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The Effects of a Very-Low-Calorie-Diet on Resting Energy Expenditure, Body Composition, and Biochemical Data in Obese Outpatients

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THE EFFECTS OF A VERY-LOW-CALORIE-DIET ON RESTING ENERGY EXPENDITURE, BODY COMPOSITION, AND BIOCHEMICAL DATA IN OBESE OUTPATIENTS

by

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ABSTRACT

The Effects of a Very-Low-Calorie-Diet on Resting Energy Expenditure, Body Composition, and Biochemical Data in Obese Outpatients

by

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Obesity is a disease of major proportion in the United States. The Surgeon General has identified obesity as a national health problem that affects approximately 34 million Americans.

The aim of this study was to investigate the very-low-calorie diet, Optifast 70. Measurements for resting energy expenditure (REE) and body composition via circumference measurements (CBF) and infrared photospectromerty (NIR) with a Futrex 5000 were collected at weeks 1, 7, 13, 19, and 25. Biochemical data, including serum chemistry panel (SMA-12) and complete blood count (CBC), were collected on weeks 1, 5, 9, 13, 17, 21, and 25. Lipid profiles were drawn on weeks 1 and 25.

Participants ranged in age from 27 to 64. Subjects' mean body mass loss was -20.4 kg ± 6.6 kg with a maximum body mass loss of -33.23 kg and minimum body mass loss of -9.770 kg. Mean loss in body fat mass using infrared photospectrometry as a measurement was -13.4 kg; mean loss of lean body mass was -4.2 kg. A significant change was noted in resting energy expenditure over the course of the diet, and a positive correlation was identified between loss of body mass and resting energy expenditure. No significant correlation was identified between the loss of lean body mass or body fat mass and its relationship to resting energy expenditure. Both circumference and infrared body fat measurements showed a positive correlation as the loss in body mass increased, making their reliability better as subjects approached desirable weight. In examining biochemical data, only cholesterol showed a significant change over the course of the diet; all other parameters remained within normal limits. Variations in patients' lipid profiles were identified, but no significant changes were noted.

(101 pages)

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Charlene A. Perkins

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INTRODUCTION

Background of the Problem

Obesity is a disease of major proportion in the United States. The U.S. Surgeon General has identified obesity as a national health problem, which affects millions of Americans, with an increase of as much as 30% in the last decade. Obesity is defined as greater than 120% of ideal body weight (1). Obesity is a multifactorial disease of which management is regularly unsatisfactory and frustrating, for both the clinician and patient. A body weight of greater than 120% of ideal has been associated with increased morbidity and mortality. Excess body weight has been recognized as a contributing factor to a wide range of chronic diseases such as diabetes, hypertension, coronary artery disease, and many orthopedic disabilities (2). A 1992 National Institutes of Health (NIH) report reinforced the concept of improved outcomes for many of the chronic diseases associated with weight loss. Obesity, when left untreated, may have serious consequences. Untreated, the average obese woman may experience a 2% percent weight gain each year, which may gradually increase an already compromised health risk. Being significantly overweight is recognized as a chronic medical problem that may require long-term and even life-long treatment. The causes of obesity are many and complex and can include genetic, physiological, and biochemical factors that work together to form a complex disorder of energy metabolism. Today obesity still has no cure. Obesity is in itself a chronic disease

as stated by Stunkard (3). The misunderstanding of the nature of obesity has contributed to a stigmatization of obese persons and to inappropriate treatment programs that lack a multidisciplinary approach to weight loss. Various regimes have come about to treat the obese individual. These include behavioral therapy, exercise, drug therapy, surgery, and very-low-calorie diets. Each affects the treatment of obesity at differing levels (3). The very-low-calorie diet (VLCD) is one of these regimens. It has been administered throughout the United States and Europe and is essentially a modification of the fasting approach to weight loss. Safe and effective long-term weight loss programs are urgently needed in light of new data which conclude the prevalence of obesity is increasing in the United States, as much as 30% in the last decade (3).

Abbreviations for specific words used throughout the text are listed in Appendix F.

Purpose of the Study

The purpose of this study was to evaluate the effects of a very-low-calorie diet program, specifically the Optifast-70 diet, on resting energy expenditure (REE), lean body mass (LBM), and fat body mass (FBM). Biochemical data including complete blood count (CBC), serum chemistry panel (SMA-12), and lipid profiles, on 17 outpatients.

Objectives

1. Examine the effects the Optifast-70 diet program has on weight loss and maintenance of weight loss through fasting, realimentation, and 6 weeks of maintenance.

2. Examine the influence of the Optifast-70 diet program on resting energy expenditure during a period of fasting, realimentation, and maintenance. Determine if there is a significant correlation between resting energy expenditure and weight loss in association with any change in lean body mass and fat body mass before, during, and after a VLCD.

3. Examine how the variation in intake and weight loss influences any change in respiratory quotients.

4. Examine body composition (% body fat) via circumference measurements (upper arm, forearm, abdomen, hip, upper thigh, and calf). Determine the relationship of change in circumference and loss in body mass during the diet.

5. Examine body composition, percent body fat, lean body mass, and fat body mass by near-infrared photospectrometry (NIR) during the course of the Optifast-70 diet. Determine the relationship of variation in body composition and loss in body mass. Determine if near-infrared photospectrometry is an accurate and reliable technique of assessing body composition in the obese population. Determine any correlation between this technique and the circumference technique in determining body composition.

6. Examine via descriptive analysis the effects of the Optifast-70 diet program on complete blood count (CBC) and serum chemistry panel (SMA-12) during fasting, realimentation, and maintenance of the Optifast-70 diet.

7. Examine by descriptive analysis the effects the Optifast-70 diet has on lipid profiles, triglycerides, total cholesterol, high-density lipoproteins (HDL), low-density lipoproteins (LDL), and very-low-density lipoproteins (VLDL) before the fast and after 6 weeks maintenance. Does the amount of body mass lost correlate to a change in lipid profiles, and does the ratio of HDL/LDL correlate with the amount of body mass lost?

Limitations

A limitation to this study was the small sample size of participants who completed the study (N=17). The second limitation was ending data collection at week 25. Continuing data collection after week 25 would have offered more insight into weight maintenance, fluctuation in resting energy expenditure, and other examined parameters.

LITERATURE REVIEW

Very-Low-Calorie Diets

The very-low-calorie diet (VLCD) has become a treatment for obesity. It is defined as any diet providing less than 800 calories per day (4). The history of the VLCD dates back to the early 1930's when patients were treated for 8 weeks with a 300-400 calorie diet with patients losing an average of 9.9 kilograms (5). Despite Frankle and Yang's great success, much of their work was forgotten until the 1950's. In 1954 the Simeons diet was developed and used extensively by the medical profession. It consisted of approximately 575 kcal and was used for 6 weeks with injections of human chorionic gonadotropin (6). Weight loss averaged between 9-14 Kg. After Simeons the next stage of treatment for obesity was the starvation diet. Bloom and his colleagues also promoted the starvation diet (7). The two major problems that lead to the abandonment of the starvation diet were physical danger to the patients, due to a loss of vital body protein stores, and poor maintenance of weight loss. By the mid-1970's, at least five deaths had been directly related to starvation. Two patients died of ventricular fibrillation. One patient with a history of hypertension and diabetes died of lactic acidosis. The last death resulted from a small bowel infarction (4). Recognizing that this loss of vital protein stores was undesirable, investigators in the late 1960's began using supplements of egg albumin as a protein source. A few years after this, Blackburn invented the term protein-sparing-modified-fast (PSMF). This terminology was later changed to

the (VLCD) (7).

Currently there are two types of VLCD's. The protein sparing modified fast (PSMF) was developed by Blackburn and colleagues (8). This diet provides 1.5 grams of protein per kilogram of ideal body weight. The protein is obtained from lean meat, fish, and fowl, consumed in three to four meals per day. No carbohydrate is permitted, and fat is limited to that present in protein foods. The other type of diet uses milk or egg based powdered protein formula consumed as a liquid three to four times per day. These commercially prepared diet products provide 30-70 grams of protein, 30-45 grams of carbohydrate, approximately 2 grams of fat, and recommended levels of all vitamins and minerals (9). The relative merits of the two programs (VLCD, PSMF) have been debated. Proponents of the PSMF argue their diet teaches patients to handle conventional foods. This better eases the transition from the diet to controlled eating. Advocates of the liquid formulas contend their diet facilitates adherence by removing the temptation of conventional food. It has been concluded that patients vary considerably in their response to these two diets, so neither can be recommended categorically.

The primary goal of a VLCD today is to lose body fat without jeopardizing the loss of lean tissue. During the early weeks of rigid dieting, approximately 3-6 grams of nitrogen are lost daily before establishing equilibrium. The loss of nitrogen during this period typically amounts to less than 4% of the total body nitrogen. Early researchers felt if this amount of nitrogen were replaced as protein, nitrogen

balance would be maintained. However, many of these studies were done immediately after a fast, and this increased the efficiency of protein use and the intensity at which nitrogen balance was retained (4). Blackburn et al. (8), and Bistrian et al. (10) recommended the use of 75 grams of protein for men and 55 grams for women. These levels produced nitrogen balance in moderately obese adults within 2 to 3 weeks. Therefore, they perceived the optimal composition of the VLCD is to provide 1.5 grams protein per kilogram ideal body weight (IBW) per day. The protein must be of high biological value, and the diet should be supplemented with the recommended dietary allowances for all vitamins and minerals, especially potassium, magnesium, phosphate, sodium, and calcium. In studies conducted by Frankle and Yang (5), nitrogen balance was maintained on as little as 31 grams and as much as 132 grams of protein daily within a period of 14 to 21 days. Therefore, the actual amount of dietary protein required to achieve nitrogen balance during caloric restriction remains controversial, and these discrepancies illustrate the vast individual differences that may be observed in response to VLCDs. The existence of these differences underscores the need for careful medical supervision.

The amount of carbohydrate used in the VLCD has caused much contention. Several researchers (6,11) favored the use of carbohydrate (30-45 grams/day). Because of its protein-sparing effect, it prevents hyperuricemia and electrolyte loss due to diuresis, and it also decreases occurrence of orthostatic hypotension and

hunger. Researchers on the opposite end of the controversy (12,13,14) have proposed a notion of protein-sparing and fat mobilization that excludes dietary carbohydrate to maintain a high degree of ketosis and low levels of plasma insulin. These researchers believe ketones will replace glucose used by the brain and thus reduce gluconeogenesis. The low levels of plasma insulin will also increase the release of free fatty acids from adipose tissue and enhance fat mobilization. Furthermore, Blackburn et al. (13,14) contended that the isocaloric substitution of carbohydrate for protein diminished the effectiveness of protein sparing therapy. Yang et al. (15) took another point of view concluding no significant difference in nitrogen loss after either diets of 132 grams protein per day or 66 grams of protein, and 59 grams of carbohydrate per day. Similarly, DeHaven et al. (16) showed no greater losses of nitrogen on a 3-week diet of 50 grams protein and 50 grams of carbohydrate than on a 100-gram protein diet. A carbohydrate intake of 30-40 grams per day can decrease amino acid requirements by half. But the provision of 80 grams or more of the carbohydrate per day can drastically reduce the amount of weight loss. The carbohydrate controversy continues.

Vitamin and mineral supplementation with the use of a VLCD is essential if the diet is not already supplemented to the Recommended Dietary Allowances (RDA) for essential vitamin and minerals. Supplements need to include a multivitamin, 3-5 grams sodium chloride to replace the sodium loss during diuresis and 3 grams (40 milliequivalents) of potassium to decrease ammonia genesis and

diminish nitrogen excretion by approximately 2 grams per 24 hours. Calcium supplementation is frequently prescribed at 400-800 milligrams per day. Fluids also play a major role, and it is recommended that patients drink a minimum of 1.5 to 2 liters of fluid per day (17).

Examples of VLCDs currently in use include Optifast-70, currently known as Promed, Medifast, Health Management Resources, Medibase, and New Directions by Ross, along with several other over-the-counter liquid formulas such as Slimfast, Sweet Success, and others (9). In selecting patients for the VLCD, most investigators limit the diet to adults between the ages of 18-70, who are moderately obese (>115 - 120% of ideal body weight [IBW]) or the morbidly obese (>200% of IBW). It appears, however, that patients over the age of 30 respond best to treatment. Patients are given a complete physical and psychological exam, including electrocardiogram, complete blood count, SMA-12, and complete urinalysis along with thyroid and lipid profiles. Contraindications for treatment include a recent myocardial infarction, cerebrovascular accident, active cancer, type I diabetes, hepatic disease, renal failure, depression, a deep vein thrombosis, and overt psychosis. Lithium therapy is now an exception, whereas it used to be a contraindication (4). Currently much emphasis is placed on cognitive and social support.

During VLCDs, patients will sometimes be started on a balanced 1200 calorie diet before beginning consumption of the formula. This may take place 2-4

weeks before beginning the diet (18). Once patients begin consumption of the formula, they consume their daily intake in five or greater feedings at regular intervals. Weight is dramatically reduced during the introduction of the liquid fast, averaging 2 to 5 kilograms in the first week. This initial weight loss is primarily due to diuresis, and afterward weight loss diminishes to average 1-2 kilograms per week for women, and 1.5-2.5 kilograms per week for men (4).

It is vital that patients be seen weekly by a physician for a clinical exam including weight and blood pressure. Ketones, urinalysis, complete blood count, and SMA-12 are drawn every 4 weeks.

Side effects of the VLCD may include postural hypotension, constipation, and cold intolerance. Other side effects such as dry skin, brittle nails, hair loss, and dizziness occur in less than 10% of all patients (19). One percent of patients complain of gall bladder symptoms. A study done by Kamrath et al. (20) concluded rapid weight loss associated with the use of a VLCD was associated with a significant incidence of gallstone formation. It is believed the weight losses experienced by VLCD reduce fatty changes in the liver, and may induce slight portal inflammation and fibrosis that may increase the incidence of gallstone formation (21). Another theory is that obese persons are at risk for cholesterol gallstones because their bile is saturated with cholesterol. Risk of gallstones is increased during the course of a VLCD because of increased bile cholesterol saturation index and gallbladder stasis. Gebbard et al. (22) found that gallstone

risk during a VLCD may be reduced by maintenance of gallbladder emptying with a small amount of dietary fat (10 grams per meal). Long-term complications may include the risk of ventricular arrhythmias. At times, large increases in a food intake or binging following severe caloric restriction can cause changes in metabolic rate and electrolyte imbalances that could result in cardiac arrhythmias. Seim et al. (23) concluded that a VLCD (800 kcal/d) for up to 3 months is not associated with significant electrocardiographic abnormalities or clinical cardiac complications, provided the patients have low cardiovascular risk at baseline of the diet. Inappropriate feeding during a course of time while following a severely restricted diet can also be associated with attacks of pancreatitis and cholecystitis (24). After the fasting phase of the diet that usually never lasts more than 3 months, the refeeding or realimentation phase begins. This process takes place over 4-6 weeks and gradually reintroduces solid food (milk, vegetables, fruits, lean meat, fish, and fowl). Carbohydrate is reintroduced gradually to prevent an abrupt increase in fluid (25). This stage of the diet is ideal for the introduction of behavioral modification and controlled eating patterns because problem foods can be reintroduced cautiously (17). Followup care is essential for maintenance of weight loss. It includes further nutrition education, behavior modification, and exercise, with special emphasis on cognitive and social support. The VLCD appears safe and effective if four specific conditions are followed.

1. The diet contains protein of high biological value.

2. Duration of the diet is brief, 3 months or less.

3. Use of the diet is restricted to patients 30% above ideal body weight, with no contraindicated physical conditions, $\geq$ 50 pounds overweight.

4. Patients receive frequent medical supervision.

The short-term effectiveness of the VLCD is dramatic, impressive, and directly associated with the duration of treatment. The mean rate of weight loss is 1.5 to 2.5 kg per week for men and 1.0 to 2.0 kg per week for women (5). Weight loss associated with the VLCD is superior to other nonsurgical treatments. However, the long-term maintenance of weight loss after this type of dietary regimen is still a criticism. The American Dietetic Association (26) believes a major limitation of the VLCD is the poor long-term weight maintenance. They concluded with the current evidence that showed a higher percentage of dieters who complete a VLCD regain over half the weight they have lost. However, the inclusion of nutrition counseling, exercise, relaxation, and behavior modification offers hope to weight loss maintenance. Genuth et al. (27) showed with their research that at 22 months, 56% of their subjects had regained more than half their weight. Hovell et al. (28) reported that maintenance at 1-2 years was apparent in only a small portion of patients completing treatment. They completed analysis of an 18-30 month weight loss maintenance program following treatment with both behavior modification and supplemented fasting procedures for 400 patients. Patients' who completed treatment lost a mean of 83.9% of their excess weight, but regained

between 59-82% of their initial weight by 30 months. Linder and Blackburn (29) reported the best results; they found that at 18-24 months follow-up, the original weight loss of 20.8 kilograms had decreased to only 14.5 kilograms. Kirshner et al. (30) examined data from 4,026 patients entering an Optifast program and found good results. Their results showed that at 18 months 58% of the men and 35% of the women who completed the treatment maintained their weight loss within 10 pounds.

Despite the diet regimen the American Medical Association Councils on Scientific Affairs have indicated a similar conclusion concerning very-low-calorie diets, that weight loss can be maintained following a controlled program of very-low caloric intake. In harmony with this belief, Wadden et al. (24) concluded that a comprehensive, long-term weight-control program is the only effective treatment for obesity.

