Enlarged fat cells seem to be common among both moderately and grossly overweight individuals, but

there is no clear evidence that increased quantities of fat cells are characteristic of the overweight (Rodin, 1977).

Hypothalamic lesions

Brobeck (1946) found that surgical lesions to the Ventromedial Nucleus of the Hypothalamus produced hyperphagia in rats. These rats, following surgery would eat voraciously until they attained a body weight much greater than their pre-surgical weights. Collateral to this marked weight gain was the emergence of hyperemotionality, and marked inactivity.

The ventromedial nucleus of the hypothalamus is seen to operate as a satiety center while another region, the lateral hypothalamus is seen as responsible for the integration of food seeking behavior. While destruction of the ventromedial nucleus produces hyperphagia, destruction of the lateral hypothalamus produces aphagia. Conversely, electrical stimulation of the ventromedial nucleus will decrease eating, and similar stimulation to the lateral hypothalamus will increase eating (Coons, Levak, & Miller, 1965).

Studies of the effects of ventromedial lesions (Miller, Bailey & Stevenson, 1950; Teitelbaum, 1955) have demonstrated that lesioned animals also tend to be finicky eaters, overeating when food is good, but tending not to eat if food is disliked. They are likely to show marked preference for food to which lard or dextrose have been added.

These animals are, as previously stated, likely to be inactive; however, when food is present they are likely to be more active. Le-

sioned rats pressed a lever for food more often than control rats when the response requirement was a fixed ratio of one, and much less than control rats when the requirement was a fixed ratio of 256. Generally, these rats would overeat when food was easily available and/or tasty, and undereat when work was required to obtain food or the food was unpleasant.

These studies have also demonstrated that lesioned rats are hyperemotional, more difficult to handle, more sensitive to pain, and better at active avoidance than non-lesioned rats.

Mrosovsky (1971) has compared the effects of hypothalamic lesioning to the behavior of hibernating animals and suggests that in the obese human the satiety centers are not functioning, though there is no evidence that they are damaged or incapable of functioning. The determinants of this hypothesized inactivity of the ventromedial nucleus are not known.

Schachter (1973) has reviewed these studies of hypothalamic lesions and compared the results to those obtained in studies of obese humans. There are marked similarities in the behavior of obese humans and rats whose ventromedial hypothalamus have been surgically destroyed. Schachter cites this as evidence of his thesis that eating in the obese is under the control of external variables. The similarities and parallels are certainly striking, but as there is no evidence of damage to the hypothalamus in obese humans, the determinants of these behavior patterns are still unclear.

Obesity and activity

Mayer (reported in Stunkard, 1976) found that rats who were required to run in a wheel to obtain food would, when required to increase their running, increase their food intake proportionately to the increased demand on energy output. However, when the running requirement was reduced below baseline requirements, food intake not only did not decrease proportionately, it actually increased sufficiently to result in obesity. When the wheel running requirement was increased for these now obese rats a subsequent decrease in feeding was observed.

Stunkard (1976) studied overweight and normal weight girls attending a girl scout camp. He compared walking rates, again as measured by a pedometer, when the girls were at home and when they were at camp. Normal weight girls tended to walk more at home than did the overweight girls. Both groups walked the same amount at the camp, much more than either at-home rate.

He found that the overweight girls tended to lose weight with this increased physical activity while the normal weight girls evidenced little weight change. Conversely, the overweight girls reported no change in appetite while the normal weight girls reported increased appetite.

Stunkard (1976) concludes that increasing activity tends to help in weight reduction by both offsetting caloric intake and also by facilitating the regulation of caloric intake.

Patterns of eating behavior

Stunkard (1976) reports a syndrome of "Night Eating" characterized by skipping breakfast and minimal hunger in the morning and at lunchtime. This is followed by a pattern of eating in the evening where the dinner meal is not satisfying, and eating progresses all evening, sometimes with the person waking up to eat in the middle of the night.

These episodes of night eating are generally accompanied by turmoil and agitation severe enough that it is sometimes described as unbearable. Stunkard reports that the proportion of the general population of obese subjects who are night eaters is small, but it is not clear that those people who did not meet the criterion of the night eating syndrome did not show some mild form of meal skipping. It is clear from his data however, that night eaters do not lose weight unless their is some change in their eating pattern.

Ross, Pliner, Nesbitt and Schachter (1971) found that the obese tend to eat fewer meals per day, but to eat more at each meal. Teitelbaum and Campbell (1958) report a similar finding for rats with hypothalamic lesions.

Schachter (1971) reports that the obese tend to be more likely than normal weight subjects to skip meals when external cues for meals are less salient. The overweight are more likely to skip breakfast, the meal for which there are the least routinized external cues and the most likely competing alternative cues. They are more likely than normal weight to skip lunch on weekends when there are less routinized lunchtime cues, and to show more variability in the time at which dinner

is eaten on weekends than on weekdays. Generally, the overweight are more likely to show variable eating times. Coincidently, Stunkard (1976) reports that differences are found when walking rates, as measured by a pedometer, are compared during weekdays and weekends, with the overweight tending to walk less on weekends than weekdays.

Stunkard (1976) distinguishes between night eating and binge eating. Binge eating is described as the ingestion of large quantities of food at a single bout. Often, these episodes follow periods of prolonged food deprivation, usually from restrictive diets. Although the night eater may ingest large quantities of food during the evening thus resembling a binge, night eating is characterized as a daily, routinized pattern, whereas binge eating is not temporally predictable.

Binge eating and night eating, when carried to the extremes reported by Stunkard, are often accompanied by serious emotional disturbance. Similar patterns of eating, though much milder in degree, often characterize the eating patterns of the overweight.

The emotional effects noticed in studies of food deprivation are similar to the dieting depression syndrome (Stunkard, 1976) which usually occurs after a subject has lost 15 to 20 pounds or has been on the weight loss regimen for more than ten days. This suggests that the emotional and behavioral abnormalities associated with dieting may have their basis in metabolic changes similar to those observed in studies of semi-starved normal weight subjects.

These patterns of eating are similar to those observed under conditions of food deprivation. Keys, et al (1950) observed that their food

deprived subjects evidenced an increased preoccupation with food, as well as increases in anxiety, irritability and overall affective lability. A marked tendency to overeat following periods of food deprivation has been observed in a number of studies (Keys et al, 1950; Rodin, 1977). Additionally, Meerloo and Klauber (1952) report that famine victims during World War Two demonstrated a desire to eat bulk and spicy foods. These effects are also similar in nature to those previously described as characteristic of obese humans and of the sequelae to hypothalamic lesions in animals.

It would appear that food deprivation might contribute to the emergence of the patterns of eating characteristic of the obese, and therefore to obesity itself. Nisbett (1972) postulated that the obese have a higher set-point, that is, that they are biologically programmed to be fat and that attempts to reduce their weight to "normal" levels essentially constituted starvation. This theory, as previously described, requires that one accept the assumption that the obese have more adipose cells than normal weight individuals, as well as, the supposition that this higher fat cell number is the determinant of obesity.

Another plausible explanation is that obesity is at least partially determined by the above described eating patterns, that is, that obesity may be an adaptive response to irregular and unpredictable cycles of deprivation and overeating. Gross (1968; reported in Rodin, 1977) conducted an experiment which examined the effect of both cyclic deprivation and unpredictability on the eating patterns of newly weaned rats. He exposed three groups of rats to one of three conditions (a) free

feeding, (b) fixed-time of 22 hours between feeding, and (c) variable-time of 22 hours, with a range of 8 to 48 hours, between feedings. All animals were maintained on this schedule for 100 days. The rats who were exposed to the variable feeding schedule were more responsive to variations in the taste and caloric density of food. At the end of 100 days when the rats were allowed to feed freely, the variable time rats continued to be preoccupied with food, and generally to demonstrate behavior which is characteristic of external cue control of eating.

It would seem that unpredictable food availability encourages dependence on external cues. It would be most adaptive to eat when food is available rather than when one was "hungry" (Rodin, 1977). It would also be likely that an organism would maintain energy stores sufficient to survive through long periods of deprivation without starving to death. Thus, it would be adaptive to eat as much as possible whenever food was available, and to show a preference for high calorie and fat-rich foods.

Research into the effects of reduced meal frequency has demonstrated that this results in elevated blood cholesterol, impaired glucose tolerance, elevated insulin levels, and increased fat synthesis in the adipose tissue (Rodin, 1977). The role of these metabolic phenomena is not clear, although each has been implicated in obesity. Woods, Decke and Vasselli (1974) found that rats injected with insulin increased their food intake and body weight, however, other studies suggest that the role of insulin levels in obesity is at best correlative (Galton, 1971). Similarly, the role of free fatty acids is unclear.

Increased levels of free fatty acids are observed in the obese and are seen to decrease when weight is lost. However, in normal weight individuals free fatty acid levels do not increase with weight gain, but do increase under conditions of semi-starvation. Schonitzer (1979) on reviewing these findings suggests that these somewhat paradoxical effects might demonstrate an effect of hunger since many obese people go without food all day and eat heavily during restricted periods.

If this is the case, then the etiology of these eating patterns are of interest. It is not clear why these eating patterns should emerge in the obese. It is also not clear whether eating patterns are a correlate of obesity or have some causal contribution to either the emergence or maintenance of obesity. There is also the possibility that these eating patterns are a consequence of obesity.

Restrained eaters

As previously described, Schachter (1971) found that subjects who had once been overweight but who were now of normal weight still manifested behavior characteristic of external cue control. Herman and Mack (1973) suggested that some normal weight subjects are actually restrained eaters. They may be externally controlled but manage to maintain a reduced body weight. They identified subjects as restrained eaters and found that these subjects ate like Schachter's externally controlled obese when restraint was experimentally undermined.

Presumably, restrained eaters exercise their restraints in response to the external social pressures directed toward the obese, to

which they, as externally controlled, would be expected to be sensitive (Rodin, 1977). It would seem that the difference between restrained eaters of normal weight and the obese is that for restrained eaters the external variables of social pressure are more salient than food.

Schonitzer (1979) suggests that many overweight subjects are restrained eaters who for some reason experience more breakdown of restraint than those who are able to maintain normal weights. Herman and Mack (1973) liken restrained eating to a trigger mechanism, in which, once the restraint has been defeated, individuals eat with little or no control. This can be seen to be similar, though on a smaller scale, to the binge eating pattern described by Stunkard (1976)

Schonitzer (1979) proposes that dieting may actually aggravate the problems of overweight restrained eaters. Weight loss diets may collaterally increase deprivation by their focus on strengthening an individual's ability to restrict caloric intake. This increased deprivation would serve to increase the salience of food related cues above the threshold where they could trigger uncontrolled eating.

Schonitzer and Harmatz (1977) designed a diet plan which minimized the "sense" of deprivation by including access to a "favorite food" as part of the weight loss plan. They compared subjects who followed this diet to subjects who followed a more restrictive, calorie counting diet. Both groups of dieters lost approximately the same amount of weight, but the "favorite food" group was better able to maintain weight loss following termination of the diet. This result was interpreted as indicating that some effects of deprivation had been attenuated.

Summary

Despite the abundance of theories and research into the causes of obesity, very little is actually known. We have little insight into causal processes in these phenomena beyond the truism that the overweight maintain a "positive caloric balance", that is, that fat people eat too much, exercise too little, or both.

The literature does provide some detailed description of behavior patterns associated with obesity and weight control. Eating may be seen to be under the control of external cues for some people. These people can be further described as exercising restraint, to varying degrees of success, over their eating. The overweight, then, are those for whom external food cues are more powerful than the restraint mechanisms.

The hypothalamus is implicated in the regulation of food intake and is suspected to be quiescent in obesity. Metabolic changes such as hyperinsulemia and increased blood levels of free fatty acids characteristic of obesity are also characteristic of starvation. Similarly, emotional reactions (dieting depression syndrome) to dieting are also similar to emotional reactions to food deprivation. Finally, activity levels, which are implicated in the regulation of caloric balance both directly and indirectly, are suppressed in the obese.

More to the point of this paper, overweight people tend to concentrate caloric intake, eating fewer meals per day than normal weight people but eating more at each meal. The overweight are more likely to

schedule meals irregularly, and to skip meals, particularly breakfast. Extreme cases of these meal patterns constitute binge eating and night eating patterns. These eating patterns seem to resemble the variable deprivation schedule which experimentally produced both obesity and external stimulus control.

Both irregular and concentrated eating have been implicated in obesity, yet very little research has been done into this area. The role of caloric distribution in weight maintenance has not been investigated. This study is designed to investigate the role that patterns of caloric distribution play in obesity and, more specifically, weight loss.

