



Determining the effectiveness of a nutrition intervention program in the treatment of bulimia
by Susan Marie Adams

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in
Home Economics

Montana State University

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Abstract:

The purpose of this study was to investigate the effectiveness of a nutrition program in the treatment of bulimia. The study measured whether treatment (a six-week nutrition program) was more effective than a control (psychotherapy only) group in: (1) decreasing the weekly frequency of binge/purge episodes; (2) increasing nutritional quality of food intake retained; (3) improving body weight and eating attitudes; and (4) maintaining body weight and percentage of body fat in bulimic subjects.

Results showed that both the treatment and control groups decreased the frequency of binge/purge episodes in the bulimic patients; however, treatment was not significantly more effective than control. Treatment was significantly more effective than control in the pre- and post-testing of body weight and eating attitudes and in improving the percentages of the Recommended Dietary Allowances for most of the nutrients analyzed. There was no significant difference found for body weight and percentage of body fat between treatment and control groups.

It was concluded that nutritional intervention helped to decrease the frequency of binge/purge episodes, changed the highly disturbed attitudes of body weight and eating to a lesser degree, and increased the nutritional quality of dietary intake of bulimic subjects without adversely affecting body weight and percentage of body fat.

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IN THE TREATMENT OF BULIMIA

by

Susan Marie Adams

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APPROVAL

of a thesis submitted by

Susan Marie Adams

This thesis has been read by each member of the author's graduate committee and has been found to be satisfactory regarding content, English usage, format, citations, bibliographic style, and consistency, and is ready for submission to the College of Graduate Studies.

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ABSTRACT

The purpose of this study was to investigate the effectiveness of a nutrition program in the treatment of bulimia. The study measured whether treatment (a six-week nutrition program) was more effective than a control (psychotherapy only) group in: (1) decreasing the weekly frequency of binge/purge episodes; (2) increasing nutritional quality of food intake retained; (3) improving body weight and eating attitudes; and (4) maintaining body weight and percentage of body fat in bulimic subjects.

Results showed that both the treatment and control groups decreased the frequency of binge/purge episodes in the bulimic patients; however, treatment was not significantly more effective than control. Treatment was significantly more effective than control in the pre- and post-testing of body weight and eating attitudes and in improving the percentages of the Recommended Dietary Allowances for most of the nutrients analyzed. There was no significant difference found for body weight and percentage of body fat between treatment and control groups.

It was concluded that nutritional intervention helped to decrease the frequency of binge/purge episodes, changed the highly disturbed attitudes of body weight and eating to a lesser degree, and increased the nutritional quality of dietary intake of bulimic subjects without adversely affecting body weight and percentage of body fat.

CHAPTER 1

INTRODUCTION

A growing incidence of eating disorders is being recognized in the United States. A consequence of living in a country with widely available food and few economic restraints, along with the desire to be thin and even underweight, could be the development of an eating disorder. In the last few years, great attention has been focused on two such disorders, anorexia nervosa and bulimia. Anorexia nervosa, described as the self-starvation syndrome, is characterized by severe emaciation and morbid fear of gaining weight. Bulimia is described as the binge/purge syndrome and is characterized by gorging or bingeing on large quantities of food in a short period of time followed by a purging method, such as self-induced vomiting, laxative or diuretic abuse, or self-imposed restrictive dieting or fasting.

Conservative estimates predict that about one-half of those suffering from anorexia nervosa will use bulimia to control or lose weight and that 40% will become bulimic. Whereas anorexia nervosa is relatively rare in our population (approximately 1%), bulimia has been found to occur much more commonly (19% in a college population) (Halmi, 1981).

Dietitians in clinical inpatient or outpatient settings are actively involved in the treatment of persons with these syndromes. Nutritional intervention is used as an adjunct to medical and/or psychological therapy because many of the patients have overt malnutrition

symptoms. Treatment of anorexia nervosa involves aggressive medical and nutritional management to deal with a life-threatening situation. Treatment with the normal weight bulimic individual more often utilizes behavioral approaches. Dietitians will most likely be involved with more bulimic clients than anorexics and will need to implement nutrition care plans with a long-term behavioral component. These plans are necessary because of a common complaint many bulimic patients have: "I have forgotten how to eat."

Purpose

The purpose of this study was to investigate the effect of a nutrition program in the treatment of bulimia. The first question to be answered as: Would nutrition intervention alone be effective in decreasing the weekly frequency of binge/purge episodes in the bulimic patient? The second question was: Would the nutrient intake of the treatment group improve more than in the control (psychotherapy only) group? The third question to be answered was: Would disturbed eating and body weight attitudes be affected by nutritional intervention? And the fourth question was: How would body weight and body fat be affected by the nutrition versus psychotherapy interventions?

CHAPTER 2

REVIEW OF RELATED LITERATURE

The bulimia literature shows that many studies have been made of its incidence and treatment. Most studies of treatment suggest that bulimia can be successfully treated by a variety of psychotherapeutic methods, nutrition counseling as an adjunct, and in some cases antidepressant therapy.

Incidence

As late as 1979, bulimia was described as a rare disorder. Casper et al. (1980) studied the various eating patterns encountered in anorexia nervosa and found that 47% of anorexic patients periodically resorted to bulimia, whereas 53% had achieved weight loss by consistently fasting. The two groups were contrasted with regard to their developmental and psycho-social history, clinical characteristics, and psychiatric symptomatology. Fasting patients were more introverted, more often denied hunger, and displayed little overt psychiatric distress. Bulimics were more extroverted, admitted more frequently to strong appetites, and tended to be older. Vomiting was frequent, and kleptomania was almost exclusively present in bulimic patients who manifested greater anxiety, depression, guilt, interpersonal sensitivity, and had more somatic complaints. These researchers were among the first to point out that bulimia forms a subgroup among patients with anorexia nervosa.

Garfinkel et al. (1980) raised the issue as to whether bulimia should be considered a distinct subgroup. They found bulimic patients had a history of greater body weight than anorexia nervosa patients and were more commonly premorbidly obese. Bulimic patients were those who vomited and misused laxatives. The bulimic group displayed a variety of impulsive behaviors including use of alcohol and street drugs, stealing, suicide attempts, and self-mutilation. With regard to family history, the high frequency of obesity in the mothers of bulimic patients was significant.

In 1981, the American Psychiatric Association included in the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), the diagnostic criteria for bulimia, thus recognizing the disorder as separate from obesity and anorexia nervosa.

The evidence regarding the incidence of bulimia is less well established than that for anorexia nervosa; most studies have relied exclusively on non-validated questionnaires (Szmukler, 1985). Simple questionnaire-based studies have indicated that a large proportion of the population will respond affirmatively to binge-eating. Far fewer fulfill variously modified DSM-III criteria for bulimia. Although these criteria have varied from study to study, 4-19% of females have been reported to meet them (Halmi et al., 1981; Pyle et al., 1981; Katzman et al., 1984; Pope et al., 1984).

Halmi et al. (1981) surveyed 355 college students and found that within the normal college population 13% experienced all of the major symptoms of bulimia as outlined in the DSM-III. Within the bulimic population, 87% were female and 13% were males. Although self-induced

vomiting may accompany other symptoms of bulimia, the results suggested that self-induced vomiting was not a necessary symptom for diagnosis. A significant relationship between laxative use and self-induced vomiting was detected. These purging behaviors occurred in 10% of the students. Individuals who experienced the symptoms of bulimia had a history of overweight or tended to be in the upper portion of their normal weight range (age, sex, and weight corrected) when compared with those individuals who had not experienced the symptoms of bulimia. No significant differences in weight history were noted between vomiters and non-vomiters.

The prevalence of binge-eating and bulimia in 1063 college students in Dublin, Ireland (361 males and 701 females) was studied by Healy et al. (1985). They found 1.1% of males and 10.8% of females met the DSM-III definition for bulimia. Hart and Ollendick (1985) determined the prevalence of binge-eating and bulimia in samples of 139 working and 234 university female students. Bulimia was found to occur in 1% of the working women and 5% of the university women. These findings are in sharp contrast to popular reports that one-half or more of young women suffer from bulimia. Humphries and Wrobel (1983) reported that bulimia occurs in roughly half of obese and anorexic patients. They discussed a study which found 19% of female and 5% of male college students to be bulimic.

The presence of bulimia in high school females was studied by VanThorre and Vogel (1985). They found that the percentages of bulimic high school females in categories of black (17%), white (17%), and other ethnic groups (15%) were equal. The incidence of bulimia among a sample

of 257 high school students was determined by Maceyko and Nagelberg (1985). Five percent were identified as bulimic, 34% as binge eaters, and 61% as normal eaters.

Fairburn and Cooper (1982) focused on self-induced vomiting and bulimia. Six-hundred and twenty women who were currently practicing self-induced vomiting to control their weight were identified with the help of a women's magazine. Nineteen women (3.1%) fulfilled the diagnostic criteria for anorexia nervosa. Of the remainder, 499 (83%) fulfilled the diagnostic criteria for bulimia. Of these, 56% practiced self-induced vomiting at least once daily; the mean duration of vomiting was 4.5 years. Most women were of normal body weight. Using standardized measures, 68% of the women showed pronounced psychiatric morbidity and 89% had profoundly disturbed attitudes to food and eating. Fifty-six percent thought they needed medical help, although only 30% had ever discussed any aspect of their eating difficulties with a doctor. This study suggests that bulimia is an important undetected source of psychiatric morbidity.

The incidence of anorexia nervosa and bulimia among 300 women suburban shoppers was studied by Pope et al. (1984). One percent reported a history of anorexia nervosa and 10.3% reported a history of bulimia. These data support the belief that these disorders may be increasing in prevalence.

Zinkand et al. (1984) studied the incidence of bulimia in a family practice setting. They found 20% of 176 participants showed abnormal eating concerns and habits as indicated by responses on the Eating

Attitudes Test (EAT-26). Eleven percent of the sample population fulfilled the DSM-III criteria for bulimia.

Bulimia has been documented in women with other medical problems. Mitchell (1985) observed bulimia with self-induced vomiting in several patients after gastric stapling. To assess the prevalence of anorexia nervosa and bulimia among young diabetic women, a questionnaire was sent to 264 young women with insulin-dependent diabetes mellitus in two clinical settings (Hudson et al., 1985). Among the women who responded (30%), 28 (35%) reported a history of bulimia. These results suggested that bulimia represents a common problem in this group of women.

Clinical Characteristics

Mitchell (1984) outlined several clinical characteristics of patients with bulimia. Patients seeking treatment were usually female, single, had some college education, and a mean age of 24 years.

The onset of the eating disorder is usually between the ages of 16 and 20. Binge-eating typically precedes onset of vomiting, laxatives, diet pills, and/or diuretic abuse. A high number of patients seeking treatment have reported a history of anorexia nervosa or a history of overweight. Among individuals diagnosed as experiencing bulimia, 60% to 100% binge-eat at least once a week; 53% to 88% vomit at least once a week; 14% to 55% use laxatives at least once a week; and a small percentage use diuretics for weight control measures. During a binge, high carbohydrate and high fat foods are usually consumed. The range of calories ingested per binge can vary tremendously; the mean caloric intake for a binge may be approximately 4000 to 5000 calories. The

duration of binge-eating episode can also vary; the average duration is under two hours. Frequent weight swings are seen in patients with bulimia because these individuals often fast or restrict food intake between binges. The clinical course of bulimia shows an initial weight loss at the onset of the disorder followed by weight gain after a period of time. The weight gain may result from increased binge episodes that typically occur during the course of the illness. Factors which seem to precipitate a binge-eating episode include: (1) negative emotional states such as tension, anxiety, boredom, and sadness; (2) craving certain foods; (3) having eaten something "forbidden"; (4) being alone; and (5) thinking of food.

The etiology of bulimia is unknown at this time. Family, stress, culture, dissatisfaction about body weight, and a high proportion of dieting behaviors have been cited as environmental factors affecting the development of bulimia. The onset of the disorder is often associated with dieting. Both psychological and physiological food deprivation which accompany diets can trigger binge-eating behavior (Keys, 1950; Mitchell, 1984; Satter, 1984; Wooley and Wooley, 1985).

Johnson and Sinnott's (1981) research found bulimia to be common, especially among young women. The researchers' work suggests some etiology possibilities as primary neurologic dysfunction (epileptic form), disturbance in the appetite/satiety center of the hypothalamus, learned abnormal behavior, or unsuppressed oral drive.

Striegel-Moore et al. (1986) examined several studies and described risk factors in the development of bulimia: (1) Bulimia is primarily a women's problem with research indicating that approximately 90% of

bulimics are female; (2) weight and dieting concerns are so pervasive among females today that they have become normative; and (3) bulimia has most affected women of this era in Western society due to developmental influences in childhood, adolescence, and adulthood. There has been a shift toward an increasingly thin standard along with the effects of media attention on dieting and bulimia. The current exercise/fitness movement and shifting sex roles have also affected women and their attitudes toward their bodies.

Polivy and Herman (1985) discussed evidence that bingeing and dieting tend to co-occur. In bulimic patients, dieting of some sort seems generally to have preceded the onset of binge-eating. Pyle et al. (1981) interviewed 34 bulimics who did not meet the criteria for anorexia and found that 30 (88%) reported a period of dieting prior to the onset of bulimia. Boskind-Lodahl and Sirlin's (1977) examination of more than 100 bulimic patients showed dieting to have preceded the binge-eating in almost every case. This evidence suggests dieting is a risk factor in the development of bulimia.

The results of a controlled comparison of bulimic, obese, and normal subjects showed that bulimics evidenced significantly more psychopathology than did normal and obese subjects (Williamson et al., 1985). In particular, bulimics were found to be more depressed, more anxious, and generally more neurotic and impulsive. Bulimics also evidenced a distorted body image in that they perceived themselves as significantly larger and desired to be significantly smaller than did matched controls. Bulimic and obese subjects showed some similarities

in eating habits and psychopathology, especially obsessiveness, impulsivity, and guilt.

A study using a questionnaire for 244 college women found 8.2% to be bulimic (Nagelberg et al., 1984). Bingers and purgers showed greater restrained eating tendencies and compulsive eating behaviors. Purgers also showed lower self-esteem, particularly in the areas of identity and family self-concept.

Fairburn and Cooper (1984), studying the clinical features of 35 bulimic patients, confirmed that these patients have grossly disturbed eating habits accompanied by morbid beliefs and values concerning their shape and weight. A history of weight disturbance was noted, although a majority had a current weight within the normal range. There was a high degree of depressive symptoms.

Researchers speculate that bulimia may be a variant of a mood disorder, especially depression and other associated features of affective disorders. Some psychological characteristics associated with bulimia include impulsivity, rebelliousness, anger, anxiety, and social withdrawal (Gwirtsman et al., 1983; Hudson et al., 1983; Johnson et al., 1982; Pope et al., 1983; Walsh et al., 1982). There is a high prevalence of substance abuse (19%) among patients with bulimia, as well as high prevalence of substance abuse among relatives of bulimic patients (Mitchell, 1984). Individuals with bulimia have been found to manifest more psychopathology and poorer social adjustment than control groups (Garner et al., 1983). The extent to which these problems precede or are consequences of the eating disorder is unknown.

Garner and Garfinkel (1985) discuss several factors predisposing one to develop bulimia. Family and peer emphasis on thinness, dieting, and appearance all affect the degree of severity of the eating disorder. Also mentioned is a family history of affective illness, multiple substance abuse, and impulse problems. The researchers found that women who binge and purge tend to report more frequent sexual activity and interest.

Johnson and Flach (1985) described the family characteristics of 105 patients with bulimia. The high level of disorganization found in the families of bulimic subjects was associated with severity of symptoms. Igoin-Apfelbaum (1985) found that occurrence of bulimia may be related to the combination of a history of violent separations (or threats of separation) in the family and the endless denial of these separations.

Kirkley (1986) postulated three models for the explanation of the etiology of bulimia. The *psychosocial model* can be traced to the pressure on young women for extreme thinness. Social pressure to be thin and consequent dieting appear to be the major reasons for bingeing. The primary role of purging in maintaining bulimia may lie in the fact that it returns the individual to a state of hunger and deprivation. Familial and personality characteristics may play a role in this model. The *biological models* show evidence relating bulimia to affective disorders (depression), neurological disturbances (epileptic form), and metabolic disturbances (faulty serotonergic feedback system). The *biopsychosocial model* suggests integrating the biological and psychosocial factors. This model proposes that bulimia stems from an

interaction between biological and psychological risk factors and environmental pressures.

Medical Complications

Bulimia is often difficult to diagnose and it is important to rule out any underlying medical causes of illness by undertaking a careful physical, neurological examination. Winstead and Willard (1983) presented ten diagnostic clues to identify bulimic patients: (1) preoccupation with weight; (2) gastrointestinal complaints; (3) dental and oropharyngeal changes; (4) salivary gland enlargement; (5) edema and bloating; (6) amenorrhea; (7) dermatological complaints; (8) substance abuse; (9) laboratory changes; and (10) serious consequences such as electrolyte imbalances, heart arrhythmias, etc. Finger calluses have also been noted, frequently caused by abrasion of the backs of hands against teeth during self-induced emesis (Wynn and Martin, 1984; Russell, 1979; Joseph and Herr, 1985).