The Optifast Program

The Optifast program came about during the late 1960's and early 1970's when Blackburn and coworkers developed the protein-sparing-modified fast. During this time Genuth and coworkers at the Mt. Sinai Medical Center were investigating the use of a protien sparing modified fast (PSMF) formula to reduce weight in obese patients (1). Genuth et al. (27) prescribed a combination of 45 grams of calcium caseinate, 30 grams of glucose, a multivitamin tablet, and folic acid supplement to a group of 75 outpatients. This regimen was continued until a

reasonable weight loss goal was achieved. Success was defined as an average weight loss of 2 pounds per week with 60% (N=45) of the patients achieving successful weight loss. No severe clinical derangements occurred during the regime. Beneficial results included lowered blood pressure, improved glucose tolerance, and improvement in cardiac and pulmonary disabilities (31).

From the research done by Genuth et al. (27) and the specific interests of Vertes, a hypertension specialist at Mt. Sinai Medical Center and member of this group, the Optifast program was developed. Since Genuth's original formula was unpalatable, Vertes and Genuth turned to the Delmark Company (formerly Sandoz Nutrition and now Novartis Nutrition Corporation) to manufacture a more palatable Optifast formula (18). Today there is only one Optifast formula in use, Optifast-800.

The Optifast-70 formula was phased out of the program, and the only formula available is the Optifast-800 formula. Novartis Nutrition Corporation rationale for one formula was:

1. The difference in weight loss between the two formulas is negligible.
2. The 800-calorie products are richer in taste.
3. The government is imposing stricter regulations on weight management and the company wishes to be proactive.

The original Optifast-70, used in this study, contained 70 grams of protein, 30 grams of carbohydrate, and 2 grams of fat and provided 420 calories per day. Past nitrogen balance studies have concluded this formula adequately spared body

protein while inducing rapid weight loss. Vitamins and minerals are supplemented adequately to meet the recommended dietary allowance, and the 2 grams of fat maintained appropriate levels of serum fatty acids. The 30 grams of carbohydrate the formula contained helped conserve lean body mass and maintained electrolyte balance (1).

The Optifast-800 formula currently provides 800 calories daily, 70 grams protein, 100 grams of carbohydrate, and 13 grams of fat along with vitamins and minerals to meet the recommended dietary allowance. At the time of this study the energy content of this formula was for individuals who had less than 50 pounds to lose or who for medical reasons did not qualify for the Optifast-70 formula (1). However, as stated earlier, the program has now changed, and studies have shown no significant difference in weight loss between the two formulas.

The Optifast program was developed for the treatment of individuals with medically significant obesity who by means of weight loss may experience improvement in obesity-related health problems (1). The fasting regimen consists of a 12-week period during which patients ingest only the Optifast formula and noncaloric beverages. The realimentation phase follows the fasting phase. During this time solid food is gradually reintroduced, and patients slowly progress to a solid diet. Rigid guidelines have been developed for acceptance into the Optifast program. To qualify, persons must be at least 130% of ideal body weight or 50 pounds overweight. Patients must have no history of a myocardial infarction,

cerebrovascular accident, diabetes with ketoacidosis, chronic steroid use, bleeding ulcers, active thrombophlebitis, or psychiatric problems. After the patients have undergone a complete physical exam, ECG, and thorough laboratory tests, they are admitted to the program (1).

Patients are under constant medical supervision during the entire diet, and the physician treats any medical problems that occur while the dietitian and behavioralist monitor diet compliance.

The Optifast program consists of several components that provide the education and support patients require for successful weight loss and maintenance with special emphasis on expanded behavioral modification and more focus on cognitive issues. The multidisciplinary approach taken provides patient education related to nutrition, behavior modification, and exercise. This teaches patients how to examine previous lifestyle behaviors, and to detect the problem or problems that may have contributed to their obesity.

Optifast centers in the U.S. are established based on regional population, usually to support at least 150 patients. In 1987, there were approximately 400 hospital-affiliated, clinic-based programs established including approximately 15 university hospital programs. More than 200,000 obese individuals have received medically supervised treatment through Optifast with 80% of those individuals losing 40 pounds or more (1). In a study conducted by Kanders et al. (32) they examined weight loss and health benefits associated with the Optifast program in

the treatment of obesity. Their conclusions support the notion that a 10-20% weight loss may be the appropriate treatment goal for a single weight loss intervention as it may improve obesity-related problems.

Participants in the Optifast programs consisted of approximately 70% women who range in age from 25 to 50 years of age (1). As stated earlier, participants over the age of 30 seem to have the best results. In 1992, Sandoz Nutrition, now Novartis Nutrition, changed the program title to Promed secondary to the bad publicity involved with the Optifast name.

Resting Energy Expenditure

During the last decade there have been many studies conducted examining the effects of dieting on resting energy expenditure (REE). People on restricted diets often show a considerable decline in REE (33, 34, 36). It was stated that if energy expenditure were reduced, it could become more difficult for the reduced obese individual to achieve further weight loss, or to maintain a reduced weight. There is much controversy about whether any body parameter can effectively explain REE (34). Bray et al. (31) concluded that oxygen consumption of obese patients is better correlated with fat tissue than with lean tissue. This means a reduction in fat must inevitably cause a reduction in energy expenditure and so make further weight loss difficult to achieve or sustain. If, however, lean tissue determines energy expenditure, the current diets that seek to spare lean tissue and preserve energy expenditures appear appropriate. Barrows and Snook (34)

investigated the long-term effects of a VLCD, specifically Optifast-70, on resting energy expenditure. They conducted three resting metabolic readings, before the fast, after the fast, and after 5 weeks of realimentation. They concluded that a significant drop in REE occurred during the diet, and REE remained lower following refeeding. They did not examine REE during the fast to see when the initial decline began nor did they examine REE during the maintenance period to see if REE returned to prediet levels.

In a study conducted by Coxon et al. (37), a comparison was made between fat free mass (FFM) and the change in REE between two groups of dieting female subjects. They found the greater the weight loss the greater the decrease in REE. The ratio of REE/FFM remained the same, and no evidence suggested the rate of weight loss was associated with any detriment to body composition or metabolic rates. Wadden et al. (4) conducted a study on the long-term changes in metabolic rates associated with consumption of a VLCD. He examined the short-term and long-term changes in resting energy expenditure associated with the consumption of a VLCD, Optifast -70, and a 1000-1200 kcal balanced deficit diet. He concluded a reduction in REE associated with the consumption of a VLCD could be limited to the actual period that the diet was consumed. The REE increased rapidly, to approximately 98% of the baseline, when the patients stopped consuming Optifast-70 and returned to a 1000-1200 kcal balanced diet.

However, other studies examining long-term energy intakes on resting

metabolisms have been conducted using other VLCD plans. Welle et al. (38) concluded that a decrease in REE of 9.4% was shown in their investigations after 5 weeks of a diet (472 kcal/d). The lowest values of REE they measured, however, were similar to REE values of lean to moderately obese women. Nevertheless, these values were not significantly lower than predicted values. Rattan et al. (39) examined REE at four intervals: 1) before the diet, 2) after 2 weeks of a VLCD, 3) at the end of 8 weeks of a VLCD, and 4) at the end of 8 weeks of maintenance. They observed that the metabolic rate decreased to 86% of the original value by the end of the 8 weeks of the diet. Resting energy expenditure then recovered to 93% of predicted values by the end of 8 weeks of maintenance on a 1500 kcal diet. No losses in REE were noted beyond that expected from the loss of fat mass itself. Elliot et al. (40) assessed the potential long-term effects of weight loss on REE and body composition in obese women. Resting energy expenditure was measured via indirect calorimetry before weight loss, during the protein sparing modified fast, and for 2 months while at a stable reduced weight. Resting energy expenditure decreased significantly with the initiation of the fast. No significance was noted between resting energy expenditure levels during the fast and at a stable reduced weight, but values were significantly less than predicted. The decrease in REE was not contributed to the decrease in lean body mass but paralleled the loss of fat body mass. Franssila-Kallanki et al. (41) reached a similar conclusion in their study that a decrease in basal energy expenditure by 10.7% with weight loss was associated

with a loss in fat mass rather than with loss of lean body mass.

Valtuena et al. (42) examined the respiratory quotient as a prognostic factor in weight-loss rebound. They concluded after a 28-day rapid weight loss diet that if the patients' respiratory quotients were in the lower range (<0.72), their more apt to maintain their weight loss. Patients in the higher respiratory quotient range (>0.75) were less able to maintain weight loss in the follow-up period. They concluded an appropriately measured respiratory quotient may be a useful indicator of the effectiveness of VLCD used to induce rapid weight loss (42).

There are various key factors in weight loss and gain that include age, fat mass, lean body mass, sex, and thyroid hormones. Therefore, the persistence of altered metabolism, which occurs with a VLCD, and the influence of refeeding and long-term, low energy intakes continue to be extensively studied. There continues to be a need to conduct further in-depth examinations of the effects of dieting on resting energy expenditure.

Anthropometrics

With the increasing use of the VLCD, there is a need to examine further the variations in body composition and pattern of weight loss that often accompany such radical treatments for obesity (43). Franssila-Kallanki et al. (41) identified in their study a decrease in body weight from 105.3 $\pm$ 4.6 Kg to 94.1 $\pm$ 4.0 Kg. This weight reduction was determined to be primarily a reduction in fat mass from 47.2 3.6 Kg to 37.7 $\pm$ 3.0 Kg. Lean body mass decreased only slightly from 58.0 $\pm$ 2.0

to 56.4 ± 1.8 Kg. Researchers have suggested that anthropometric measures may be inadequate for the clinical assessment of body composition in obese patients (43). Barrows and Snook (35) examined the effects of high protein, VLCD on anthropometric parameters of obese women. They used the Optifast-70 diet specifically, and tested the validity of nine previously published equations for predicting the body fat of obese women before and after weight loss. In addition, various anthropometric parameters (circumference and skinfolds) were used to develop multiple regression equations that would better predict the body composition of patients before and after weight loss. They concluded that body weight and circumference measurements were the variables most strongly associated with body composition before weight loss. After weight loss, only those circumference measures in the body trunk region (abdomen, maximum abdomen, and buttocks) remained significantly correlated with body composition. Katch and Katch (44) also contended these circumferences should be the preferred method to predict percent body fat when a one-time, accurate assessment is needed. Equations are available for both men and women.

Near-infrared photospectrometry (NIR), based on the principle of light interaction with organic material, has been used for assessing body composition (% lean body mass, % fat mass, and % body water). Kamrath et al. (20) used infrared photospectrometry to assess body composition in 11 moderately obese patients treated for 8 weeks with a diet providing 605 calories. They found no excess loss

of fat free mass. Davis et al. (45) evaluated the infrared technique in 85 subjects, differing in age, gender, skin color, and body composition. They were evaluated with the following results: a test-retest reliability of 94% based on intraclass correlation. The mean of the measures yielded a reliability coefficient of 98%. This preliminary analysis suggests that body composition can be assessed with excellent reliability and good validity (46). Dotson (47) estimated the body composition in children by near-infrared photo spectrometry. A heterogeneous group of students (320 males and 272 females) from 5 to 13 years old was randomly sampled. Data were collected for age, height, waist and neck circumferences, weight, skinfold measurements, and NIR. Coxon et al. (37) used both bioelectrical impedance and infrared (Futrex-5000) to study body composition in 26 patients on a VLCD. They found both techniques correlated very well ($r = 0.95$-0.99) on weekly analysis. These methods were validated with a subset of 16 patients who were measured with hydrodensitometry and by the skinfold technique. The correlation among all techniques was quite satisfactory. Brodie et al. (48) examined body fat estimation methods. They compared hydrodensitometry, bioelectrical impedance, BC-3000 analyzer, infrared (Futrex-5000), and skinfold measurements. There was a nonsignificant difference between the means. The infrared technique had a standard of error of the mean approximately half the values of the others. Infrared was also found to have a low operator error, was quick and reliable, but expensive. Conway et al (49) estimated body composition in 53 adults by infrared and

compared results from deuterium oxide dilution (r = 0.94), skinfold (r = 0.90), and ultrasound (r = 0.89) measurements. They concluded infrared was a safe, noninvasive, rapid, easy to use method, and could prove successful in predicting percent body fat, specifically in the obese. This is a relatively new technique and research is limited, especially in the obese population. Through infrared photospectrometry and body circumference measurements the effects of Optifast-70 diet on body composition will be examined to see if a correlation exists between the two techniques. A determination of the accuracy of infrared photospectrometry in measuring body composition of obese women will also be examined.

Biochemical

It is crucial that the VLCD be supplemented with vitamins and minerals to meet the Recommended Dietary Allowances. A diminished dietary intake can induce major changes in electrolyte balance. Weight loss on a VLCD is initially greater than can be accounted for from endogenous and exogenous protein and fat metabolisms secondary to diuresis (50). Sodium loss exceeds that produced by dietary sodium. Sodium is the major extracellular cation, and the reduction in extracellular fluid volume that occurs via diuresis may result in decreased blood volume, and decreased serum sodium. It has been reported the major side effect experienced by patients on a very-low-calorie-diet is mild postural lightheadedness occurring within the first 2 weeks, perhaps related to diuresis and a loss of sodium. Adequate potassium is necessary to achieve optimal protein-sparing therapy during

a VLCD. The protein sparing characteristics of the VLCD can increase potassium requirements (9). Sapir et al. (50) reported nitrogen wasting caused by losses of urinary ammonia during starvation could be eliminated by adequate potassium supplementation and urinary alkalinization, thus supporting the conclusion that potassium is necessary for optimal protein sparing-therapy. Recommendations for potassium intakes are between 2 and 3 grams per day. Calcium ions have many physiological functions besides being a principal component of skeletal tissue. It plays a vital role in a variety of essential physiologic and biochemical processes such as blood coagulation, neuromuscular excitability, transmission of nerve impulses and activation of enzyme reactions, and hormone secretion (50). Because of the major importance of calcium in the diet it is essential that the liquid VLCD contain the Recommended Dietary Allowance. In addition, an extra 400 to 800 milligrams of calcium per day should be provided. The phosphate ion is essential for the metabolism of carbohydrate, lipids, and protein. It functions as a cofactor for a multitude of enzyme systems and contributes to metabolic potential as a high-energy phosphate compound (50). The average daily requirement for phosphorous is between .8 and 1.2 milligrams per day. A source for dietary phosphorous in the American diet is in nonnutritious soft drinks. The individuals on a liquid VLCD are allowed to consume noncaloric beverages such as soft drinks. Therefore, phosphorous requirements may not be important.

Data are limited on the effects of the VLCD on serum electrolytes. Volioritch

et al. (52) examined biochemical data of patients involved in a clinical trial VLCD (Cambridge diet of 330 kcal/day). No change was seen in hematocrit, hemoglobin, or serum electrolytes [sodium (Na), potassium (K), chloride (Cl), calcium (Ca), phosphorous (P), magnesium (Mg), zinc (Zn)]. Significant changes occurred within the normal ranges of globulin, creatinine, uric acid, albumin, cholesterol, triglycerides, and iron. Kreitzman et al. (51) concluded the initial shift in lab values may reflect homeostatic adjustments to a new dietary regimen, and the one medical concern is the elevation of serum uric acids that may predispose a patient to gout. Commercially prepared liquid formula diets (such as Optifast-70) may contain some or all of the major electrolyte supplements. Descriptive analysis of serum electrolyte levels during the very-low-calorie regimen, realimentation, and maintenance will provide a more thorough understanding of the metabolic effects VLCDs have on electrolyte balance.

Elevated plasma cholesterol (values >220 mg/dL) according to American Heart Association guidelines have been associated with an increased risk of coronary heart disease. Cholesterol levels can be responsive to changes in body weight, diet, and exercise. It has been concluded that weight loss can have a significant impact on serum lipid levels, including triglycerides, total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (LDL).

Several studies have confirmed that plasma lipid values decrease during a VLCD, with serum triglycerides falling significantly in both men and women after

weight loss (53). In an investigative study conducted by Inoue et al. (54), reductions in body weight from 87.3 ± 3.8 Kg to 76.4 ± 2.8 Kg in 4 weeks significantly decreased fasting blood glucose, serum triglycerides, and serum total cholesterol levels during a VLCD. Weight loss was associated with decreases in fasting glucose from 110.4 ± 7.1 mg/dL to 80.8 ± 2.0 mg/dL, tryglycerides from 150.1 ± 8.3 mg/dL to 107.2 ± 4.3 mg/dL and cholesterol from 204.6 ± 6.5 mg/dL to 157.1 ± 6.0 mg/dL, respectively, in 4 weeks. Studies evaluating the influence of weight loss on high-density lipoprotein, cholesterol, and other lipoproteins are somewhat limited. The studies conducted yielded conflicting results. Weight loss has been reported to increase high-density lipoprotein cholesterol levels, decrease low-density lipoprotein cholesterol levels, or produce no change (55).