Speculation

Eating infrequent but calorie dense meals may be seen to constitute a form of variable deprivation cycle. It is possible that these eating patterns replicate internal phenomena characteristic of food-scarce environments, triggering metabolic and behavioral processes which are adaptive to such environments. This hypothesis is consistent with what is known about the correlates of obesity.

Beller (1977) has provided an exhaustive and detailed review of the literature supporting an account of obesity based in principles of evolution and natural selection, documenting the influence of what Skinner (1953) might call the "Contingencies of Survival". She makes a convincing case for the hypothesis that obesity may be consistent with behavioral and metabolic patterns that have evolved as survival mechan-

isms during the history of our species.

Extrapolating from this one might speculate that mechanisms which minimize control of eating by internal state and maximize control of eating by the environment would be adaptive in a food scarce environment. These mechanisms would be likely to maximize synthesis of fats by the body, thus explaining the metabolic changes observed in the obese. Under these conditions the body would also be expected to minimize expenditures of energy which were not directly food related, and generally to defeat the mechanisms which normally balance caloric intake and energy output.

It is possible that weight loss dieting actually aggravates this process by increasing both deprivation and variability in caloric intake. Meal skipping is commonly employed by dieters as a method of weight reduction. Structured weight loss programs often recommend regular meals and recommend against large, sudden reductions in caloric intake (Stuart & Davis, 1972) and these usually report success while the individual is following the program. However, it is likely that when the individual completes the diet there is a return to irregular eating patterns. This might partially account for the tendency to regain weight following the termination of weight loss dieting.

Emotional Disorders induced by dieting may be due to metabolic factors associated with food deprivation, and thus these reactions might also be seen to be a function of restricted meal frequency. The emotional responsiveness of the overweight may be exaggerated by increased deprivation and variability introduced by weight loss dieting. Like-

wise, the probability of losing control of eating and engaging in binge-like eating would be increased in this process, as would be the likelihood of a preference for bulk and high-caloric foods.

Continuing this speculation, the body is behaving as though it were in a food scarce environment, so it would be expected that receding fat stores would signal replacement of a low activity level (energy conservation) with a high level of activity directed toward obtaining food (agitation, preoccupation with food). Caloric restriction under these conditions could be seen as self-defeating, the harder one worked to lose fat, the harder the body would work to conserve and acquire more fat. Alternatively, one might contrive to replicate conditions of a food-dense environment in order to defeat the mechanisms triggered by this state of pseudo-deprivation. Weight loss dieters who ate more frequent but smaller meals could be expected to experience decreased incidence and intensity of these emotional reactions to dieting, whereas dieters who further decrease frequency of eating would be expected to experience increased emotional lability.

The problem

A review of the literature on behavioral interventions with the overweight revealed no attempts to increase frequency of eating as a treatment for obesity. This is not surprising given that the conventions which guide most studies of obesity posit eating too much as a cause of obesity, and it would seem counterintuitive to prescribe eating more as a treatment. It seems true that caloric saturation is a

problem, but this refers to the intensity of the response. What is proposed here is that caloric saturation is a function of the frequency of eating and that, as such, increases in the frequency of eating will attenuate saturation.

The question remains as to the validity of these speculations. If temporal variability of caloric intake does contribute to weight gain or high-weight maintenance, then regulating the frequency of caloric intake ought to contribute to weight loss, and to the maintenance of that weight loss.

The Present Experiment

The present experiment is designed to investigate the hypothesis that regulation of the frequency of caloric intake contributes to weight loss and the maintenance of weight loss. The test of this hypothesis was effected by monitoring a group of dieters under varied conditions of caloric restriction and frequency regulation.

Four groups will be created out of a sample of weight loss dieters who are at least 10 per cent overweight. One group will be instructed to restrict caloric intake by means of an "exchange diet" (Stuart & Davis, 1972) with no instructions to regulate the frequency of intake (Restrict Calorie Group). A second group will be instructed to eat at regular times each day and to distribute caloric intake across the day, but will be given no instructions to limit caloric intake (Distribute Calorie Group). A third group will be instructed to both restrict caloric

intake and to distribute and regulate caloric intake (Both Instructions Group). The fourth group will be instructed to monitor their caloric intake on a daily basis, with no instructions to either restrict or regulate intake (Monitor Only Group). Subjects will remain on these regimes for at least eight weeks.

Subjects will maintain a daily diary of their eating and their mood. Additionally, they will be weighed twice a week for the duration of the experiment.

Supplemental to these hypotheses is the more theoretical question of whether the increased sensitivity to external cues found to characterize the obese (Rodin, 1977; Schachter, 1973) is a partial function of irregular caloric intake. This may be evaluated by comparing relative changes in cue salience between the four groups.

Cue salience has been shown to facilitate learning of cognitive tasks (Trabasso & Bower, 1968). Levitt and Epstein (1962) obtained greater rates of recall to food related cues in a paired-associate memory task when the subjects were hungry during the learning trials. If sensitivity to control by external cues occurs in response to variable deprivation schedules then changes should be observed in differential learning to food and non-food cues in the different groups.

Subjects in this experiment will be administered a Cue Salience test three times during the experiment. The first administration will occur during the introductory meeting prior to the experiment. The second administration will take place following the eight week experimental period, and the final administration will be held after the

eight weeks of follow-up.

Hypotheses

If this analysis is correct, and the distribution of caloric intake contributes to overweight maintenance, then we may state the following expectations:

(1) Subjects in the Restrict Calories group, the Distribute Calories, and the Both Instructions group should show more weight loss than subjects in the Monitor Only group as measured by percentage weight lost.

(2) Subjects in the Restrict Calories group would be expected to have regained more weight than the subjects in the other two experimental groups at the end of the eight week follow-up period.

(3) It is expected that the Restrict Calories group will evidence either a greater frequency, a greater intensity, or both a greater frequency and intensity of off-diet eating episodes when compared to the Both Instructions group.

(4) The Distribute Calories group would be expected to show increased spontaneous modulation of caloric intake compared to the Monitor Only group

(5) Subjects in the Restrict Calories group should show more negative affect than the other groups, and a greater degree of fluctuation in mood than either the Distribute Calories group or the Both Instructions group

(6) The Distribute Calories group and the Both Instructions group should show a greater decrease in the recognition and recall of food related words, or an increase in the recognition and recall of non-food related words, when compared with the Monitor Only group. The Restrict Calories group should show a greater effect of food-cue salience when compared to the Monitor Only group and the other groups.

CHAPTER II

METHOD

Subjects

Subjects for this study were recruited during the first three weeks of the Spring, 1983 semester at the University of Massachusetts. One group of prospective subjects was identified through a questionnaire on dieting habits developed by Harmatz (note 1). The Questionnaire was distributed to undergraduates at the University of Massachusetts during the Fall, 1982 semester and potential participants in this study were identified by the criteria of (1) being at least 10% overweight, (2) reporting that they intended to go on a diet in the near future, and (3) agreeing to be contacted about future studies of weight loss diets. Additional subjects were recruited by means of posters in the Psychology Department Building inviting potential subjects to participate in diet research.

All prospective subjects were screened at meetings designed to introduce subjects to the procedures, and those prospective subjects meeting the criteria for participation were invited to join the formal diet program. All meetings were held during the evening hours. Twen

ty-three prospective subjects from the questionnaire pool and sixteen subjects who responded to the posters agreed, at one of the evening meetings to participate in the project yielding a starting sample of thirty-nine subjects.

Of the initial 39 subjects five dropped out during the first week, with three of them not attending their first scheduled weigh-in. One more subject dropped out during the baseline phase, prior to receiving a diet. Of the remaining 33 subjects, 5 dropped out during the diet phase leaving a sample of 28 subjects completing the program. The five subjects who dropped out during the diet phase were evenly distributed among groups, with one each from the Restrict, Distribute and Monitor groups and two from the Both Instructions group. An analysis of the existing data for these subjects revealed no systematic reasons for their dropping out. Three of the five had lost 2.0% or more weight and the other two had shown minimal or no weight loss when they dropped out.

Of the remaining 28, one subject was reassigned from the Distribute to the Both Instructions group when it was discovered that she was following the diet assigned to her friend.

Instruments

Daily record form

Subjects recorded data on food intake and mood using a record form which consisted of two sections. These were:

Daily eating diary. This instrument was designed for the recording

of daily food intake. It consisted of a record form where all food eaten, and the circumstances and time when it was eaten, could be recorded by the individual subject.

Daily mood diary. This ten item Likert-type scale, developed by Epstein (1982) was designed to detect changes in mood over time. Normative data are available (1979) which demonstrate that this scale measures transient mood on a day-to-day basis. The mood factors are organized into ten bipolar dimensions where the subject could endorse either pole, a neutral position or some tendency toward either pole.

Cue salience test

This consisted of two lists of 40 words each. The words were presented for very brief durations such that they were about at the threshold for recognition. Differences in correct recognition and recall could be attributed to the relative salience of the particular words. Each list contained ten food related words (steak, apple), ten evocative words (scum, hate), and ten neutral words (wave, tree). Each word was followed immediately by a masking stimulus which consisted of a random jumble of letters. Ten additional words were presented at a brighter light intensity with a duller intensity masking stimulus (details of administration are described later in this section).

Weight, in pounds, was assessed using a medical-type, balance scale. This scale was reset to "zero weight" prior to each bi-weekly weigh-in and sometimes was reset during the weigh-in day to insure reliable measurement.

Procedure

Subjects who met the criteria for participation in the study were contacted at the beginning of the Spring, 1983 semester. At this time they were invited to participate in a formal diet program. If they were interested they were invited to an informational meeting scheduled on weekday evenings during the first three weeks of the semester.

The first meeting

The subjects were informed that the experiment was designed to investigate the relative effects of various weight loss procedures, each of which had been shown to be effective with some people, on aspects of behavior other than weight loss itself. They were told that they would be asked to monitor their eating behavior and their mood. In this meeting the subjects received the first administration of the Cue Salience Test.

Cue salience. The subjects were told that this was a measure of attention and reaction ability and that we would be monitoring this at various times during the course of the experiment. This probe was administered in a group format. The stimulus word lists and order of presentation may be found in Appendix A.

The subjects were told that the words were to be projected at varying durations so that we could measure their reaction times and that their job was to write down each word that they recognized, or their best

guess, on a record form numbered to correspond to the order of word presentation. Actually the thirty words were presented at the same very short duration (approximately 50msec) such that they were just about at recognition threshold. Each word was immediately followed by the presentation of a masking stimulus (a jumble of letters). Ten neutral distractors were presented at a higher light intensity with a less intense masking stimulus such that they would definitely be recognized and recorded. This was designed to minimize the chance that subjects would detect the fact that the words which they were recognizing were food related. Subjects wrote down what they thought each word was after it was presented. Immediately following presentation of the entire list the subjects were asked to write down as many words as they recalled.

Data recording. Following administration of the Cue Salience Test, the subjects were instructed in maintaining the daily diet diary and the daily mood diary. They were instructed as to the procedure for the biweekly weigh-in and were introduced to the research assistants who were to be their contact persons for the weigh-in and for the collection of the daily records. The subjects were again asked if they wished to continue with the program and, on their agreement, a baseline weight (B0) was obtained.

At this time a specific appointment was made for each subject to weigh-in on the weigh-in days (Monday and Friday). Each subject was asked to keep the daily records and to attend their scheduled weigh-ins until the specific diet instructions were assigned (baseline phase). They were told that this would allow them to become used to the diaries

and weigh-ins. At this time informed consent and a medical history screening inventory was obtained from each subject.

The baseline phase

During the baseline phase, the subjects were randomly assigned to one of the four experimental groups by means of the roll of a four-sided die (available from TSR Hobbies, Lake Geneva, WI). The results of the Cue Salience Test were subjected to an Analysis of Variance procedure on the variable of experimental group to identify any pre-existing differences which might potentially confound the results of this study. The results of this indicated no group differences but a decidedly higher recall rate for food and emotion words as compared to neutral and baseline words. The first batch of daily records was examined in order to identify gross differences in eating pattern and to identify problems in recording intake such that feedback could be provided prior to assignment to groups.

The diet phase

After approximately seven to twelve days of baseline recording each subject was given a set of diet instructions. At this time, the principal experimenter met with each subject individually to explain their diet and to answer any questions the subject had about the diet or the procedures. The subjects were reminded not to compare diet plans with each other as this would defeat the purpose of identifying differential effects of the procedures.

The subjects were also reminded to be accurate in reporting their food intake and mood as this would help us to identify problems which they might have with dieting. They were assured that at the end of the experiment we would make the hypotheses and preliminary results available to them.

Diet instructions

The following is a summary of the instructions given to each of the groups (the full text of the instructions may be found in Appendix B):

Restrict calories. Each subject was given an "exchange diet" (Stuart & Davis, 1972) with instructions to eat as they normally would while restricting their intake to the number of units specified in the diet. They were told that this diet was successful because it was so easy to follow and did not deprive them of their favorite foods.