Mitchell (1984) described several medical consequences of bulimic behavior: (1) electrolyte imbalances; (2) renal changes; (3) gastrointestinal complications; (4) endocrine problems; (5) central nervous system effects; and (6) dental problems. Fluid and electrolyte imbalances are related to vomiting and/or laxative and diuretic abuse. Renal characteristics may include elevated blood urea nitrogen with a normal creatinine. Self-induced vomiting is the purging method most frequently used (Johnson et al., 1982) with spontaneous regurgitation occurring over time (Humphries and Wrobel, 1983). Metabolic alkalosis, especially hypochloremia, is found, whereas hyponatremia and hypokalemia

are more infrequent laboratory results. Jacobs and Schneider (1985) found a significant association between low body weight and hypokalemia. Elevated serum amylase values were also noted in 15 of 24 patients (62%). Dehydration is a common complication. Parotidomegaly may be seen (Harris, 1983; Berke and Calcaterra, 1985).

Endocrine symptoms which arise are normal thyroid T3 and T4, but a blunted thyroid-stimulating hormone (TSH) response to thyrotropin-releasing hormone (TRH) (Gwirtsman et al., 1983). Growth hormone studies show a pathological responsiveness to increased TRH levels in blood. These abnormal growth hormone levels in the bulimic are similar to neurochemical changes seen in patients with affective disorders.

Rau and Green (1978) found electroencephalogram (EEG) abnormalities common in women with bulimia, whereas Mitchell (1984) found them infrequently. Phenytoin (a drug used to treat epileptics) use is controversial as a treatment measure for bulimia.

Finger clubbing or swelling has been observed in several cases as a consequence of chronic laxative abuse (Malmquist et al., 1980; Prior and White, 1978; Silk et al., 1975). Deaths from fluid and electrolyte imbalances, gastric dilation or rupture, and cardiac arrhythmias have been reported (Mitchell, 1984; Isner et al., 1984; Matikainen, 1979; Saul et al., 1981; Edwards, 1985).

Bulimia and Its Relationship to Affective Disorders

Many studies have strongly suggested that bulimia is a type of affective disorder. This knowledge is helpful in understanding the treatment of bulimia, the implications being that long-term therapy will

be necessary and the use of antidepressants may be helpful. The dexamethasone suppression test (DST) and thyrotropin-releasing hormone (TRH) test have been used to determine the family history of major depression in depressed and bulimic groups. There is conflicting evidence that neuroendocrine abnormalities such as DST and TRH non-suppression are similar in both groups and thus link bulimia to affective disorders. Other studies using depression scores have also reported data both supporting and rejecting the theory that bulimia is a type of affective disorder.

Evidence linking affective disorder with bulimia was presented by Hudson et al. (1983a), Mitchell et al. (1984), Piran et al. (1985), and Lindy et al. (1985). Using the DST, all four studies showed bulimics failed to exhibit suppression of cortisol on the DST; these findings were similar for patients with major depression in other studies. A substantial level of depressive symptomatology was documented in patients with bulimia in these studies.

Musisi and Garfinkel (1985) studied 20 depressives and 20 normal control subjects using the DST. They found that bulimic subjects had a rate of DST non-suppression of 20% and that 20% had a past history of major depression. Forty percent had a history of alcoholism and/or antisocial personality in a first-degree relative, but only 5% had a positive family history of affective disorder. These rates were significantly lower than those found in the depressed group except for alcoholism and antisocial personality for which there was no significant difference. A subgroup of bulimic DST non-suppressors was identified who were characterized by past history of major depression and high

rates of family history of affective disorder. This group responded to antidepressant therapy in a manner similar to depressed patients.

Gwirtsman et al. (1983) studied neuroendocrine abnormalities in bulimia. The author examined the relationship of family history and neuroendocrine function in 18 normal weight bulimic patients. Twelve of 18 patients (67%) showed abnormalities of cortisol suppression, and 8 of 10 (80%) showed blunted thyrotropin-releasing hormone (TRH) tests. This evidence indicates that neuroendocrine abnormalities, previously found in anorexia nervosa, are not present simply because of low body weight and, further, that eating disorders and affective disorders may share neurochemical illnesses. Norris et al. (1985) reported a controlled study on TRH testing in bulimia and anorexia nervosa. Serum TRH levels were examined in 10 bulimic outpatients, 9 anorexia nervosa inpatients, and a group of age-matched healthy subjects. There were no group differences between bulimic subjects and the healthy comparison group. Anorexic subjects at low weight showed a delayed response to TRH, which tended to normalize with weight gain. Bulimic subjects showed varied patterns of response and it remains possible that a subgroup of bulimic patients may have an abnormal TRH response, especially those at low body weights.

Hudson et al. (1983b) studied 420 first-degree relatives of 14 patients with anorexia nervosa, 55 patients with bulimia, and 20 patients with both disorders. These relatives were evaluated for the presence of psychiatric illness, using DSM-III criteria, by the family history method. The morbid risk for affective disorder in the families of the eating disorder-tested subjects was similar to that found in the families of

patients with bipolar (manic-depressive) disorder, but significantly greater than that found in families of patients with schizophrenia or borderline personality disorder. These data add to the evidence that anorexia nervosa and bulimia are closely related to affective disorders.

A study designed to evaluate the frequency of affective illness among 50 women with bulimia was reported by Walsh et al. (1985). Seventy percent of the patients had, at some time during their lifetimes, met Research Diagnostic Criteria (RDC) for an episode of major depression and 88% had met RDC at some time during their lives for some affective disturbance.

One-hundred and eight women with bulimia were evaluated for affective disorder and alcohol or drug abuse (Hatsukami et al., 1984). Forty-three percent had a history of affective disorder and 18.5% had a history of alcohol or drug abuse. Fifty-six percent of the bulimic patients scored within the moderate to severe range of depression.

Stern et al. (1984) reported nonsignificant differences when studying the lifetime prevalence of affective disorder in the first- and second-degree relatives of 27 bulimic women and 27 women with no history of an eating disorder.

Daily measurements of mood and the occurrence of specific eating behaviors were assessed concurrently over an eight-week period in 50 bulimic subjects (Johnson-Sabine et al., 1984). Negative mood states were more marked when binge-eating, vomiting, or purging occurred, and they increased as abnormal eating behaviors accumulated. It was suggested that the accompanying dysphoric mood states of bulimia are likely

to be secondary related to the presence of abnormal eating symptoms and do not constitute a primary depressive illness.

Fairburn et al. (1985) found a significant association between outcome in terms of eating habits and the severity of neurotic symptoms after treatment. Their findings suggested that the neurotic symptoms seen in bulimia are likely to constitute a secondary psychological reaction due to malnutrition rather than primary depression.

The above research findings suggest that there may be a distinct subgroup of bulimic patients who have a hereditary predisposition to affective illness and, hence, will most likely respond positively to antidepressant therapy. However, it is also evident that other bulimic patients become depressed as a result of disordered eating patterns and/or malnutrition, and in these cases antidepressant therapy may not be relevant.

Hunger and Self-Awareness

Kim Chernin (1981), a recovered anorexic and bulimic, described bulimic behavior as an obsession. During the course of the eating disorder Chernin stated, "My hunger filled me with despair." Although Chernin resolved to control this hunger, it always returned. The efforts to kill off appetite were unsuccessful and the bingeing continued. During this time, Chernin awoke around midnight feeling hungry, even though a great deal had been eaten that day. After spending several minutes staring at foods in the refrigerator, suddenly for the first time, Chernin realized that what was felt was not hunger at all. Instead, the feelings of loneliness, sadness, and being frightened were

recognized. What food had to offer was companionship, comfort, reassurance, a sense of warmth and well-being. It was a hunger, but food apparently was only symbolic of that for which Chernin was hungering.

Hilde Bruch (1973), one of the foremost clinicians in the field of eating disorders, has identified two traits as being of fundamental significance in the development of severe eating disorders: (1) the inability to recognize hunger, and (2) lack of awareness of living one's own life. The inability to recognize hunger involves a functional defect according to Bruch. This defect in the perceptual and conceptual awareness of hunger was identified over a period of time during psychiatric and psychoanalytic treatment of a large number of severely obese and anorexic patients. At first the symbolic meanings of disturbed food intake were recognized, both in uncontrollable quantities as well as in the rigid refusal to eat. Some of the symbolic meanings that food was thought to express were an insatiable desire for unobtainable love, an expression of rage and hatred, a substitute for sexual gratification, an indication of ascetic denial, a wish to be a man, the wish to be pregnant (or fear of it), a sense of spurious power, and/or it may serve as a defense against adulthood and subsequent responsibility. Symbolic significance of food may carry an enormous variety of different, often contradictory connotations. Bruch also describes the lack of awareness of living one's own life as significant in those with eating problems. Bulimic individuals often express low self-esteem, lack of self-identity and lack of self-awareness. These feelings may have developed from inappropriate responses during infancy, childhood, and/or adolescence.

Implications of Starvation and
Appetite to Bulimic Behavior

The study of starvation, fasting, and the use of consistent dieting may provide insight into the treatment of bulimia. The great majority of women in our society feel too fat and in an effort to reduce the discrepancy between their perceived body size and ideal weight, nearly all of them diet (Thompson and Schwartz, 1982). Eating disorders may occur when the rational attempt to exert self-control over food intake fails and the individual loses control entirely or cannot give up control.

The Minnesota Study (Keys et al., 1950), a classic study of physical and mental affects of semistarvation (approximately 1600 calories/day for six months for male volunteers), shows that starvation creates conditions which could foster bulimia.

The study showed four important consequences of semistarvation:

(1) The self-starving men became profoundly "neurotic." These men were conscientious objectors, of apparently sterling character, who tested out as high in "ego strength." After losing 25% of their body weight, MMPI (a mental illness test) results showed highly significant increases in hypochondriasis, depression, and hysteria.

(2) It showed that constant, tormenting, obsessive thoughts of food are an inevitable result of starvation. Food in all its ramifications became the principal topic of communication, reading, and daydreams for almost all the subjects.

(3) Starvation predisposes one to binge, to lose control of eating, and having done so, to vomit, to purge oneself of the "sinful" food which caused an excess of the daily 1600-calorie allotment.

(4) Once a starvation period is over, the predisposition to binge becomes an uncontrollable reality. In the re-feeding ("rehabilitation") part of the Minnesota study, all subjects showed a loss of control of appetite (gorging and sometimes vomiting).

It was observed that without any improvement in nutritional status, subjects tended to stop losing or even to gain weight, as a result of edema. The effect of accumulation of fluid in cells was thought to be especially damaging to heart and brain tissue.

Of particular significance to eating disorders was the compensatory adaptation to starvation. Metabolic rate (oxygen consumption) was lowered 33% by the end of the starvation period in the Minnesota study. Abnormal levels of fat were deposited during rehabilitation. These effects are experienced as disasters by those with eating disorders. Many anorexics ("starvers") become bulimic as a result of wanting to get well. Once she/he decides to eat and regain some weight, she/he is subject to the rapid weight gain and disinhibition of appetite as seen in the Minnesota study. Bulimia resolved itself spontaneously in the subjects of the Minnesota study when they had eaten enough to return to (or exceed) original weights. In vulnerable women, however, a vicious cycle of starve versus binge may begin.

Tepperman and Tepperman (1964) also reported edema during the starvation period, slowing of the metabolic rate, and attendant "hyperlipogenesis" during the re-feeding period.

Wooley and Wooley (1985) have described the progression of bulimic development. In the bulimic woman, the first purge usually occurs after an unintentional lapse at a special occasion where prolonged exposure or

close proximity to food cannot be avoided. The young woman remembers or gets the idea to vomit or take laxatives to rid herself of the food ingested. ^xFor a period of time, this pattern of purging to rid oneself of food is used to control weight. In time, as hunger ensues and techniques of purging are learned, a subtle change occurs: binges are planned with the intent of purging. Binge/purge episodes become major ways to deal with hunger and to meet nutritional needs because considerable food can be absorbed despite purging. The dilemma faced by the bulimic individual is the fact that she cannot eat satisfying amount of food and remain at an acceptable weight. As long as food intake (absorption) is sufficiently low to maintain her low weight, she will be continuously subject to voracious appetite and lapses of control. Even when normal weight has been restored, abnormal appetite and lapses of control increase over time in the bulimic individual.

The progression in appetite may occur in the following ways:

(1) Over time, the body adapts to an abnormal diet in ways that perpetuate the problem. Most bulimics eat little between binges. Foods consumed during binges are usually high in sugar content, and it is primarily sugar which can be absorbed before purging. The subsequent dramatic rise in blood sugar after such a sugar load could result in the release of large amounts of insulin shortly after initiation of eating. Insulin increases appetite and promotes fat storage. In addition, after months or even years on such a pattern of eating, the body's insulin response to small amounts of sugar (one cookie, for example) could be affected. This, paired with the disinhibition of cognitive restraint caused by eating a "forbidden" food, guarantees a binge.

(2) There may be a conditioning process by which satiety is progressively impaired. In binge/purging the body receives continuous misinformation. Attempts to eat normal amounts of food usually will provide very little satiety, especially foods low in sugar or carbohydrates (e.g., proteins and fats). This may explain both the voracious appetite and strong preference for sweets. The process of regaining normal satiety and appetite may take several weeks or months.

The Dieting Dilemma in Bulimia

Wooley and Wooley (1985) have taken the position that voluntary caloric restriction (dieting, starvation) causes eating disorders. It causes weight loss, which in the present climate of internal fear and hatred of fat, acts as a strong reward or reinforcement of further caloric restrictions. Dieting and management of the effects of food deprivation (starvation) become an obsessive integral part of the young woman's life. One of the symptoms of starvation is the virtually uncontrollable urge to binge-eat. To manage this symptom, one or more purgative measures is used. Whatever shame is experienced is preferable (at least in the short term) to the alternative (weight gain).

Further clinical evidence that dieting predisposes individuals to bingeing may be derived from patients with anorexia nervosa, in which self-imposed starvation is a primary feature. Studies have found that 30 to 50% of patients referred for anorexia nervosa also have bulimia (Beumont et al., 1976; Garfinkel et al., 1980; Hsu et al., 1979). Garfinkel et al. (1980) found that dieting preceded bingeing, on the average, by more than one and one-half years in their sample of bulimic

anorexic patients. Pyle et al. (1981) observed that 88% of their sample of bulimic patients began dieting prior to the onset of binge-eating. Fairburn and Cooper (1982) found that over 80% of their sample of bulimic women reported that they were attempting to lose weight when their bingeing began. Bingeing is also common among obese dieters. In a study of 102 obese individuals seeking treatment for overweight, Gormally et al. (1980) found that 78% were having moderate to severe problems with bingeing.

Awareness of this way of thinking is helpful to the clinician. In treatment approaches, it appears that offering the patient an explanation of abnormal hunger and appetite and the effects of restrictive dieting may help the individual realize that his/her dilemma is not only psychological, but physical as well.

Nutrition Counseling for Bulimia

Willard et al. (1983), Goff (1983), and Huse (1982) have described nutrition programs using nutrition counseling as an adjunct to individual and group psychotherapy. These programs, behavioral in nature, stressed development of individual responsibility in controlling food intake, resumption of healthy eating behavior, nutrition information, and initially avoiding binge foods. Neuman and Halvorson (1983) also recommended using a dietitian, along with the efforts of a mental health professional in the treatment of bulimia.

Boutacoff et al. (1984) developed a nutrition manual for bulimics outlining three parts:

(1) *Food: An Eating Disorder Perspective.* Hunger, the need for food, the role of nutrients in the diet, and the misuse of eating are discussed.

(2) *A Meal Planning System for Healthy Eating.* An individualized meal pattern form is provided by the dietitian. Each patient is instructed on the use of food lists and meal plan worksheets. Group sessions are then used for menu planning and nutrition education.

(3) *Shaping and Maintaining Healthy Eating Patterns.* This section describes such issues as continuing to plan meals, developing a healthy attitude toward food and eating, regulating eating and activity patterns, and listing resources for further reference.

Satter (1984) developed her treatment strategy on the premise of establishing normal eating patterns without specific structure. Satter believes that anything which resembles a diet is just that in the mind of the woman with bulimia. Her materials include a focused eating exercise, ways to analyze and satisfy appetite, regulating food intake, controlling the eating environment and determining why one eats for emotional reasons, and the definition of normal eating. She emphasizes discontinuing any type of diet(s) (food deprivation) and establishing healthy eating by asking oneself, "What do I really want to eat?" No outcome information on this treatment program has been reported.