Kreitzman et al. (51) showed conclusive evidence of decreased serum cholesterol and triglyceride levels after weight loss on a VLCD with serum electrolytes and serum protein levels remaining stable. Wadden et al. (4) saw a significant decrease in total serum cholesterol in 20-25% of patients on a VLCD. The largest reduction was seen in low-density lipoprotein (29% decrease); some decrease was also noted in high-density lipoprotein (28% decrease). Typically, women have a decrease in high-density lipoproteins following a VLCD, but men have an increase in high-density lipoprotein. Brownell and Stunkard (56) concluded women show a significant decrease of 3.3% in high-density lipoprotein levels after weight loss, with a 4.7% decrease in levels. Parenti et al. (57) analyzed total

cholesterol, high and low-density lipoproteins, and triglycerides every week for 8 weeks of a VLCD. They concluded all values decreased. However, low-density lipoprotein fell, then returned to normal at the end of the program. Consequently, the HDL-LDL ratio did not change. Gebbard et al. (22) concluded weight loss reduced bile cholesterol saturation and improved high-density lipoprotein (HDL) levels. Because of the variable influences weight loss has on serum lipids along with the few prospective studies of the effects of weight loss changes in plasma lipid levels, evaluating the specific effects the Optifast-70 diet on plasma lipids is important.

METHODOLOGY

Restatement of Purpose

The purpose of this study was to evaluate the effects of a very-low-calorie diet program, specifically Optifast-70, on resting energy expenditure, body composition including percent body fat, lean body mass (LBM), and fat body mass (FBM), using body circumference measurements and near-infrared photo spectrometry. Also examined were the effects the diet program had on biochemical data, including complete blood count (CBC), serum chemistry panel (SMA-12), and lipid profile, on 17 outpatients during an Optifast-70 diet treatment program.

Subjects and Recruitment

Subjects for this study had participated in the McKay-Dee Hospital Optifast Program, Ogden, Utah. They included 25 volunteers from the outpatient population attending the Optifast Diet Program. The participant letter and consent form are noted in Appendix B, and Appendix C. The study was approved by the Utah State University Review Committee on Human Research and by the Institutional and Medical Review Board at McKay-Dee Hospital. The review board letter is shown in Appendix A.

Procedures

Before being accepted into the diet study, all subjects were given a preliminary physical and psychological screening. Primary subject selection criteria

were as follows:

1. Females aged 20-70 years
2. A typically sedentary lifestyle
3. A normal electrocardiogram (EKG)

After successfully completing the initial health screening process, each subject was seen on an appointment basis for the initial measurement of resting energy expenditure, body circumference measurements, and near-infrared photo spectrometry. Measurements for each of these parameters were collected:

1. Before the fast (week 1)
2. During the fast (week 7)
3. After the fast (week 13)
4. After realimentation (week 19)
5. After six weeks of maintenance (week 25).

Biochemical data including a serum chemistry panel (SMA-12) and complete blood count (CBC) were collected before the fast, and on weeks 5, 9, 13, 17, 21, and 25. Normal laboratory values for the serum chemistry panel and complete blood count are noted in Appendix G.

Fasting lipid profiles were drawn before the fast (week 1) and after 6 weeks of maintenance (week 25).

Each participant's height was taken to the nearest 0.1 centimeters before the fast, and weights were taken weekly to the nearest 0.001 kilograms (Kg).

Body composition was determined by using circumference measurements and following standard procedure (46). Measurements were taken to the nearest 0.1centimeter with a cloth measuring tape pulled tautly but not tight against the skin at designated points on the right side of the body:

1. Bicep - the midpoint between the shoulder and the elbow with the arm straight, palm up and extended in front of the body.
2. Forearm - widest point between the wrist and elbow.
3. Abdomen - one inch above the umbilicus
4. Upper thigh - just below the buttocks
5. Calf - widest point midway between the calf and ankle.

Body composition of each patient was determined by near-infrared photospectrometry using a Futrex-5000 fitness computer. Two measurements were taken from the prominent arm, midway between the shoulder and the elbow (bicep), and these were averaged.

Resting energy expenditure was determined by indirect calorimetry with a Beckman Metabolic Cart through the Respiratory Therapy Department at McKay-Dee Hospital. Actual resting energy expenditure (AREE) was calculated from oxygen consumption (VO2) and carbon dioxide production (VCO2). Predicted energy expenditure (PREE) was calculated using the Harris-Benedict equation for women (655 + (9.6) x (WT (Kg)) + (1.7 x HT (cm)) + (4.7 x Age). A respiratory quotient (RQ) was determined following the required standardized conditions for

subject preparation and data analysis. Subjects were post-absorptive before testing, and each subject was encouraged to have the measurement taken at the same time of day during each week the data were collected to decrease variability. Measurements were made in a quiet thermoneutral environment with patients resting in a supine position. Subjects had no voluntary muscle activity during the measurement. All data used to derive resting energy expenditure were taken during a "steady state."

After the initial measurement was collected at week 1, all subjects began consuming the liquid fasting supplement, Optifast-70. Water, noncaloric beverages, and 25 optional calories were allowed besides the Optifast-70 formula.

During the study the subjects were required to attend the clinic weekly to obtain weight, blood pressure, and pulse measurements and to insure dietary compliance. All subjects remained on the diet until they attained their desired goal weight, completed the fasting phase, or noncompliance was evident. After realimentation the maintenance period began. During this 6-week period of time each subject was gradually reintroduced to solid food. This entailed behavior modification, and identifying eating patterns necessary for maintenance of weight loss.

Statistical Analysis

Analyses of variance (ANOVA) were completed for each parameter investigated using time as the dependent variable. By-variant plots and frequency

data tables were completed on all data examined in the study. Stepwise regression was completed to identify how the change in weight related to all variables being examined in the study over time.

RESULTS

Body Mass

Mean body mass lost during the VLCD program was -20.44 Kg ± 6.62 Kg with a maximum loss of -33.23 Kg and a minimum loss of -9.77 Kg. Multivariate and univariate tests of significance reflected a significant ($p < .001$) change in body mass over time both between subjects and within subjects. Table 1 reflects the mean body mass of subjects before, during, and after the fast, 6 weeks into realimentation and 6 weeks into maintenance. Figure 1 represents a descriptive picture of the mean change in body mass of subjects over time.

TABLE 1.
The mean body mass (Kg) of subjects over the course of the diet program.

	Variable			
Week	Body Mass (Kg)	SD	N	95%CI
1	100.96	13.770	17	93.637-108.313
7	89.23	12.374	17	82.641- 95.828
13	83.09	13.262	17	76.022- 90.156
19	79.61	11.772	17	73.334- 85.880
25	80.53	11.527	16	74.391- 86.675

($p < .001$)

Resting Energy Expenditure

MANOVA measurements for the analysis of variance were completed on 5 points of actual resting energy expenditure (AREE) and predicted resting energy expenditure (PREE) over time (weeks 1, 7, 13, 19, 25). Multivariate and univariate

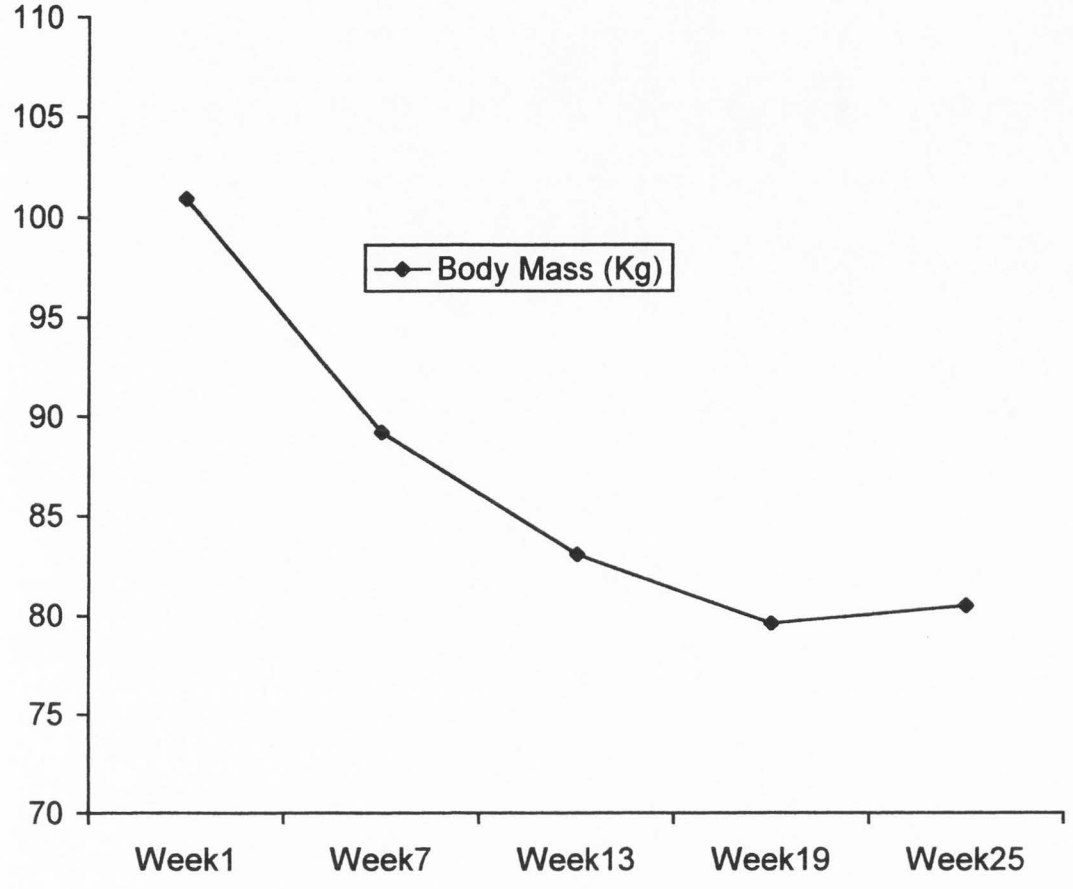

FIG. 1. The change in body mass in (Kg) over the course of the diet program.

TABLE 2.
The actual resting energy expenditure of subjects over the course of the diet.

Variable				
Week	Mean (Kcal)	SD	N	95% CI
1	1657	212	17	1543-1770
7	1503	203	17	1394-1611
13	1441	207	17	1330-1551
19	1429	167	17	1340-1519
25	1500	211	16	1387-1613

($p<.001$)

tests of significance (s = 1, m = 1, n = 5) both reflected significant changes ($p < .001$) in actual and ($p < .001$) predicted resting energy expenditure over the course of the diet program, as identified in Tables 2 and 3. However, when examining the mean difference between actual and predicted resting energy expenditure no significant change ($p < .409$) was noted over the course of the diet. Table 4 identifies the relationship between the difference in means for actual and predicted resting energy expenditure over the course of the diet. When comparing the mean values of actual resting energy expenditure and predicted energy expenditure per subject, using univariate and multivariate tests, a significant change ($p < .002$) was noted in actual and predicted resting energy expenditure over the course of the diet. Figure 2 outlines the relationship between actual and predicted resting energy expenditure over the course of the diet. Table 5 identifies the positive correlation between the amount of body mass lost and actual resting energy expenditure over the course of the diet. When examining the relationship between actual resting energy expenditure per kilogram of body mass [AREE/BM (Kg)], a significant difference was noted over the course of the diet ($p < .001$). Repeated measures of analysis of the variance identified a significant change between weeks 1 and 7. However, no significant change was noted between weeks 7 and 13, and between weeks 13 and 19. The relationship between weeks 19 and 25, however, did approach significance ($p < .05$). The significant values between actual resting energy expenditure and body mass over time are identified in Table 6.

TABLE 3.
The predicted resting energy expenditure of subjects over the course of the diet.

Variable				
Week	Mean (kcal)	SD	N	95% CI
1	1715	147	17	1636-1793
7	1622	133	17	1549-1692
13	1547	127	17	1479-1615
19	1514	118	17	1451-1577
25	1523	121	16	1458-1587

($p < .001$)

TABLE 4.
The difference in means between actual and predicted resting energy expenditure of subjects over the course of the diet.

Variable				
Week	Mean(kcal)	SD	N	95% CI
1	58	146	17	-19-135
7	118	115	17	56-179
13	106	128	17	38-175
19	85	126	17	17-152
25	22	152	16	-59-103

($p < .409$)

TABLE 5.
The correlation between body mass (Kg) and resting energy expenditure (REE) over course of the diet.

Variable			
Weight 7	Weight 13	Weight 19	Weight 25
.0960	.1686	.0752	.0989
17	17	17	16
p=.357	p=.259	p=.387	p=.358
REE 7	REE 13	REE 19	REE 25

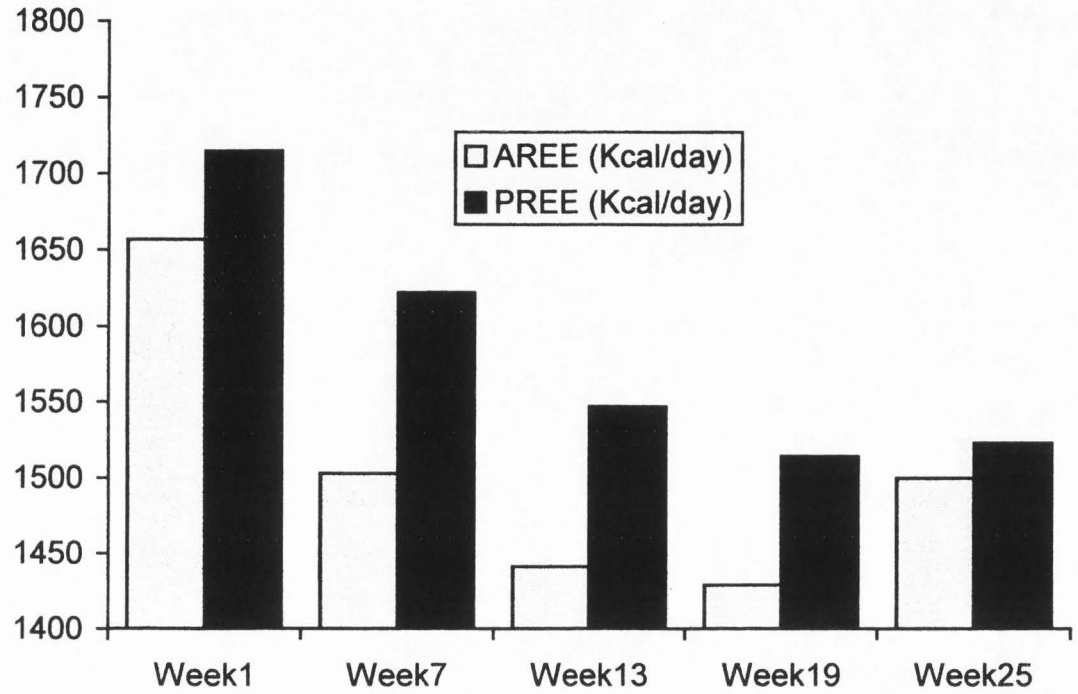

FIG. 2. The relationship between actual resting energy expenditure (AREE) and predicted resting energy expenditure (PREE) over the course of the diet.

TABLE 6.
The relationship between actual resting energy expenditure (AREE) and body mass (Kg) over the course of the diet.

	Variable			
Week	AREE/BM	AREE/BM	AREE/BM	AREE/BM
1-7	p=.001			
7-13		p=.095		
13-19			p=.609	
19-25				p=.095

Respiratory Quotient

Repeated measures of analysis of variance examining the degree at which the variation of intakes affected changes in respiratory quotient (RQ) showed a statistically significant value ($p < .001$) for variation in RQ within subjects and between subjects over time. Table 7 identifies the change in RQ during the fasting, realimentation, and maintenance phases of the program. Figure 3 visualizes the change in RQ over the course of the diet.

TABLE 7.
The mean change in respiratory quotient over the course of the diet.

Variable Week	Mean	SD	N	95% CI
1	.82	.07	17	.77-.86
7	.74	.02	17	.73-.76
13	.78	.05	17	.75-.80
19	.78	.03	17	.76-.80
25	.82	.08	16	.78-.86

($p < .001$)

Anthropometrics

In examining the variability of measurements between circumference body fat (CBF) and infrared body fat (IBF), a significant correlation ($p < .000$) was noted between the percent body fat measured via circumference measurements and those done via infrared photospectrometry during the diet. The correlation between the two measurements became more significant as body mass loss progressed over the

course of the program. These are reflected in Tables 8 and 9. Figure 4 visualizes the positive correlation between circumference and infrared body fat measurements over time. Descriptive analysis was done on the fat body mass (FBM) and lean body mass (LBM) lost over the course of the diet. Table 10 identifies the mean differences between circumference and infrared body fat measurements over time. Pearson correlations reflected no significant change in the relationship between fat body mass and lean body mass over the course of the diet. This is identified in Table 11. Tables 12 and 13 identify the mean loss in fat and lean body mass during the course of the diet program. These losses are identified and compared to the

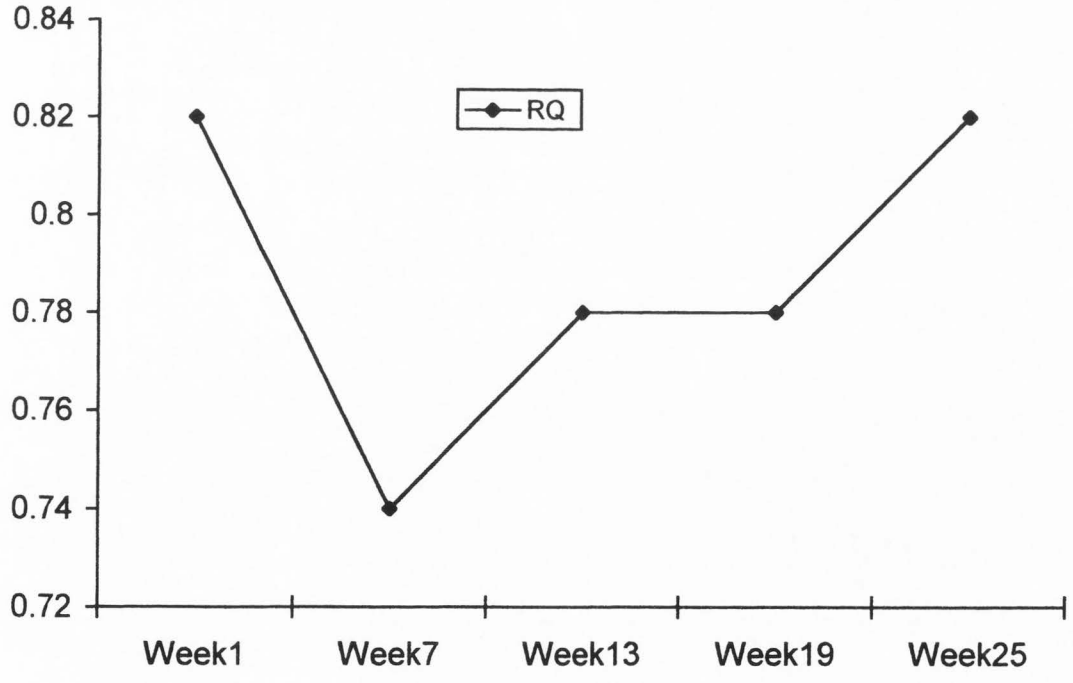

FIG. 3. The change in respiratory quotient (RQ) over the course of the diet.