Distribute calories. Subjects were instructed to eat three, or more, full meals per day at specified times, with the first meal of the day eaten within one hour of waking. They were told that their tendency to gain weight was the result of irregular eating and that getting in the habit of eating regularly would result in the body spontaneously regulating weight. They were instructed not to count calories or restrict their eating during meals, but rather to let their bodies restore the natural regulation of eating.

Both Instructions. Subjects in this group were given the exchange diet with additional instructions to eat three or more meals per day at specified times, with the first meal of the day eaten within one hour of

waking. They were told that their bodies would begin to regulate food intake once they were in the habit of eating regularly but that they would have to restrict their caloric intake to lose weight at first.

Monitor Only. Subjects in this group were told that they knew what to do in order to lose weight and that they should design a diet for themselves and follow it. They were told that monitoring the circumstances and times of eating as well as what they ate would result in an increased awareness of their eating and that this would result in weight loss.

Weigh-ins

The subjects reported on a biweekly basis to turn in their daily records and to be weighed. The subjects were weighed by undergraduate research assistants who were blind to the experimental hypotheses. The research assistants also did not know to which group a subject was assigned. Each research assistant was assigned a fixed time to be in the weigh-in room and these differed on the two weigh-in days. As a result, all subjects were weighed by two different research assistants. Subjects were instructed to contact the principal experimenter (this writer) if they had questions specific to their diet plan.

Half-way follow-up

At approximately the halfway point in the diet phase each subject was contacted by the principal experimenter. At this time, each subject was asked how they felt about their diet and how they felt about the

amount of weight they were losing. The experimenter then told them that he did not have his list of group assignments and asked them to remind him what diet they were following. This was completed as a check against whether the subjects understood their instructions and were actually following the assigned diet. This procedure identified one subject who was following the diet assig to her neighbor. This subject was reassigned from the Distribute Calories group to the Both Instructions group. Each subject was then encouraged to continue with their diet and encouraged not to lose faith in their ability to lose the weight they wished.

The formal diet phase continued for approximately seven weeks (spring break intervened in this period, so there are thirteen weigh-in sessions recorded as part of the diet phase). Due to the staggered starting times for the subjects (one or two weeks apart) the weigh-in data was analyzed to check for time effects. This failed to reveal any differences so the data was pooled according to time from the start of the diet. Similarly, a check for effects due to vacation week was completed and this also revealed no systematic effects. As a matter of record, the weights remained remarkably stable over the vacation week. At the end of the formal seven-week period, subjects were informed that they might, if they wished, discontinue keeping the daily records, but that we would like them to continue to attend the weigh-ins until the end of the semester. These weigh-ins were treated as post-intervention or "follow-up" weights.

The Final meeting

When all of the subjects had completed the formal diet phase, they were invited to return to a follow-up feedback meeting, scheduled during the same evening hours as the original group meetings in order to control for effects on Cue Salience due to time of day or latency since eating. At these meetings a parallel form of the Cue Salience test was administered to 22 of the 28 subjects who completed seven weeks of the diet phase.

As there was determined to be no systematic group effect on weight loss, the decision was made to share the hypotheses of the study and request that all subjects continue to come in for weigh-ins but to make their own decisions regarding how they would continue to diet.

Data management

Each item recorded on the daily record had to be assigned a calorie and carbohydrate value in order to be translated into an analyzable form. This task, which turned out to be monumental, was completed by a team of undergraduate research assistants directed by the principal experimenter. The details of this process are described in the "Reliability" subsection. In addition to these conversions, a rating was assigned to each group of items falling within a one-hour interval. These were assigned according to a subjective categorization of the quality of the group of items. These categories are described in detail in the "Carbohydrates" subsection of the Results.

In order to handle the massive amount of data generated for each subject (approximately 3900 observations per subject) a sequence of programs were written for the entry and retrieval of the data sets. A program was also written which would read the created data files and check them for the most common coding and data-entry errors. This program automatically corrected errors due to entry and identified and informed the operator of suspicious calorie, carbohydrate or rating codes, allowing the operator to check the raw data and input the correct data.

Much of the data analysis was completed using the "Microstat" program (available from the Ecosoft Corporation), with the exception of the repeated-measures Analysis of Variance which was accomplished using a program written specifically for this study. This program was "benchmarked" against both Microstat and a known data set whose results were obtained from the Statistical Package for the Social Sciences (SPSS). Listings of all programs are available from this writer.

Reliability

The most clearly reliable data generated during this project is that of weight change. This was partially insured by the accuracy of the scale, which was balanced at the beginning of each weigh-in day and at least once again during each day, and also by the frequency of measurements through which discrepancies due to measurement would easily be identified.

The calorie and carbohydrate data is not so clearly reliable or valid. There were a number of sources which introduced the potential for error or bias. First, subjects were responsible for recording their own food intake. There is always the likelihood that they were not completely honest. This might stem from a deliberate failure (or embarrassment) to record all that was eaten or a from more subtle minimizing of quantity. In any event, the measures which reflect quantity of intake are suspect, in their absolute form, in that one may not be confident that between-subject differences in actual quantity eaten are consistent or systematic in any way. One can, however, be more confident that bias within-subjects is consistent, that is that each subject would minimize or omit reports of intake in much the same way throughout the study. This source of bias is the most troubling as it is the most difficult to assess, let alone control. The subjects were encouraged to record everything they ate and they were encouraged to record both quantity and type of food with as much detail as possible. Nonetheless, one must assume that there were occasions of misrepresentation and the data should be considered with this likelihood in mind.

Another source of bias and/or error was introduced through the process of obtaining calorie and carbohydrate equivalents to the recorded foods. As reported earlier, these conversions were made by undergraduate research assistants from each subject's record using a text listing. This was a time consuming, tedious and frustrating job and, as is to be expected from this type of task, there was a likelihood of errors. Additionally, to some degree the coding required many

"judgement calls" on occasions where the brand of a food, or an exact quantity was not specified. Thus the errors were likely to fall into two categories, either glaring errors due to inattention and frustration (i.e.:"donut" being coded for the caloric equivalent of a dozen doughnuts) or disagreements (i.e.: "apple" was coded for the caloric equivalent of a "medium apple" by one assistant and "large apple" by another).

There is one final class of inaccuracy present in the coding and this arises from occasions when no listing of a conversion was available. On some occasions, the equivalents were obtained by visits to the supermarket, phone calls to cocktail lounges (for some of the more exotic mixed drinks) and phone calls to friend's mothers to obtain ethnic recipe's; however, there were a few occasions where no coding could be obtained and in these instances a code for a similar item was used (i.e.: "Babka" became "coffee cake")

Quality control on this process was effected by a series of techniques. First, the assistants were divided into teams of two and each team was given the responsibility of coding specific folders. The teams overlapped as each person was on two teams and hence worked with two different raters. This procedure decreased the likelihood of gross misrepresentation as each partner provided a check on the other. This also served as a check on the quality of "judgement calls" by having the decision made among the two raters. Additionally, the fact that each folder was completed by the same team increased the likelihood of within-subject consistency and the overlapping teams increased the

likelihood of consistent coding between subjects.

An additional check on this process was provided when the coded numbers were entered into the computer. At this time, the operator could notice conversions that did not agree with his or her own estimates. These could then be checked and remediated. Errors made during the data entry process were identified by a program, described earlier in this section, which was designed to read the disk files created on each subject, identify the most common data entry errors, and prompt for correction. This program also picked up a few coding errors which had escaped earlier detection. The process of handling a data set of this size, approximately averaging 3900 observations for each of 28 subjects, was certain to produce a some degree of error. It is believed that the error trapping process described here identified and corrected the bulk of these.

Reliability checks. A reliability check was made by this writer at the point where the data were ready to be entered into computer files. This was effected by selecting (at random) one daily record from each of ten folders (each coded by a different team). These were rescored by this writer and the results compared with the original. A total of 126 items were compared and 13 disagreements obtained yielding, by the method of agreement, a Reliability score of approximately 0.89. The greatest magnitude of disagreement was approximately 0.20 with an average at approximately 0.13. As a further test, the items on which disagreement was obtained were checked throughout the rest of the folder in order to see if the disagreed upon coding was consistent. Except for

one occasion, the coding was found to be consistent throughout the folder. Similar results were obtained on the occasion when the same foodstuff had been coded in two separate folders.

While this magnitude and rate of disagreement may appear acceptable, it must be remembered that this is a relatively small sample (ten out of approximately 1800 records) and that the error rate in coding, however small, was multiplied by the existing and nonassessable error rate introduced in the subject's recording. As such, the use of any absolute statistics derived from these data and used in between-subject analyses were seen to be suspect and a decision was made to limit the analyses and the subsequent conclusions to statistics that could be derived from within-subject analyses, where one could be more confident that the numbers were legitimately comparable. For example, while we may not be confident that the average number of calories reported as ingested by one subject is directly comparable to that of another subject, we may be more confident that the standard deviation of a subject's calorie ingestion, which is based on fluctuations in biased data, but is largely independent of that bias, can be used in a meaningful between-subject comparison. As such, tests of the hypotheses of this study were designed that would maximize the use of statistics which were more likely to be independent of bias.

C H A P T E R III

RESULTS

The results of each class of hypothesis in this project will be reported in a separate subsection. These will include an analysis of the actual eating patterns observed in the subjects, as well as, findings regarding the following: actual weight lost, calorie and carbohydrate intake, fluctuation in calorie and carbohydrate intake, mood changes and finally, an analysis of changes in cue salience. Where appropriate, these sections will be followed by a summary of the major findings.

Unless otherwise noted, the criterion for statistical significance (Alpha) was set at $p. = 0.05$ for the analyses to be reported here; however, for the readers information the probability of the obtained statistic will be reported when results are claimed as significant using the stated criterion.

Eating Patterns

The primary hypotheses of this study are based in the theoretical notion that eating patterns have an effect on various aspects of weight loss and gain. It was reasoned that if the experimental

findings regarding group differences were to be considered valid then each diet group should evidence eating patterns consistent with their Diet instructions. Toward this end, the average caloric intake within each one-hour period, expressed as a percentage of daily caloric intake, was obtained for each subject. A repeated measures Analysis of Variance procedure was then performed on these data. The results of this analysis are shown in Table 1. Figure 1 shows a comparison of the percentage of daily caloric intake for each Diet group (Group).

Table 1

Results of repeated measure Analysis of Variance on Average Caloric Intake by One-Hour-Interval (TRIAL) and by Diet Instructions Group (GROUP)

SOURCE	DF	SS	MS	MS ERR	F
GROUP	3	0.010	0.003	SUBJ	0.762 (3,24)
TRIAL	18	5330.520	296.140	S/R	17.773 (18,432)
GT	54	1620.270	30.005	S/R	1.801 (54,432)
SUBJ	24	0.103	0.004		
S/T	432	7197.960	16.662		

The repeated measures Analysis of Variance revealed a significant main effect of one-hour interval (Trial) (f = 17.774, p. = 0.0000), and a significant effect of the Diet Group by Hour (GT) interaction (f = 1.808, p. = 0.0008). This would seem to indicate that the different groups followed different patterns in their daily eating, as per the design of the experiment. However, an examination of Figure 1 suggests that all four groups essentially followed the same pattern of massing their intake in two two-hour intervals beginning at approximately Noon