Dalvitt-McPhillips (1984) studied a dietary approach to bulimia treatment. A treatment group of 10 bulimic women were placed for six weeks on a nutrient-dense diet containing no fewer than 1400 calories and free of suspected blood sugar-insulin level destabilizers. Simultaneously, a control group of 10 bulimic were placed on a Sham Food Plan

which allowed unknowing duplication of their pre-study food patterns. After three weeks they were switched to the nutrient-dense diet for the remainder of the study. No psychiatric counseling was given to either group. The treatment group assigned to the nutrient-dense diet ceased to binge. The control group on the sham diet binged steadily until switched to the nutrient-dense diet, after which all bingeing ceased. Subjects lost weight (or maintained weight by choice) and have remained binge-free for over two and one-half years. Evidence suggests that malnutrition and blood sugar-insulin level destabilizers may play a role in precipitating the bulimic condition.

Treatment Approaches

A number of recent studies have been reported which discuss appropriate treatment strategies for bulimia. Some treatment methods include: behavioral techniques, group therapy, antidepressant and anticonvulsant medication, psychotherapy, and cognitive behavior therapy.

Cinciripini et al. (1983) demonstrated that a behavioral program used with inpatients resulted in increased body weight and caloric consumption. A follow-up study two years later indicated that improved eating behaviors were maintained. Lacey (1983) described a short-term outpatient behavioral treatment program designed for use by a non-specialist staff, under medical supervision. In 30 women with bulimia, the program significantly reduced their incidence of bingeing and vomiting without producing weight gain, weight disorder, or neurotic illness. After treatment, all the women had fewer symptoms. A pretreatment history of alcohol abuse or anorexia nervosa were indicators of

poorer prognosis. Mitchell (1984) and Neuman and Halvorson (1983) described behavioral programs as effective in the treatment of bulimia. Dixon and Kiecolt-Glaser (1984) studied behavioral techniques and insight-oriented discussion group therapy for bulimia. Two comparison groups consisted of bulimic women who received either no treatment or individual psychotherapy. The differences on parameters measured in subject groups suggested that a group therapy approach held promise for successful treatment.

Fairburn (1981) found promising results using a cognitive behavioral approach with eleven bulimic women. Treatment consisted of (1) interrupting the vicious cycle of overeating and vomiting by helping the patient to control her food intake, and (2) helping the patient to modify her abnormal attitudes to food, eating, body weight and shape, and to maintain progress following treatment. Nine patients reduced their frequency of binge/purge episodes to less than once a month. This change was accompanied by improved attitudes regarding food, eating, and body weight and shape. The mean duration of treatment was seven months. Follow-up data for six patients (twelve months after treatment) showed continued improvement with two patients no longer bingeing or purging and four patients doing so every two to three months. Another study examining the effectiveness of a group cognitive behavioral treatment found significant pre- and post-treatment changes (Schneider and Agras, 1985). Vomiting frequency decreased by 91% and depression, eating attitudes, and assertiveness were improved.

Boskind-White and White (1983) studied women who binged and self-induced vomiting in order to avoid gaining weight. Their work with group

therapy proved much more productive than previous one-to-one psychotherapy because approximately half of the group members had eliminated or significantly reduced their bingeing behavior within six months. Furthermore, the group experience was cited as critical in helping patients to recognize their problem and in leading them to deal with it more actively. Connors et al. (1984) studied the treatment of bulimia with brief psychoeducational group therapy. Two groups of ten normal-weight bulimic women received short-term, structured group therapy beginning three weeks apart in a multiple-baseline design. Techniques incorporated in the program included education, self-monitoring, goal setting, assertion training, relaxation, and cognitive restructuring. Results showed an overall reduction of 70% in binge/purge episodes. There were significant improvements in psychological functioning, including self-esteem, depression, assertiveness, and pathological attitudes toward eating.

Inbody and Ellis (1985) reported that using group therapy in the treatment of eating-disordered patients can be particularly difficult because of their unique dynamics, including their stubborn resistance to change, cognitive distortions, and counterdependency. Mitchell et al. (1984) are currently studying the effectiveness of an intensive outpatient group treatment for bulimia. Two major influences can be discerned in this treatment: (1) the insistence upon abstinence from self-induced vomiting upon entry into the program, and (2) behavioral psychology, both cognitive and operant. A survey of 104 patients who completed the program revealed that 47% reported being abstinent throughout the program, 25% reported between one and three episodes of

vomiting during the program, and 11% reported four or more episodes. Huon and Brown (1985) described a multi-faceted group based approach. The treatment focused on control issues, food and eating behaviors, and interpersonal relationships. Results of this study indicated that 36 of 40 participants (90%) reduced bingeing and vomiting behaviors.

Wooley and Wooley (1985) described two separate programs of treatment for bulimic women. The first, in existence since 1974, was an outpatient program in which treatment focused on individual therapy, group therapy, family therapy, and a newly developed body image treatment group. The second program, begun in 1983, was a three and one-half week residential program for groups of six women whose symptoms were severe enough to warrant concentrated attention, but whose level of functioning was high enough to permit them to be treated outside a hospital. This patient selection allowed intense, multidimensional, multitherapist attention to be focused on the bulimic symptoms. The main goal of treatment was a permanent resolution of the patient's eating/weight problem. The effort was made to transform a moral problem into a psychological one. Outcome studies were not reported.

Because studies have found a possible link between affective disorder and bulimia, the use of antidepressant therapy with drugs such as imipramine, cloripramine, amitriptyline, and nortriptyline has been investigated. Speculations have been raised that the depressive effects seen may reflect serotonin deficiencies or problems in the dopamine system within the brain (Johnson et al., 1983). Many bulimics often alternate between severe carbohydrate restriction to high-carbohydrate binge eating. Such dietary habits may produce abnormal fluctuations in

brain serotonin levels as well as depressive symptoms. Serotonin, a neurotransmitter, when present in high concentrations in the brain, has been shown to decrease appetite and may increase appetite when present in low levels. These relationships are not fully understood, but the similarities between depressed and eating-disordered patients have led investigators to study the possible benefit of the use of antidepressants. The usefulness of L-tryptophan, a serotonin precursor, in relieving the binge/purge symptom in bulimia was studied by Krahn and Mitchell (1985). A double-blind, placebo-controlled trial was conducted with 13 bulimic patients of normal weight. They found no clinically significant improvement in study patients. L-tryptophan, at a dose of 3 gm/day, was not significantly better than a placebo at improving mood or decreasing binge-eating.

Mitchell and Groat (1984) reported a placebo-controlled, double-blind trial of amitriptyline hydrochloride in a series of 32 female outpatients with bulimia. The results indicated that the group given amitriptyline (150 mg at bedtime) had significantly more antidepressant activity than the placebo group. However, both groups demonstrated considerable improvement in eating behavior. Patients in both placebo and active drug group also received a minimal behavioral treatment program. Amitriptyline (a serotonin inhibitor) was not associated with weight gain or increased carbohydrate craving.

In a retrospective study, 22 patients with bulimia were treated with at least one therapeutic trial of antidepressants (Brotman et al., 1984). Most patients were clinically depressed (77%) before the drug trial. Bingeing frequency decreased in 59% of all cases, and depression

improved in 58%, after a four-week trial of antidepressants. Only 27% of all patients showed both an antibinge and an antidepressant response. After a three-month follow-up period, several patients relapsed despite continuing medication. These data suggest that medication may have a separate and possible unrelated antibinge and antidepressant effect in bulimia.

Walsh et al. (1982) described six patients with bulimia who responded dramatically with monoamine oxidase inhibitors (MAOI's). Five of six subjects were treated with phenelzine (an MAOI and tranquilizer). All six patients experienced immediate improvement in mood and in eating behavior. It was noted that clinicians who are considering using MAOI's should keep in mind that such drugs will be hazardous for patients unwilling to comply with dietary restrictions because of severe reactions of the medication with certain foods.

Twelve bulimic women with depression and anxiety were treated with MAOI's (Stewart et al., 1984). A dramatic response was seen in 10 cases. Improvement in depression and anxiety were reported. The number of binge episodes was significantly decreased. Walsh et al. (1984) studied 20 bulimic women of normal weight who participated in a double-blind trial studying the effects of an MAOI. Nine women received phenelzine sulfate and 11 received placebos. The phenelzine-treated patients reported significantly fewer binges per week and had a lower Eating Attitudes Test score. This treatment may be useful in bulimic patients capable of maintaining a tyramine-free diet. These results were confirmed by Walsh et al. (1985) who demonstrated a significant therapeutic advantage of phenelzine over placebo in a double-blind,

placebo-controlled trial involving 30 normal weight bulimic women. Preliminary data suggest that phenelzine may be of benefit even to non-depressed patients with bulimia. Pope et al. (1983) treated eight bulimics with tricyclic antidepressants and found moderate or marked improvement in bingeing behavior in six of the eight.

Some available studies indicate phenytoin may be a helpful drug in treating some patients with eating disorders; however, there needs to be more research to help identify specific patient characteristics which would distinguish those patients who will be more responsive to this anticonvulsant medication (Rau and Green, 1978; Mitchell, 1984). Studies with lithium carbonate have been reported to improve mood swings and affective disorders in some isolated cases of anorexia nervosa (Johnson et al., 1984). The authors caution about the clinical dangers of using lithium in eating-disordered patients. There is considerable risk of lithium intoxication in patients who are known to severely restrict food intake, are self-induced vomiters, and/or abuse laxatives or diuretics.

Fifteen patients with bulimia received fenfluramine (an appetite suppressant) or placebo under double-blind, randomly-ordered conditions (Robinson et al., 1985). Findings suggested that, in patients with bulimia, hunger was abnormally reported and eating was suppressed. Bulimic symptoms were probably reduced by fenfluramine, which may be a useful treatment measure.

In Fairburn's (1985a) view, for the majority of patients, psychological treatments are to be preferred to treatment with antidepressant drugs. Antidepressant drugs are indicated in the minority of cases in

which there appears to be a coexisting depressive disorder. "Biological" symptoms of depression are not a useful guide because appetite, weight, energy, and gastrointestinal function are all directly affected by the eating disorder itself. Although treatment of depressed bulimic patients with antidepressants often substantially alleviates depressive symptoms, the eating disorder tends to persist unchanged. Preliminary studies suggested that both individual cognitive therapy (Fairburn, 1981) and combined individual and group therapy (Lacey, 1983) achieve positive results. Both treatments have yet to be satisfactorily investigated.

Conclusion

Bulimics are usually young (typically 24 years of age) female patients, make up 4 to 19% of our population, and may exhibit anorexia nervosa tendencies as well. Bulimia has serious medical consequences; particularly common are electrolyte and dehydration problems. The etiology of bulimia is unknown at the time; however, family problems, stress, cultural influences, dissatisfaction with body weight, and restrictive dieting behaviors have been cited in the literature as environmental factors.

Other research has indicated that bulimia may be a variant of an inherited mood disorder, especially depression. The relationship of bulimia to affective disorders is an area of much current investigation. Studies reported to date both support the claim that bulimia is a type of affective disorder and refute the claim. The underlying question still to be resolved is: Does the depression experienced by most

bulimics represent a primary (biochemical or genetic makeup) or secondary (malnutrition or disordered eating patterns) depressive illness? Most literature suggests bulimia can be subgrouped; a minority of patients have a primary depression and will respond to antidepressant therapy, whereas most patients will benefit once eating patterns are resolved and some form of psychotherapy is used.

Attempts to control body weight through severe dieting and/or vomiting behavior lead to known and predictable starvation effects. Starvation creates conditions which foster bulimia. Noteworthy symptoms are "neurotic" behavior (hypochondriasis, depression, and hysteria); obsessive thoughts of food; food preoccupation; physiological urge to binge-eat; edema or fluid retention; and a lowering of metabolism with attendant hyperlipogenesis.

Most treatment strategies report favorable short-term outcomes for their bulimic patients. Programs which integrate behavioral techniques, group therapy, psychotherapy, and cognitive therapy appear to be most useful. For the majority of patients, psychological and nutritional treatment is preferred to antidepressant drugs unless evidence of a coexisting depressive disorder is found. Nutrition counseling, used by most program centers specializing in treatment of eating disorders, which tries to correct abnormal eating patterns and to establish healthy eating guidelines, is essential to the bulimic's recovery.

CHAPTER 3

RESEARCH DESIGN

The quasi-experimental design was chosen over a strictly experimental design for practical and ethical reasons. Random assignment of subjects would have been very difficult to achieve because of limited population size. Six subjects were accepted into the nutrition treatment group and five were assigned to the comparative or control group. The comparative group consisted of subjects who were in conventional treatment; that is, this group was seen by psychotherapists or qualified counselors and did not receive nutrition intervention. Denial of any type of treatment (a true control group) to women seeking help with this eating disorder could have been harmful to their health.

Subjects

Treatment and control subjects selected fulfilled the same diagnostic criteria for bulimia as set by the researcher and also as having met the criteria for bulimia as defined by the Diagnostic and Statistical Manual for mental Disorders (1980) (Appendix A). These subjects were referred to the investigator by medical doctors at the Student Health Services at Montana State University and community physicians or psychologists in Bozeman, Montana. Subjects were between the ages of 17 and 29 years. Written permission was obtained from the Human Subjects Research Committee at Montana State University and each

subject signed a contract agreeing to participate anonymously in the research project (Appendix B). Each subject was allowed to terminate participation at any time during the study. Two subjects did not complete the time period for the control group and chose to receive nutrition therapy and discontinued the study. Each subject was medically evaluated by their medical doctor at the outset of treatment and was allowed to participate in the study.

Procedure

Each subject was evaluated for anthropometric data at the beginning and end of the study period. These measurements included height, weight (taken weekly), and skinfold measurement. The author hoped to maintain the treatment subjects' body weights as part of the nutrition program. The skinfold measurements were evaluated by a chart which gives an approximate percent of body fat based on the sum of three areas of tricep, suprailiac, and thigh body fat (Pollack et al., 1980). These measurements were taken with Lange calipers which measure skinfold adiposity in millimeters.

Each subject was given the Eating Attitudes Test (EAT-26) before and after the treatment or control period (Appendix B). This test was developed by Garner et al. (1983) to be used as a screening for disturbed eating and dieting behaviors or attitudes. It is a self-report measure useful in determining the severity of an eating disorder, particularly anorexia nervosa and bulimia. High scores on EAT are closely associated with various clinical and psychometric variables measuring psychopathology. EAT-26 (a shortened version of the original

EAT) has been demonstrated to be a highly valid, reliable, and economical instrument which may be useful as an objective measure of the symptoms of bulimia (Garner et al., 1982). While EAT-26 may indicate the presence of disturbed eating patterns, it does not reveal the motivation or possible psychopathology underlying the manifest behavior. In this study, the EAT-26 instrument was used as an outcome measure in a nonclinical setting to determine whether the nutrition program helped to improve eating and body weight attitudes or behaviors. Total scores of 11 or more indicate disturbed eating patterns. A score of 3 was used for the response "always," a score of 2 was used for "usually," and a score of 1 was given for the response "often." The responses "sometimes," "rarely," and "never" received no score.

In the nutrition treatment group, weekly food intake records and weekly binge/purge episodes were recorded and given to the investigator at the weekly session. Each nutrition session was approximately one hour in length. In the control group, food intake records were kept before psychotherapy and from week five to week six (the same period for the treatment group). A weekly record of binge/purge episodes was also assigned to each control subject. Food records for each subject were analyzed for nutritional adequacy pretreatment and week six (Appendix B). All data were collected by the same investigator.

Treatment

A nutrition program developed by the researcher, a Registered Dietitian, was used. This program emphasized patient participation in planning appropriate nutritional needs. The treatment sessions were

adapted from Satter's (1984) viewpoint of teaching the patient specific eating behaviors, nutrition education, cognitive change, conceptual awareness, and shaping aerobic exercise (Appendix C). The Healthy Eating Manual, developed by Boutacoff et al. (1984) was also used to provide meal planning structure. An outline was developed and given to each treatment participant listing responsibilities of the subject and the investigator each week (Appendix C).

Analysis of Data

The analysis of variance model used for the data in this study was interpreted with a repeated-measures design. This type of correlated-samples design is called a repeated-measures design because more than one measure is taken on each subject. It often takes the form of a before-and-after experiment. A pretest(s) is given, some treatment is administered, and a post-test(s) is given. The mean of the scores on the post-test is compared with the mean of the scores on the pretest to determine the effectiveness of the treatment. Using the repeated-measures terminology, there is, for each of the 20 analyses in this study, a two-group (control versus treatment) design with one repeated measure. The repeated measures are the number of times (weeks) the dependent variable is measured in each analysis. (The number of repeated measurements varies from 8 for binge/purge episodes to 2 for most of the nutrition measurements.) More technically, the design is a three-factor, mixed-model analysis of variance with a fixed grouping factor (dependent variables), a fixed-measurement factor (week of measurement), and a random factor (subjects) nested within groups and crossed with weeks.

In each analysis of variance, the interaction effect of treatment/control and week was chosen to describe the results of the study. The F test for the main effect of treatment/control was not of much interest. It tested the null hypothesis of no difference between treatment and control groups in the average or means over time of the 2 to 8 responses on each dependent variable. A significant effect here might mean only that there were significant differences between the two groups even before the treatment was applied.