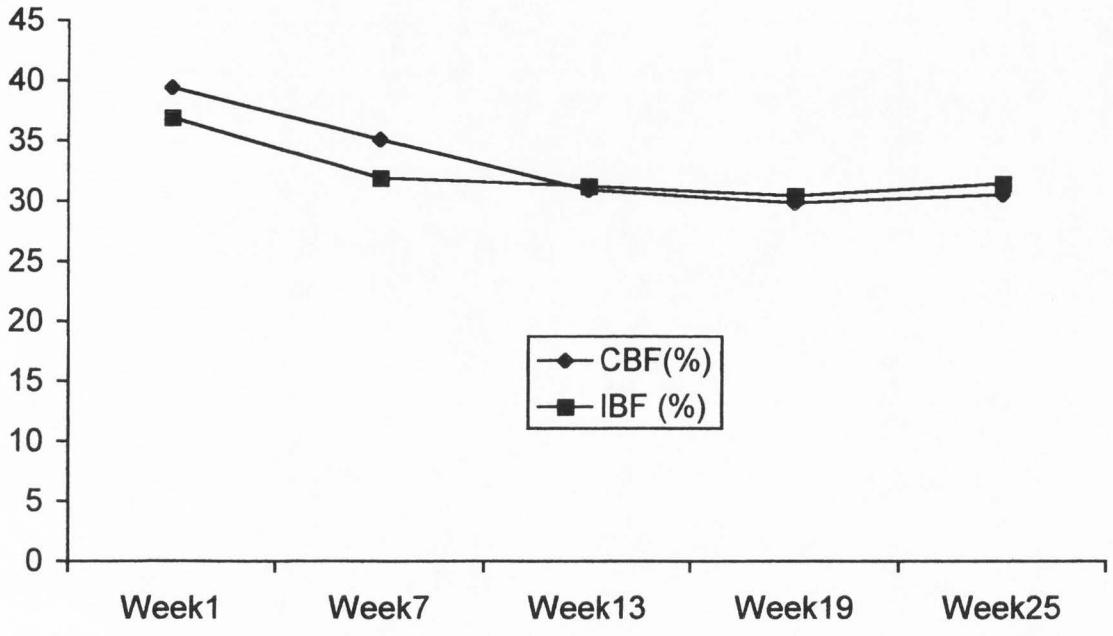

FIG. 4. The comparison of the circumference body fat (%) and infrared body fat (%) over the course of the diet.

loss of total body mass in Figure 5. The relationship between body mass and percent body fat became more highly correlated over the course of the diet. This is identified in Table 14. No correlation was found between body fat mass and actual resting energy expenditure over time as noted in Table 15. When the relationship between lean body mass and resting energy expenditure was examined, a significant correlation ($p = .001$) was noted between the two parameters over time. Significance was noted between weeks 1 and 7. No significance was identified between weeks 7 and 13, and 13 and 19. The relationship approached significance between weeks 19 and 25, noted in Table 16.

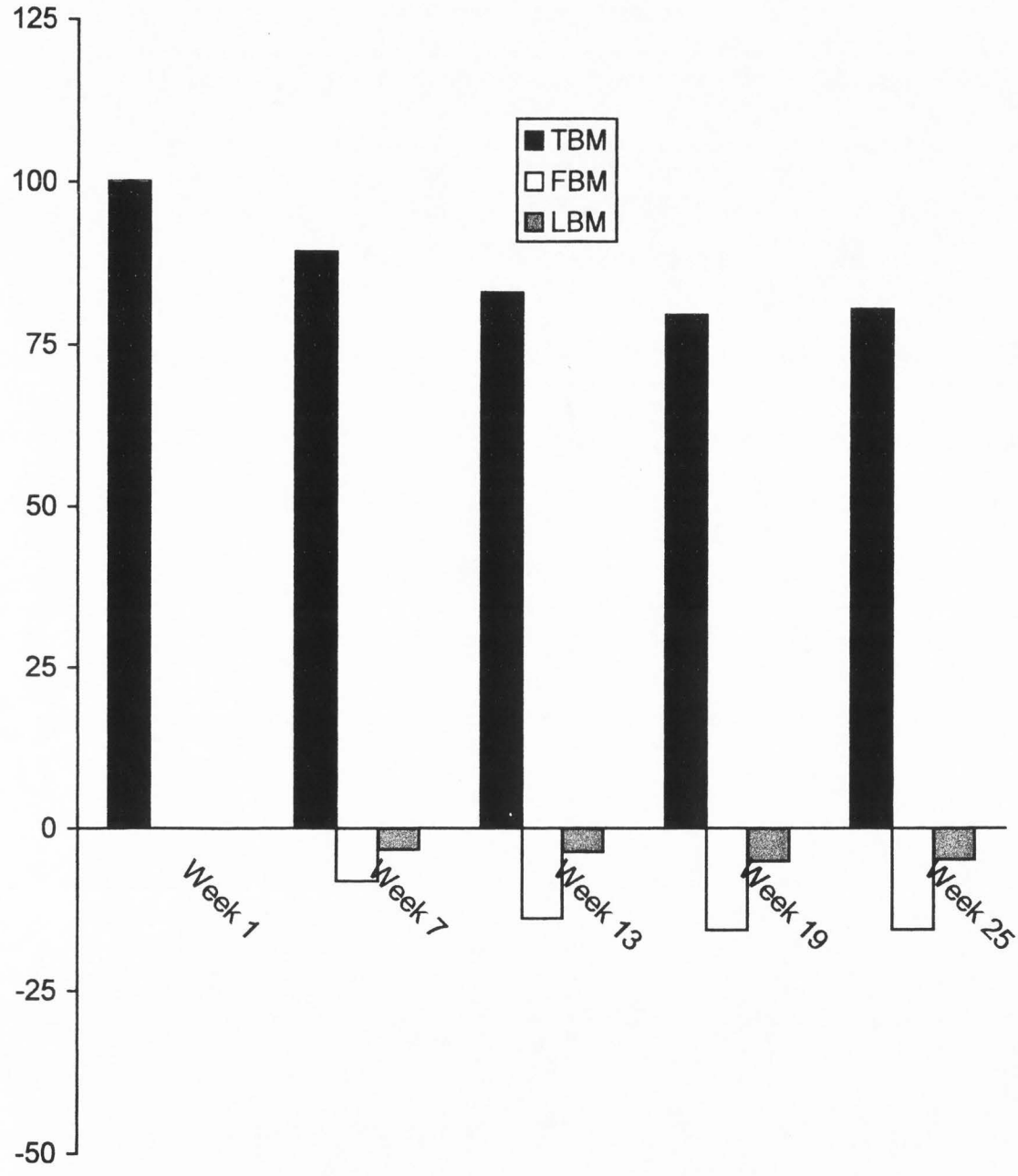

FIG. 5. The relationship between total body mass (TBM), lean body mass (LBM), and fat body mass (FBM) over the course of the diet.

TABLE 8.
The t-test comparison between circumference and infrared body fat measurements over the course of the diet.

Variable	N	Mean(%)	SD	Correlation	Two Tail Probability
CBF1	17	39.4	5.0	.452	.11
IBF1		36.9	6.4		
CBF7	17	35.1	5.0	.598	.02
IBF7		31.9	6.5		
CBF13	17	30.9	5.0	.587	.77
IBF13		31.2	2.9		
CBF19	17	29.8	5.2	.727	.55
IBF19		30.4	3.4		
CBF25	16	30.5	4.7	.568	.40
IBF25		31.4	3.2		

CBF - circumference body fat IBF - Infrared body fat

TABLE 9.
The correlation between infrared body fat (IBF) and circumference body fat (CBF) measurements over the course of the diet program.

	Variable				
	IBF1	IBF7	IBF13	IBF19	IBF25
	N=17	N=17	N=17	N=17	N=16
CBF1	.4523 p=.034				
CBF7		.5981 p=.006			
CBF13			.5872 p=.007		
CBF19				.7271 p=.000	
CBF25					.5680 p=.011

CBF - circumference body fat IBF - infrared body fat

TABLE 10.
The difference between circumference body fat (%) and infrared body fat (%) means over the course of the diet.

Variable	N	Mean(%)	SD	Minimum	Maximum
CBF1					
IBF1	17	2.4	6.1	-14.1	9.8
CBF7					
IBF7	17	3.2	5.3	-3.4	18.7
CBF13					
IBF13	17	-.29	4.1	-6.6	7.4
CBF19					
IBF19	17	-.52	3.6	-7.0	6.5
CBF25					
IBF25	16	-.85	3.9	-9.6	6.5

CBF - Circumference body fat IBF - Infra-red body fat

TABLE 11.
The correlation between the change in fat body mass and lean body mass over the course of the diet.

	Variable				
	LBM1	LBM7	LBM13	LBM19	LBM25
	N=17	N=17	N=17	N=17	N=16
FBM1	0				
FBM7		-.7281 p=.000			
FBM13			-.2672 p=.148		
FBM19				.1172 p=.327	
FBM25					.0155 p=.477

FBM - fat body mass LBM - lean body mass

TABLE 12.
The mean loss of fat body mass (FBM) over the course of the diet using infrared photospectrometry as a measurement.

Variable

	N	Mean(Kg)	SD	Minimum	Maximum
FBM1	17	0.0	0.0	0.0	0.0
FBM7	17	-8.2	3.0	-3.3	-13.9
FBM13	17	-13.9	4.5	-7.7	-21.7
FBM19	17	-15.7	4.8	-8.8	-24.0
FBM25	16	-15.6	5.7	-6.7	-27.8

TABLE 13.
The mean loss of lean body mass (LBM) over the course of the diet using infrared photospectrometry as a measurement.

Variable

	N	Mean(Kg)	SD	Minimum	Maximum
LBM1	17	0.0	0.0	0.0	0.0
LBM7	17	-3.3	3.0	1.9	-9.2
LBM13	17	-3.7	2.9	1.0	-10.2
LBM19	17	-5.1	3.2	1.8	-12.8
LBM25	17	-4.8	3.1	-.4	-13.0

TABLE 14.
The correlation between body mass (Kg) and percent body fat (%BF) over the course of the diet.

BM/%BF 7	BM/%BF 13	BM/%BF 19	BM/%BF 25
-.1364	.5635	.4750	.6558
N=17	N=17	N=17	N=16
p=.301	p=.009	p=.027	p=.003

TABLE 15.
The correlation between percent body fat (%BF) and resting energy expenditure (REE) over the course of the diet.

BF%/REE7	BF%/REE13	BF%/REE19	BF%/REE25
-.0615	.0490	-.2211	-.0522
N=17	N=17	N=17	N=16
p=.407	p=.426	p=.197	p=.424

TABLE 16.
The relationship between actual resting energy expenditure (AREE) and lean body mass (LBM(Kg)) over the course of the diet.

	Variable			
Week	AREE/LBM(Kg)	AREE/LBM(Kg)	AREE/LBM(Kg)	AREE/LBM(Kg)
1-7	p=.001			
7-13		p=.138		
13-19			p=.582	
19-25				p=.060

Biochemical

The biochemical data by week using chi-square charts indicated no significant change in any biochemical parameter over the course of the diet regimen. However, secondary to the increased number of missing data, trusting the results is difficult. Cholesterol did show a significant change over the course of the diet ($p \leq .020$). Uric acid approached significance with ($p \leq .076$). Table 17 identifies the mean biochemical profiles of subjects at baseline weeks. Figures 10 through 26, as shown in Appendix D, illustrate the small variation in biochemical

parameters over the course of the diet.

Lipid profiles, independent of body mass, taken before the start of the fast and after 6 weeks of maintenance showed no significance, but variations were identifiable. Tables 18 and 19 show the correlation between the difference of the two measurements with each parameter, and the mean differences. In Table 19, the DHL variable identified the difference in the ratio of high-density lipoproteins (HDL) to low-density lipoproteins (LDL).

TABLE 17.
The biochemical profile of subjects at baseline week, before the fast.

Variable	Mean	SD	Minimum	Maximum
Sodium (mmol/L)	140	2.0	36	143
Potassium (mmol/L)	4.0	.3	3.4	4.7
Chloride (mmol/L)	107	2.7	103	111
Glucose (mg/dL)	94	49	66	281
BUN (mg/dL)	12	2.0	8	19
Creatinine (mg/dL)	0.9	.2	.6	1.3
Uric Acid (mg/dL)	5.6	1.1	3.5	7.7
Total Protein(gm/dL)	7.0	.3	6.3	7.6
Albumin (gm/dL)	4.2	.2	3.8	4.7
Calcium (mg/dL)	9.2	.4	8.7	10.1
Phosphorus (mg/dL)	3.4	.5	2.1	4.5
Cholesterol (mg/dL)	202	30	143	245
White Blood Cells 10^3/uL	7.5	1.4	5.2	10.0
Red Blood Cells 10^6/uL	4.3	1.1	3.0	5.2
Hemoglobin (gm/dL)	13.9	.9	12.0	15.8
Hematocrit %	41.9	2.7	36.0	47.0
Total Lymphocyte Count	2159	462	404	3171
Count %				

TABLE 18.
The correlation between the differences of measurements for each parameter examined in the lipid profile before the fast and after maintenance.

DChol	DTG	DHDL	DVLDL	DLDL	DHL
-.1407	-.2910	-.1266	.1037	.2659	.0963
N=13	N=13	N=13	N=13	N=13	N=10
p=.323	p=.167	p=.340	p=.368	p =.190	p=.396

D - difference, Chol - cholesterol, TG - triglycerides, HDL - high-density lipoproteins, VLDL - very-low-density lipoproteins, LDL - low-density lipoproteins, DHL- difference in the ratio of high-density lipoproteins to low-density lipoproteins

TABLE 19.
The mean changes in lipid profile parameters over the course of the diet.

Variable	Mean	SD	Minimum	Maximum
Cholesterol	17	24	-23	51
TG	45	48	-.5	155
HDL	-1.6	8.5	-16	13
VLDL	6.7	13	-26	31
LDL	-20	67	-132	88
DHL	-.5	1.8	-5.8	.2

TG - triglycerides, HDL - high-density lipoproteins, VLDL - very-low-density lipoproteins, LDL - low - density lipoproteins, DHL - difference in ration between high-density lipoproteins and low-density lipoproteins.

DISCUSSION

Body Mass

A significant loss in body mass of -20.442 $\pm$ 6.609 Kg was noted over the course of the diet program. These findings are consistent with investigators who associated the loss of body mass on a VLCD as superior to other nonsurgical treatment (5). The most significant loss in body mass occurred during the first 7 weeks of the diet, or the fasting stage. This dramatic early loss of body mass could also have been attributed to diuresis. Subjects' loss of body mass appeared to slow after the fast and stabilized at week 25. Examining these subjects for a regain in body mass would be interesting and would determine the percentage of subjects who maintained a portion of their weight loss.

Resting Energy Expenditure

Both actual and predicted resting energy expenditure showed a significant decrease with body mass loss over the course of the diet. This is in agreement with the data obtained by Barrows and Snook (34), who identified a significant reduction in resting energy expenditure with weight loss using the Optifast-70 formula. Assuming the reduction in resting energy expenditure was caused by a loss in total body mass would be reasonable, as Coxon et al. (37) determined, the greater the weight loss the greater the decrease in resting energy expenditure. A significance was identified between actual resting energy expenditure per kilogram of body

mass between weeks 1 and 7, and weeks 19 and 25. The initial drop in actual resting energy expenditure during the initial phase of the fast relates to loss in body mass. However, the relationship between actual resting energy expenditure (AREE) and body mass [BM(Kg)] during stabilization may be conclusive with the data of Wadden et al. (4), who concluded a reduction in resting energy expenditure associated with the consumption of a VLCD may be limited to the actual period of the diet. Coxon et al. (37) concluded the change in resting energy expenditure was not associated with any specific component of body composition, as their data suggested no change in the ratio of resting energy expenditure (REE) to lean body mass (LBM), REE/LBM(Kg). However, this study did identify significance between the loss in lean body mass and the change in resting energy expenditure. This does not relate to the findings of Franssila and Kallunki (41), who related the decrease in actual resting energy expenditure paralleled the loss in fat body mass, not lean body mass. Perhaps a larger sample size would have concluded otherwise. Exercise plays a major role in maintaining muscle mass during weight loss. This would have been an area of interest to be investigated as some subjects did not participate actively in exercise programs. It appeared patients' resting energy expenditures were on an upward trend from week 19 through week 25. Perhaps with further study the same conclusion could be drawn as that of other investigators (38, 39, 40) in which a reduction in resting energy expenditure was associated with a VLCD. However, the resting energy expenditure increased rapidly to 93-98% baselines when patients reached a stable reduced weight.