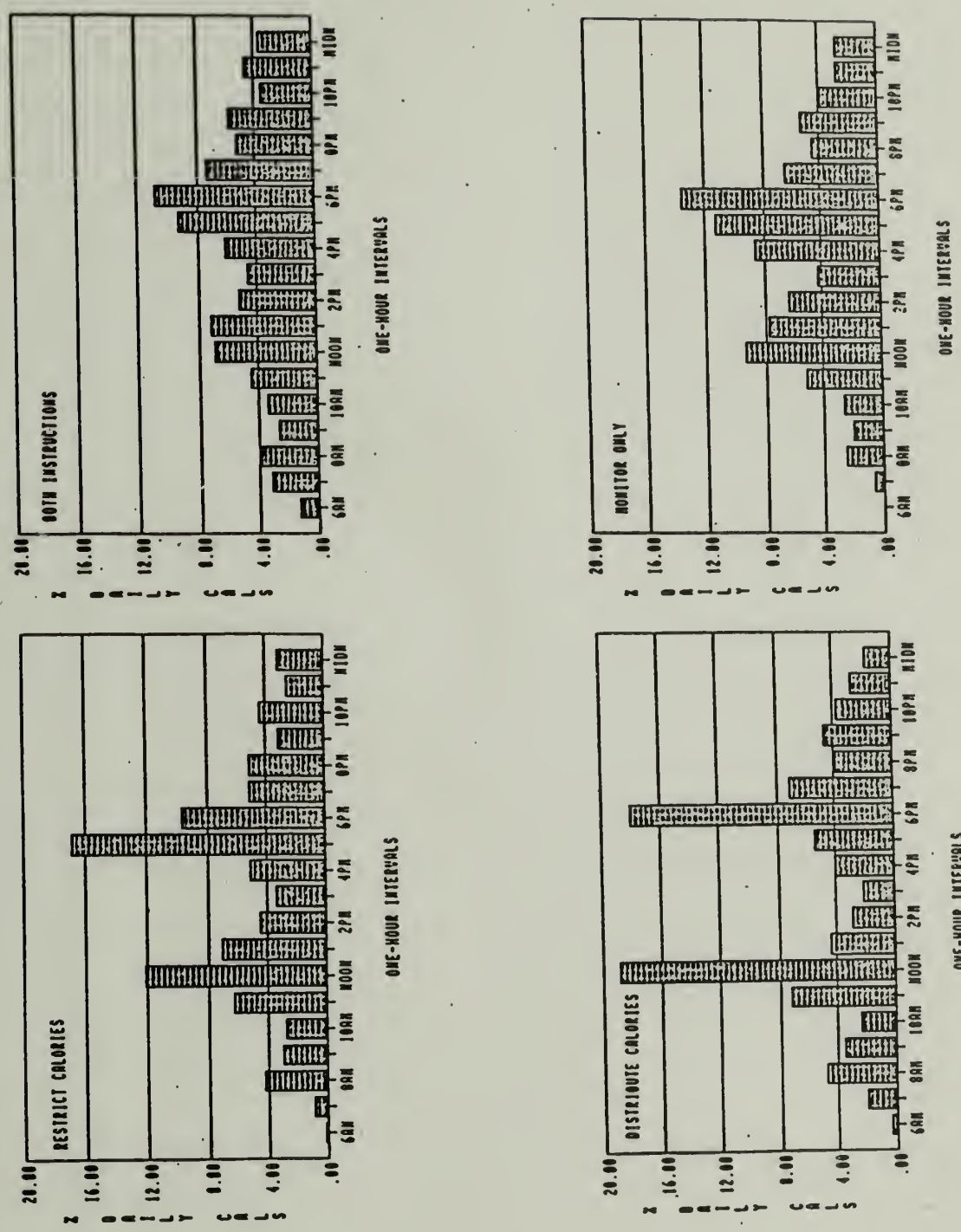

Figure 1. Hourly Caloric Intake for Diet Groups.

and 5:00 P.M., with the smallest percentage (average of all groups = 11.7%) being consumed prior to 11 AM. The interaction may best be explained by the degree to which each group massed calories in the later periods.

These findings would indicate that the two groups (Distribute Calories and Both Instructions) who were instructed to consume 25% to 33% of their daily caloric intake within an hour of rising did not comply with those instructions. An examination of Figure 1 suggests that the Distribute Calories group did evidence more regular meal patterning and that the Both Instructions group tended to disperse intake to a greater degree than the other groups. It is to be noted that these two groups did consume higher percentages of calories prior to 11:00 AM (Space = 13.24% and Both = 14.52%) than the Monitor group (7.87%); however, these percentages do not differ significantly.

An examination of each individual subject's average hourly intake revealed only one subject, a member of the Distribute Calories group, who had at least consumed 25% of her calories prior to 11:00 A.M. This subject did lose weight during the study and evidenced a lower percentage of evening eating than average in her group.

Another analysis was performed by dichotomizing the sample according to degree of weight change at the end of the seven week study. The criterion established for classification was -1.0 percent weight change. According to this classification: 13 subjects were classified as having shown no weight loss and 15 subjects were classified as having shown weight loss. The results of this analysis are shown in

Table 2.

Table 2

Results of repeated measures Analysis of Variance
on Average Caloric Intake
by One-Hour-Interval (TRIAL) and by Loss Status (LOSS)

SOURCE	DF	SS	MS	MS ERR	F
LOSS	1	0.001	0.001	SUBJ	0.224 (1,26)
TRIAL	18	5330.520	296.140	S/R	16.305 (18,468)
LT	18	318.325	17.685	S/R	0.974 (18,468)
SUBJ	26	0.113	0.004		
S/T	468	8499.910	18.162		

The repeated measures Analysis of Variance using these groups showed a significant main effect for one-hour interval (Trial) but no significant interaction effect, suggesting that the pattern of eating actually followed by the subjects in this study was irrelevant to weight loss. The actual average hourly percentage of intake are shown in Figure 2.

Weight Loss

Table 3 shows the distribution of subjects who completed the seven week study divided according to Diet Group membership and whether or not they lost weight. It is interesting to note that the Monitor Only control group, contrary to expectations, was the group in which the most members lost weight. A contingency table analysis of these frequencies yielded a chi-square value of 4.60 (d.f. = 3) which is not significant.

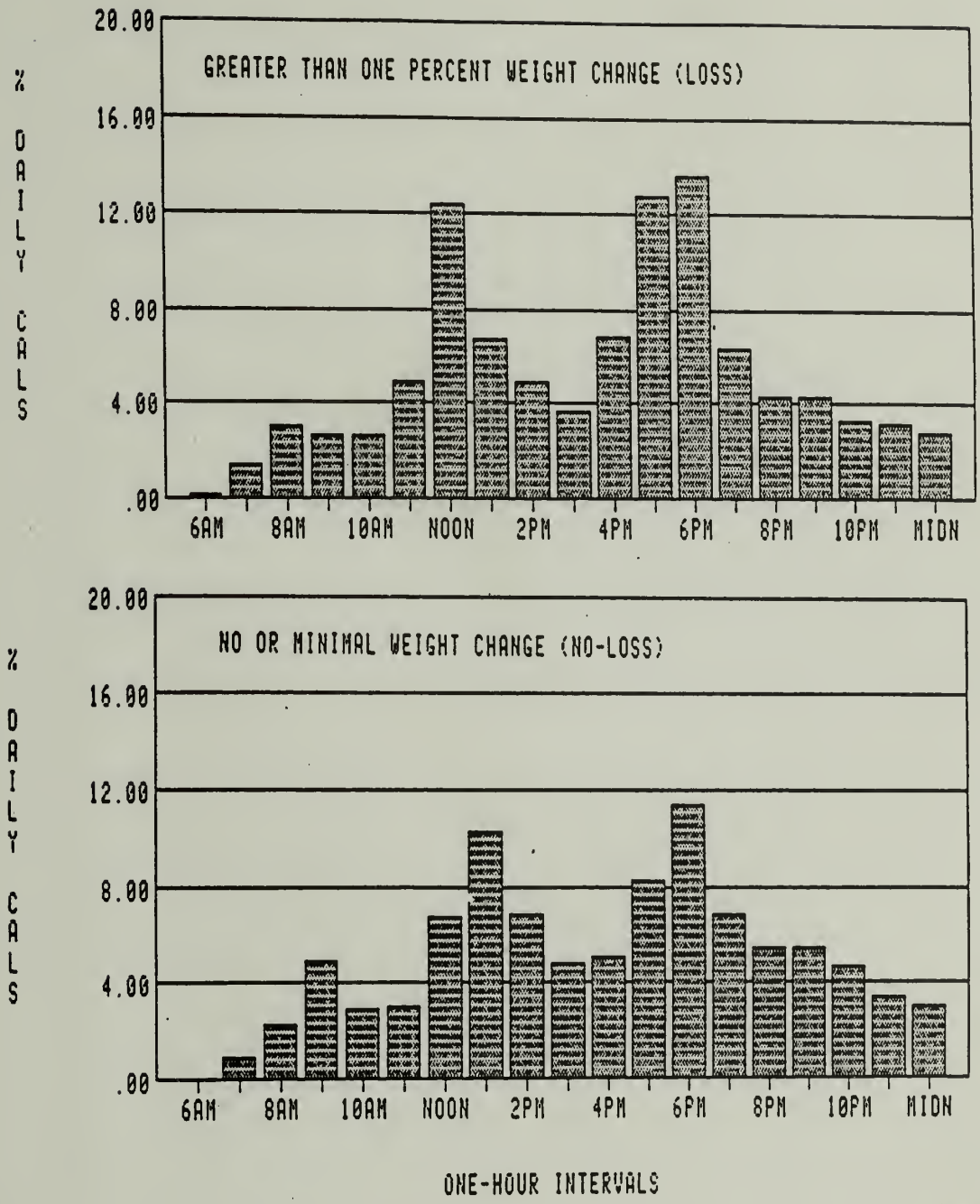

Figure 2. Hourly Caloric Intake for Loss Groups.

The degree of actual change in weight was assessed by taking the difference between the weight obtained at each weigh-in and the subject's weight when they joined the study (the baseline or B0 weight) and expressing this as a percentage of the baseline weight. This procedure sets the baseline (B0) weight at zero and renders the derived percentage change score as a deviation from zero, with positive scores indicating weight gain and negative scores indicating weight loss.

Table 3

Frequency distribution on Weight Loss Status
by Diet Group Instructions

	REST.	SPACE	BOTH	MON.	TOTAL
NO-LOSS	4	4	4	1	13
LOSS	3	2	4	6	15
TOTAL	7	6	8	7	28

CHI-SQUARE = 4.260 D.F. = 3

Table 4

Results of repeated measure Analysis of Variance
on Percentage of Weight Change
by Weigh-in (TRIAL) and by Diet Instructions Group (GROUP)

SOURCE	DF	SS	MS	MS ERR	F
GROUP	3	342.302	114.101	SUBJ	0.987 (3,24)
TRIAL	14	306.442	21.889	S/R	0.919 (14,336)
GT	42	1018.530	24.251	S/R	1.018 (42,336)
SUBJ	24	2774.390	115.600		
S/T	336	8001.850	23.815		

Table 4 shows the results of a repeated measures Analysis of Variance procedure performed on the percentage weight change at each

weigh-in. This yielded no significant effects for either the Diet group (Group) or the Weigh-in (Trial) main effect, as well as, a similarly non-significant effect for the interaction between the two (GT). This procedure failed to provide support for the major hypothesis that the Distribute Calories and/or the Both Instructions Diet Group would more effectively lose weight. In fact the obtained results do not support the existence of any difference between Diet Groups on the weight loss dimension as an additional analysis of the percentage change in weight at termination of the formal study also revealed no effect for Group.

Follow-up data obtained approximately four weeks following termination of the study also failed to yield any significant differences attributable to Diet Group membership.

In order to test the possibility that the findings so far reported were due to an overall failure to obtain significant differences in weight loss even among those who did lose weight, a similar analysis was obtained on a two group comparison where the subjects were divided on the criterion of showing at least a one percent overall loss in weight (Loss). Ideally, a two-way ANOVA would have yielded this comparison more effectively and additionally allowed an assessment of the differences in loss as they interacted with Diet Group instructions. However, owing to the nature of the frequencies obtained for the interaction between the two variables (see Table 3) a two-way ANOVA was impossible, as the interaction cell frequencies vary too greatly to allow meaningful analysis (Myers, 1966).

A one-way repeated measures Analysis of Variance was performed on

A one-way repeated measures Analysis of Variance was performed on the dichotomy of Loss status and the results of this analyses are shown in Table 5.

Table 5

Results of repeated measures Analysis of Variance on Percentage of Weight Change by Weigh-in (TRIAL) and by Loss Status (LOSS)

SOURCE	DF	SS	MS	MS ERR	F
LOSS	1	1356.810	1356.810	SUBJ	20.045 (1,26)
TRIAL	14	306.442	21.889	S/R	0.924 (14,364)
LT	14	399.286	28.520	S/R	1.204 (14,364)
SUBJ	26	1759.880	67.688		
S/T	364	8621.090	23.684		

The results of this analysis yield a significant main effect for weight loss status (Loss) (f = 20.045, p. = .0001) but no significant effect for either Weigh-in (Trial) or the Loss by Weigh-in (LT) interaction. Figure 3 provides a graphic representation of these findings. As is easily noted, the two groups do differ from each other. The lack of a significant interaction effect fails to provide support for the hypothesis that the two groups differed in their pattern of weight loss. We may be confident that the lack of significant effects for Diet Group membership are not due to an overall failure to lose weight. It is also worth noting that the two Loss groups were clearly beginning to differ when they received the Diet Group instructions suggesting that their weight loss was independent of those instructions.