It was the interaction effect that indicated most strongly whether the treatment was more effective than the control group. For example, for the number of binge/purge episodes, observing a significant main effect of week would have suggested that both groups changed significantly over time, but not whether the treatment group improved significantly more than the control group. In other words, the control group could have improved equally on its own without benefit of treatment. If a significant interaction effect were present (and the change was in the appropriate direction), then it could be correctly concluded that the treatment was helpful as desired. Specifically, the interaction effect tests the null hypothesis that the treatment produces no more change over time than that which occurred in the control group.

It should be noted that for the two analyses (binge/purge episodes and body weight) where more than two repeated measures were made, adjusted probabilities (the Geisser-Greenhouse 1958 adjustments) were used for the tests of week and the interaction effect. This adjustment compensates for the intercorrelations between the weekly measurements which otherwise tend to give erroneous results. When only two

measurements were made for each subject, the analysis of the repeated measures factors was similar to a paired comparison t test.

The level of significance was set at $p \leq 0.05$. In addition to the statistical report presented in the results section of this study, some pertinent individual clinical results have been outlined in the results section.

CHAPTER 4

RESULTS

Binge/Purge Episodes

Both treatment and control groups decreased the number of weekly binge/purge episodes over the eleven-week period (Table 1 and Figure 1). The treatment/control and week or time interaction effects were non-significant, indicating that there was no difference in the amount of decline of binge/purge episodes due to the treatment ($p = .21$). The psychotherapy control group was as successful as the nutrition treatment group in decreasing the level of binge/purge episodes.

Table 1. Summary of mean binge/purge episodes.

Week	Control Group	S.D.*	Treatment Group	S.D.*
Baseline	22.2	13.07	12.7	5.13
1	11.0	6.12	11.0	7.13
2	13.2	3.90	7.7	5.61
3	11.0	4.06	8.2	6.43
4	12.4	2.07	8.7	7.26
5	11.4	5.68	6.0	6.16
6	9.8	3.03	3.2	4.45
Follow-up	7.0	6.28	5.0	4.82

*S.D. = Standard Deviation

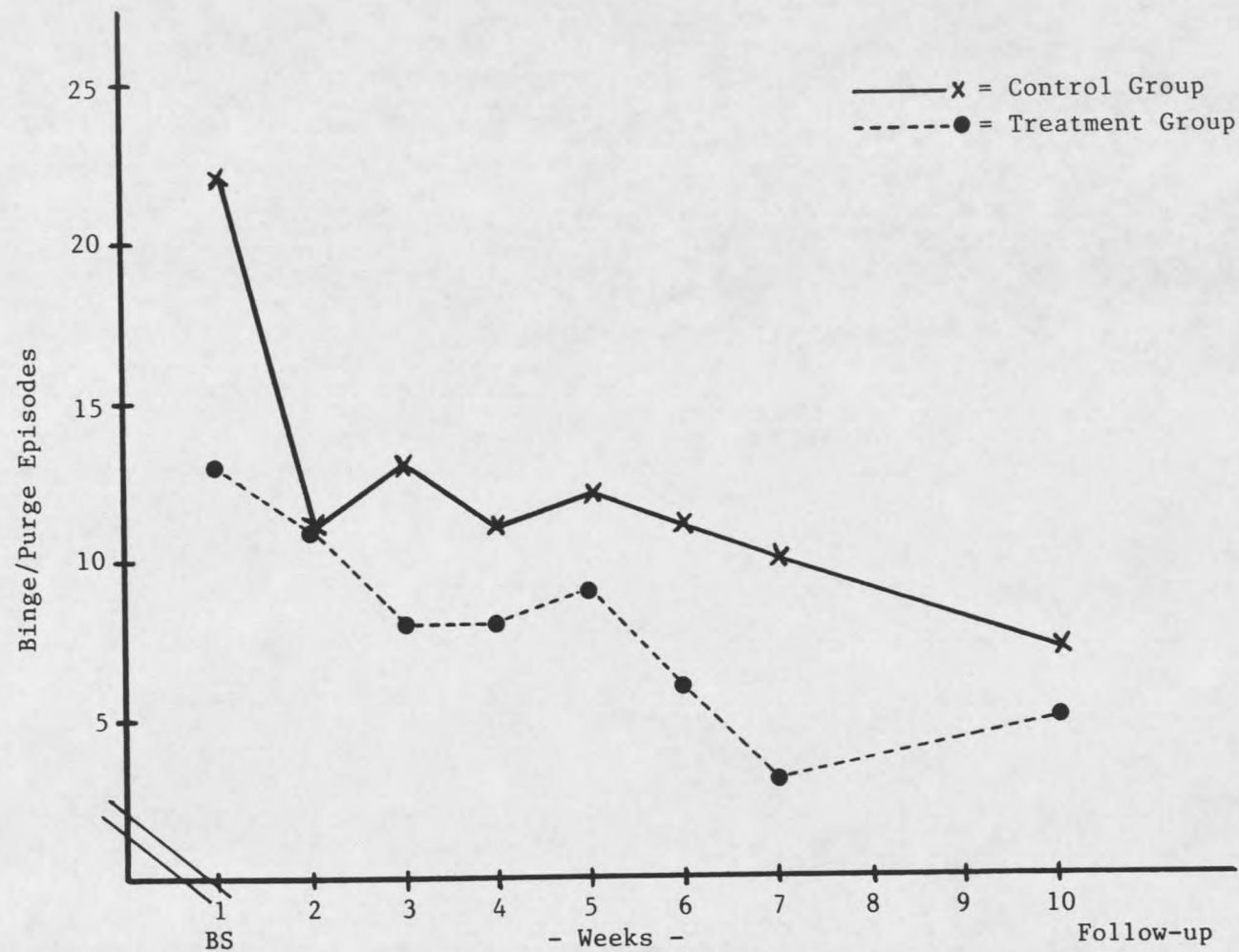


Figure 1. Mean weekly frequency of binge/purge episodes in treatment and control groups.

Individual binge/purge frequencies for the subjects are listed in Appendix D (Raw Data).

Body Weight

Weekly measures of body weight were taken in the study. The treatment and control groups showed non-significant changes in body weight, with the interaction effect being $p = .35$ (Table 2 and Figure 2). However, it was observed that treatment subjects slightly decreased body weight while control subjects slightly increased their body weights. The treatment group had a higher standard deviation for body weight at the beginning and throughout the study. The non-significant change in body weight was a desired goal of the nutrition program. However, Subject 4 in the treatment group lost twenty pounds from pre- to post-treatment (Table 3). This subject lost weight even though she was keeping down more food during the treatment program.

Table 2. Summary of mean body weights.

Week	Control Group	S.D.*	Treatment Group	S.D.*
Baseline	132.9	4.08	132.3	14.72
1	130.8	4.44	130.7	13.37
2	132.2	4.56	129.6	13.01
3	132.9	5.45	128.9	13.05
4	132.5	6.69	128.5	12.74
5	133.4	3.80	128.9	13.45
6	134.1	5.01	129.1	13.07

*S.D. = Standard Deviation

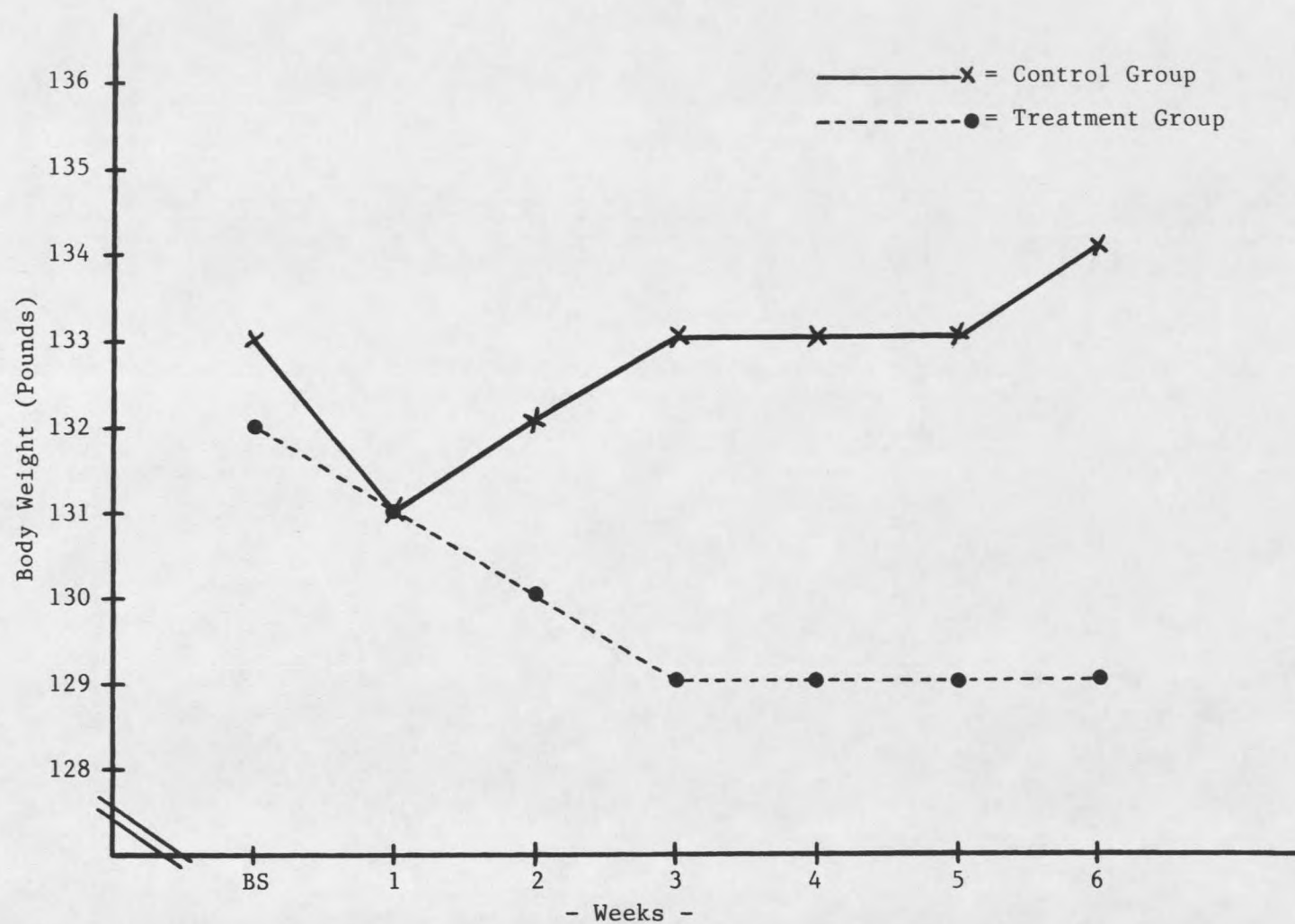


Figure 2. Mean weekly body weights for control and treatment groups.

Table 3. Body weights of bulimic subjects.

Treatment Subjects	Body Weight		Control Subjects	Body Weight	
	Pre	Post		Pre	Post
1	123.0	120.0	7	138.0	141.8
2	137.0	138.5	8	136.0	132.5
3	144.0	148.5	9	128.5	133.0
4	153.0	133.5	10	129.5	135.0
5	114.8	116.5	11	132.5	128.0
6	122.3	117.8			

Other Measurements

Body Fat

The percentages of body fat in both the treatment and control groups changed at a non-significant level ($p = .27$) (Table 6). No subject in either group had an excessive amount of body fat. Subject 4, who lost twenty pounds in the nutrition program, had a 9.7% decrease in body fat (Table 4).

Eating Attitudes Test (EAT)

The EAT reflects the disturbed eating and body shape attitudes of patients with bulimia; the higher the score, the more disturbed the attitudes. The EAT results before and after treatment in this study showed that the treatment subjects changed more profoundly than the bulimic subjects in the control group (Table 5). However, Subject 4, who lost twenty pounds, showed the least improvement on EAT scores in

Table 4. Percentages of body fat of bulimic subjects.

Treatment Subjects	% Body Fat		Control Subjects	% Body Fat	
	Pre	Post		Pre	Post
1	15.5	16.0	7	21.7	21.7
2	24.2	23.2	8	18.3	20.6
3	18.3	19.5	9	18.5	18.3
4	25.7	16.0	10	17.2	18.3
5	15.3	15.3	11	20.6	18.3
6	20.6	18.3			

Table 5. EAT scores of bulimic subjects.

Treatment Subjects	EAT Scores		Control Subjects	EAT Scores	
	Pre	Post		Pre	Post
1	36	7	7	42	38
2	36	8	8	46	39
3	41	25	9	43	42
4	40	36	10	26	15
5	35	17	11	35	32
6	49	35			

the treatment group. The reason for this effect of the treatment program could have been: (1) the behavioral component where positive thinking was stressed; (2) the educational component, where appropriate body weights and percentage of body fat were discussed; or (3) the improved nutritional intake during the program enabling treatment subjects

to have more rational thinking. It appears probable that a combination of any or all three components was helpful. The difference in pre- and post-test scores (Table 6) between the treatment and control groups was significant ($p < .05$). The treatment group scored much lower on post-scores compared to pre-scores.

Nutrient Analysis

Although the difference between treatment and control groups (interaction effect) was statistically non-significant for calories, a clinical significance can be argued (Table 6). The quality of the increased caloric intake in the treatment subjects positively affected the total overall nutrient intakes. Treatment subjects consumed more nutrient-dense foods, thereby increasing their protein, vitamin, and mineral intakes.

Carbohydrate and fat measurements are in grams rather than percentage of Recommended Dietary Allowances (RDA). No RDA's have been established for these two nutrients because their recommended intakes are dependent upon total caloric intake and need rather than on amounts needed to prevent dietary deficiencies. Changes in both carbohydrate and fat intake were non-significant in the results of treatment group versus control group (Table 6) ($p = .68$ and $p = .48$, respectively).

Most nutrient levels decreased from pre- to post-treatment in the control group. The treatment group increased protein, calcium, Vitamin A, thiamine, riboflavin, niacin, and Vitamin B12 from below 67% of their RDA to above that level (67% of the RDA is set as the level to prevent a deficiency state for those nutrients). The control group failed to

Table 6. Summary of means for other measurements.

Measurement	Control Group				Treatment Group				p Value
	Pre	S.D.*	Post	S.D.*	Pre	S.D.*	Post	S.D.*	
% Body fat	19.26	1.84	19.44	1.61	20.02	4.28	17.97	3.06	p = .27
EAT scores	38.40	8.02	33.20	10.60	39.50	5.24	21.30	12.79	p ≤ .05
Calories (% RDA)	36.60	30.08	38.60	13.79	38.50	24.17	58.40	18.64	p = .27
Carbohydrates (gm)	81.00	48.58	95.60	38.39	106.50	62.83	136.50	18.54	p = .68
Fat (gm)	38.60	44.35	38.00	17.69	28.00	17.43	41.20	21.12	p = .48
Protein (% RDA)	59.60	55.20	56.60	22.28	52.40	34.53	127.30	31.23	p ≤ .01
Calcium (% RDA)	39.00	35.95	37.60	7.30	48.40	35.62	83.60	34.46	p ≤ .05
Phosphorus (% RDA)	40.90	40.95	13.90	13.91	48.20	48.21	45.90	45.90	p ≤ .05
Iron (% RDA)	20.60	19.77	17.80	8.50	28.70	23.16	47.40	13.92	p ≤ .04
Potassium (% RDA)	30.80	31.40	26.60	10.43	34.20	23.89	52.50	11.43	p = .08
Vitamin A (% RDA)	64.60	60.29	29.00	17.44	55.90	35.90	147.80	57.15	p ≤ .01
Thiamine (% RDA)	46.40	46.07	36.40	7.30	46.60	35.98	85.50	22.65	p ≤ .02
Riboflavin (% RDA)	57.20	53.08	45.20	11.37	61.10	44.43	101.70	29.07	p ≤ .03
Niacin (% RDA)	43.40	45.79	51.00	17.36	48.20	33.21	114.50	39.24	p ≤ .03
Vitamin C (% RDA)	54.00	44.18	44.40	47.35	101.20	77.19	153.20	77.47	p = .22
Folacin (% RDA)	21.90	21.89	10.50	10.52	20.40	26.39	14.10	14.07	p ≤ .01
Vitamin B12 (% RDA)	33.60	30.39	19.80	5.26	37.00	39.88	73.80	36.55	p ≤ .02
Zinc (% RDA)	15.40	17.08	12.80	7.16	21.40	16.73	37.70	10.05	p ≤ .03

*S.D. = Standard Deviation

improve any nutrient levels to above the 67% RDA level. The percentages of RDA's (adjusted for age and sex) for protein, calcium, phosphorus, iron, Vitamin A, thiamine, riboflavin, niacin, folacin, Vitamin B12, and zinc were all significant ($p < .05$) interaction effects between the treatment and control groups (Table 6), with treatment improving the nutritional quality of the diets. Differences in potassium and Vitamin C were non-significant ($p = .08$ and $p = .22$, respectively) (Table 6). Individual nutrient intakes for all subjects are listed in Appendix D (Raw Data).