Respiratory Quotient

Patients' respiratory quotients paralleled each stage of the diet program. Results suggested patients initially were consuming a mixed balanced diet and as fasting began, values dropped to those values equal to patients being underfed, or those mobilizing fat for energy. These values returned to baseline when patients began the maintenance portion of the program.

Anthropometrics

The variation in body composition associated with weight loss during a VLCD has prompted many researchers to suggest that anthropometric measurements could be inadequate for clinical assessment of body composition in obese patients. Circumference measurements may be more effective at estimating body fat as weight loss progresses. This is in agreement with the findings of other researchers (34, 43). Near-infrared photospectrometry measurements showed a significant correlation with circumference measurements. The correlation between the two became more significant as weight loss progressed. Therefore, near-infrared photospectrometry is more accurate as weight loss progresses and as patients reach desirable weight. The loss in body mass progressed with losses in fat body mass (~15.6 Kg) being greater than those of lean body mass (~5.0 Kg). These findings could point to the fact that perhaps the change in actual resting energy expenditure was associated with total body mass lost, rather than an individual parameter.

Biochemical

No significant changes were noted in complete blood count over the course of the diet program. Only cholesterol reflected a significant change ($p \leq .020$) in the SMA-12, with uric acid being close to significance ($p \leq .076$). These are in agreement with Kreitzman et al. (51) and Voliovitch et al. (52) who saw no change in hematocrit, hemoglobin, or any serum electrolytes (Na, K, Cl, Ca, P, Mg, Zn). The one major medical concern with VLCD is the elevation in uric acid. This may predispose patients to gout. This could be a legitimate concern with the change in uric acid approaching significance in this study. No subjects were identified with gout during the diet. More conclusive data, however, may have yielded different results. The data suggest changes in cholesterol levels can be in response to a loss in total body mass. This is in agreement with Wadden et al. (4). No significance was noted in the lipid profiles of patients before the fast and six weeks into maintenance. Variations in values appeared to fit those of other investigators (55, 56). Perhaps initial values fell during the diet and returned to normal at the end of the diet, consequently showing no change in the ratio of high- density lipoproteins (HDL) to low-density lipoproteins (LDL), (HDL/LDL). This would be in agreement with Parenti et al. (57).

CONCLUSION

Obesity remains a chronic disease of major proportion in the U.S. and it continues to be a disease with no cure. Because obesity is a complex problem, a multidisciplinary approach to weight loss is still the best overall method in its management. VLCDs have received their share of criticism over the past decade. Complaints related to weight regain have been justified but not always presented in an unbiased manner. Other criticisms include the diet's adverse effects on resting energy expenditure and body composition but the criticisms have not been supported by the literature. Others criticisms require further study, such as the risk of gallstone formation and complications associated with gout. Despite the rave of criticism, VLCDs remain a highly effective option in the treatment of obesity, if accompanied by a multidisciplinary approach to patient care. They induce weight loss and control health complication in the obese.

Current research has lead to new conclusions concerning the clinical use of VLCD.

1. Studies have concluded no significant difference in weight loss between 420 kcal/d and 800 kcal/d regimens.
2. Success may be more directly related to portion and calorie controlled food then severe caloric restriction.
3. Better effects may be noted by combining liquid meal supplements with solid food options.

From these conclusions, the new Optifast program "Promed" has been redesigned and changed from the old rigorous methods to more individualized regimens that cater to each patient's specific needs. It is anticipated that these changes will eliminate much of the controversy surrounding VLCDs and improve long-term maintenance of weight loss. The primary goal of the new concepts is to improve long-term maintenance of weight loss and the medical complications. An increasing awareness of the chronic, multifactorial nature of obesity will lead to the development of safe and effective long-term treatment programs (3).

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APPENDICES

Appendix A

Review Board Letter

All data except the body circumference measurements and NIR photospectrometry will be collected by the Optifast program at McKay-Dee Hospital. Circumference and photospectrometry (NIR) readings will be collected by the investigator, with supervision by the Optifast program director. The Optifast program is already conducting resting metabolic readings via indirect calorimetry through respiratory therapy, the additional Beckman readings will also be collected by respiratory therapy. Research subjects will be at minimal risk besides those outlined by the Optifast program. Subjects will not be identified by name but by number to safeguard confidentiality.

Funding is needed for the three additional Beckman readings only; and will be provided by Utah State University Department of Nutrition & Food Sciences. Therefore, will be no extra cost to the subjects.

Appendix B

Participant Letter

Dear Participant:

A study is being conducted by Charlene Perkins a graduate student in Nutrition at Utah State University, evaluating the Optifast 70 diet and it's effects on resting metabolic rate, body composition, blood electrolytes and cholesterol. All tests involved in the study are included in the Optifast Program except the body composition measurements. Your participation would be appreciated.

Factors to be considered in the study:

1. Resting metabolic rate (RMR) will be measured with a Beckman Metabolic cart, which determines RMR by measuring oxygen consumption through a mask while resting in a quiet environment. This procedure will be done during weeks one (1), seven (7), thirteen (13), nineteen (19), and twenty-five (25). It will take approximately 45 minutes and an appointment should be made no less than 7 days in advance. Measurements must be taken during the same time of day for each consecutive week. This information will help to determine the effects calorie restriction has on resting metabolic rate.

2. On the same weeks as above, body composition will be evaluated by taking circumference measurements of the arm, waist buttocks, thigh and calf. Body composition will also be examined using a Futrex-500 Fitness Computer, which assesses body composition using light beams. The measurement will be taken on the prominent arm midway between the shoulder and elbow, and will only takes a couple of minutes. These measurements will help to evaluate the effects of calorie restriction on body composition particularly fat and muscle.

3. On the same weeks as above your blood chemistry results will be evaluated concerning electrolytes and cholesterol. These results will give a basic overview of how the Optifast diet effects your blood chemistry.

Appendix C

Consent Form

It has been explained to me that Miss Perkins will conduct the body composition measurements and the Optifast Program will conduct the remaining tests, including the RMR, blood electrolytes and blood minerals.

I understand the information obtained in this study will be held in strict confidence. If this study is published I have been assured there will be no information disclosed which identifies me as a participant.

I understand there is no perceived risk to me participating in this study. Any questions that I have regarding the study have been answered to my satisfaction.

I understand I am free to withdraw from the study, at any time, for any reasons of my choosing, without my standard of care being affected.

I understand that there is no additional cost to me for participating in this study.

Any questions may be answered by calling Miss Perkins at 801/750-2139 or 801/753-4595.

_____ _____

Date Signature of Participant

_____ _____

Signature of Investigator Signature of Principal Investigator

_____

Signature of Optifast Program Director

Appendix D

Changes in Biochemical Data over the Course of the Diet

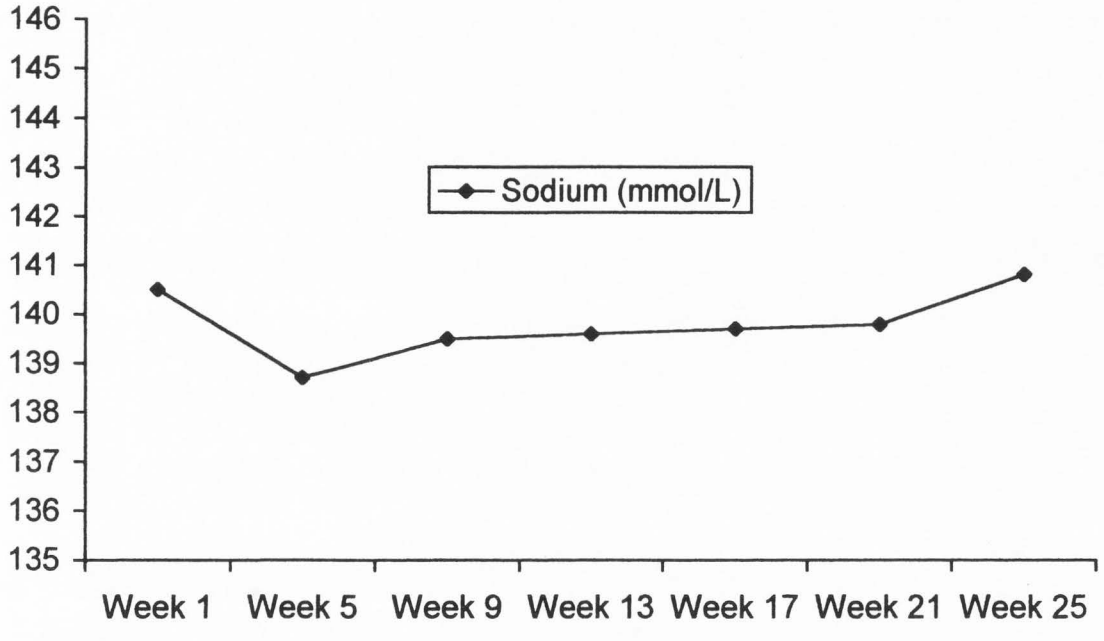

FIG. 6. The change in serum sodium over the course of the diet.

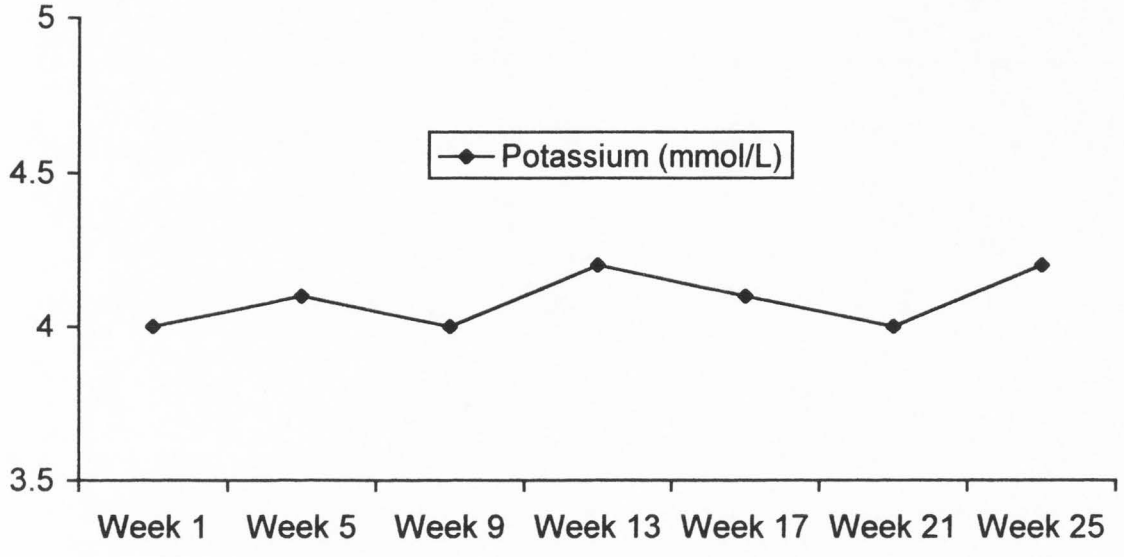

FIG. 7. The change in serum potassium over the course of the diet.

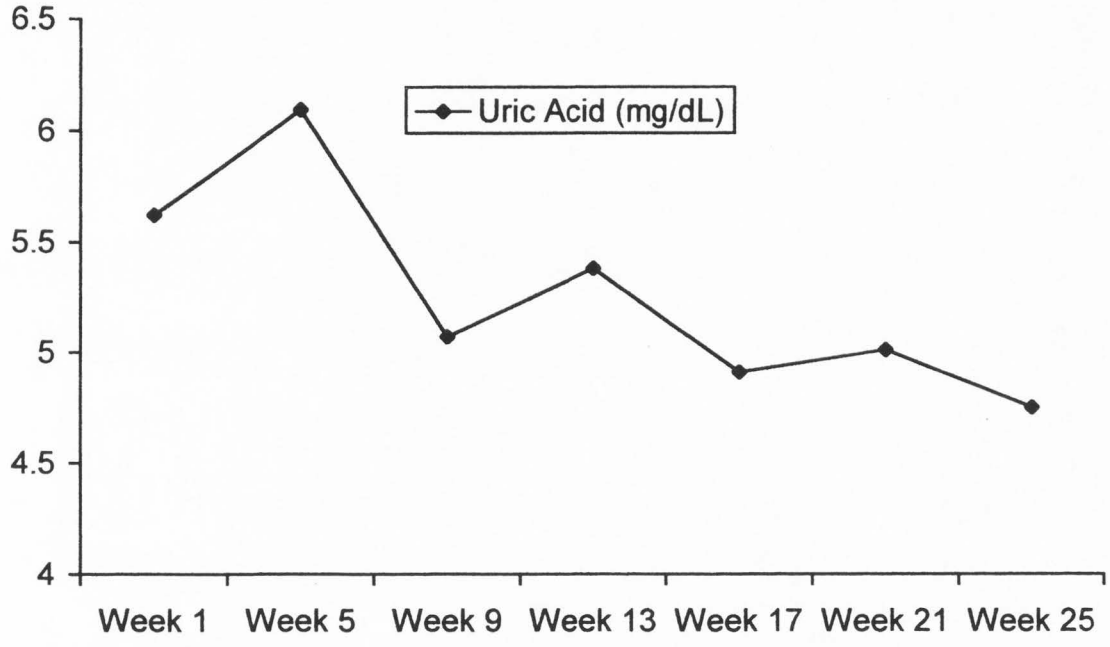

FIG. 8. The change in serum uric acid over the course of the diet.

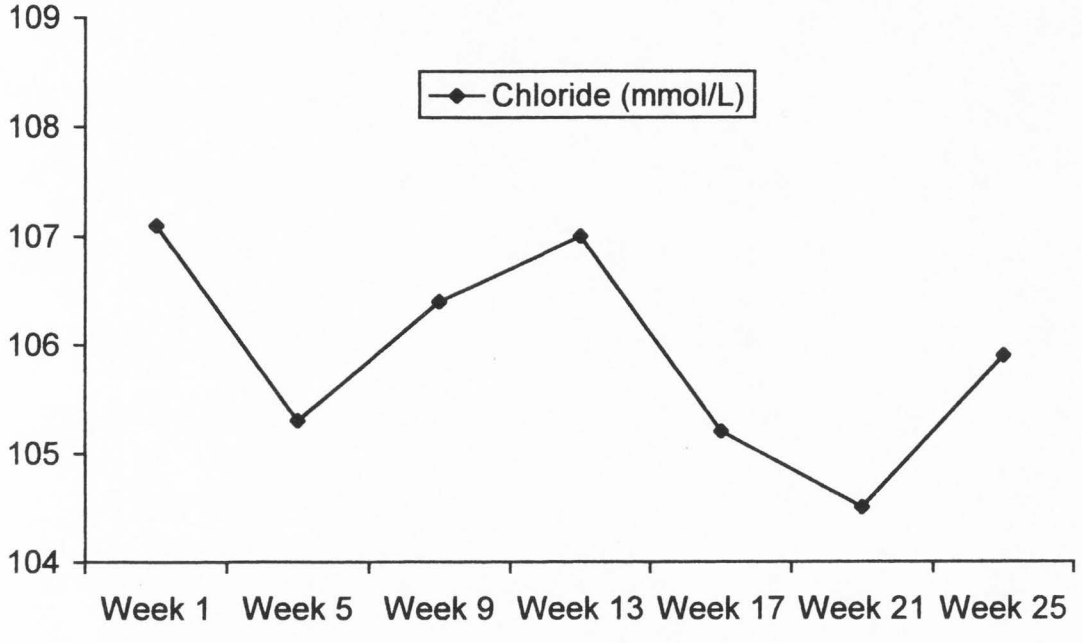

FIG. 9. The change in serum chloride over the course of the diet.

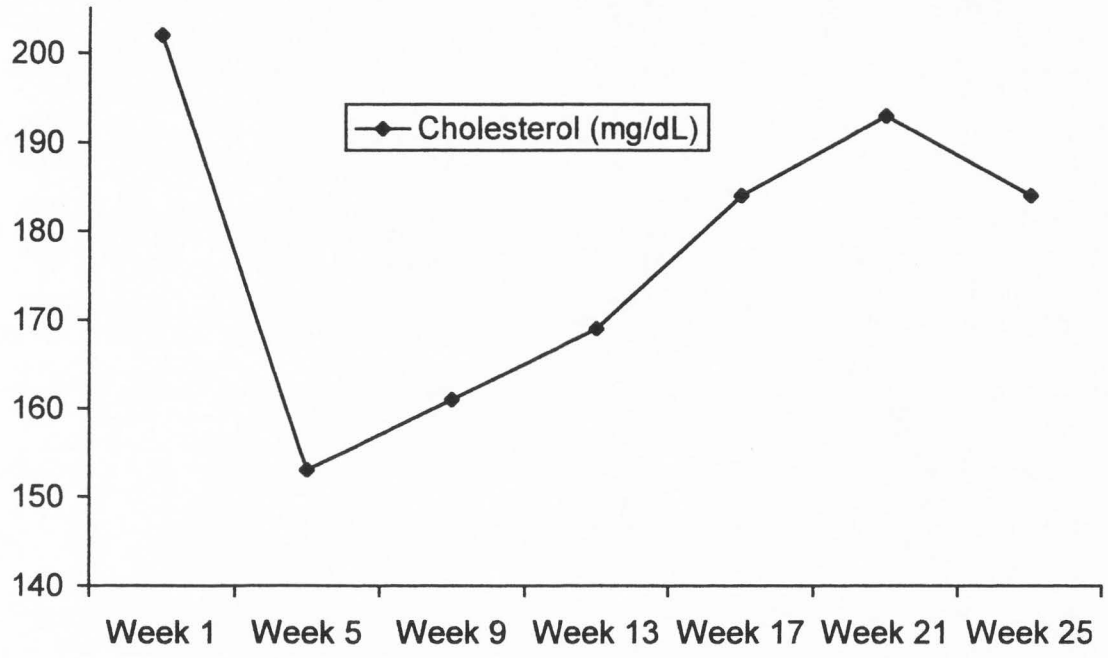

FIG. 10. The change in serum cholesterol over the course of the diet.