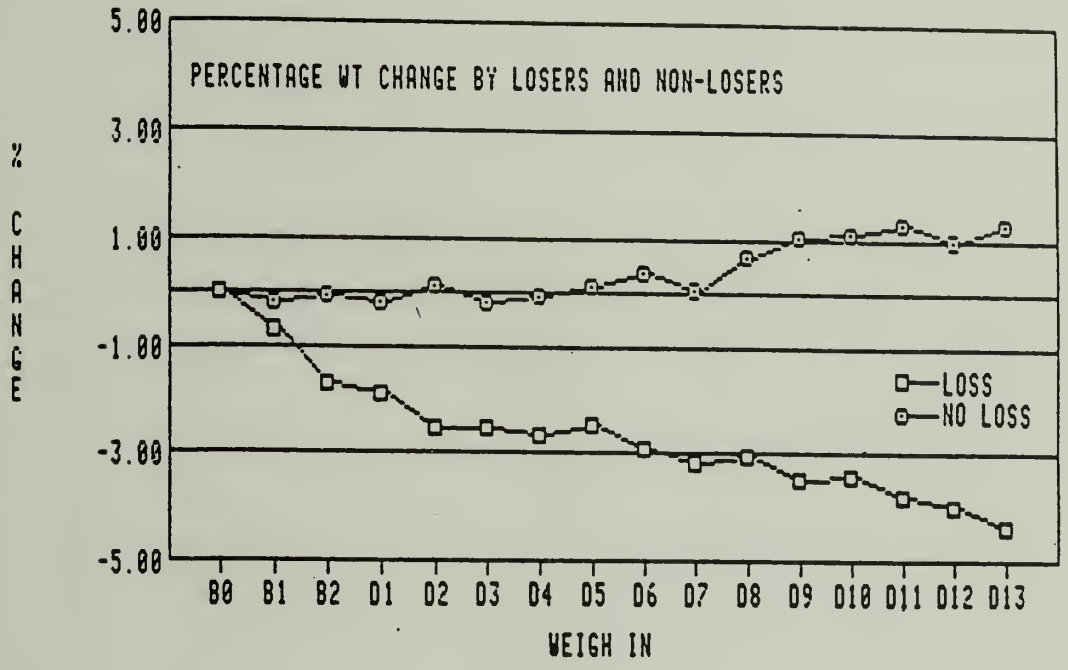

Figure 3. Percentage Weight Loss for Loss Groups.

The hypothesis that fluctuations in weight differed according to Diet was tested by obtaining the standard deviation of weight change for each subject and submitting these to an Analysis of Variance procedure. This analysis was completed both for Diet groups and Loss Groups, yielding non-significant effects for each.

Summary of Weight Loss Findings

No findings were obtained which would indicate any differential effect of Diet Group instructions on actual weight loss. The finding that the Loss groups, created by dividing the sample on the criterion of 1% weight loss, did differ significantly on percentage weight loss suggests a potentially useful classification scheme for additional post-hoc analysis. This further suggests that the failure to obtain significant findings regarding Diet group differences is not due to an overall failure to lose weight. Despite the lack of significance, the fact that all but one of the seven Monitor Only group members did successfully lose weight is consistent with the finding that the subjects who lost weight seemed to do so independently of Diet Group instructions.

A Change in Method

At this point, it was decided that subsequent tests of the formal hypotheses could be augmented by an investigation of possible differ-

ences in the data which might emerge from analyses on the Loss criterion. This would have the dual benefit of validating the particular variables chosen for the analyses as actually being relevant to weight loss, and be a potential source of hypotheses for future research. Toward this end all subsequent analyses will be completed on both Diet group (Group) and Loss Status (Loss).

Calorie and Carbohydrates

Caloric intake

An Analysis of Variance was performed on the average reported daily caloric intake during the baseline and diet phases and on the difference between the phases comparing both Diet and Loss Groups. These findings are of interest primarily because of the stated concerns regarding the reliability of subject self-report.

The Analysis of variance showed no significant effects by Diet Group during either the baseline or the diet phase, or for the difference in average daily caloric intake between the phases.

The results for the Loss groups are shown in Table 6. The loss groups did show significant differences in reported caloric intake during the diet phase (f = 5.699; $p.$ = 0.024). No difference was obtained for reported intake during the baseline phase but the difference in caloric intake between phases was found to be significant (f = 4.681; $p.$ = 0.039). It is reassuring that the subjects who lost weight do report a lower caloric intake during the diet phase and a

greater decrease from baseline intake than do the subjects who did not lose weight. This finding allows somewhat greater confidence regarding the validity of the self-report data.

Table 6

Results of Analyses of Variance on Average Daily Caloric Intake by Loss Status

	BASELINE	DIET	DIFFERENCE
NO LOSS	1777.85	1821.27	43.42
LOSS	1589.46	1368.75	-220.71
GRAND MN.	1676.43	1578.85	-98.08
f (1,26)	0.89	5.67	4.68
p.	NS	0.024	0.039

Fluctuation in caloric intake

The hypothesis that the Diet Groups would differ on the degree of fluctuation in caloric intake was tested by obtaining the standard deviations of each subject's reported daily caloric intake and submitting them to an Analysis of Variance procedure. No difference was observed in calorie fluctuation for Diet Groups during either the baseline phase or the diet phase. Additionally there were no systematic effects by Diet Group on the difference in calorie fluctuation between the diet and baseline phases.

The results shown in Table 7 suggest no difference in fluctuation by Loss category during the baseline phase, however, a significant difference was obtained between the Loss Groups during the diet phase

with the group which did lose weight showing less fluctuation (f = 6.347; $p.$ = 0.017). There was no difference between Loss Groups in the degree of fluctuation reduction from the baseline to the diet phase.

Table 7

Results of Analyses of Variance on Standard Deviation of Caloric Intake by Loss Status

	BASELINE	DIET	DIFFERENCE
NO LOSS	648.11	650.05	1.94
LOSS	494.56	474.91	-20.24
GRAND MN.	565.85	555.91	-9.94
f (1,26)	1.94	6.35	0.06
$p.$	NS	0.017	NS

A second hypothesis involved differences in modulating intake at a level which would be likely to produce weight loss. Toward this end, the average difference between the reported calorie intake and a 1200 calorie ideal was computed and these differences were subjected to an Analysis of Variance procedure.

No significant effects by Diet Group were obtained during the baseline phase, the diet phase or the difference between the two phases. Table 8 shows the results of the analysis for the Loss Groups. There was no observed difference in the two groups during the baseline phase but a difference was observed during the diet phase (f = 5.709; $p.$ = 0.024). There was also a significant effect by Loss group observed for the difference between the two phases (f = 4.51; $p.$

= 0.041).

The standard deviation of this statistic was obtained as an additional measure of fluctuation, in this case the degree to which subjects fluctuated in their deviations from a 1200 calorie ideal. Results of this analysis demonstrated no significant differences between Diet groups on this measure.

Table 8

Results of Analyses of Variance on Average Daily Deviation from 1200 Calories by Loss Status

	BASELINE	DIET	DIFFERENCE
NO LOSS	584.95	618.90	33.96
LOSS	395.61	167.12	-228.64
GRAND MN.	483.50	376.88	-106.64
f (1,26)	0.88	5.71	4.51
p.	NS	0.024	0.040

Table 9

Results of Analyses of Variance on Standard Deviation of Average Daily Deviations from 1200 Calories by Loss Status

	BASELINE	DIET	DIFFERENCE
NO LOSS	638.18	656.97	18.79
LOSS	487.36	476.16	-11.20
GRAND MN.	557.38	560.11	2.73
f (1,26)	2.15	6.71	0.13
p.	NS	0.015	NS

On repeating this procedure for the Loss groups, no difference was found during the baseline phase and a significant effect was ob-

tained during the diet phase (f = 6.705; p. = 0.0153). The result of the Analysis of Variance on the difference between the two phases was not significant. These results are shown in Table 9.

Carbohydrate intake

Carbohydrate Intake was assessed by obtaining the average percentage of daily caloric intake which was accounted for by carbohydrate content and subjecting these percentages to an Analysis of Variance procedure. This analysis revealed no significant effects due to Diet Group in either the baseline or diet phase. Similarly, no significant effect was observed in the difference between the two phases.

Results of Loss group analyses are shown in Table 10.

Table 10

Results of Analyses of Variance by Loss Status on Percentage of Daily Caloric Intake Accounted for by Carbohydrate Content

	BASELINE	DIET	DIFFERENCE
NO LOSS	47.41	45.56	-1.85
LOSS	44.01	44.79	0.78
GRAND MN.	45.59	45.15	-0.44
f (1,26)	5.07	0.22	3.36
p.	0.033	NS	0.077

Here we see a reversal of the usual trend in that a significantly smaller proportion of caloric intake is accounted for by carbohydrate content in the weight losers group during the baseline phase (f = 5.067; p. = 0.033). No significant effect is observed on this measure during the diet phase. The analysis of the between-phase differences

suggests a reduction among the non-losers on this measure and a slight increase among the losers which approaches but does not obtain statistical significance (f = 3.36; p. = .07). It would appear that the existing difference in the proportion of caloric intake accounted for by carbohydrate content somehow disappeared during the diet phase.

Types of carbohydrate

It was reasoned that these results might be due to differences in the quality of carbohydrate which was being consumed. Therefore, a breakdown of carbohydrate percentages was calculated on the basis of ratings of the individual meals.

The ratings on which this breakdown is based were assigned after the coding process had been completed. Each meal was rated as either a good diet meal (balanced healthy food, usually no white sugar products), a mixed meal (generally balanced but with questionable components, i.e. sugar deserts; or, generally substantial food but not balanced, i.e. "a burger and fries"). A third category was that of junk food, and included candy bars, chips, etc.. A fourth category of beverages, included coffee, tea, diet soda (regular soda was classified as "junk" because of its sugar content), fruit juices, etc.. Finally, alcoholic beverages constituted a fifth category of their own.

The results of the Analysis of Variance by diet group on the average percentage of calories accounted for by carbohydrates of the good, mixed, junk, beverage and alcohol categories revealed no effect

by Diet group.

Table 11 shows the results of the same analyses performed for the Loss Groups. We see that a higher percentage of calories was accounted for by the "good" category of carbohydrates among the "losers" (f = 6.885; $p.$ = 0.014). In the other categories, intake was approximately the same for both groups, although there is a trend suggesting a lower percentage of caloric intake accounted for by the "junk" category among the losers which approaches but does not obtain significance (f = 3.98; $p.$ = 0.058).

Table 11

Results of Analyses of Variance by Loss Status on Types of Carbohydrate Content

	GOOD	MIXED	JUNK	BEV.	ALC.
NO LOSS	17.88	16.00	7.04	1.18	2.65
LOSS	24.86	11.98	3.71	1.39	1.74
GRAND MN.	21.62	13.85	5.26	1.29	2.17
f (1,26)	6.89	3.10	3.92	0.14	1.28
$p.$	0.014	0.090	0.058	NS	NS

Similarly, a somewhat lower proportion is obtained by the losers in the "mixed" category which again approaches but does not obtain significance (f = 3.104; $p.$ = 0.0899). It would appear that the finding of no change in intake of carbohydrates among the "losers" and the finding of no difference between loss groups on Carbohydrate percentage during the diet phase may be tempered by appeal to the type of carbohydrate consumed.

Summary of Calorie and Carbohydrate Intake

No effects by Diet group were observed on any of the measures of Calorie and Carbohydrate intake. Analysis of the differences between Losers and Non-losers suggest that, in general, weight loss was associated with reduce calorie consumption, reduced fluctuation in calorie consumption, and reduced deviation from an intake of 1200 calories. Additional findings indicated that weight loss was associated with a higher proportion of "good" quality carbohydrate intake.

Moods and Mood Fluctuation

The hypothesis that differences in affect would be observed among the Diet Groups was tested by obtaining the average mood rating for each subject and submitting these to an Analysis of Variance procedure. No effect by Diet group was observed on any of the ten mood dimensions. A similar analysis by Loss status also revealed no significant effect.

The hypothesis that mood would fluctuate differently among the different groups was tested by calculating the Standard Deviations of each of the ten mood dimensions for each subject. These standard deviations were then compared using an Analysis of Variance procedure. No differences by Diet Group were obtained on any of the mood dimensions. A similar analysis was completed for the Loss groups and, again, no significant effect was obtained on the ten mood dimensions.

One difference which approached significance was obtained on the "Worried/Secure" dimension (f = 3.957; p. = 0.057). The non-losers average S.D. was 1.63 and the Losers was 1.31. However gratifying this particular glimmer of significance might seem, in the context of the number of analyses (20) completed on this particular subset of the data, this one difference is probably best seen as occurring by chance.

Correlation coefficients of mood and mood fluctuation with calorie intake, calorie fluctuation and carbohydrate content were obtained in order to test a sub-hypothesis that these might be related. Because of the large number of correlations being performed the criterion for acceptability was set at $p.< 0.01$. The results of these correlations failed to support the existence of a relationship between these variables.