The variability of caloric content in binge/purge episodes was found to be substantial. Especially noteworthy was the difference among subjects (Table 7). Subject 4 reported consuming the highest number of calories in binges. Caloric content ranged from 8321 to 3388 and from 8025 to 3348 in the pre/post phases of the study. When Subject 4 entered the nutrition program, she was not keeping down any meals. During the program, she began keeping down some food and decreased the bingeing/purging frequency. She lost twenty pounds in the program. This weight loss was probably due to body fluid loss and less overall caloric consumption (binge/purge episodes were a substantial source of calories in this subject). Subject 9 purged one slice of white toast and a diet pop (61 calories). Her "binges" were generally the lowest among the eleven subjects in both pre- and post-phases of the study, indicating more anorexia nervosa tendencies. Mitchell (1984) estimated the mean caloric intake for a binge to approximately 4000 to 5000 calories; whereas in the present study, the bulimic subjects averaged about 2600 to 2800 calories in binge/purge episodes.

Table 7. Calories consumed during binge/purge episodes in bulimic subjects.

Subject	Pre-Phase		Post-Phase	
	Highest	Lowest	Highest	Lowest
1	1082	965	914	N/A*
2	1852	743	1494	N/A
3	3653	1037	N/A	N/A
4	8321	3388	8025	3348
5	2243	671	N/A	N/A
6	2912	615	N/A	N/A
7	1031	248	969	368
8	2482	403	2262	348
9	1216	61	652	163
10	2283	1535	2363	1073
11	1536	667	5812	631
Average	2601	969	2811	989

*N/A = No data available

The actual foods consumed during the binge/purge episodes listed above are found in Appendix E. Although the types and amounts of foods consumed varied greatly among subjects, some common foods included candy bars, ice cream, pizza, French fries, cereals, breads, butter, mayonnaise, potato or corn chips, sugar, and alcoholic beverages. Diet pop and milk were often used to "help bring food up."

CHAPTER 5

DISCUSSION

According to Fairburn (1985b), four criteria constitute the minimum requirements for inferences to be drawn concerning the effectiveness of the treatment in question. These criteria include: (1) the treatment of at least eight patients is described, (2) the patients have (or seem likely to have) fulfilled diagnostic criteria for DSM-III bulimia or for bulimia nervosa; (3) a single treatment was applied in a standard fashion and there was no other concurrent treatment (or if there was concurrent treatment, this was the same for all patients); and (4) data are presented on patients' eating habits, both at the beginning and end of treatment. This present study fulfilled these criteria. It should be noted that it was difficult to find a large population sample. In particular, control subjects tended to drop out of psychotherapy. Two subjects dropped out toward the end of the study and their data were used without one last session of psychotherapy. It was interesting to note that their binge/purge frequency actually decreased upon discontinuing psychotherapy. Two other subjects in the control group dropped out after the first session and their data were not used in the study.

Seeking help itself appeared to be effective in decreasing the frequency of self-induced vomiting in the treatment and control subjects. This may be explained by the cyclical nature of psychological illness where a person seeks help when his/her problem is at its worst.

The severity of the problem may be alleviated by time or by talking to someone and admitting the problem for the first time. In this study, the subjects may have improved because treatment was promised (either nutrition or psychotherapy).

The treatment subjects all reported that their binges were decreased in the amounts of foods consumed, even when purging persisted. It was also noted that the higher the caloric content of binges, the higher the frequency of binge/purge episodes. When these subjects "lost control," they tended to continue bingeing, and consequently, purging. Helping the bulimic patient to decrease the amounts of foods consumed during binges may be useful in helping her/him gain control over the disordered eating. Eventually, as the patient gains more confidence with eating, the urge to vomit may lessen. There was no evidence found in the literature to support this postulation.

Some of the subjects in the study used concurrent purging techniques to control body weight. Subject 2 used diuretics (no more than two over-the-counter pills per day); however, she discontinued using them during the nutrition program. Subjects 3 and 5 used laxatives occasionally (no more than seven on any one day), but this behavior also discontinued during the nutrition program. Other researchers have noted that more than one purging method may often be used by bulimic patients (Mitchell, 1984; Garner and Garfinkel, 1985). All of the bulimic subjects in this study admitted to stringently dieting prior to developing bulimia. This concurs with other findings in the literature. The more the bulimic tries to diet, the more propensity he/she has toward bingeing (Mitchell, 1984; Polivy and Herman, 1985; Wooley and Wooley, 1985).

The nutrition program was developed to promote increased knowledge of nutrition as well as to develop healthy eating patterns. Attitudes about body weight and dieting should have been improved as well. The Eating Attitudes Test (EAT) was used as a measure in this study to determine whether a highly disturbed attitude toward food and eating could be modified. The highest scores in this test indicate more anorexia nervosa tendencies. Subject 4, who lost twenty pounds during the nutrition program, had the least improvement on the EAT measure. She often binged on fewer calories and purged less during the treatment program while actually increasing the amount of food kept down; however, her attitudes toward weight and eating remained disturbed. She changed her disordered eating patterns from totally out of control to a type of control similar to anorexia nervosa patients. The youngest patient, Subject 6 in the treatment group, had the highest score on EAT. This may have been an indication of her immaturity and/or more propensity toward anorexia nervosa. Although changing attitudes does not necessarily change behavior, the subjects in the treatment group had lower scores on the EAT from pre- to post-treatment. These lower scores reflected more confidence about their bodies, less preoccupation about eating and weight, fewer urges to binge and purge, and that body weight and eating were still important, but not all-consuming.

The nutrition program used in this study resulted in an effective and significant improvement of nutritional intake for treatment subjects. Most authorities in the field of bulimia research stress the importance of improving nutritional intake as part of the recovery process in the treatment of bulimic patients (Fairburn et al., 1985;

Garner and Garfinkel, 1985; Mitchell et al., 1985; Wooley and Wooley, 1985).

In a study conducted by Dalvitt-McPhillips (1984), nutrient-dense, pre-planned diets were shown to eliminate binge/purge behavior. The food plan was conducted so that over each 24-hour period, no individual could consume fewer than 1400 calories, the minimum level set for this study based upon the investigator's observation that diets under 1400 calories precipitated bingeing in patients. No upper limit was put on caloric intake. This investigator's treatment plan was totally free of alcohol, caffeine, refined sugar, white flour products, cigarettes, all medications, and salt except for the salt occurring in several of the foods listed on the food plan.

In the present nutrition intervention study the frequency of binge/purge episodes was decreased, not eliminated. One important difference in the present study, as opposed to the Dalvitt-McPhillips methodology, was that in this study individual recommendations and guidelines were given by the nutritionist; but the actual meal planning was performed by the treatment subjects after instruction. Flexibility in food selection was stressed so that binge foods could be eventually viewed as "safe" in moderate amounts. The consumption of binge foods was not encouraged until the treatment subject no longer looked at that food as "bad." Alcohol consumption was discouraged because of its depressant nature; however, it was not prohibited.

CHAPTER 6

CONCLUSIONS

As a result of this study, it can be concluded that the nutrition intervention group was as effective as the psychotherapy group in decreasing the weekly frequency of binge/purge episodes in women with bulimia. However, the effectiveness of treatment versus control was nonsignificant.

Both the nutrition therapy and the psychotherapy groups were helpful in maintaining body weight which is deemed as important by several researchers as part of the recovery process. The percentage of body fat also showed nonsignificant changes over the experimental period. Exercise was stressed in the treatment group; however, all of the women had already established adequate activity patterns.

The nutrition group had a more significant change in attitudes toward their body weights and eating patterns than the control group. This change could reflect a longer lasting effect to clients because attitudes can often directly influence behavior.

The most profound result of this study was the nutritional adequacy of food intake retained (food kept down). The treatment group showed more significant increases of nutrients than the control group. The nutrition education component of the treatment group helped in achieving improved nutritional intake which should help positively affect recovery outcome since malnutrition has been shown to have a direct influence on

depression, fluctuations in blood sugar and insulin levels, and neurotic symptoms (Wooley and Wooley, 1985; Fairburn et al., 1985; Keys et al., 1950).

Future research into the helpfulness of nutrition education is warranted. In particular, this researcher feels that a study should be done which tests psychotherapy in conjunction with nutrition intervention along with a control group using a larger population size.

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APPENDICES

APPENDIX A

SUBJECT CRITERIA;
DIAGNOSTIC CRITERIA FOR BULIMIA

SUBJECT CRITERIA

Subjects must meet each of the following criteria in order to be included in the study:

- (1) The subject reports incidents of compulsive or binge eating (defined as eating when not hungry or with a sense of loss of control).
- (2) The subject counteracts any possible weight gain from binge eating by vomiting.
- (3) The pattern of bingeing and purging is repetitive.
- (4) The subject is preoccupied with weight, diet, and/or body size as evidenced by excessive, expressed concern about these variables.
- (5) The subject is within a normal weight range for height and age (not more than 20% above normal or more than 25% below normal).
- (6) The subject has not received psychotherapy, professional counseling, or nutrition intervention in the last six months prior to this study.
- (7) Binge/purge episodes are not less than a frequency of four episodes per week.
- (8) The subject has had no prior hospitalization(s) for suicidal attempts or poor medical status including electrolyte imbalances, heart arrhythmias, etc.
- (9) Subjects are not currently (or within the last month) using any type of antidepressant medication.

DIAGNOSTIC CRITERIA FOR BULIMIA*

- (1) Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually less than two hours).
- (2) At least three of the following:
 - (a) consumption of high-caloric, easily ingested food during a binge;
 - (b) inconspicuous eating during a binge;
 - (c) termination of such eating episodes by abdominal pain;
 - (d) sleep, social interruption, or self-induced vomiting;
 - (e) repeated attempts to lose weight by severely restrictive diets, self-induced vomiting, or use of cathartics or diuretics;
 - (f) frequent weight fluctuations greater than 10 pounds due to alternating binges and fasts.
- (3) Awareness that the eating pattern is abnormal and fear of not being able to stop eating voluntarily.
- (4) Depressed mood and self-depreciating thought following eating binges.
- (5) The bulimic episodes are not due to anorexia nervosa or any known physical disorder.

Source: Diagnostic and Statistical Manual for Mental Disorders, 3rd edition (DSM III). Washington, D.C.: American Psychiatric Assn., 1980.

*Bulimia Definition: Deriving from the words "ox" and "hunger," is a term used to describe an eating disorder that occurs when an individual has repeatedly lost control of the impulse to binge and engages in the rapid ingestion of a large quantity of food, followed by attempts to avoid weight gain through self-induced vomiting. The diagnosis of bulimia in contrast to anorexia nervosa does not require a specific degree of weight loss and now is recognized to exist at any weight level from extremely starved to normal to overweight states.

APPENDIX B

CONSENT FORM; EATING ATTITUDES

TEST; NUTRIENTS ANALYZED

CONSENT FORM

Montana State University
Committee on Human Subjects in Research

Title of Project: _____

Name of Researcher: _____

Name of Person Briefing Subject: _____

I, _____, am a willing participant in this project and
[Please print name of participant]
have been informed of the following items:

- I. I have read or had the opportunity to read the proposal which includes the general description of this research project, its purpose and benefits;
- II. I have read the research document and been given an explanation of all procedures to be followed and why I have been asked to participate;
- III. I have been given an explanation of my specific involvement and any foreseeable risks or discomfort;
- IV. I have been given a description of any benefits which may be expected from the research;
- V. I have been given a disclosure of appropriate alternative procedures or courses of treatment that might be advantageous;
- VI. I have been assured of confidentiality of records identifying me as a subject;
- VII. I have been given assurance that minimal risk is involved in my participation in this study;
- VIII. I understand as a voluntary participant that I may withdraw from the experiment at any time that I desire without any loss of benefits to which I am entitled;
- IX. I have been given the opportunity to ask questions at any time about the experiment, my rights and whom to contact in the event of research-related injury to me and all questions have been answered to my satisfaction.

SIGNATURE: _____

If the person giving consent is not the participant, a statement that he/she is legally authorized to represent the participant must be included, e.g.:
I am the parent or legal guardian of _____

If drugs are involved, this form will not be used as we need specific information about the drug being used.

EATING ATTITUDES TEST
(EAT-26)

Name: _____ Date: _____ Age: _____

Present Weight: _____ (lbs) Height: _____ Sex: _____

Highest Past Weight: _____ How Long Ago? _____

Lowest Past Adult Weight: _____ How Long Ago? _____

Instructions: Please place an (X) under the column which best applies to each of the numbered statements. All of the results will be strictly confidential. Most of the questions directly relate to food or eating, although other types of questions have been included. Please answer each question carefully. Thank you.

ALWAYS	USUALLY	OFTEN	SOMETIMES	RARELY	NEVER	
☐	☐	☐	☐	☐	☐	(1) Am terrified about being overweight.
☐	☐	☐	☐	☐	☐	(2) Avoid eating when I am hungry.
☐	☐	☐	☐	☐	☐	(3) Find myself preoccupied with food.
☐	☐	☐	☐	☐	☐	(4) Have gone on eating binges where I feel that I may not be able to stop.
☐	☐	☐	☐	☐	☐	(5) Cut my food into small pieces.
☐	☐	☐	☐	☐	☐	(6) Am aware of the calorie content of foods that I eat.
☐	☐	☐	☐	☐	☐	(7) Particularly avoid foods with a high carbohydrate content (e.g., bread, rice, potatoes, etc.)
☐	☐	☐	☐	☐	☐	(8) Feel that others would prefer if I ate more.
☐	☐	☐	☐	☐	☐	(9) Vomit after I have eaten.
☐	☐	☐	☐	☐	☐	(10) Feel extremely guilty after eating.

Eating Attitudes Test (EAT-26)

Page 2

- | ALWAYS | USUALLY | OFTEN | SOMETIMES | RARELY | NEVER | |
|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--|
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (11) Am preoccupied with a desire to be thinner. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (12) Think about burning up calories when I exercise. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (13) Other people think that I am too thin. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (14) Am preoccupied with the thought of having fat on my body. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (15) Take longer than others to eat my meals. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (16) Avoid foods with sugar in them. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (17) Eat diet foods. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (18) Feel that food controls my life. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (19) Display self-control around food. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (20) Feel that others pressure me to eat. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (21) Give too much time and thought to food. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (22) Feel uncomfortable after eating sweets. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (23) Engage in dieting behavior. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (24) Like my stomach to be empty. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (25) Enjoy trying new rich foods. |
| ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | (26) Have the impulse to vomit after meals. |

Source: Garner, D.M., and Garfinkel, D.E. EAT. Toronto, Canada: Toronto General Hospital, 1979.

Table 8. Nutrients analyzed by computer.

Nutrients Analyzed	
Calories	Vitamin A
Carbohydrates	Thiamin
Protein	Riboflavin
Fat	Niacin
Calcium	Ascorbic Acid
Phosphorus	B12
Iron	Zinc
Potassium	Folacin

APPENDIX C

NUTRITIONAL TREATMENT FOR BULIMIA;
OUTLINE OF RESPONSIBILITIES

NUTRITIONAL TREATMENT FOR BULIMIA: SCOPE & SEQUENCE OUTLINE

Eating	Education	Cognitions	Concepts	Exercise
Build awareness of internal cues for hunger & satiety regulation.	Provide information to support behavior changes.	Make self-talk positive.	Permission is the paradox that gives control.	Shape aerobic exercise by increasing frequency & duration, if needed.
Goal setting.	Explain futility of binge/purge cycle.	"I don't have to hound myself about dieting/weight."	Taking the responsibility & making the choice to binge and/or purge.	
Build experience of positive, deliberate, & focused eating.	Record keeping.	"I can find attractive clothing that fits me <u>now</u> so I can get the pressure off my weight."	Appetite & hunger are equally important in food regulation.	Goal setting.
Weight loss will not be undertaken in the program. In fact, the client may gain weight if binge/purge episodes decrease (most likely due to rehydration).	Nutrition education: - Why do you need food? - Key roles of carbohydrates, proteins & fats. - Use & misuse of food. - What foods stay with you? - Normal eating. - Take pressure off weight. - Exercise & hunger.	"A degree of change is success."	Make the decision to eat what you really want and to control impulses. - "I don't want that now, but I can have it later if I want it then."	Use heart rate for monitoring & assessing intensity.
Focused eating exercises.	Food intake regulation.		Mental or actual food deprivation leads to overeating or overcompensation.	
	Physiology of weight regulation.		Eating is flexible.	
	Healthy eating instruction: - Food exchange lists - Menu planning		Tight-fitting clothing puts pressure on eating & weight.	
	Provide list of alternative actions for eating.			

OUTLINE OF RESPONSIBILITIES

Subject	Investigator
Makes and keeps appointment with investigator.	Interviews subject. Provides assessment measure to determine whether or not project is appropriate to participate in the study. Confers with subject's M.D.
<i>Pre-Baseline Phase</i>	
Has urinalysis and blood testing done by M.D. or Student Health Service.	Administer EAT-26.
<i>Baseline Phase</i>	
Patient will record the number of binge/purge episodes daily and keep food record.	Plot information on M-B* graph.
Meets with investigator in one week.	Take weight, height, and % body fat (using three sites: triceps, thigh, and suprailiac). Record this information.
Negotiates contract with investigator concerning commitment to program.	Set up appointment for next week.