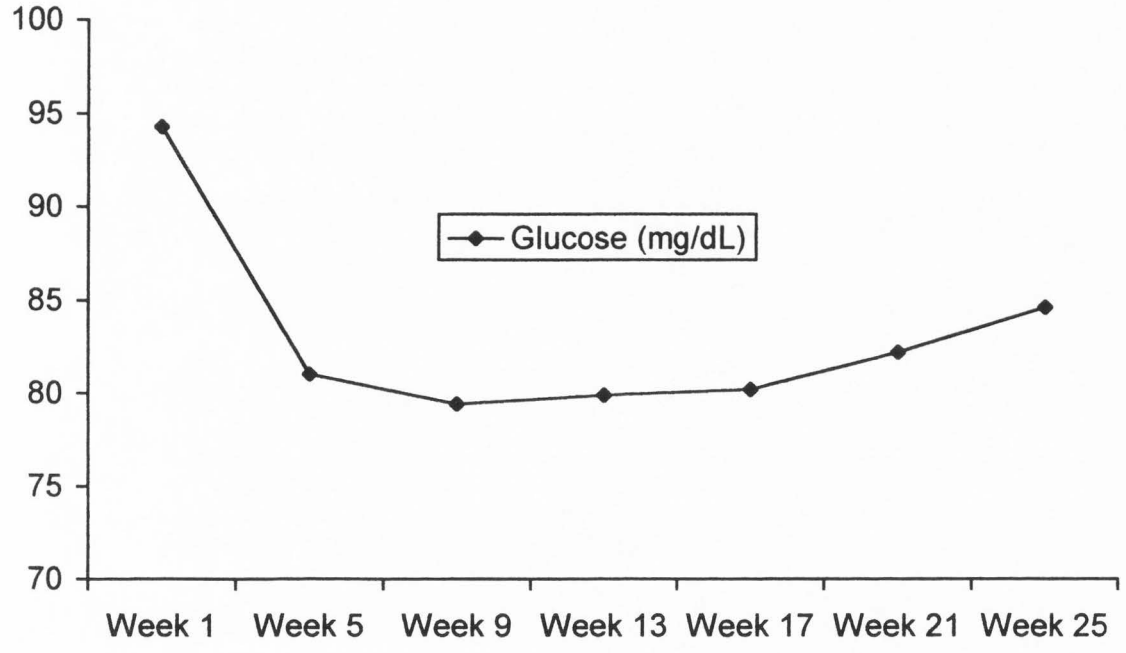

FIG. 11. The change in serum glucose over the course of the diet.

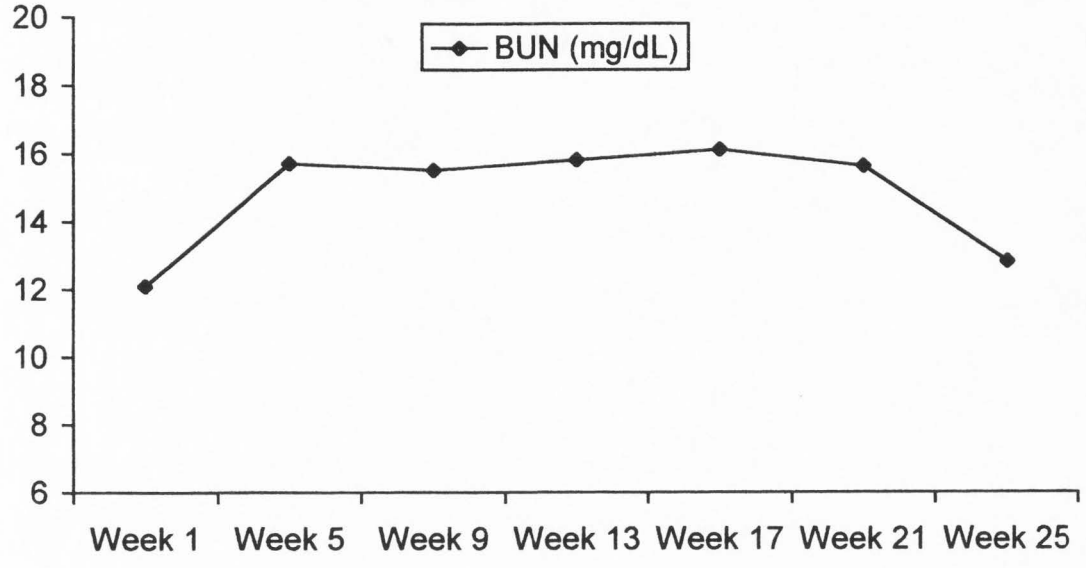

FIG. 12. The change in serum blood urea nitrogen (BUN) over the course of the diet.

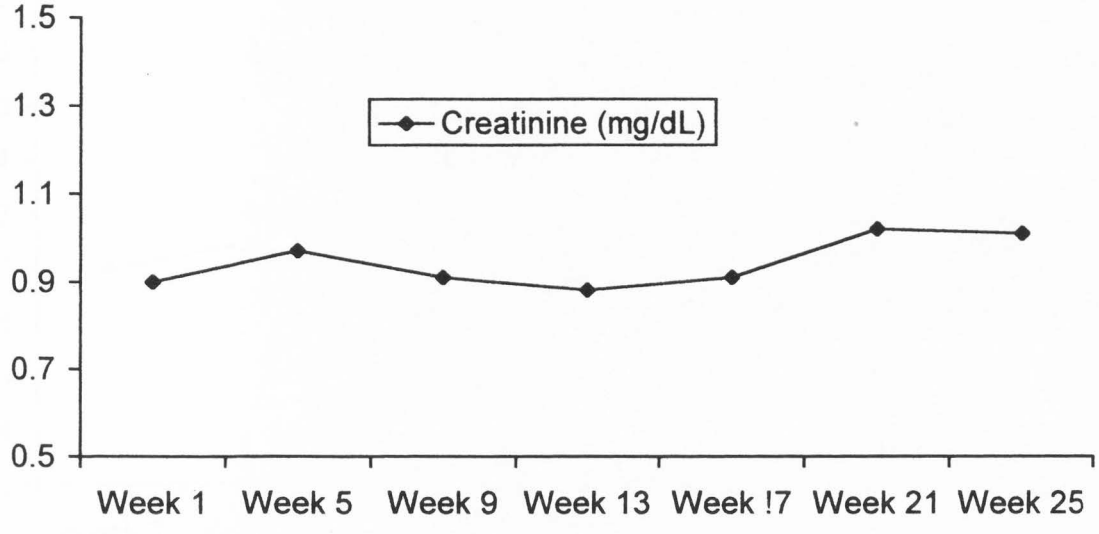

FIG. 13. The change in serum creatinine over the course of the diet.

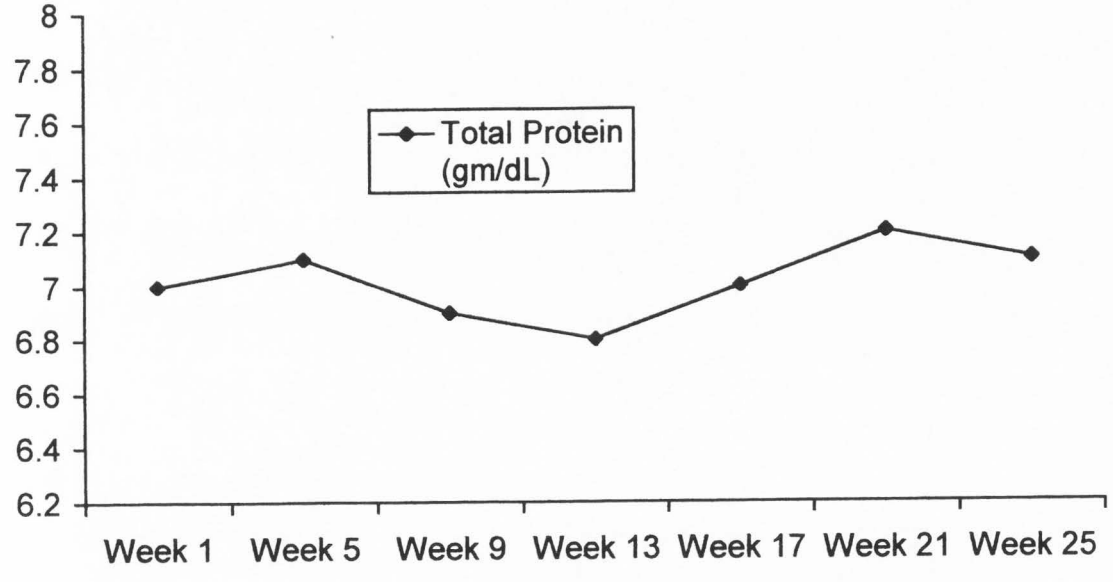

FIG. 14. The change in serum total protein over the course of the diet.

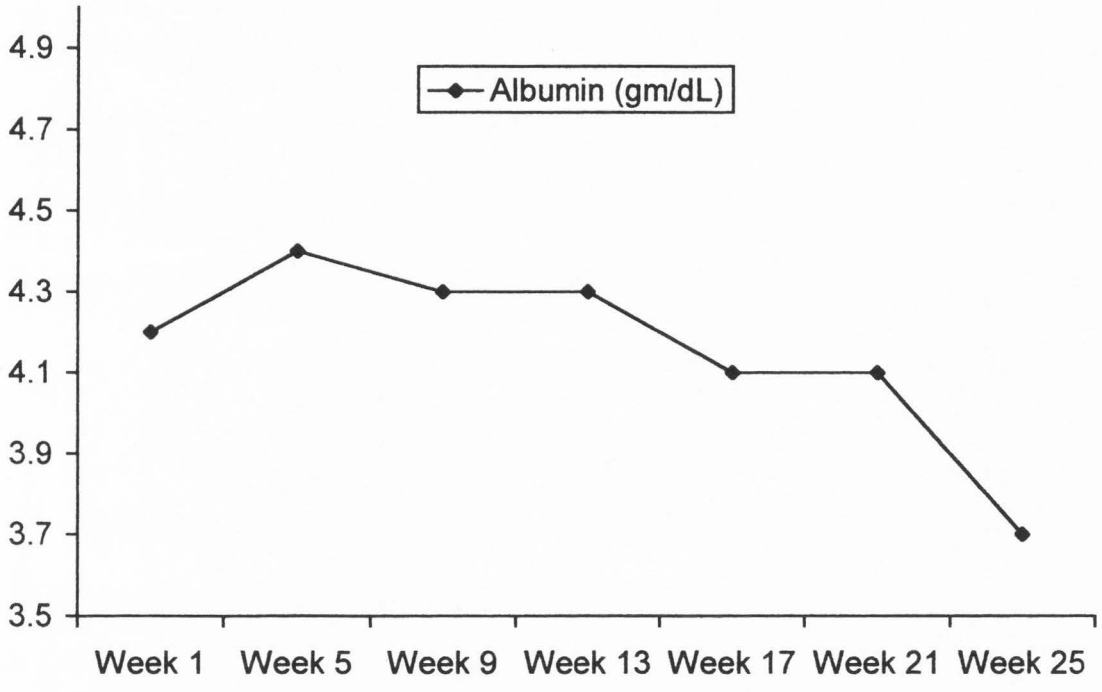

FIG. 15. The change in serum albumin over the course of the diet.

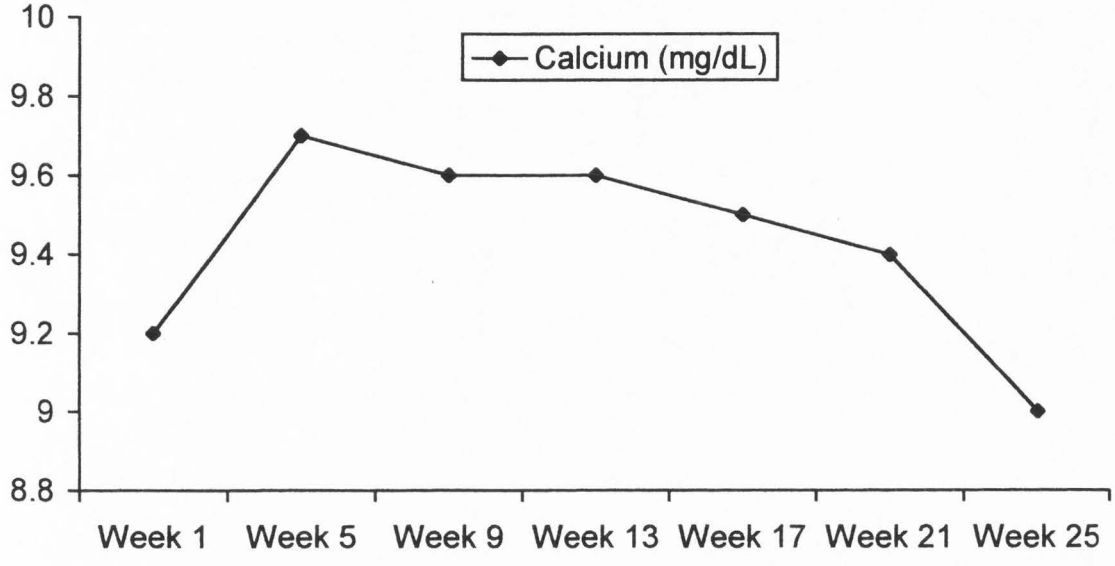

FIG. 16. The change in serum calcium over the course of the diet.

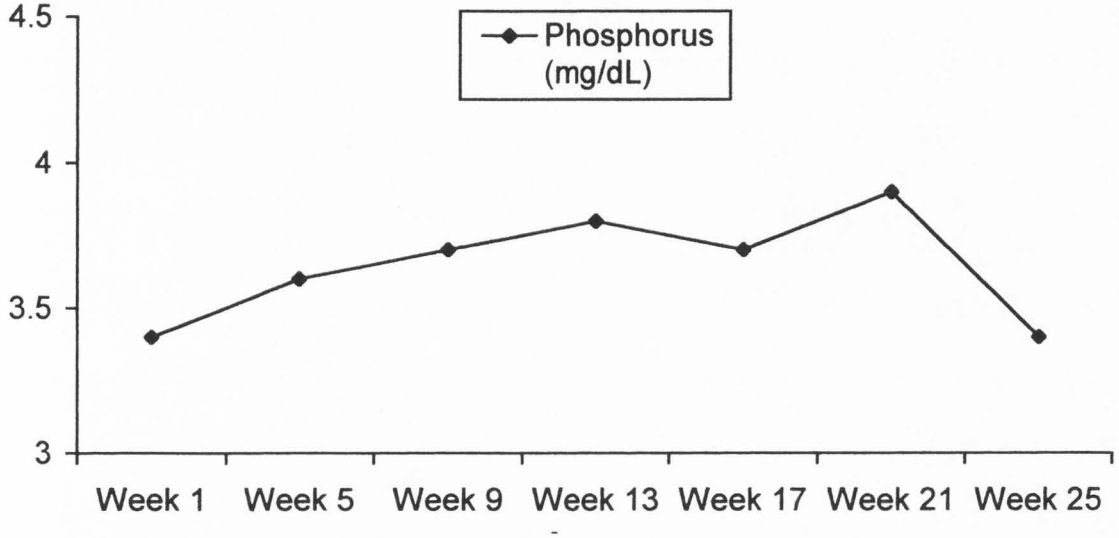

FIG. 17. The change in serum phosphorus over the course of the diet.