Cue Salience

Twenty-two of the 28 subjects who completed the diet study also completed the second administration of the Cue Salience test. Of these, six were from the Restrict Calories group, three from the Distribute Calories group, seven from the Both Instructions group and six from the Monitor Only group. On the Loss dimension, the sample divided evenly with eleven subjects in each group.

Results of the Cue Salience test were analyzed by obtaining a "Recall Percentage" score in each Cue category for each subject. This

was accomplished by dividing the number of words recalled in a particular category by the number of words recognized in that category. This calculation was completed separately for each administration of the test. A new statistic, the "Recall Difference Percentage", was generated by subtracting the Recall Percentage for each Cue category on the second administration from its complementary Recall Percentage obtained from the first administration. This procedure yielded scores wherein a positive number indicated increased salience and a negative number indicated decreased salience. A repeated measures Analysis of Variance procedure was then performed on the Recall Difference Scores.

It should be pointed out that the Analysis of Variance by Diet group could be expected to yield suspect results owing to the degree of inequality in the cell frequencies (Myers, 1966). As it turned out, no main effect for Diet group was obtained in this procedure, nor was an effect obtained for the Diet by Cue interaction. An examination of the cell means confirmed that this finding was most likely an accurate description of the obtained differences. A significant main effect was observed for Cue category (f = 2.91; p. = 0.043). These data, thus, fail to support the hypothesis of changes in Cue salience associated with Diet Group.

Table 12 shows the results of the Analysis of Variance procedure completed on Loss status.

A significant main effect for Cue category was obtained (f = 3.33; p. = 0.025). The main effect for Loss approached but did not obtain significance (f = 3.05; p. = 0.096). No effect was obtained for the

Loss by Cue category interaction.

Table 12

Results of repeated measure Analysis of Variance on Recall Difference Percentage by Cue Type (CUE) and Loss Status (LOSS)

SOURCE	DF	SS	MS	MS ERR	F
LOSS	1	2033.280	2033.280	SUBJ	3.043 (1,20)
CUE	3	5419.310	1806.440	S/R	3.327 (3,60)
LC	3	2255.940	751.979	S/R	1.385 (3,60)
SUBJ	20	13365.200	668.259		
S/C	60	32577.000	542.950		

Table 13 shows the cell means obtained from this analysis.

Table 13

Recall Difference Percentage Broken Down by Cue Type and Loss Status.

		FOOD	EMOTION	NEUTRAL	BASELINE	MEAN
NO-LOSS	(11)	-15.455	-9.091	0.818	-4.727	-7.114
LOSS	(11)	4.273	-12.455	20.091	-1.909	2.500
MEAN	(22)	-5.591	-10.77	10.45	-3.32	-2.307

Post-hoc comparisons of these differences were obtained for each of the cue categories using the Newman-Keuls test (Pagano, 1981). The criterion of acceptability was set at 0.05 and the critical value of "Q" was determined to be 2.83 for adjacent means and 3.40 for non-adjacent means. The observed increase in the recall of Neutral words was significant as compared with Emotion words (Q = 3.02), and all other comparisons were determined to be non-significant. It is worth

noting that the Loss and No Loss groups differed in the category of words where decrease was noted. The No-Loss group decreased its rate of recall on all categories with the largest reduction in Food related words, whereas the Loss group shows its greatest reduction in recall to Emotion related words with an almost equal increase in recall of Neutral words. The Loss group, in contrast, showed a slight increase in recall rate across all categories. While these differences are interesting, it must be remembered that the interaction effect was not significant and as such these findings are, at best, inconclusive.

Halfway Follow-up

Each subject was contacted by telephone at the halfway point in the study in order to assess the subject's understanding of and compliance with the diet instructions and to provide a qualitative assessment of each subject's progress and subjective reactions to the diet. Inquiry included the subject's satisfaction with rate of weight loss and the specific diet plan they were following. All of the calls were made by this writer, and the inquiry was completed without access to information regarding either Diet group membership or actual weight lost.

No differences were obtained as to reported compliance with the diet instructions. All but one of the subjects claimed to follow the assigned diet but to "occasionally go off". Subjects uniformly were dissatisfied with their degree of weight loss, regardless of how much

weight they had lost (or gained).

A number of subjects whose instructions included eating a morning meal reported that they had noticed that on days when they ate breakfast they felt very hungry both at lunch-time and dinner-time. They reported that they "felt as though they ate more" on these days even though an examination of their records indicated that they had eaten less and they "didn't seem to be hungry at night as much".

The only group which consistently reported liking their diet was the Monitor Only group. This is not surprising as the diet this group followed was of their own design.

CHAPTER IV

DISCUSSION

The study undertaken here had two major goals. The first was to develop and validate a method for the study of eating patterns. The second was to test some specific hypotheses about the effect of some specific eating patterns in facilitating the weight loss process. The argument will be made that this study was successful inasmuch as concerns the first goal. That is, that the method was developed and validated and that it holds promise for providing results which will constitute a substantive contribution to the study of eating.

As regards the second goal, that of testing the specific substantive hypotheses, it may be seen that these did not yield the expected information. Simply put, there were no effects due to Diet group observed on any of the variables under investigation. This is not to say that nothing was learned, only that the questions which this study was designed to answer were not answered. It is argued here that the questions raised and clarified by these results compensate for the failure to answer the stated questions.

At least two explanations are possible for this lack of experimental effect. The first and most obvious explanation is that patterns of eating have no effect on weight loss or any other aspect of the eating process. The second explanation is that this experiment

did not actually evaluate eating patterns and that these results are not relevant to the theoretical notions involved. Support for this second explanation is provided by the analysis of the obtained eating patterns.

It seems clear that the subjects in the Distribute Calorims and Both Instructions groups did not adhere to the instructions that they received. In fact, the Distribute Calories group evidenced the greatest degree of calorie massing. Neither group evidenced much caloric intake in the morning hours, as they were instructed, and showed no difference from the other groups in the percentage of calories consumed prior to 11:00 AM. Overall the intake patterns of all four groups were similar and differed only in the degree of massing during the two-hour intervals beginning at Noon and 5:00 PM.

The Diet group instructions were designed to produce different and specific eating patterns, the true independent variable in this study, in the different groups. As there were effectively no between-groups differences in eating patterns, relevant to the theoretical concerns at issue in this experiment, it does not seem at all surprising that little, if any, effects emerged in the dependent variables. As such it would appear that the most conservative conclusion which may be drawn about these data is that this is not a sufficient or legitimate test of the theoretical hypotheses.

It is of interest, however, that the one subject who did consume, on the average, approximately 25% of total caloric intake prior to 11:AM did lose weight, and reported a lower rate of intake after 7:00

PM than was average for her group (Distribute Calories). This, taken with the subjective reports obtained at the halfway point, which indicated that subjects experienced internal hunger sensations more acutely when they had eaten breakfast, might suggest that further investigation of these variables is warranted.

It would appear that compliance with a dietary regimen has emerged as a determining feature of this study and this will be discussed in detail in a later section. Little attempt, other than encouragement, was made to insure compliance and it is not clear what else might have been done.

For reasons of experimental control, the research assistants conducting the weigh-ins and collecting the records did not know what diet a given subject was following and did not know the rationale behind the diet. The principal experimenter, this writer, knew which diet a given subject was on, but had as little contact with the daily records and bi-weekly weigh-ins as possible, again for reasons of experimental control.

Future studies designed to investigate these issues should build in some method of ongoing assessment and control of regimen compliance in such a way as to not bias the results. A post-hoc validation of the independent variable, while necessary, can be seen to be insufficient.

One potentially useful method for assessing and for potentially intervening in compliance with eating pattern instructions would be to include in the standard set of self-report data a report of waking

time. This would allow a computation of latency to the first meal, in itself a useful statistic, on the basis of which compliance with this instruction could be monitored on an ongoing basis. This tactic would allow the calculation of the interval between meals, another potentially useful statistic, using waking time as a starting point in the calculation. This statistic was impossible to compute in this study because the differences were washed out by the time spent sleeping which was figured into the calculation.

The Alternative Experiment

Although no meaningful test may be made of the stated hypotheses, it remains that 15 of the 28 subjects completing the study did lose at least some weight and it is interesting to speculate as to this finding. Given the homogeneity of the formal independent variable, it was reasoned that the twenty-eight subjects behaved as a single sample and that analyses based on other classification schemes could be independently implemented.

This was the rationale taken in postdictively dividing the sample along the empirically derived dimension of actual weight loss. It was clear that this, at least, was a valid independent variable. The method was similar to that of a longitudinal study where a group is followed over a period of time and hypotheses are empirically derived. The weakness of this type of study and by extension of the Loss results in this study is that causal relations are difficult, if not

impossible, to demonstrate. As regards this experiment, the effects obtained from the analyses based on the loss/no loss dimension might, at most, be considered as associated with weight loss. These findings were to be considered as the source of hypotheses to be submitted to formal test in future research.

An objection can be raised, on methodological grounds, to this tactic in that by effectively doubling the number of analyses performed on the same data set one increases the probability of Type 1 error. Alternatively, it can be argued that, with the exception of the Cue Salience data, the analyses to be considered here were performed on statistics (either means or standard deviations) derived from repeated measurements on the same subject. As such the numbers submitted to the analyses were very stable. On this basis, one might argue that the .05 criteria for acceptability be relaxed.

As it turns out, most of the analyses on the Loss dimension reported here would have met a .025 criteria for acceptability and there were only rare cases where comparisons came close but did not obtain significance. In general, where no difference was found the obtained *f* statistic was extremely low and often less than 1.0. This may be seen to be an effect of using such stable numbers in the analyses.

Given these considerations, the criterion for acceptability was maintained at 0.05 for all analyses, except the correlations attempted for the mood variables.

Factors Associated with Weight Loss

It is no surprise that the group which lost weight also evidenced a lower average daily calorie intake. This finding is consistent with the conventional wisdom that "you lose weight by eating fewer calories", adding little more than this to our understanding of weight loss and gain. Similarly, the finding that weight losers evidenced less fluctuation in their daily intake and deviated from 1200 calories to a lesser degree than those who did not lose weight does little to improve our knowledge base. This latter finding is, however, potentially of interest in that presumably this includes deviations which fall below the 1200 calorie mark. It seems plausible that the weight losers are less likely to deprive themselves as well as less likely to gorge. It would seem from these data that there exists a relationship between the two extremes.

It would be interesting to study the modulation process specifically, both tc confirm these suspicions and to understand better the processes involved in caloric intake modulation. It seems that the weight losers do, in some way, modulate their caloric intake, but it is beyond the scope of this project to identify the relevant factors.

What is useful about these findings is that they confirm the ability of the methods and techniques devised for this experiment to identify and demonstrate relations which may be of interest in further study. Clearly, stable and reliable effects were generated and confirmed by use of these methods. From this standpoint, it is possible

to view the Loss findings as a sort of "benchmark" test of this approach. It was not only possible to identify differential effects due to an independent variable with parameters known to be valid (Loss), but it was also possible to identify the invalidity of an independent variable for which the parameters were unknown.

It is also useful to know that despite the potential for bias and poor reliability introduced by using self-report in the sampling process, findings could be derived which were consistent with what was known about the weight loss process.

Carbohydrates

The findings regarding the carbohydrate component of caloric intake are of some interest. Conventional wisdom, at least in some quarters, has it that weight loss requires a reduction in carbohydrate intake. These data would seem to support the contrary notion that an increase, at least in certain kinds of carbohydrate content is associated with weight loss. These findings are equivocal in that what was measured was the percentage of total intake accounted for by the carbohydrate component. Given this, it is also possible that the operational factor is not carbohydrate increase, but a reduction in either fat or protein content (whose sum is the reciprocal of carbohydrate proportion). A reduction in either of these would have the effect of increasing the proportion of caloric intake attributable to carbohydrates.

It is true that the weight losers evidenced a greater degree of intake of "good" carbohydrates and that a larger proportion of daily caloric intake was accounted for by carbohydrate content, but it is also the case that many of the foods classified as "good" also have lower fat contents. Alternatively, while some of the foods classified as "junk" are almost totally composed of carbohydrate (i.e. sodas, candy) there are also a number which have high fat contents (i.e. potato chips, "twinkies") so an appeal to the difference between the rate of "junk" and that of "good" food consumption will still not resolve the issue.

The use of absolute measures of carbohydrate intake also would not resolve the issue as this approach could not control for overall caloric intake which has been seen to differ between groups. Additionally, the variable of interest here is the proportion of caloric intake which is accounted for by carbohydrates rather than the carbohydrate magnitude . A more appropriate answer would be to develop a rating system which took fat content into account (i.e.: subdivide "junk" foods into "high fat junk" and "low fat junk" categories) or to obtain a measurement, complementary to the carbohydrate proportion, of proportion of caloric intake accounted for by fat content.