Outline of Responsibilities
Page 2

Subject	Investigator
<i>Post-Baseline Phase</i>	
<u>Week One</u> -- Treatment: Phase I. Information giving & nutrition education.	
Patient will define her own problem. What expectations does patient have of treatment? What is the dietary concern of utmost importance?	Provide list of questions to determine treatment subgoals.
Define hunger; appetite. Why do you eat?	Explain binge/starve cycle & physiological basis for overeating in response to food deprivation.
Record the Food Intake Form until next session.	Explain that weight loss cannot be undertaken in this study and that weight gain may occur due to the body's rehydration (especially if laxatives, diuretics, or self-induced vomiting are used).
Record frequency of binge/purge episodes weekly.	Explain that food intake records will be gathered at each session. Explain importance of accuracy of these records for research purposes.
Establish one realistic goal for next session.	Distribute reading packet.
	Plot information on binge/purge graph; record weight.
	Analyze food intake record.

Outline of Responsibilities

Page 3

Subject	Investigator
<u>Week 2</u> -- Treatment: Phase I. Information giving & nutrition education.	
Discuss past week; goal accomplishment. Discuss food records. Discuss reading packet.	Food records gathered.
Record Food Intake Forms.	Weight recorded.
Plan one week's menus.	Plot information on binge/purge graph.
Establish one realistic goal.	Nutrition education lecture.
Record frequency of binge/purge episodes weekly.	Provide <u>Guide to Wise Food Choices</u> handout. Topics covered:
	(1) Why do you need food? (2) Key roles of carbohydrates, protein, & fat in the body. (3) What foods stay with you? (4) Issues concerning normal eating and giving permission. (5) Taking pressure of weight to establish normal, individual eating patterns. (6) Exercise & hunger -- working on increasing exercise frequency & duration, if needed. (7) The misuse of food.

Outline of Responsibilities

Page 4

Subject	Investigator
<u>Week 3</u> -- Treatment: Phase I. Information giving & nutrition education.	
Discuss past week; goal accomplishment. Discuss food records. Note improvements. Discuss planning menu and its helpfulness.	Food records gathered. Observe menu plans. Weight recorded.
Define & explain good nutrition examples in past week's diet.	Plot information on binge/purge graph.
Record Food Intake Forms.	Question client about feelings concerning eating and weight.
Plan one week's menu.	Go over menu plans & point out positive aspects.
Establish one realistic goal.	Was client able to <u>implement</u> menu?
Record frequency of binge/purge episodes weekly.	Use checklist to determine behaviors which could be changed.
	Provide feedback & suggestions for menus which were written.
	Provide handout on <u>Control Your Eating Environment</u> . Discuss this handout.
	Give examples of positive self-talk. Reemphasize giving permission and making choices concepts.

Outline of Responsibilities

Page 5

Subject	Investigator
<u>Week 4</u> -- Treatment: Phase II. Dietary Instructions.	
Discuss past week; goal accomplishment.	Record weight.
Discuss food records and menus.	Plot information on binge/purge graph.
Note improvements.	Provide feedback & suggestions for menus which were written.
Record frequency of binge/purge episodes weekly.	Investigator provides dietary instruction:
Record Food Intake Forms.	(1) Normal body composition. (Provide feedback on client's % body fat.)
Prepare meal plan worksheets.	(2) Discussion on very low-calorie diets and/or fasting.
Bring individualized meal pattern.	(3) Discussion on activity patterns & body composition.
Practice a day's menu on worksheet.	(4) Establish patient's caloric needs together.
Establish one realistic goal.	(5) Provide a <u>Meal Planning System for Healthy Eating</u> manual.
Bring a <u>feared</u> food to next session.	(6) Provide the meal plan worksheets for one week & the individualized meal pattern.

Outline of Responsibilities
Page 6

Subject	Investigator
<u>Week 5</u> -- Treatment: Phase II. Dietary Instructions.	
Discuss past week; goal accomplishment. Discuss food records and menu plans. Note improvements.	Record weight. Gather food records and menus.
Provide list of questions of problems encountered with menu planning assignments.	Plot information on binge/purge graph.
Patient will identify intervention points & alternatives to binge, overeat, and/or purge.	Provide feedback & suggestions for menus which were written.
Record Food Intake Forms.	Provide feedback to patient about accuracy of forms completed. Note discrepancies.
Practice on meal planning worksheets.	Discuss "cue" recognitions.
Bring individualized meal pattern.	Provide list of alternative actions.
Participate in Focused Eating Exercise.	Discuss making the choice to binge. Client must accept responsibility for actions.
Record frequency of binge/purge episodes weekly.	To aid in helping client view food more positively, investigator to use Focused Eating Exercise.
Establish one realistic goal.	Emphasize internal vs. external eating cues.
Practice Focused Eating Exercise at least once during week.	

Outline of Responsibilities

Page 7

Subject	Investigator
<u>Week 6</u> -- Treatment: Phase III. Shaping & Maintaining Healthy Eating Patterns.	
Discuss past week; goal accomplishment. Discuss food records and menu plans. Note improvements.	Record weight and % body fat.
Review of checklist of behavior changes.	Gather food forms & meal plans.
Discussion on handouts concerned with maintaining healthy eating patterns.	Plot information on binge/purge graph.
Respond to the following questions:	Provide feedback on forms gathered.
(1) What is normal now compared to one month ago? (2) What has happened to the binge/purge frequency since you started? (3) How has the quality of your diet changed? (4) What eating behaviors have changed? (5) What is hunger; appetite? (6) Are you more aware of cues to binge? (7) Have you discovered ways to intervene or to select alternative actions to binges and/or purges? (8) What has happened to your food/weight preoccupation?	Provide the following handouts: (1) Five Steps to Healthy Eating (2) Ascribe to Healthy Eating & Activity Patterns. (3) Activity Patterns. (4) Resources. (5) Most Frequently Asked Questions About Food & Meal Planning. Evaluate program by asking several questions. EAT-26 given. Schedule follow-up in 4 weeks.

APPENDIX D

RAW DATA

Table 9. Frequency of binge/purge episodes in treatment subjects.

Subjects	Baseline	Weeks						Follow-up
		1	2	3	4	5	6	
1	10	10	4	4	4	2	1	2
2	7	7	5	5	4	3	1	2
3	14	9	3	5	8	0	0	2
4	12	10	10	8	8	11	6	7
5	22	25	18	21	23	16	11	14
6	11	5	6	6	5	4	0	3

Table 10. Frequency of binge/purge episodes in control subjects.

Subjects	Baseline	Weeks						Follow-up
		1	2	3	4	5	6	
7	17	7	9	7	14	9	6	0
8	45	8	16	14	15	21	14	17
9	14	16	17	16	10	11	10	7
10	14	5	9	7	12	10	8	4
11	21	19	15	11	11	6	11	7

Table 11. Nutrient intake of treatment subjects.

Subjects	Nutrients Analyzed							
	Calories (kcal)/ % RDA*		Carbohydrate (gm)/% RDA*		Protein (gm)/ % RDA*		Fat (gm)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	989/49	1160/55	120	131	28.5/64	51.7/117	44.1	48.2
2	1308.5/65	1541/76.5	171.5	158	57.5/79.3	74.1/168	38.7	62.95
3	1224/58	1294/61	162	158	42.5/96	67.9/154	42.4	45.1
4	5.87/0	835/39	0	138	0/0	41.5/94	0	15.7
5	737/36	1642/82	76.5	118	15.8/35	61.0/138	27.4	60.2
6	656/23	783/37	109	116	22.8/40	43.1/93	15.6	15.1

*RDA = Recommended Dietary Allowance.

Table 12. Nutrient intake of control subjects.

Subjects	Nutrients Analyzed							
	Calories (kcal)/ % RDA*		Carbohydrate (gm)/% RDA*		Protein (gm)/ % RDA*		Fat (gm)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
7	854/42	401/20	127	60.4	27/58	10.3/23	31.4	14.3
8	552/26	1131/53	82.3	159	13.6/29	23.6/51	21.2	46.6
9	203/9	969/46	25.4	97.5	2.8/6	28.5/64	4.18	53.6
10	1819/86	946/45	131	90.3	69.9/151	38.8/84	116	51.5
11	432/20	599/28	39.4	71	24/54	27.2/61	20.2	24.2

*RDA = Recommended Dietary Allowance.

Table 13. Vitamin intake of treatment subjects.

Subjects	Nutrients Analyzed							
	Vitamin A (IU)/ % RDA*		Thiamin (mg)/ % RDA*		Riboflavin (mg)/ % RDA*		Niacin (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	2663/66	2875/71	.44/44	.93/84	.61/50	1.18/90	5.96/45	11.2/80
2	2660/66.5	7426.5/185	.725/72.5	.91/91	1.345/111.5	1.585/131	10.195/78	17.55/135
3	4439/110	8705/217	1.09/99	1.37/124	1.5/115	1.76/135	13.1/93	18.2/130
4	0/0	.5651/141	0/0	.65/59	0/0	.74/56	0/0	7.54/53
5	1847/46	3705/92	.17/17	.67/67	.49/40	1.23/102	5.11/39	20.3/156
6	2380/47	7274/181	.65/46	.97/88	.85/50	1.25/96	6.28/34	18.7/133

*RDA = Recommended Dietary Allowance.

Table 13--Continued

Subjects	Nutrients Analyzed					
	Vitamin C (mg)/ % RDA*		Folacin (ug)/ % RDA*		Vitamin B12 (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post
1	46.0/76	162/270	91.6/22	221/55	.54/18	1.87/62
2	97.7/162.5	77.8/130	149.5/37	230.5/57	2.24/74	2.16/71.5
3	130/216	87.7/146	238/59	230/57	2.97/99	3.77/8.04
4	0/0	51.6/85	0/0	105/26	0/0	1.26/42
5	34.4/57	42.1/70	85.2/21	209/52	.28/9	.99/33
6	57.6/96	131/218	168/42	275/68	.68/22	3.27/109

*RDA = Recommended Dietary Allowance.

Table 14. Vitamin intake of control subjects.

Subjects	Nutrients Analyzed							
	Vitamin A (IU)/ % RDA*		Thiamin (mg)/ % RDA*		Riboflavin (mg)/ % RDA*		Niacin (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
7	2491/62	1575/39	.9/81	.42/42	.85/65	.53/44	8.19/58	3.83/29
8	476/11	580/14	.17/15	.34/30	.62/47	.58/44	1.01/7	6.59/47
9	126/3	425/10	.11/9	.46/41	.06/4	.6/46	.85/6	8.52/60
10	5835/145	2097/52	1.21/110	.47/42	1.86/143	.81/62	16.3/116	10.5/75
11	4114/102	1227/30	.19/17	.3/27	.36/27	.39/30	4.25/30	6.18/44

*RDA = Recommended Dietary Allowance.

Table 14--Continued

Subjects	Nutrients Analyzed					
	Vitamin C (mg)/ % RDA*		Folacin (ug)/ % RDA*		Vitamin B12 (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post
7	69.3/115	75/125	123/30	121/30	1.08/36	.61/20
8	13.6/22	24.6/41	10.8/2	34.5/8	.73/24	.66/22
9	10.9/18	1.05/1	13.2/3	15.3/3	.04/1	.52/17
10	52.4/87	15.6/26	217/54	80.1/20	2.5/83	.83/27
11	17.3/28	17.5/29	55.5/13	60/15	.72/24	.41/13

*RDA = Recommended Dietary Allowance.

Table 15. Mineral intake of treatment subjects.

Subjects	Nutrients Analyzed									
	Calcium (mg)/ % RDA*		Phosphorus (mg)/ % RDA*		Iron (mg)/ % RDA*		Potassium (mg)/ % RDA*		Zinc (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	382/47	738/92	478/59	926/115	5.36/29	7.51/41	1195/31	1971/52	5.36/36	5.23/34
2	646.5/80.5	801/99.5	978.5/121.5	1257.5/157	8.22/45	11.5/63.5	2263/60	2193/58	4.2/27.5	6.66/44
3	791/98	1069/133	999/124	1374/171	11.6/64	11.8/65	2157/57	2469/65	6.58/43	8.04/53
4	0/0	491/61	0/0	691/86	0/0	5.6/31	0/0	1255/33	0/0	4.02/26
5	272/34	679/84	514/64	1143/142	2.12/11	6.93/38	525/14	1787/47	1.44/9	6.0/40
6	381/31	394/32	458/38	604/50	4.31/23	8.44/46	1315/43	1850/60	1.99/13	4.36/29

*RDA = Recommended Dietary Allowance.

Table 16. Mineral intake of control subjects.

Subjects	Nutrients Analyzed									
	Calcium (mg)/ % RDA*		Phosphorus (mg)/ % RDA*		Iron (mg)/ % RDA*		Potassium (mg)/ % RDA*		Zinc (mg)/ % RDA*	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
7	383/31	242/30	498/41	234/29	4.0/22	1.19/6	1063/34	701/18	2.49/16	1.04/6
8	425/35	430/35	396/33	538/44	1.55/8	2.41/13	662/21	878/28	.71/4	1.72/11
9	20.5/2	320/40	35.1/4	538/67	.97/5	4.09/22	102/2	894/23	.22/1	1.07/7
10	1188/99	598/49	1391/115	640/53	9.87/54	5.09/28	2556/83	1368/44	6.66/44	3.58/23
11	228/28	272/34	360/45	416/52	2.56/14	3.61/20	555/14	783/20	1.88/12	2.69/17

*RDA = Recommended Dietary Allowance.

APPENDIX E

FOODS CONSUMED DURING
BINGE/PURGE EPISODES

Table 17. Foods consumed in pre- and post-phases, treatment group, subject 1.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Cereal-oats-cooked	2	cups
Sugar-brown-pressed down	6	tablespoons
Butter-regular-tablespoon	2	tablespoons
Bread-whole wheat-firm	2	slices
Butter-regular-tablespoon	1	tablespoon
Oranges-raw-all varieties	1	item

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1082 Kc	51	Niacin	3.053 mg	21
Carbohydrate	167.9 gm	--	Folacin	86.56 ug	21
Protein	18.41 gm	41	Phosphorus	529.4 mg	66
Fat	41.34 gm	--	Zinc	3.251 mg	21
Potassium	883.8 mg	23	Vitamin C	69.70 mg	116
Iron	7.899 mg	43	Riboflavin	0.299 mg	23
Vitamin A	1629 IU	40	Vitamin B12	0.000 ug	0
Thiamine	0.821 mg	74	Calcium	208.2 mg	26

Protein: 7% Carbohydrate: 60% Fat: 33% Alcohol: 0%

Table 17--Continued

Pre-Phase Lowest Binge		
Food Name	Serving	Portion
Porkchop-lean-broiled	1	item
Potato-bake-peel after	1	item
Butter-regular-tablespoon	1	tablespoon
Rice-Spanish-home recipe	.5	cup
Roll-whole wheat-homemade	1	item
Cobbler-Weight Watcher	6	ounces
Ice cream-van-hard-10% fat	.5	cup

Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	964.5 Kc	45	Niacin	8.133 mg	58
Carbohydrate	123.8 gm	--	Folacin	20.12 ug	5
Protein	31.05 gm	70	Phosphorus	478.2 mg	59
Fat	40.14 gm	--	Zinc	3.092 mg	20
Potassium	1403 mg	37	Vitamin C	39.05 mg	65
Iron	2.792 mg	15	Riboflavin	0.566 mg	43
Vitamin A	1515 IU	37	Vitamin B12	1.023 ug	34
Thiamine	1.002 mg	91	Calcium	155.4 mg	19

Protein: 13% Carbohydrate: 50% Fat: 37% Alcohol: 0%

Table 17--Continued

Post-Phase Highest Binge		
Food Name	Serving	Portion
Cheese-cream	2	servings
Egg-scrambled-milk/butter	2	items
Cheese-American-processed	.5	piece
Orange juice-frozen-diluted	2	cups
Bagel-water	1	item
Milk-nonfat-fluid	1	cup

Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	914.0 Kc	43	Niacin	3.314 mg	23
Carbohydrate	100.9 gm	--	Folacin	297.2 ug	74
Protein	37.07 gm	84	Phosphorus	723.4 mg	90
Fat	40.50 gm	--	Zinc	3.651 mg	24
Potassium	1656 mg	44	Vitamin C	196.5 mg	327
Iron	4.635 mg	25	Riboflavin	1.067 mg	82
Vitamin A	2492 IU	62	Vitamin B12	2.541 ug	84
Thiamine	0.783 mg	71	Calcium	596.1 mg	74

Protein: 16%	Carbohydrate: 44%	Fat: 40%	Alcohol: 0%
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Table 18. Foods consumed in pre- and post-phases, treatment group, subject 2.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Bread-whole wheat-firm	2	slices
Peanut butter-low sodium	2	tablespoons
Milk-chocolate-2% lowfat	2	cups
Nut-walnut-Persian/English	6	ounces
Sugar-white-granulated	2	tablespoons