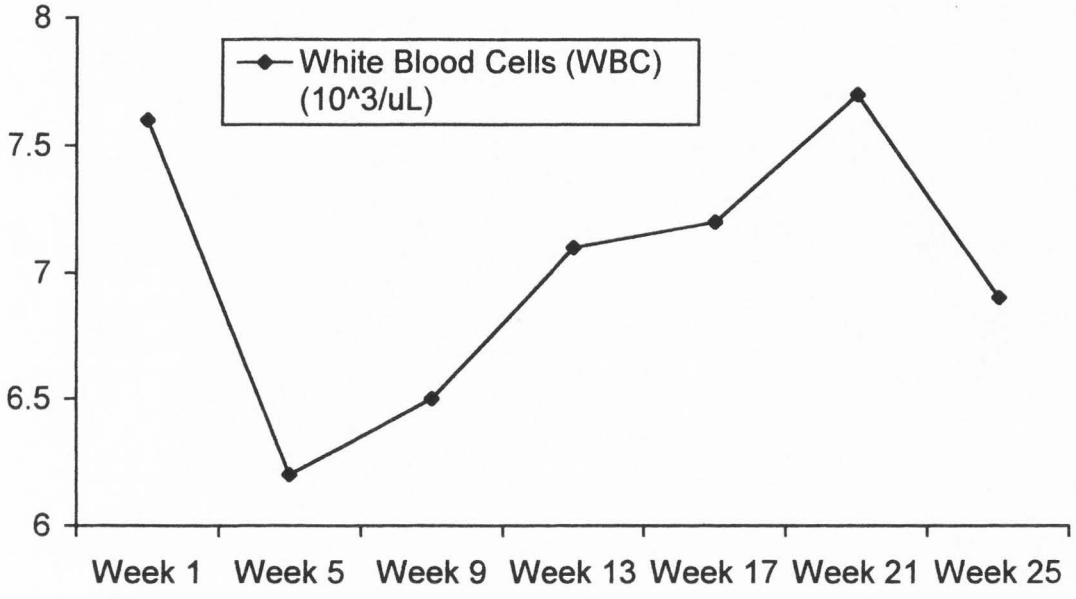

FIG. 18. The change in serum white blood cells (WBC) over the course of the diet.

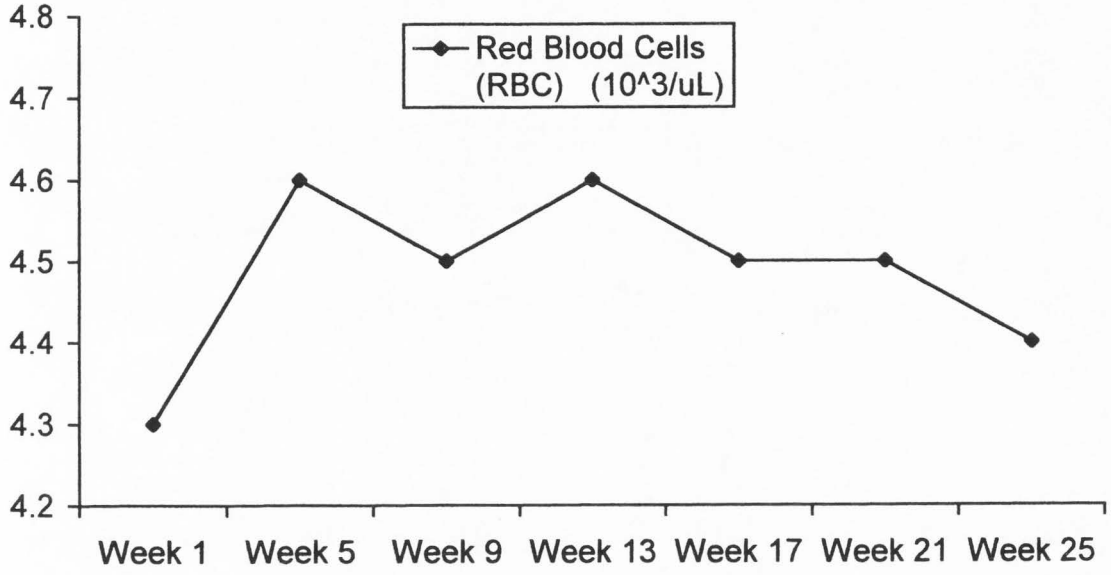

FIG. 19. The change in serum red blood cells (RBC) over the course of the diet.

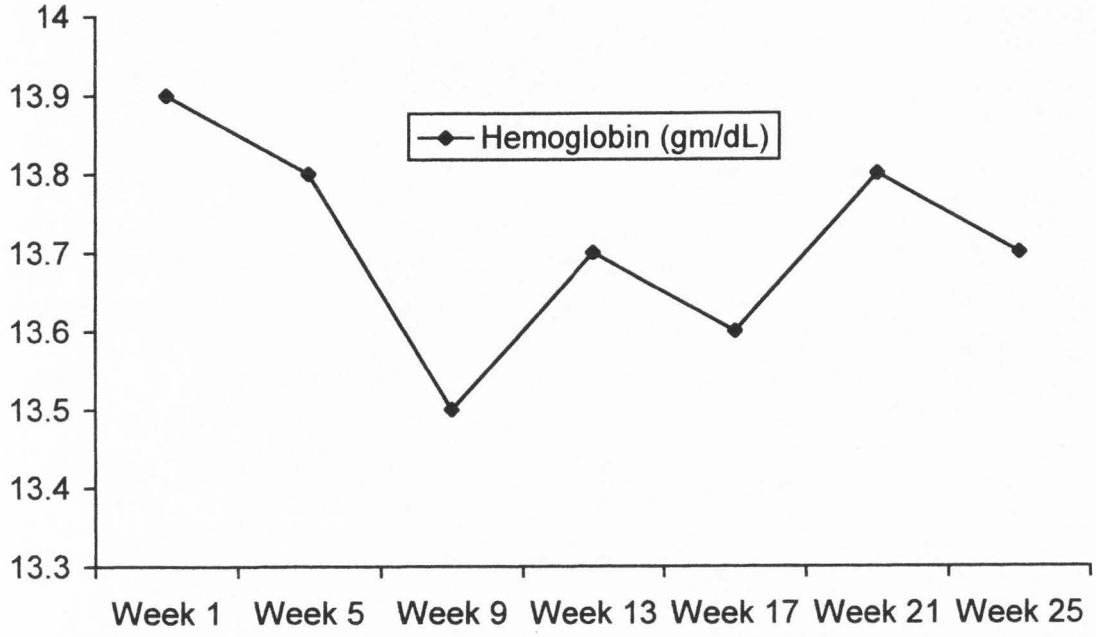

FIG. 20. The change in serum hemoglobin over the course of the diet.

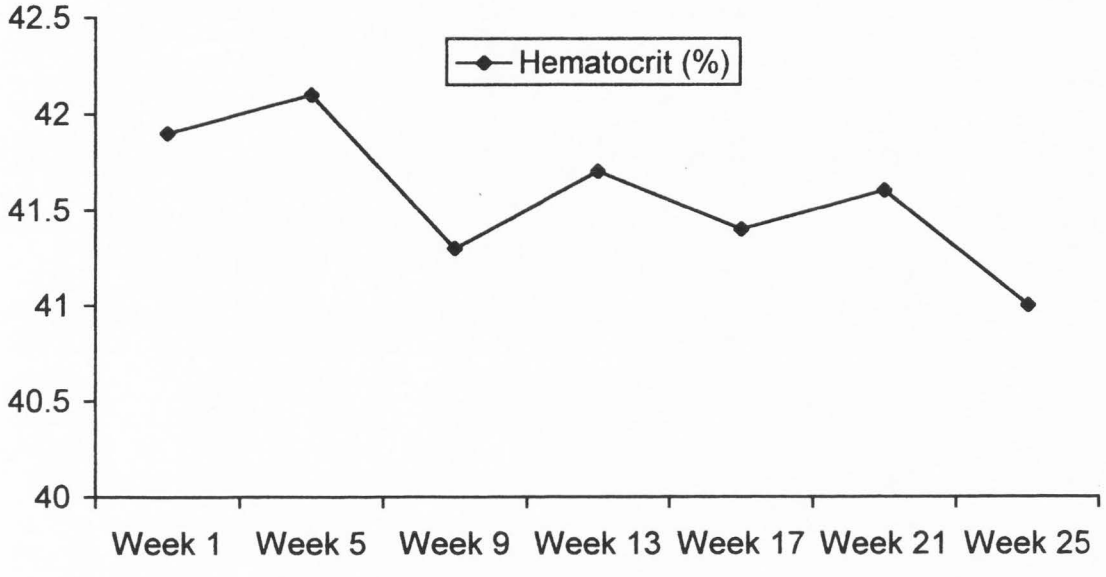

FIG. 21. The change in serum hematocrit over the course of the diet.

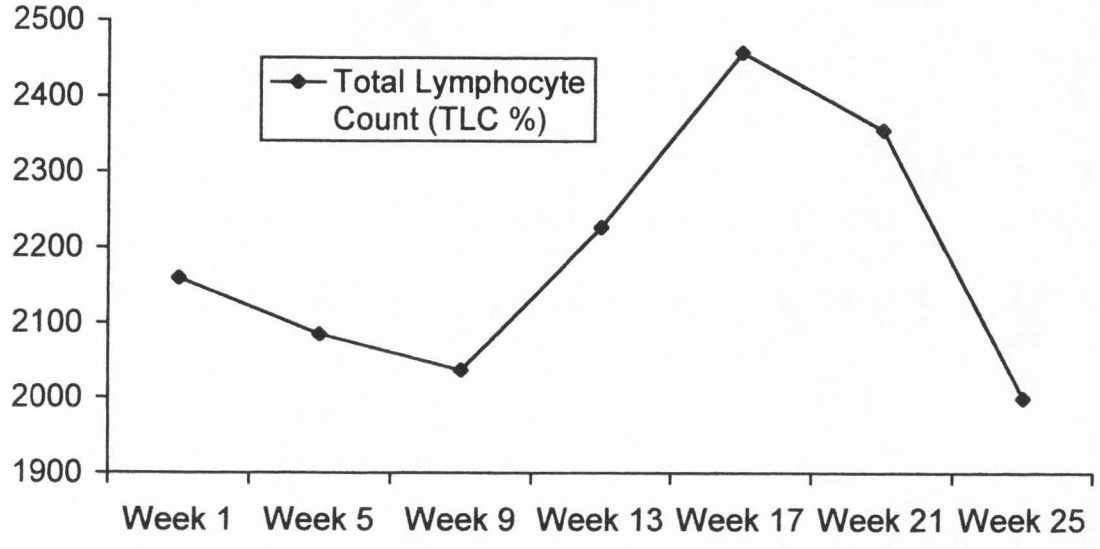

FIG. 22. The change in serum total lymphocyte count over the course of the diet.

Appendix E

SUBJECT DATA

Age	Ht	Weight	CBF	IBF	FBM	LBM	TBM	AREE	PREE	REE/Kg	RQ
A 54	160	109.698	45.4	36.3	39	70	49.6	1867	1724	17.4	.89
		95.227	37.7	36.6	35	60	49.3	1574	1625	16.0	.75
		85.227	32.6	32.4	27	57	51.8	1383	1512	16.0	.75
		80.545	33.9	31.1	26	55	52.2	1472	1472	18.0	.80
		77.681	28.2	30.8	24	53	52.8	1585	1446	21.0	.82
B 33	167	90.454	32.3	31.1	28	62	52.7	1607	1645	18.4	.87
		76.409	28.4	28.8	22	54	54.2	1386	1563	17.0	.74
		67.272	21.4	28.0	19	48	57.4	1269	1463	18.6	.72
		65.500	21.2	25.6	16	48	56.4	1380	1428	21.0	.81
		71.681	24.7	27.9	20	51	54.9	1420	1467	21.0	.81
C 60	164	99.227	37.4	35.6	35	64	49.7	1639	1614	16.7	.73
		86.818	37.2	35.5	30	56	49.9	1518	1514	17.0	.74
		79.636	27.5	32.9	26	56	49.9	1240	1436	15.6	.73
		77.227	26.8	31.8	25	53	52.3	1492	1401	20.0	.75
		77.272	26.8	31.9	25	53	52.2	1381	1414	18.0	.74
D 27	173	114.181	39.9	34.0	37	75	50.9	1822	1944	15.9	.73
		99.363	37.6	31.9	31	68	52.5	1727	1818	17.3	.70
		88.772	34.2	29.4	26	62	53.6	1653	1700	18.6	.77
		82.727	32.6	28.6	23	59	54.4	1532	1631	19.0	.73
		80.954	31.4	27.0	21	60	55.9	1584	1613	20.0	.83
E 48	165	75.909	32.7	30.8	23	53	52.9	1305	1462	17.2	.91
		68.272	28.2	29.2	20	48	54.2	1162	1388	17.0	.80
		65.000	26.2	28.5	19	46	54.2	1081	1349	17.0	.82
		64.500	24.8	27.3	17	46	55.3	1011	1332	16.0	.81
		62.272	25.8	35.5	22	40	49.9	1176	1323	19.0	.81
F 47	174	115.360	38.8	38.0	44	71	47.6	1854	1850	16.0	.75
		102.727	40.3	30.6	31	71	53.2	1660	1752	15.9	.72
		97.090	34.1	28.9	28	69	54.2	1638	1678	17.0	.71
		94.091	31.0	29.3	27	66	53.9	1683	1644	18.0	.73
		95.540	33.5	29.6	28	67	53.7	1941	1648	21.0	.93
G 41	160	112.682	48.0	38.2	44	70	48.0	1864	1833	16.6	.84
		103.181	45.9	37.4	38	65	48.6	1554	1750	15.0	.76
		98.636	43.5	36.1	35	63	49.6	1858	1707	19.0	.82
		96.818	42.3	35.8	35	62	49.7	1488	1690	15.0	.81
		96.818	42.2	35.8	35	62	49.7	1510	1690	16.0	.80
H 59	160	76.909	33.0	31.8	24	52	52.3	1331	1401	17.5	.84
		67.727	29.5	31.5	21	46	52.7	1280	1461	16.7	.87
		62.272	27.5	30.1	19	43	53.4	1145	1311	18.3	.94
		62.272	26.6	30.0	19	45	53.6	1340	1271	22.0	.91

NO DATA FOR WEEK 25

Age	Ht	Weight	CBF	IBF	FBM	LBM	TBM	AREE	PREE	REE/Kg	RQ
I 64	160	83.181	37.1	51.2	42	40	39.7	1552	1447	20.0	.93
		75.000	29.8	33.2	25	50	51.5	1113	1378	15.0	.72
		70.090	27.3	32.3	22	47	52.0	1268	1326	18.0	.68
		72.181	25.8	32.8	23	48	51.6	1360	1326	19.0	.74
		72.500	29.2	32.4	23	49	51.9	1376	1348	18.0	.67
J 35	163	99.864	45.0	53.1	53	48	38.6	1475	1748	15.0	.75
		87.863	35.1	33.9	30	58	51.0	1653	1647	19.0	.71
		77.954	29.6	30.1	23	54	53.3	1540	1540	21.0	.79
		72.909	29.6	29.0	20	52	54.3	1573	1488	22.0	.73
		74.545	28.5	30.1	22	52	53.6	1390	1509	20.0	.82
K 45	163	96.364	42.0	35.7	34	63	50.1	1650	1667	17.0	.76
		83.954	36.5	32.4	27	56	51.8	1256	1558	15.0	.76
		76.318	32.4	31.3	24	52	52.6	1161	1476	15.0	.83
		75.454	29.5	30.6	23	52	53.3	1236	1463	17.0	.82
		77.273	30.9	31.6	24	53	52.4	1371	1458	18.0	.98
L 57	155	130.454	45.2	42.6	55	75	45.4	2149	1936	16.0	.74
		116.363	41.9	41.5	48	68	46.0	1658	1840	14.0	.73
		113.409	37.3	39.1	44	69	47.7	1478	1749	14.0	.76
		108.409	38.5	39.3	42	65	47.3	1616	1710	15.0	.79
		108.636	38.6	39.0	42	66	47.4	1850	1749	17.0	.87
M 44	170	95.772	38.6	32.5	31	69	51.9	1433	1655	15.0	.76
		85.181	35.5	30.6	26	59	53.1	1505	1590	17.O	.75
		80.318	33.2	29.0	23	56	54.0	1306	1529	16.0	.77
		79.545	32.8	28.6	23	56	54.2	1365	1521	18.0	.86
		80.000	32.6	29.6	23	56	53.8	1222	1525	16.0	.73
N 28	172	95.591	36.8	32.5	30	65	52.1	1703	1717	18.0	.95
		82.909	32.5	29.9	25	58	53.5	1534	1619	18.0	.76
		76.454	27.5	28.0	21	55	54.8	1530	1532	21.0	.80
		68.636	23.8	25.3	17	51	56.4	1513	1472	22.0	.79
		69.090	24.7	26.0	18	51	56.2	1622	1476	23.0	.84
O 42	155	88.409	33.5	35.4	31	57	49.9	1446	1582	17.0	.91
		78.545	29.6	32.9	25	53	51.6	1334	1525	17.0	.78
		75.454	27.8	32.7	25	50	51.7	1512	1482	21.0	.82
		72.500	28.2	31.6	22	48	52.8	1209	1464	17.0	.77
		76.545	29.9	33.0	25	51	51.5	1376	1473	20.0	.82
P 28	165	102.090	39.4	33.6	34	67	51.1	1540	1803	16.0	.82
		91.318	34.4	31.4	29	62	52.4	1782	1699	20.0	.76
		84.727	30.2	29.8	24	58	53.5	1484	1612	18.0	.77
		82.272	30.2	29.8	24	58	53.5	1484	1612	18.0	.77
		84.818	30.6	30.9	25	59	53.1	1488	1634	18.0	.96
Q 32	164	106.409	45.6	36.9	30	76	00.0	1608	1815	15.0	.80
		94.636	38.2	35.0	33	61	50.2	1543	1715	16.0	.80
		85.727	31.4	33.0	28	57	51.5	1572	1632	18.0	.87
		80.227	30.5	30.9	25	55	53	1458	1580	19.0	.78
		82.909	32.1	31.5	25	57	52.6	1773	1597	21.0	.75