An equally good case may be, and has been, made for the hypothesis that either fat reduction or increased consumption of complex carbohydrates (usually identified as the "good" carbohydrates) is the relevant factor in weight loss. Further research is needed to clarify this finding and it would seem that this is a finding that is worth

investigation.

Moods

The failure to find any differential effect on moods is surprising given the reports of dieting depression syndrome and the conventional wisdom that dieting is an emotionally wrought undertaking. A number of interpretations of this lack of effect are possible. The first is, of course, the null hypothesis that weight loss dieting and weight loss itself has no differential effect upon mood. The argument here would be that this is an explanatory fiction designed to excuse lack of weight loss. That is, that it is less upsetting, both to the dieter or the weight loss-counselor, to attribute failure to emotional factors as opposed to lack of compliance or discipline on the part of the dieters, or lack of knowledge on the part of those claiming expertise in the weight loss process.

Another possible explanation has to do with the method of measurement of mood. It is possible that the ten-dimension scale used in this study, either fails to record mood changes with enough sensitivity to allow detection of fluctuation patterns. Alternatively, it is possible that the dimensions measured by this scale are simply the wrong ones, that is, they are not appropriate to the mood or emotional dimensions actually experienced by the dieters. The former explanation seems unlikely in that these dimensions have been used previously with some success to monitor day to day fluctuations in mood in other

populations (Epstein, 1979). As to the latter speculation, at least in face validity these dimensions do seem to comprehensively address the emotional dimensions reported as constituting the emotional changes reported by dieters.

Some variation in mood did occur and it is unlikely, although possible, that this variation is completely random. This raises a third possibility and that is that the degree of mood fluctuation is identical among dieters regardless of whether or not they effectively lose weight. If this is the case then one would expect no difference. The question posed by this speculation would have to be addressed through comparison to a group of non-dieters, or even to a group who were not overweight. While it is probable that mood fluctuation is not random, the observed fluctuations may not differ from that fluctuation which is appropriate to everyday life amongst the general population and as such is not a special property of the dieting process.

An alternative approach, beyond the scope of the present study, would be to look within-subject for systematic relations between mood and other factors which vary on a daily basis. For instance, mood might vary with caloric intake, with carbohydrate content or particular carbohydrate type. It might also vary with fluctuations in caloric intake, or with particular numbers of days at a particular level of calorie or carbohydrate intake. These questions might all be addressed in a data set such as the one obtained during this study, but would be rendered more powerful if comparison could be made to a non-dieting group. Clearly, questions related to emotional factors in

weight control constitute an area of investigation much in need of empirical research.

Compliance

It was rather naively assumed that subjects who stated that they wished to lose weight and that they wished assistance in doing so, would also be willing to comply with a diet once one was given. As such, the subjects were provided with a set of instructions and allowed to "free run", with the expectation that this process would provide the independent variation necessary to test the hypotheses stated in this study. This, obviously, was not the case. A number of factors can be identified which might have compromised compliance.

First, the subjects in this study were all students and most of them lived in dormitories. As such, they were locked into eating schedules which were defined by Dining Common policy. A special effort would be required to comply with instructions regarding meal frequency, particularly those involving a morning meal. For the student in the habit of waking only in time to make a mad dash for an early class, the effort might be too much to be expected without some specific intervention. This situation would require a greater commitment to following the regimen than was likely given the minimal external controls provided by this study's program.

A related issue is that we are attempting to intervene in established and well-practiced patterns of behavior related to eating; hab-

its, as it were. As such, the conventional wisdom of old habits dying hard might be seen to apply. It might not have been wise to attempt to provide experimental control at the expense of direct feedback regarding compliance and direct interventions to support behavior change. It would seem that behavior change must be explicitly programmed into a study such as this in order to assess effectively outcomes related to those specific changes in behavior. An explicit program of feedback and direct intervention would more likely provide motivation to overcome the behavioral inertia observed in this study.

Toward this end, a revised procedure might be proposed in which the subjects were "proctored" throughout the study. This could be provided through the use of a standard form (separately prepared for each group) which listed the specific areas of compliance and would be checked off following a review of the subjects daily record. Use of a standard form would minimize differential feedback owing to experimenter expectation. The weigh-ins could still be completed by a different person who did not know the diet specifics or the hypotheses at issue. These strategies would provide at least minimal experimental control. The specifics of these procedures can be worked out at a future date, but it remains obvious that an investigation of this type is impossible without some ongoing validity check of the independent variables.

Cue Salience

Results of the Cue Salience test suggest that the subjects in this study, regardless of Loss category were less sensitive to emotion cues and more sensitive to neutral cues at the second administration of the test than they were at the first administration. There was also a trend suggesting differential changes in cue sensitivity associated with weight loss status. These results would suggest that cue sensitivity findings, such as those reported by Schachter (1973;1971) and Rodin (1977), represent a dynamic process rather than a static characteristic or trait of the overweight dieter.

These conclusions must be tempered with the recognition of certain methodological constraints on interpretation of these data. First, the cue words in the two lists employed in this study may have had different properties which might account for the differences. This possibility is being investigated in a separate study. A second objection might be that the subjects determined the hypothesis of the study and specifically attempted to recall the neutral words. This objection, while plausible and difficult to directly assess, is not consistent with the minimal change in recall of baseline words (the words projected at an increased light intensity with a reduced intensity masking stimulus). If subjects were actively attempting to respond only to neutral stimuli, one would expect an increased recall rate for the baseline words collateral with the increase in rate for the neutral words. While this is not a conclusive test, it does ren-

der the results somewhat more credible.

Of additional interest here is the almost significant trend toward a difference in recall rate on the food cue words between the Loss groups. The weight losers showed a trend toward slightly increased recall, accompanied by a decrease in recall of the emotion words, while the non-losers showed a sizable decrease in recall of the food words. This finding, while certainly not conclusive, is consistent with the hypothesis that increased deprivation (presumably existing in the weight-losers group) would lead to higher salience.

It is not clear what might account for the increase in recall rate to neutral words following seven weeks of dieting. One possibility is that by this time in the process, the subjects had satiated in their preoccupation with food and were thus simply less susceptible to food related stimuli. This would not explain the reduction in recall rate to the emotional words.

Another possible explanation, along similar lines, is that as the second administration occurred at the end of the semester, when stress levels presumably were high, neutral words might have been more salient because the subjects were most deprived of neutral stimuli.

Admittedly, this speculation is a bit far-fetched, but some hypothesis regarding environmental or experiential control of cue susceptibility remains as the most plausible explanation of these findings. In any event, these data seem to confirm the notion that cue susceptibility is not a static process intrinsic to the individual, but is rather a dynamic process most likely under the control of envi-

ronmental factors.

These results are not directly comparable to those reported by Schachter (1973;1971) and Rodin (1977) inasmuch as the studies there reported did not use a recall rate measure but rather used simple measures of recognition or recall. Additionally, the data obtained in this study are not based on comparison with normal weight subjects as are the studies reported by those authors. A study designed to provide comparisons similar to those reported in the above noted literature has been separately undertaken and should yield findings which will resolve some of the concerns raised regarding these findings as well.

Conclusions

In retrospect, the present study seems to have been premature. There is too little known of the parameters of eating behavior and weight change to allow for substantial and meaningful conclusions to be drawn. An alternative, more appropriate, preliminary study would have involved an attempt to identify and define some base-rates or parameters of the eating process amongst weight loss dieters as compared to normal weight individuals.

A review of the literature suggests no systematic attempts to study the differences among normal and overweight subjects with the goal of clearly identifying behavioral and situational differences between the two groups. We do not know much about how normal weight

people eat, how much they eat, when they eat or what they eat. Until we do, we will probably not understand much about the overweight individual or the process of weight loss.

A simple study which would have yielded the same validation of the method as well as some useful parametric data would have been to simply follow a group of overweight subjects and a group of normal weight subjects, using much the same procedure as was followed in this study, with a goal of identifying some factors on which the two groups differed. A study such as this would provide both a firm empirical basis for further research and a set of relevant dimensions from which to derive more productive hypotheses.

This study has yielded a method which has been shown to be capable of addressing the intricacies of eating behavior and providing a relatively fine-grained analysis of that behavior. Continued development of these tools shows promise of eventually allowing the identification and definition of the relations between aspects of the eating process and between those aspects and the environmental influences which might affect them. While it is true that the method failed to successfully address the stated hypotheses, it was clearly successful in identifying the constraints which prevented more definitive tests of those hypotheses. There is a great potential for future research even within the data-base generated during the present study.

Additional to this is the evolution of a microcomputer implementation which makes feasible both the management and analysis of the massive quantities of data which can be generated while not sacri-

ficing grain or accuracy. These programs are largely portable between systems and therefore allow for easy distribution to other investigators who may wish to obtain the listings.

There is a need in the field of eating research for advanced methods which are capable of identifying systematic relations in more exact detail. Too little is known about the processes of eating and weight maintenance. It is often not clear what questions need to be asked and often difficult to answer adequately questions which have been identified. It is hoped that the methods developed for this study constitute a step in the direction of remediating that situation.

REFERENCE NOTES

1. Harmatz, M. G. Diet History, emotional state, and range of reinforcers. Unpublished research, University of Massachusetts, Amherst, 1982.

REFERENCES

Abramson, E. E. A review of behavioral approaches to obesity. In J. P. Foreyt (Ed.), Behavioral Treatments of obesity. New York: Pergamon, 1977. (a)

Abramson, E. E. Behavioral approaches to weight control. New York: Springer, 1977. (b)

Beller, A. S. Fat and thin. New York: Farrar, Straus and Giroux, 1977

Bliss, E. L. & Branch, C. H. Anorexia nervosa. New York: Hoeber Co., 1960.

Brobeck, J. R. Mechanisms of the development of obesity in animals with hypothalamic lesions. Physiological Reviews, 1946, 26, 541-559.

Bruch, H. Transformation of oral impulses in eating disorders: A conceptual approach. Psychiatric Quarterly, 1961, 35, 458-481.

Bruch, H. Eating Disorders. New York: Basic Books, 1973.

Cautela, J. R. & Kastenbaum, R. A reinforcement survey schedule for use in therapy, training, and research. Psychological Reports, 1967, 20, 1115-1130.

Coons, E. E., Levak, M., & Miller, N. E. Lateral hypothalamus learning of food-seeking response motivated by electrical stimulation. Science, 1965, 150, 1320-1321.

Curtis, H. Biology (2nd ed.). New York: Worth, 1975.

Dwyer, J. H., Feldman, J. J., & Mayer, J. The social psychology of dieting. In N. Kiell (ed.) The Psychology of obesity: Dynamics and treatment. Springfield, IL: Charles C. Thomas, 1973.

Epstein, S. The stability of behavior across time and situation. In A. I. Rabin, J. Aronoff, A. M. Barclay, & R. Zucker (Eds.) Further explorations in personality (Vol. 2). New York: Wiley, 1982.

Ferster, C. B., Nurnberger, J. L., & Levitt, E. B. The control of eating. Journal of Mathetics, 1962, 1, 87-109.

Foreyt, J. P. Behavioral Treatments of obesity. New York: Pergamon, 1977.

Galton, D. J. The human adipose cell: A model for errors in metabolic regulation. London: Butterworths, 1971.

Garner, D. M., & Garfinkel, P. E. The eating attitudes test: An index of the symptoms of anorexia nervosa. Psychological Medicine, 1979, 9, 273-279.

Goldman, R., Jaffa, M., & Schachter, S. J. Yom Kippur, Air France, dormitory food, and the eating behavior of obese and normal persons. Journal of Personality and Social Psychology, 1968, 10, 117-123.

Griggs, R. C., & Stunkard, A. J. The interpretation of gastric motility: II. Sensitivity and bias in the perception of gastric motility. Archives of General Psychiatry, 1964, 11, 82-89.

Gross, L. The effect of early feeding experience on external responsiveness. Unpublished doctoral dissertation, Columbia University, 1968.

Hashim, S. A. & Van Itallie, T. B. Studies in normal and obese subjects with a monitored food dispensing device. Annals of the New York Academy of Sciences, 1965, 131, 1965, 654-661.

Herman, C. P., & Mack, D. L. Restrained and unrestrained eating. Journal of Personality, 1973, 43, 647-660.

Hutchinson, R. R. By products of aversive control. In W. K. Honig & J. E. R. Staddon (Eds.), Handbook of operant behavior. Englewood Cliffs, NJ: Prentice-Hall, 1977.