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1852 Kc	88	Niacin	4.318 mg	30
Carbohydrate	134.8 gm	--	Folacin	163.9 ug	40
Protein	55.24 gm	125	Phosphorus	1177 mg	147
Fat	134.4 gm	--	Zinc	7.541 mg	50
Potassium	1785 mg	47	Vitamin C	10.13 mg	16
Iron	7.063 mg	39	Riboflavin	1.178 mg	90
Vitamin A	1210 IU	30	Vitamin B12	1.694 ug	56
Thiamine	1.009 mg	91	Calcium	764.2 mg	95

Protein: 2%	Carbohydrate: 5%		Fat: 11%	Alcohol: 81%	

Table 18--Continued

Pre-Phase Lowest Binge							
Food Name			Serving		Portion		
Milk-1%-milk solids added			2		cups		
Cheese-cottage-4%-small curd			1		cup		
Raspberries-raw			.5		cup		
Cereal-oats-cooked			1		cup		
Sugar-brown-pressed down			1		tablespoon		
Milk-2% fat-lowfat-fluid			.75		cup		
Pre-Phase Lowest Binge Nutrient Values							
Nutrient		Value	%RDA	Nutrient		Value	%RDA
K Calories		742.5 Kc	37	Niacin		1.742 mg	13
Carbohydrate		84.36 gm	--	Folacin		73.00 ug	18
Protein		55.91 gm	127	Phosphorus		1129 mg	141
Fat		20.48 gm	--	Zinc		4.888 mg	32
Potassium		1527 mg	40	Vitamin C		22.04 mg	36
Iron		3.029 mg	16	Riboflavin		1.602 mg	133
Vitamin A		1835 IU	45	Vitamin B12		3.848 ug	128
Thiamine		0.591 mg	59	Calcium		1020 mg	127

Protein: 30%		Carbohydrate: 45%		Fat: 25%		Alcohol: 0%	

Table 18--Continued

Post-Phase Highest Binge					
Food Name			Serving		Portion
Candy-milk chocolate-plain			8		ounces
Cola-type-soda			24		fl ounces
Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1494 Kc	71	Niacin	0.810 mg	5
Carbohydrate	211.0 gm	--	Folacin	15.88 ug	3
Protein	16.20 gm	36	Phosphorus	650.6 mg	81
Fat	72.90 gm	--	Zinc	0.312 mg	2
Potassium	896.9 mg	23	Vitamin C	0.000 mg	0
Iron	2.790 mg	15	Riboflavin	0.810 mg	62
Vitamin A	648.0 IU	16	Vitamin B12	0.000 ug	0
Thiamine	0.162 mg	14	Calcium	548.5 mg	68

Protein: 4%		Carbohydrate: 54%		Fat: 42%	
				Alcohol: 0%	

Table 19. Foods consumed in pre- and post-phases, treatment group, subject 3.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Cheese-cheddar-cut pieces	14	ounces
Corn chips	6	servings
Cream-sour-cultured	.67	cup
Sauce-Picante-canned	1	cup
Cereal-Cracklin Bran	1.5	cups
Milk-nonfat-fluid	1	cup
Raisins-seedless	.25	cup
Apple juice-frozen-diluted	1.5	cups

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	3653 Kc	182	Niacin	22.06 mg	169
Carbohydrate	272.0 gm	--	Folacin	420.9 ug	105
Protein	135.8 gm	308	Phosphorus	2918 mg	364
Fat	237.5 gm	--	Zinc	21.51 mg	143
Potassium	2897 mg	77	Vitamin C	125.4 mg	209
Iron	12.26 mg	68	Riboflavin	3.747 mg	312
Vitamin A	11780 IU	294	Vitamin B12	4.705 ug	156
Thiamine	1.962 mg	196	Calcium	3502 mg	437

Protein: 14%	Carbohydrate: 29%	Fat: 57%	Alcohol: 0%
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Table 19--Continued

Pre-Phase Lowest Binge							
Food Name			Serving		Portion		
Beer-Michelob			12		fl ounces		
Whiskey/gin/rum/vodka-80 proof			13.5		fl ounces		
Pre-Phase Lowest Binge Nutrient Values							
Nutrient		Value	%RDA	Nutrient		Value	%RDA
K Calories	1037 Kc	49	Niacin	0.000 mg	0		
Carbohydrate	15.96 gm	--	Folacin	0.000 ug	0		
Protein	1.104 gm	2	Phosphorus	0.000 mg	0		
Fat	0.000 gm	--	Zinc	0.000 mg	0		
Potassium	13.50 mg	0	Vitamin C	0.000 mg	0		
Iron	0.135 mg	0	Riboflavin	0.000 mg	0		
Vitamin A	0.000 IU	0	Vitamin B12	0.000 ug	0		
Thiamine	0.000 mg	0	Calcium	405.0 mg	50		

Protein: 0%		Carbohydrate: 4%		Fat: 0%		Alcohol: 96%	

Table 20. Foods consumed in pre- and post-phases, treatment group, subject 4.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Carrot-raw-whole-scraped	5	items
Crackers-graham-sugar/honey	8	ounces
Milk-2% fat-lowfat-fluid	4	cups
Potato chips-salt added	1.5	pounds
Cream-sour-cultured	16	ounces
Cheese-cheddar-shredded	13	ounces
Pie-apple-home rec	10	ounces

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	8321 Kc	396	Niacin	43.02 mg	307
Carbohydrate	739.3 gm	--	Folacin	566.1 ug	141
Protein	208.3 gm	473	Phosphorus	4749 mg	593
Fat	529.9 gm	--	Zinc	26.52 mg	176
Potassium	13154 mg	350	Vitamin C	333.3 mg	555
Iron	21.72 mg	120	Riboflavin	4.724 mg	363
Vitamin A	110791 IU	2769	Vitamin B12	7.962 ug	265
Thiamine	3.102 mg	281	Calcium	4752 mg	593

Protein: 10% Carbohydrate: 35% Fat: 56% Alcohol: 0%

Table 20--Continued

Pre-Phase Lowest Binge		
Food Name	Serving	Portion
Crackers-saltines	.5	pound
Crackers-graham-sugar/honey	.5	pound
Butter-regular-tablespoon	8	tablespoons
Butter-regular-tablespoon	2	tablespoons
Popcorn-popped-plain	8	cups
Milk-2% fat-lowfat-fluid	1.5	cups

Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	3388 Kc	169	Niacin	16.49 mg	126
Carbohydrate	397.5 gm	--	Folacin	92.51 ug	23
Protein	58.80 gm	133	Phosphorus	990.0 mg	123
Fat	165.4 gm	--	Zinc	8.614 mg	37
Potassium	1249 mg	33	Vitamin C	3.480 mg	5
Iron	18.24 mg	101	Riboflavin	2.422 mg	201
Vitamin A	5030 IU	125	Vitamin B12	1.332 ug	44
Thiamine	11.24 mg	1123	Calcium	614.5 mg	76

Protein: 7%	Carbohydrate: 48%		Fat: 45%	Alcohol: 0%	

Table 20--Continued

Post-Phase Highest Binge		
Food Name	Serving	Portion
Lettuce-iceberg-raw-chopped	3	cups
Soup-chili-beef-can-water	2	cups
Roll-hamburger/hotdog	2	items
Cheese-cheddar-shredded	4	cups
Cream-sour-cultured	1	cup
Corn chips	3	servings
Cheese-cottage-4%-small curd	3	cups
Salad-potato	2	cups
Potato-scallop-home rec	2	cups
Seeds-sunflower-oil roast	1	cup
Cauliflower-frozen-boil	3	cups
Sauce-white-mix/milk	1	cup
Doughnuts-cake-plain	6	items
Ice cream-van-hard-10% fat	1	quart

Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	8025 Kc	401	Niacin	29.09 mg	223
Carbohydrate	540.0 gm	--	Folacin	946.6 ug	236
Protein	327.3 gm	743	Phosphorus	7097 mg	887
Fat	514.4 gm	--	Zinc	40.20 mg	268
Potassium	8847 mg	235	Vitamin C	292.5 mg	487
Iron	31.84 mg	176	Riboflavin	7.088 mg	590
Vitamin A	15285 IU	382	Vitamin B12	12.97 ug	432
Thiamine	3.072 mg	307	Calcium	5867 mg	733

Protein: 16%	Carbohydrate: 27%		Fat: 57%	Alcohol: 0%	

Table 20--Continued

Post-Phase Lowest Binge		
Food Name	Serving	Portion
Potato-boil-peel before	2	items
Butter-regular-tablespoon	8	tablespoons
Popcorn-popped-plain	10	cups
Cereal-100% bran	1	pound
Milk-nonfat-fluid	.5	gallon
Corn chips	1	serving

Post-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	3348 Kc	167	Niacin	150.5 mg	1157
Carbohydrate	546.7 gm	--	Folacin	131.2 ug	32
Protein	141.1 gm	320	Phosphorus	7785 mg	973
Fat	126.7 gm	--	Zinc	53.52 mg	356
Potassium	9826 mg	262	Vitamin C	472.2 mg	786
Iron	59.62 mg	331	Riboflavin	15.33 mg	1277
Vitamin A	7424 IU	185	Vitamin B12	50.71 ug	1690
Thiamine	12.02 mg	1202	Calcium	2789 mg	348

Protein: 15%	Carbohydrate: 56%	Fat: 29%	Alcohol: 0%
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Table 21. Foods consumed in pre- and post-phases, treatment group, subject 5.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Cereal-Wheaties	2	cups
Milk-2% fat-lowfat-fluid	1	cup
Bananas-raw-peeled	2	items
Sugar-white-granulated	2	tablespoons
Cake-coffee-mix/prep	8	ounces
Pumpkin-can	.75	cup
Margarine-unspec ingr-soft	1	tablespoon
Sugar-brown-pressed down	1	tablespoon
Bread-white-firm	3	slices
Margarine-unspec ingr-soft	2	tablespoons
Butter-regular-tablespoon	2	tablespoons
Sugar-white-granulated	2	tablespoons

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2243 Kc	106	Niacin	19.10 mg	136
Carbohydrate	343.3 gm	--	Folacin	123.4 ug	30
Protein	40.23 gm	91	Phosphorus	1029 mg	128
Fat	88.80 gm	--	Zinc	3.405 mg	22
Potassium	2307 mg	61	Vitamin C	61.63 mg	102
Iron	18.87 mg	104	Riboflavin	2.252 mg	173
Vitamin A	46404 IU	1160	Vitamin B12	3.924 ug	130
Thiamine	1.819 mg	165	Calcium	702.4 mg	87

Protein: 7%	Carbohydrate: 59%		Fat: 34%	Alcohol: 0%	

Table 21--Continued

Pre-Phase Lowest Binge		
Food Name	Serving	Portion
Pork-loin-lean-roasted	2	ounces
Salad-potato	.5	cup
Spinach-raw-chopped	2	cups
Mushrooms-raw-chopped	.25	cup
Tomato-raw-red-ripe	.5	item
Salad dressing-thous island	1	tablespoon
Beer-light	12	fl ounces
Biscuits-home recipe	1	item
Butter-regular-tablespoon	.5	tablespoon

Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	670.5 Kc	33	Niacin	6.850 mg	52
Carbohydrate	43.02 gm	--	Folacin	235.3 ug	58
Protein	26.18 gm	59	Phosphorus	331.9 mg	41
Fat	34.57 gm	--	Zinc	2.466 mg	16
Potassium	1402 mg	37	Vitamin C	56.75 mg	94
Iron	5.524 mg	30	Riboflavin	0.630 mg	52
Vitamin A	8814 IU	220	Vitamin B12	0.532 ug	17
Thiamine	0.837 mg	83	Calcium	182.7 mg	22

Protein: 16% Carbohydrate: 26% Fat: 46% Alcohol: 13%

Table 22. Foods consumed in pre- and post-phases, treatment group, subject 6.

Pre-Phase Highest Binge					
Food Name			Serving		Portion
Pizza-pepperoni-baked			6		slices
Ice cream-van-hard-10% fat			1		quart
Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2912 Kc	138	Niacin	31.44 mg	224
Carbohydrate	347.0 gm	--	Folacin	480.0 ug	120
Protein	97.20 gm	220	Phosphorus	536.0 mg	67
Fat	126.2 gm	--	Zinc	5.640 mg	37
Potassium	2324 mg	61	Vitamin C	17.20 mg	28
Iron	15.60 mg	86	Riboflavin	3.044 mg	234
Vitamin A	5364 IU	134	Vitamin B12	4.660 ug	155
Thiamine	2.152 mg	195	Calcium	1880 mg	235

Protein: 13%	Carbohydrate: 48%		Fat: 39%		Alcohol: 0%

Table 22--Continued

Pre-Phase Lowest Binge					
Food Name		Serving		Portion	
Yogurt-fruit flavor-lowfat		1		cup	
Noodles-saimin-wheat-dry		.25		pound	
Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	615.8 Kc	30	Niacin	0.216 mg	1
Carbohydrate	126.6 gm	--	Folacin	21.00 ug	5
Protein	21.26 gm	48	Phosphorus	271.0 mg	33
Fat	2.855 gm	--	Zinc	1.680 mg	11
Potassium	1459 mg	38	Vitamin C	1.500 mg	2
Iron	0.160 mg	0	Riboflavin	0.404 mg	33
Vitamin A	104.0 IU	2	Vitamin B12	1.060 ug	35
Thiamine	0.084 mg	8	Calcium	345.0 mg	43

Protein: 14%	Carbohydrate: 82%	Fat: 4%	Alcohol: 0%		

Table 23. Foods consumed in pre- and post-phases, control group, subject 7.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Potato puffs-frozen-heated	12	items
Corn fritter	.5	item
Frankfurter-hot dog-no bun	1	item
Sandwich-club	1	item

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1031 Kc	49	Niacin	13.81 mg	98
Carbohydrate	75.67 gm	--	Folacin	16.40 ug	4
Protein	46.14 gm	104	Phosphorus	506.0 mg	63
Fat	50.15 gm	--	Zinc	1.290 mg	8
Potassium	442.5 mg	11	Vitamin C	48.50 mg	80
Iron	6.580 mg	36	Riboflavin	0.573 mg	44
Vitamin A	1787 IU	44	Vitamin B12	0.740 ug	24
Thiamine	0.691 mg	62	Calcium	144.0 mg	18

Protein: 20%	Carbohydrate: 32%	Fat: 48%	Alcohol: 0%		

Table 23--Continued

Pre-Phase Lowest Binge					
Food Name			Serving		Portion
Milk-chocolate-2% lowfat			1		cup
Cookie-gingersnap-home recipe			2		items
Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	247.8 Kc	12	Niacin	0.559 mg	4
Carbohydrate	35.44 gm	--	Folacin	13.12 ug	3
Protein	8.672 gm	19	Phosphorus	261.4 mg	32
Fat	8.240 gm	--	Zinc	1.082 mg	7
Potassium	449.4 mg	11	Vitamin C	2.300 mg	3
Iron	0.922 mg	5	Riboflavin	0.434 mg	36
Vitamin A	505.0 IU	12	Vitamin B12	0.859 ug	28
Thiamine	0.120 mg	12	Calcium	289.2 mg	36

Protein: 14%		Carbohydrate: 57%		Fat: 30%	
				Alcohol: 0%	

Table 23--Continued

Post-Phase Highest Binge		
Food Name	Serving	Portion
Roll-submarine-hoagie-enrich	.5	item
Beef-lean/fat-simm/roast	3	ounces
Cheese-cheddar-shredded	2	ounces
Lettuce-butterhead-head	.5	item
Tomato-raw-red-ripe	.5	item
Pickle-dill-cucumber-med	.5	item
Cola-type-soda	12	fl ounces
Salad dress-mayonnaise type	2	tablespoons

Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	969.0 Kc	46	Niacin	6.547 mg	46
Carbohydrate	91.22 gm	--	Folacin	82.19 ug	20
Protein	44.96 gm	102	Phosphorus	575.7 mg	71
Fat	46.95 gm	--	Zinc	11.34 mg	75
Potassium	722.3 mg	19	Vitamin C	20.40 mg	34
Iron	5.887 mg	32	Riboflavin	0.641 mg	49
Vitamin A	2285 IU	57	Vitamin B12	2.630 ug	87
Thiamine	0.415 mg	37	Calcium	504.7 mg	63

Protein: 19% Carbohydrate: 38% Fat: 44% Alcohol: 0%

Table 23--Continued

Post-Phase Lowest Binge							
Food Name			Serving		Portion		
Soup-bean/bacon-can-water			1.25		cups		
Milk-2% fat-lowfat-fluid			10		fl ounces		
Post-Phase Lowest Binge Nutrient Values							
Nutrient		Value	%RDA	Nutrient		Value	%RDA
K Calories		367.5 Kc	18	Niacin		0.971 mg	7
Carbohydrate		43.13 gm	--	Folacin		54.88 ug	13
Protein		20.01 gm	45	Phosphorus		455.0 mg	56
Fat		13.27 gm	--	Zinc		2.475 mg	16
Potassium		975.0 mg	26	Vitamin C		4.900 mg	8
Iron		2.713 mg	15	Riboflavin		0.545 mg	45
Vitamin A		1736 IU	43	Vitamin B12		1.110 ug	37
Thiamine		0.230 mg	23	Calcium		472.5 mg	59

Protein: 22%		Carbohydrate: 46%		Fat: 32%		Alcohol: 0%	

Table 24. Foods consumed in pre- and post-phases, control group, subject 8.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Candy-peanut butter cup	1.6	ounces
Cola-type-soda	6	fl ounces
Pizza-pepperoni-baked	6	slices
Cookie-choc chip-home recipe	3	items
Milk-2% fat-lowfat-fluid	1.5	cups

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2482 Kc	118	Niacin	33.79 mg	241
Carbohydrate	300.5 gm	--	Folacin	488.7 ug	122
Protein	97.55 gm	221	Phosphorus	513.6 mg	64
Fat	98.34 gm	--	Zinc	2.275 mg	15
Potassium	2108 mg	56	Vitamin C	17.88 mg	29
Iron	16.78 mg	93	Riboflavin	2.458 mg	189
Vitamin A	3964 IU	99	Vitamin B12	3.522 ug	117
Thiamine	2.265 mg	205	Calcium	1676 mg	209

Protein: 16%	Carbohydrate: 49%	Fat: 36%	Alcohol: 0%
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Table 24--Continued

Pre-Phase Lowest Binge					
Food Name		Serving		Portion	
Cookie-choc chip-home recipe		6		items	
Beer-regular		10		fl ounces	
Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	402.8 Kc	20	Niacin	2.706 mg	20
Carbohydrate	50.16 gm	--	Folacin	22.10 ug	5
Protein	3.830 gm	8	Phosphorus	140.4 mg	17
Fat	16.08 gm	--	Zinc	0.414 mg	2
Potassium	198.0 mg	5	Vitamin C	0.000 mg	0
Iron	1.584 mg	8	Riboflavin	0.180 mg	15
Vitamin A	25.80 IU	0	Vitamin B12	0.060 ug	2
Thiamine	0.100 mg	10	Calcium	34.80 mg	4

Protein: 3%	Carbohydrate: 46%		Fat: 33%		Alcohol: 18%

Table 24--Continued

Post-Phase Highest Binge					
Food Name			Serving		Portion
Pizza-pepperoni-baked			6		slices
Cola-type-soda			32		fl ounces
Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2262 Kc	113	Niacin	30.90 mg	237
Carbohydrate	328.7 gm	--	Folacin	468.0 ug	117
Protein	78.00 gm	177	Phosphorus	165.4 mg	20
Fat	69.00 gm	--	Zinc	0.416 mg	2
Potassium	1315 mg	35	Vitamin C	14.40 mg	24
Iron	15.60 mg	86	Riboflavin	1.728 mg	144
Vitamin A	3192 IU	79	Vitamin B12	2.160 ug	72
Thiamine	1.944 mg	194	Calcium	1205 mg	150

Protein: 14%		Carbohydrate: 58%		Fat: 28%	
				Alcohol: 0%	

Table 24--Continued

Post-Phase Lowest Binge							
Food Name			Serving		Portion		
Apple-raw-peeled			1		item		
Cola-type-soda			12		fl ounces		
Salami-cooked-beef			2		slices		
Post-Phase Lowest Binge Nutrient Values							
Nutrient		Value	%RDA	Nutrient		Value	%RDA
K Calories		347.6 Kc	17	Niacin		1.686 mg	12
Carbohydrate		60.82 gm	--	Folacin		0.500 ug	0
Protein		6.950 gm	15	Phosphorus		117.0 mg	14
Fat		9.640 gm	--	Zinc		1.186 mg	7
Potassium		255.0 mg	6	Vitamin C		11.10 mg	18
Iron		1.190 mg	6	Riboflavin		0.131 mg	10
Vitamin A		56.00 IU	1	Vitamin B12		2.220 ug	74
Thiamine		0.080 mg	8	Calcium		20.00 mg	2

Protein: 8%		Carbohydrate: 68%		Fat: 24%		Alcohol: 0%	

Table 25. Foods consumed in pre- and post-phases, control group, subject 9.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Potato-baked-peel after	1	item
Cream-sour-cultured	1	ounce
Butter-regular-tablespoon	1	tablespoon
Steak-sirloin-lean	4	ounces
Cocktail-gin rickey	1	item
Cocktail-daiquiri	1	item
Bread-French-enriched	2	ounces
Butter-regular-tablespoon	.5	tablespoon
Lettuce-butterhead-head	.5	item
Tomato-raw-red-ripe	.5	item
Carrot-raw-whole-scraped	.5	item
Croutons-herb seasoned	.25	cup
Salad dress-thousand island	2	tablespoons

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1216 Kc	57	Niacin	11.26 mg	80
Carbohydrate	88.26 gm	--	Folacin	118.0 ug	29
Protein	45.32 gm	102	Phosphorus	511.1 mg	63
Fat	49.15 gm	--	Zinc	7.309 mg	48
Potassium	1662 mg	44	Vitamin C	53.99 mg	89
Iron	6.619 mg	36	Riboflavin	0.661 mg	50
Vitamin A	12667 IU	316	Vitamin B12	2.414 ug	80
Thiamine	0.714 mg	64	Calcium	170.1 mg	21

Protein: 15% Carbohydrate: 29% Fat: 36% Alcohol: 21%

Table 25--Continued

Pre-Phase Lowest Binge					
Food Name		Serving		Portion	
Bread-whole wheat-firm		1		slice	
Tab-low cal cola		12		fl ounces	
Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	61.30 Kc	2	Niacin	0.958 mg	6
Carbohydrate	11.30 gm	--	Folacin	13.80 ug	3
Protein	2.410 gm	5	Phosphorus	110.0 mg	13
Fat	1.090 gm	--	Zinc	0.420 mg	2
Potassium	44.00 mg	1	Vitamin C	0.000 mg	0
Iron	0.855 mg	4	Riboflavin	0.053 mg	4
Vitamin A	0.000 IU	0	Vitamin B12	0.000 ug	0
Thiamine	0.088 mg	8	Calcium	18.00 mg	2

Protein: 15%		Carbohydrate: 70%		Fat: 15%	
				Alcohol: 0%	

Table 25--Continued

Post-Phase Highest Binge		
Food Name	Serving	Portion
Corn chips	1	ounce
Bread-whole wheat-firm	2	slices
Cheese-cheddar-shredded	2	ounces
Mustard-yellow-prepared	2	tablespoons
Apple juice-canned/bottled	1	cup

Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	651.6 Kc	31	Niacin	2.762 mg	19
Carbohydrate	69.20 gm	--	Folacin	38.34 ug	9
Protein	20.77 gm	47	Phosphorus	462.5 mg	57
Fat	30.23 gm	--	Zinc	3.105 mg	20
Potassium	481.7 mg	12	Vitamin C	2.300 mg	3
Iron	3.616 mg	20	Riboflavin	0.387 mg	29
Vitamin A	602.6 IU	15	Vitamin B12	0.469 ug	15
Thiamine	0.291 mg	26	Calcium	484.9 mg	60

Protein: 13%	Carbohydrate: 44%	Fat: 43%	Alcohol: 0%		

Table 25--Continued

Post-Phase Lowest Binge		
Food Name	Serving	Portion
Bagel-egg	1	item
Tab-low cal cola	1.5	cups

Post-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	163.0 Kc	8	Niacin	1.940 mg	14
Carbohydrate	30.90 gm	--	Folacin	13.20 ug	3
Protein	6.020 gm	13	Phosphorus	81.90 mg	10
Fat	1.410 gm	--	Zinc	0.286 mg	1
Potassium	40.70 mg	1	Vitamin C	0.000 mg	0
Iron	1.460 mg	8	Riboflavin	0.160 mg	13
Vitamin A	17.60 IU	0	Vitamin B12	0.052 ug	1
Thiamine	0.209 mg	20	Calcium	23.10 mg	2

Protein: 15%	Carbohydrate: 77%	Fat: 8%	Alcohol: 0%		

Table 26. Foods consumed in pre- and post-phases, control group, subject 10.

Pre-Phase Highest Binge		
Food Name	Serving	Portion
Chicken-breast-no skin-fri	3	ounces
Lettuce-butterhead-head	.5	item
Salad dress-ranch style	2	tablespoons
Corn-cream-can-low sodium	.25	cup
Potato-mashed-milk/butter	.25	cup
Gravy-chicken-canned	.25	cup
Bread-white-firm	2	slices
Butter-regular-tablespoon	3	tablespoons
Milk-2% fat-lowfat-fluid	2	cups
Cola-type-soda	12	fl ounces
Cookie-choc chip-home recipe	2	items
Cheese-cheddar-shredded	1	ounce
Pie-apple-home recipe	1	slice
Corn chips	1.5	ounces
Candy-Snickers bar	1	item

Pre-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2283 Kc	108	Niacin	18.80 mg	134
Carbohydrate	234.2 gm	--	Folacin	151.6 ug	37
Protein	73.27 gm	166	Phosphorus	1083 mg	135
Fat	121.6 gm	--	Zinc	6.235 mg	41
Potassium	1752 mg	46	Vitamin C	19.32 mg	32
Iron	5.580 mg	30	Riboflavin	1.483 mg	114
Vitamin A	3800 IU	94	Vitamin B12	2.337 ug	77
Thiamine	0.854 mg	77	Calcium	966.2 mg	120

Protein: 13%	Carbohydrate: 40%		Fat: 47%	Alcohol: 0%	

Table 26--Continued

Pre-Phase Lowest Binge		
Food Name	Serving	Portion
Bread-white-firm	4	slices
Beef-lean/fat-simm/roast	8	ounces
Cheese-cheddar-shredded	3	ounces
Salad dress-mayonnaise type	2	tablespoons
Lettuce-butterhead-head	.25	item
Tomato-raw-red-ripe	.5	item
Popcorn-popped-oil & salt	4	cups

Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1535 Kc	73	Niacin	14.45 mg	103
Carbohydrate	76.76 gm	--	Folacin	98.70 ug	24
Protein	95.44 gm	216	Phosphorus	949.2 mg	118
Fat	92.56 gm	--	Zinc	29.27 mg	195
Potassium	923.2 mg	24	Vitamin C	15.15 mg	25
Iron	12.18 mg	67	Riboflavin	1.182 mg	90
Vitamin A	2205 IU	55	Vitamin B12	6.467 ug	215
Thiamine	0.627 mg	57	Calcium	783.1 mg	97

Protein: 25%	Carbohydrate: 20%	Fat: 55%	Alcohol: 0%
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Table 26--Continued

Post-Phase Highest Binge					
Food Name		Serving	Portion		
Cereal-wheat flakes-sugar		6	cups		
Cake-cheesecake-commercial		1	slice		
Crackers-wheat thins		.5	pound		
Cheese-cheddar-shredded		3	ounces		
Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	2363 Kc	118	Niacin	32.26 mg	248
Carbohydrate	326.9 gm	--	Folacin	85.11 ug	21
Protein	59.51 gm	135	Phosphorus	1009 mg	126
Fat	88.62 gm	--	Zinc	7.013 mg	46
Potassium	652.8 mg	17	Vitamin C	100.3 mg	167
Iron	29.79 mg	165	Riboflavin	3.130 mg	260
Vitamin A	9037 IU	225	Vitamin B12	10.66 ug	355
Thiamine	2.449 mg	244	Calcium	733.0 mg	91

Protein: 10%	Carbohydrate: 56%	Fat: 34%	Alcohol: 0%		

Table 26--Continued

Post-Phase Lowest Binge		
Food Name	Serving	Portion
Taco Bell-beefy tostado	2	items
Milk-chocolate-2% lowfat	1	cup
Potato-French fried-frozen	4	ounces
Tomato catsup	4	tablespoons

Post-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	1073 Kc	51	Niacin	10.32 mg	73
Carbohydrate	122.6 gm	--	Folacin	33.82 ug	8
Protein	49.94 gm	113	Phosphorus	913.5 mg	114
Fat	44.93 gm	--	Zinc	1.632 mg	10
Potassium	1711 mg	45	Vitamin C	48.17 mg	80
Iron	9.320 mg	51	Riboflavin	1.035 mg	79
Vitamin A	8240 IU	206	Vitamin B12	0.847 ug	28
Thiamine	0.588 mg	53	Calcium	721.1 mg	90

Protein: 18%	Carbohydrate: 45%	Fat: 37%	Alcohol: 0%		

Table 27. Foods consumed in pre- and post-phases, control group, subject 11.

Pre-Phase Highest Binge							
Food Name			Serving		Portion		
French toast-home recipe			5		slices		
Orange juice-frozen-diluted			1		cup		
Milk-chocolate-2% lowfat			1		cup		
Syrup-pancake-karo			.5		cup		
Pre-Phase Highest Binge Nutrient Values							
Nutrient		Value	%RDA	Nutrient		Value	%RDA
K Calories		1536 Kc	76	Niacin		5.868 mg	45
Carbohydrate		258.0 gm	--	Folacin		209.0 ug	52
Protein		38.05 gm	86	Phosphorus		740.5 mg	92
Fat		38.79 gm	--	Zinc		3.915 mg	26
Potassium		1333 mg	35	Vitamin C		99.20 mg	165
Iron		13.94 mg	77	Riboflavin		1.270 mg	105
Vitamin A		1249 IU	31	Vitamin B12		2.302 ug	76
Thiamine		0.909 mg	90	Calcium		739.0 mg	92

Protein: 10%		Carbohydrate: 67%		Fat: 23%		Alcohol: 0%	

Table 27--Continued

Pre-Phase Lowest Binge		
Food Name	Serving	Portion
Fish-shrimp-French fried	4	ounces
Potato-French fried-frozen	1	item
Lettuce-iceberg-raw-chop	2	cups
Tomato-raw-red-ripe	.5	item
Carrot-raw-whole-scraped	.5	item
Salad dress-blue cheese	2	tablespoons
Roll-hard-enriched	1	item
Butter-regular-tablespoon	.5	tablespoon

Pre-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	667.1 Kc	33	Niacin	5.836 mg	44
Carbohydrate	54.81 gm	--	Folacin	116.0 ug	29
Protein	31.43 gm	71	Phosphorus	344.4 mg	43
Fat	36.58 gm	--	Zinc	0.711 mg	4
Potassium	776.2 mg	20	Vitamin C	20.70 mg	34
Iron	4.604 mg	25	Riboflavin	0.346 mg	28
Vitamin A	11532 IU	288	Vitamin B12	0.000 ug	0
Thiamine	0.372 mg	37	Calcium	167.4 mg	20

Protein: 19% Carbohydrate: 33% Fat: 49% Alcohol: 0%

Table 27--Continued

Post-Phase Highest Binge		
Food Name	Serving	Portion
Waffles-enr-home recipe	16	items
Bananas-raw-peeled	1	item
Syrup-pancake-Karo	1	cup
Cereal-wheat chex	2.5	cups
Milk-2% fat-lowfat-fluid	1	cup
Grapefruit-can/juice	24	fl ounces

Post-Phase Highest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	5812 Kc	290	Niacin	44.46 mg	342
Carbohydrate	852.4 gm	--	Folacin	668.5 ug	167
Protein	136.7 gm	310	Phosphorus	3009 mg	376
Fat	210.3 gm	--	Zinc	15.23 mg	101
Potassium	4623 mg	123	Vitamin C	326.0 mg	543
Iron	56.78 mg	315	Riboflavin	4.922 mg	410
Vitamin A	2836 IU	70	Vitamin B12	12.73 ug	424
Thiamine	4.745 mg	474	Calcium	3071 mg	383

Protein: 9%	Carbohydrate: 58%		Fat: 32%	Alcohol: 0%	

Table 27--Continued

Post-Phase Lowest Binge		
Food Name	Serving	Portion
Meatloaf-frozen dinner-Banquet	3	ounces
Potato-au gratin-home recipe	4	ounces
Lettuce-butterhead-head	.5	item
Tomato-raw-red-ripe	.5	item
Carrot-raw-whole-scraped	.5	item
Salad dress-ranch style	2	tablespoons
Bread-whole wheat-firm	2	slices
Butter-regular-tablespoon	1	tablespoon

Post-Phase Lowest Binge Nutrient Values					
Nutrient	Value	%RDA	Nutrient	Value	%RDA
K Calories	631.3 Kc	30	Niacin	5.175 mg	36
Carbohydrate	52.95 gm	--	Folacin	108.1 ug	27
Protein	19.19 gm	43	Phosphorus	380.7 mg	47
Fat	40.44 gm	--	Zinc	1.914 mg	12
Potassium	1132 mg	30	Vitamin C	35.18 mg	58
Iron	4.375 mg	24	Riboflavin	0.406 mg	31
Vitamin A	12989 IU	324	Vitamin B12	0.228 ug	7
Thiamine	0.418 mg	37	Calcium	240.1 mg	30

Protein: 12% Carbohydrate: 32% Fat: 56% Alcohol: 0%

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