Keys, A., Brozek, J., Henschel, A., Michelson, O., & Taylor, H. L. The biology of human starvation,. Minneapolis, MN: University of Minnesota Press, 1950.

Levitt, H. & Epstein, S. Influence of hunger on learning and recall of food related words. Journal of Abnormal and Social Psychology, 1962, 64, 130-135.

Meerloo, J. A. M., & Klauber, L. D. Clinical significance of starvation and oral deprivation. Psychosomatic Medicine, 1952, 14, 491.

Miller, N. E., Bailey, C. J., & Stevenson, J. A. F. Decreased "hunger" but increased food intake resulting from hypothalamic lesions. Science, 1950, 112, 256-259.

Mrosovsky, N. Hibernation and the hypothalamus. New York, Appleton-Century-Crofts, 1971.

Myers, J. M. Fundamentals of experimental design: Second Edition. Boston: Allyn and Bacon, 1966

Nisbett, R. E. Taste, deprivation and weight determinants of eating behavior. Journal of Personality and Social Psychology, 1968, 10, 107-116.(a)

Nisbett, R. E. Determinants of food intake in human obesity. Science, 1968, 159, 1254-1255.(b)

Nisbett, R. E. Hunger, obesity and the ventromedial hypothalamus. Psychological Review, 1972, 79, 433-453.

Pagano, R. R. Understanding statistics in the behavioral sciences. St. Paul, MN: West Publishing Co., 1981

Rimm, D. C., & Masters, J. C. Behavior therapy (2nd ed.). New York: Academic Press, 1979.

Rodin, J. Bidirectional influences of emotionality, stimulus responsiveness, and metabolic events in obesity. In J. D. Maser & M. E. P. Seligman (Eds.), Psychopathology:Experimental Models. San Francisco: Freeman & Co., 1977.

Ross, L. Cue and cognition controlled eating among obese and normal subjects. Unpublished doctoral dissertation, Columbia University, 1969.

Ross, L. D., Pliner, P., Nesbitt, R. & and Schachter, S. J. Patterns of externality in the eating behavior of obese and normal college students. In S. J. Schachter Emotion, obesity, and crime. New York: Academic Press, 1971.

Schachter, S. J. Emotion, obesity, and crime. New York: Academic Press, 1971.

Schachter, S. J. Some extraordinary facts about obese humans and rats. In N. Kiell (ed.) The Psychology of obesity: Dynamics and treatment. Springfield, IL: Charles C. Thomas, 1973.

Schachter, S. J., Goldman, R., & Gordon, A. Effects of fear, food deprivation, and obesity on eating. Journal of Personality and Social Psychology, 1968, 10, 91-97.

Schachter, S. J. & Gross, L. Manipulated time and eating behavior. Journal of Personality and Social Psychology, 1968, 10, 98-106.

Schonitzer, D. L. Stimulus and affective control in weight reduction:Evaluation of spouse participation, restraint and bingeing. Unpublished doctoral dissertation, University of Massachusetts, Amherst, 1979

Schonitzer, D. L. & Harmatz, M. G. A weight loss program aimed at weight maintenance. Current Concepts in Psychiatry, 1977, , 14-20.

Sidman, M. Tactics of scientific research. New York: Basic Books, 1960.

Skinner, B. F. Science and human behavior. New York: MacMillan, 1953.

Staddon, J. E. R. Schedule induced behavior. In W. K. Honig & J. E. R. Staddon (Eds.), Handbook of operant behavior. Englewood Cliffs, NJ: Prentice-Hall, 1977.

Stuart, R. B. Behavioral control of overeating. Behavior Research and Therapy, 1967, 5, 357-365.

Stuart, R. B. & Davis, B. Slim chance in a fat world: Behavioral control of obesity. Champaign, IL: Research Press, 1972.

Stunkard, A. J. Obesity and the denial of hunger. Psychosomatic Medicine, 1959, 21, 281-289.

Stunkard, A. J. The pain of obesity. Palo Alto, CA: Bull Publishing Co., 1976.

Stunkard, A.J., & Mendelsohn, M. Obesity and body image. In N. Kiell (ed.) The Psychology of obesity: Dynamics and treatment. Springfield, IL: Charles C. Thomas, 1973.

Stunkard, A. J.,Van Itallie, T. B., & Reiss, B. B. The mechanism of satiety: Effects of glucagon on gastric hunger contractions in man. Proceedings of the Society of Experimental Biology and Medicine, 1955, 89, 258-261.

Teitelbaum, P. Sensory control of hypothalamic hyperphagia. Journal of Comparative and Physiological Psychology, 1955, 48, 156-163.

Teitelbaum, P. & Campbell, B. A. Ingestion patterns in hyperphagic and normal rats. Journal of Comparative and Physiological Psychology, 1958, 51, 135-141.

Trabasso, T., & Bower, G. H. Attention in learning: Theory and research. New York: Wiley, 1968

Woods, S. C., Decke, E., & Vasselli, J. R. Metabolic hormones and regulation of body weight. Psychological Review, 1974, 81, 26-43.

APPENDIX A

List 1

1. CAKE :
2. CARPET :
3. CONTROL :
4. CHAIR :
5. SPACE :
6. ESCAPE :
7. READ :
8. WORSHIP :
9. NUTS :
10. RETURN :
11. DOOR :
12. SPEAK :
13. CANDY :
14. GORGE :
15. SALAD :
16. ENJOY :
17. DESK :
18. LOOK :
19. CHEESE :
20. LADDER :
21. CATCH :
22. GRASS :
23. LUST :
24. APPLE :
25. STREET :
26. RESPECT :
27. PIE :
28. HATE :
29. TREE :
30. WALK :
31. COAT :
32. BACON :
33. STEAK :
34. SCUM :
35. WAVE :
36. LISTEN :
37. LOVE :
38. BUILDING :
39. BUTTER :
40. ENTER :

List 2

1. BUSH:
2. GLUT:
3. VIEW:
4. BREAD:
5. FEEL:
6. CRACKER:
7. UNCLE:
8. PRAYER:
9. DRIVE:
10. FLEE:
11. FUDGE:
12. SUNSET:
13. ROAD:
14. POWER:
15. FOLDING:
16. CONCERN:
17. PEACH:
18. SHIRT:
19. LOATHE:
20. BEEF:
21. DISTANT:
22. DELIGHT:
23. SAUSAGE:
24. LAMP:
25. STEP:
26. SLIME:
27. COOKIE:
28. THROW:
29. MILK:
30. ENVY:
31. ARRIVE:
32. STAND:
33. TABLE:
34. LETTUCE:
35. SODA:
36. CONSTANT:
37. VISIT:
38. WALL:
39. EASE:
40. FLOOR:

APPENDIX B

Insructions Common to All Groups

As you may have noticed by now, this program focuses on recording your eating patterns, as well as other aspects of your daily life, in great detail. You may have noticed also that simply keeping a detailed record of your eating and coming in to share these records and be weighed has already had an effect on your eating patterns.

This process may have already resulted in some weight loss. We believe that this is an important part of the weight loss process, at least in the early stages, in that you are provided with an opportunity for increased awareness of your eating, as well as an opportunity to share these insights and the resulting progress. It is also important that you record your occasions of failure, as in many cases these will result in even greater awareness of the role that food plays in your life. Additionally, this will allow us to begin to understand more clearly the difficulties in the dieting process, so that we can design procedures that minimize these difficulties.

Distribute Calories Group Instructions

This plan differs from most diet plans in that we will be making no specific recommendations regarding what foods you should eat. We are assuming that you already know how to eat a balanced diet which will result in weight loss but that you have difficulty doing so. As such we suggest that you plan what you will eat but that you follow our recom-

mendation as to when and how you eat.

The most important step that you can take in making this diet work is to minimize your hunger. We have found that your craving for high calorie foods is most easily controlled when you are not hungry. One of the best ways to do this is to divide your food intake into three (or four) regularly scheduled meals. The first of these should be within one half of an hour after waking and should consist of at least one third of your daily eating. Skipping meals is a poor way to lose weight as it intensifies hunger, which intensifies your cravings for food, usually rich food. If you follow a regular meal schedule (a rigid schedule, at first) you may find yourself, after a few days, only thinking about food at your now regular mealtime and you may find yourself fully enjoying, even preferring a low calorie diet.

This may seem awfully simple, perhaps too simple, to work, but we suggest that you give it a try. The evidence suggests that constant hunger and the feelings of having to deprive yourself are related to irregular eating patterns, and further that erratic eating patterns contribute to the maintenance of overweight. Put simply, your feelings of constant hunger are probably real. As such your body may be storing food because it thinks that you are starving, your job is to convince your body that there is plenty of food around.

Monitor Only Group Instructions

This plan differs from most diet plans in that we will be making no

specific recommendations regarding what foods you should eat. We are assuming that you already know what you should eat in order to lose weight but that you have difficulty keeping to that plan. As such we suggest that you plan your own diet and follow it as best you can, while continuing to record your eating behavior and to visit us twice weekly.

Restrict Calories Instructions

As you can see, this is a very flexible, easy to follow diet which allows you to eat some of your favorite high calorie foods. The following exchange lists are provided as a guide to help you in planning your diet. Please, experiment with this diet and generate a plan which is individually tailored to meet your personal preferences, and which works best for you.

Remember, you may make the exchanges you wish, even between categories, and distribute these as you wish (for instance, you may wish to save all of your meat exchanges for dinner, or trade a fruit exchange for syrup on your pancakes). How you distribute your exchanges is up to you, do what works best for you.

Both Instructions Group

As you can see, this is a very flexible, easy to follow diet which allows you to eat some of your favorite high calorie foods. The following exchange lists are provided as a guide to help you in planning your diet.

Please, experiment with this diet and generate a plan which is individually tailored to meet your personal preferences, and which works best for you.

The most important step that you can take in making this diet work is to minimize your hunger. We have found that your craving for high calorie foods is most easily controlled when you are not hungry. One of the best ways to do this is to divide your food intake into three (or four) regularly scheduled meals. The first of these should be within one half of an hour after waking and should consist of at least one third of your daily eating. Skipping meals is a poor way to lose weight as it intensifies hunger, which intensifies your cravings for food, usually rich food. If you follow a regular meal schedule (a rigid schedule, at first) you may find yourself, after a few days, only thinking about food at your now regular mealtime and you may find yourself fully enjoying, even preferring a low calorie diet. This will help immensely in getting the most out of the exchange plan.

Exchange Diet for Restrict Calories and Both Instructions Groups

Food Exchanges: Foods have been divided into five lists: meats, cereals, milk, vegetables, and fruits. Each of the foods within a list, in the amount recommended, is similar in nutrient content and caloric value to all other foods in the same list. For example, both one slice of bread 1/2 cup of cooked rice are on the cereal exchange list. Each of these supplies approximately 70 calories of energy, and they contain similar amounts of carbohydrates, proteins, vitamins, and minerals. Either may be chosen as one cereal exchange.

In addition to the five exchange lists based on food groups, there is a sixth food exchange list- the miscellaneous foods. These include concentrated sources of fats and sugars, as well as alcoholic beverages, which are commonly eaten and enjoyed in modern societies.

It must be pointed out that the nutrient and caloric values of the foods within a food exchange list are not identical; to a degree, exactness is sacrificed for simplicity and convenience. Caloric values, are always approximations.

Until portions of food can be estimated accurately, it is essential to measure or weigh the food before eating. Most foods on the exchange lists can be measured with regular kitchen measuring spoons, measuring cups, or a ruler. Many meat exchanges, however, are listed by weight and may present a problem. A postal scale is inexpensive and easy to use, alternatively, portion weights may be estimated from the weights listed

on the meat packages (for instance, if you by an eight-ounce package of ground beef from the market, and divide it into two roughly equal portions, each portion would be four uncooked ounces, from this you can estimate the cooked weight). The weights the exchanges are for cooked meat, with four ounces of raw meat weighing approximately three ounces when cooked. Most meat exchanges weigh one ounce; however, an average serving portion of meat for dinner weighs three to four ounces, thus equalling three to four meat exchanges.

In summary, then, the six food exchange lists are as follows:

1. Meat exchange list
2. Cereal exchange list
3. Milk exchange list
4. Vegetable exchange list
5. Fruit exchange list
6. Miscellaneous exchange list

Your Basic Food Plan

6 meat exchanges

4 cereal exchanges

2 milk exchanges

3 vegetable exchanges

3 fruit exchanges

3 exchanges from the miscellaneous list

These diets may be modified somewhat to suit individual preferences and budgets. As can be seen in the food exchange lists which follow, each meat exchange provides approximately 75 calories, while each cereal ex-

PLEASE NOTE:

Appendix B is incomplete.
Filmed as received.

UNIVERSITY MICROFILMS INTERNATIONAL.