	Na	K	Cl	gluc	BUN	Creat	UA	TP	alb	Ca	P	Chol
A1	142	3.9	104	78	12	.9	5.7	7.0	4.3	10	3.6	234
A5	137	4.2	103	90	16	1.0	5.7	6.8	4.4	9.6	3.5	166
A9	137	3.9	104	85	13	.9	4.6	6.5	4.2	9.5	3.8	143
A13	140	4.2	105	76	16	.9	55	6.5	4.1	9.5	4.0	166
A17	no data											
A21	140	3.9	107	76	12	.9	5.2	6.5	4.3	9.2	4.1	195
A25	141	4.1	107	78	11	1.0	5.3	6.3	4.1	8.9	4.1	187
B1	140	4.1	105	66	10	.7	5.2	7.6	4.4	9.5	4.5	175
B5	139	4.1	105	72	14	.9	7.1	7.4	4.4	9.8	3.9	112
B9	139	3.9	105	68	13	.8	5.6	7.4	4.3	9.6	4.0	107
B13	140	4.1	107	61	13	.7	5.5	7.6	4.5	9.8	4.3	109
B17	138	4.2	106	71	15	.8	4.3	7.3	4.4	9.7	4.5	148
B21	139	4.3	105	69	12	.9	4.7	7.2	4.5	9.4	4.4	144
B25	141	4.2	116	85	12	.8	5.7	7.1	4.1	8.8	4.2	164
C1	141	4.0	106	75	19	.9	6.1	7.1	4.2	9.2	4.2	231
C5	139	3.9	106	91	20	1.0	5.8	6.8	4.3	9.6	4.3	229
C9	140	4.0	107	90	20	.9	5.2	6.5	4.1	9.6	4.3	171
C13	142	4.3	109	85	26	.9	5.6	7.1	4.4	9.8	4.6	195
C17	142	4.3	110	95	21	.9	5.8	6.7	4.1	9.3	4.3	218
C21	139	4.3	106	99	18	1.0	5.9	6.7	4.0	9.2	4.3	224
C25	141	4.5	108	85	17	.8	5.8	6.7	4.1	9.1	4.1	235
D1	143	4.2	110	77	10	1.0	7.7	7.6	4.7	9.7	3.6	245
D5	139	3.9	107	64	13	1.0	9.6	7.0	4.6	9.6	2.9	158
D9	140	4.0	109	72	12	.9	7.0	7.2	4.7	9.6	3.1	161
D13	138	4.1	109	90	18	1.0	6.7	6.9	4.6	9.7	3.1	178
D17	139	4.1	110	87	9	.9	6.0	6.7	4.5	9.8	3.1	213
D21	139	4.0	107	116	9	.9	6.8	7.0	4.7	9.7	3.4	209
D25	139	4.4	108	91	11	1.0	5.6	6.7	4.5	9.5	2.8	188
E1	137	4.2	104	68	13	1.2	7.1	6.5	4.0	9.6	2.8	178
E5	139	4.2	107	78	22	1.1	5.4	6.8	4.3	10.3	3.3	129
E9	136	4.5	106	70	18	1.2	5.3	6.7	4.4	10.3	2.7	137
E13	137	4.6	108	74	20	1.1	5.3	6.9	4.4	10.4	3.2	137
E17	137	4.7	107	77	20	1.2	6.3	6.7	4.4	10.3	2.7	117
E21	139	4.6	108	83	21	1.3	6.0	7.0	4.5	10.6	3.4	123
E25	140	4.3	105	74	13	1.4	6.1	7.3	3.8	10.1	2.0	142
F1	138	3.7	103	97	12	.6	4.7	6.9	4.1	8.9	3.0	200
F5	138	4.6	105	78	14	.7	4.0	7.4	4.4	9.9	3.9	177
F9	141	4.6	106	82	14	.8	3.4	7.2	4.3	10.0	4.2	179
F13	138	4.6	107	85	14	.7	3.7	7.1	4.2	9.7	3.9	186
F17	139	4.4	106	82	20	.3	4.7	7.4	4.4	9.7	4.3	197
F21	141	4.4	105	87	20	.8	4.1	7.7	4.6	10.0	4.6	187
F25	141	4.4	103	87	16	1.0	4.0	7.7	3.4	9.1	3.5	185
G1	140	4.0	107	96	13	.8	4.3	7.2	4.6	9.1	2.8	182
G5	139	4.3	106	75	14	.8	4.6	6.8	4.5	9.8	3.6	149
G9	143	4.4	110	78	11	.8	3.5	7.1	4.7	9.7	3.3	169
G13	140	4.4	108	75	9	.7	3.9	6.9	4.6	9.6	3.2	170
G17	141	4.4	111	73	10	.8	3.9	7.0	4.7	9.8	3.2	177

	Na	K	Cl	gluc	BUN	Creat	UA	TP	alb	Ca	P	Chol
G21	no data											
G25	138	4.2	101	68	11	1.0	3.5	7.4	4.2	8.9	3.0	186
H1	142	4.3	107	92	17	1.2	1.0	7.3	4.4	9.1	3.2	324
H5	139	4.3	103	83	24	1.2	5.7	6.9	4.2	9.6	3.4	185
H9	142	4.1	107	92	30	1.1	4.9	6.8	4.2	9.6	4.5	194
H13	140	4.4	108	89	24	.9	5.0	6.4	3.9	8.9	4.4	211
H17	138	4.5	106	90	29	.9	5.6	6.2	3.7	8.8	3.7	221
H21	137	4.4	105	87	24	1.0	5.2	6.7	4.0	9.3	4.0	247
H25	no data											
I1	143	3.4	111	75	15	1.1	4.7	6.6	4.1	8.7	2.1	223
I5	141	4.0	107	80	20	1.2	5.5	7.4	4.6	9.8	2.7	191
I9	140	4.7	107	82	22	1.2	4.6	6.9	4.4	9.8	2.6	194
I13	140	4.4	108	79	20	1.2	4.3	6.7	4.2	9.4	2.3	220
I17	140	4.2	105	74	16	1.2	5.2	6.8	4.3	9.7	2.1	242
I21	141	4.1	102	61	23	1.4	5.1	7.8	3.7	9.6	2.6	252
I25	142	3.9	106	95	19	1.3	4.5	7.0	3.3	8.2	2.3	211
J1	138	3.9	106	84	14	.6	4.3	7.2	4.3	8.8	3.5	215
J5	143	3.8	109	70	11	.9	6.0	6.6	4.3	9.3	3.9	155
J9	140	3.7	108	74	13	.8	5.1	6.6	4.2	9.4	3.7	151
J13	139	4.0	106	78	14	.6	5.1	6.6	4.2	9.5	3.8	155
J17	137	3.7	109	78	11	.8	4.8	6.3	4.2	9.6	3.2	170
J21	141	3.3	103	74	9	1.0	4.8	7.1	3.7	9.5	3.3	187
J25	141	3.9	104	74	11	.9	4.2	7.0	3.7	9.0	3.5	189
K1	143	3.8	109	107	12	.9	6.5	7.3	4.6	10.1	4.0	225
K5	139	3.8	103	80	16	1.0	7.1	7.8	4.8	10.3	4.1	157
K9	139	4.1	104	83	20	.8	5.2	7.3	4.7	10.5	4.6	162
K13	143	3.9	108	79	12	.8	4.7	6.7	4.4	10.2	4.2	169
K17	143	4.1	102	80	14	1.0	5.2	7.6	4.1	10.4	4.2	229
K21	no data											
K25	146	4.6	106	91	12	1.0	5.4	7.2	3.6	9.5	3.4	203
L1	141	4.3	105	281	9	.7	6.3	6.9	4.0	9.1	3.7	225
L5	140	4.2	104	99	13	.8	9.1	7.3	4.4	9.9	4.0	190
L9	140	3.9	104	85	12	.8	8.7	7.2	4.4	9.4	3.7	220
L13	142	4.0	108	87	21	.9	10.0	7.0	4.2	9.8	4.3	204
L17	143	4.3	101	74	19	1.0	8.1	7.8	3.8	9.0	4.1	204
L21	no data											
L25	143	4.0	103	118	15	.9	6.4	7.5	3.4	9.1	3.8	229
M1	136	4.5	104	89	13	1.3	4.8	7.0	3.9	8.7	3.2	191
M5	139	4.2	105	75	13	1.1	5.1	7.7	4.7	9.9	3.5	156
M9	139	4.2	106	70	13	1.1	5.0	7.1	4.5	9.7	3.4	170
M13	no data											
M17	139	3.9	103	67	17	1.0	4.9	7.1	4.4	9.4	3.3	190
M21	139	4.4	100	69	19	1.2	4.8	8.4	4.3	9.9	3.4	194
M25	138	4.1	103	84	14	1.1	4.1	7.2	3.7	9.0	3.4	189
N1	141	4.6	110	81	10	.7	3.5	6.9	4.4	9.2	3.1	163

	Na	K	Cl	gluc	BUN	Creat	UA	TP	alb	Ca	P	Chol
N5	137	4.2	107	81	12	.9	4.5	6.6	4.3	9.5	3.5	108
N9	138	4.2	106	69	9	.8	4.1	6.5	4.3	9.5	3.9	109
N13	139	4.0	107	73	9	.8	3.5	6.5	4.5	9.8	3.9	123
N17	141	4.1	102	65	11	.8	2.5	7.4	4.3	9.7	3.7	139
N21	no data											
N25	139	3.9	102	81	9	.9	3.4	7.2	3.9	9.1	4.0	148
O1	140	3.9	111	78	8	.9	4.7	6.9	4.2	9.1	3.6	242
O5	135	4.5	106	88	19	1.0	5.3	7.3	4.5	9.6	3.2	155
O9	138	4.2	107	93	19	.9	4.1	7.0	4.4	9.5	4.5	202
O13	138	4.2	105	84	15	1.0	6.3	7.3	4.8	9.9	3.9	195
O17	139	4.0	101	88	12	1.0	4.0	8.2	4.2	9.6	4.0	220
O21	no data											
O25	139	4.6	108	68	13	1.1	4.4	7.5	3.8	8.6	3.7	206
P1	142	3.8	110	88	8	.9	6.5	7.1	4.4	9.2	3.4	143
P5	140	4.6	105	79	16	.9	7.6	7.2	4.5	10.0	3.7	102
P9	142	4.5	107	78	17	.8	5.1	6.9	4.4	9.7	4.0	126
P13	141	4.4	105	92	13	1.0	6.7	7.5	4.6	10.1	4.6	131
P17	144	3.8	102	85	13	1.1	5.3	7.6	3.9	8.9	4.3	113
P21	143	3.8	108	78	14	1.1	4.3	7.6	3.8	8.8	4.1	113
P25	143	4.5	106	84	12	.9	4.4	7.2	3.7	8.7	4.0	110
Q1	141	4.7	109	93	12	.9	5.2	6.3	3.8	8.9	3.7	175
Q5	136	4.0	104	85	11	.9	5.5	6.4	4.0	9.4	3.6	156
Q9	138	3.7	106	79	9	.8	4.8	6.4	4.1	9.2	3.6	149
Q13	138	4.1	108	85	8	.9	5.0	6.0	3.8	8.8	3.9	140
Q17	139	4.0	100	81	18	1.0	3.7	7.0	3.5	9.0	3.5	169
Q21	139	3.9	102	89	12	1.0	4.2	7.0	3.5	8.9	4.2	174
Q25	no data											

	WBC	RBC	Hgb	Hct	TLC
A1	8.6	4.6	13.9	42	1522
A5	6.2	4.6	13.9	42	1457
A9	5.3	4.2	12.9	40	1071
A13	4.8	4.4	13.3	41	1344
A17	5.9	4.2	13.1	59	1381
A21	5.9	4.2	13.1	40	1381
A25	5.1	4.4	14.1	42	995
B1	10.5	4.8	14.7	43	3171
B5	8.0	4.5	13.7	41	2704
B9	7.6	4.5	13.4	40	2592
B13	7.8	4.6	13.8	42	2449
B17	9.0	4.5	13.3	41	3100
B21	8.8	4.6	13.8	42	2693
B25	11	4.2	12.8	38	3025
C1	8.0	4.7	13.8	41	2688
C5	6.5	4.5	13.2	40	2305
C9	6.2	4.4	12.6	39	2151

	WBC	RBC	Hgb	Hct	TLC
C13	6.3	4.6	13.2	41	2388
C17	8.1	4.5	13.2	40	2680
C21	8.2	4.4	12.5	38	2640
C25	6.9	4.5	13.4	39	1925
D1	8.3	5.1	14.4	43	2473
D5	6.4	5.1	13.9	42	2086
D9	6.6	5.2	14.1	44	1940
D13	8.9	5.2	14.3	44	2198
D17	7.4	4.9	13.7	43	2494
D21	7.3	4.9	14.3	43	2175
D25	6.8	4.8	14.5	43	1584
E1	9.0	4.3	13.3	41	1782
E5	7.6	4.3	13.6	41	2265
E9	8.6	4.3	13.3	41	2167
E13	8.9	4.7	14.1	44	2456
E17	6.9	4.3	13.4	41	2063
E21	no data				
E25	7.9	4.9	15.4	46	2054
F1	7.3	4.3	12.7	39	2679
F5	5.9	4.2	12.5	38	2572
F9	6.0	4.2	12.1	38	2628
F13	7.0	4.1	11.8	37	2541
F17	5.1	4.1	12.1	37	2621
F21	6.3	4.4	13.2	40	2848
F25	4.8	4.1	12.3	36	2140
G1	7.4	4.9	14.7	44	2042
G5	6.3	4.8	14.2	43	1852
G9	6.1	4.9	14.4	44	1537
G13	7.7	4.9	14.4	44	2202
G17	no data				
G21	no data				
G25	5.8	4.9	14.4	43	1833
H1	6.9	4.8	14.5	43	1987
H5	6.1	4.6	13.7	42	2220
H9	5.7	4.5	13.4	41	1870
H13	6.8	4.3	12.8	39	2264
H17	7.0	4.4	13.1	40	2142
H21	7.7	4.9	14.3	43	2349
H25	no data				
I1	6.0	4.4	13.0	39	2268
I5	5.7	4.8	14.2	43	2234
I9	5.2	4.6	13.6	42	2210
I13	5.6	4.5	13.5	41	2442
I17	6.6	4.5	13.8	42	2653
I21	7.7	4.6	14.4	43	2333
I25	6.0	4.1	12.7	38	2016

	WBC	RBC	Hgb	Hct	TLC
C13	6.3	4.6	13.2	41	2388
C17	8.1	4.5	13.2	40	2680
C21	8.2	4.4	12.5	38	2640
C25	6.9	4.5	13.4	39	1925
D1	8.3	5.1	14.4	43	2473
D5	6.4	5.1	13.9	42	2086
D9	6.6	5.2	14.1	44	1940
D13	8.9	5.2	14.3	44	2198
D17	7.4	4.9	13.7	43	2494
D21	7.3	4.9	14.3	43	2175
D25	6.8	4.8	14.5	43	1584
E1	9.0	4.3	13.3	41	1782
E5	7.6	4.3	13.6	41	2265
E9	8.6	4.3	13.3	41	2167
E13	8.9	4.7	14.1	44	2456
E17	6.9	4.3	13.4	41	2063
E21	no data				
E25	7.9	4.9	15.4	46	2054
F1	7.3	4.3	12.7	39	2679
F5	5.9	4.2	12.5	38	2572
F9	6.0	4.2	12.1	38	2628
F13	7.0	4.1	11.8	37	2541
F17	5.1	4.1	12.1	37	2621
F21	6.3	4.4	13.2	40	2848
F25	4.8	4.1	12.3	36	2140
G1	7.4	4.9	14.7	44	2042
G5	6.3	4.8	14.2	43	1852
G9	6.1	4.9	14.4	44	1537
G13	7.7	4.9	14.4	44	2202
G17	no data				
G21	no data				
G25	5.8	4.9	14.4	43	1833
H1	6.9	4.8	14.5	43	1987
H5	6.1	4.6	13.7	42	2220
H9	5.7	4.5	13.4	41	1870
H13	6.8	4.3	12.8	39	2264
H17	7.0	4.4	13.1	40	2142
H21	7.7	4.9	14.3	43	2349
H25	no data				
I1	6.0	4.4	13.0	39	2268
I5	5.7	4.8	14.2	43	2234
I9	5.2	4.6	13.6	42	2210
I13	5.6	4.5	13.5	41	2442
I17	6.6	4.5	13.8	42	2653
I21	7.7	4.6	14.4	43	2333
I25	6.0	4.1	12.7	38	2016

	WBC	RBC	Hgb	Hct	TLC
P17	8.4	5.1	15.4	46	2814
P21	8.8	5.1	15.4	46	2939
P25	8.0	4.9	14.5	43	2216
Q1	6.5	4.4	13.0	40	1970
Q5	4.7	4.5	13.2	41	1673
Q9	5.8	4.4	13.2	39	1380
Q13	4.5	4.7	13.5	41	1735
Q17	6.2	4.4	13.3	40	1953
Q21	7.4	4.5	13.4	40	2220
Q25	6.2	4.2	12.3	38	1476

	CHOL	TG	HDL	VLDL	LDL
A1	220	138	84	28	108
A25	196	117	82	23	91
B1	147	60	54	12	81
B25	170	65	46	13	111
C1	209	148	65	30	114
C25	245	127	73	25	147
D1	213	116	68	23	122
D25	212	91	58	18	36
E1	164	219	49	44	71
E25	131	64	36	13	88
F1	183	85	70	17	96
F25	182	69	75	14	93
G1	170	121	49	24	97
G25	181	122	46	24	117
H1	310	245	63	49	212
H25	/	/	/	/	/
I1	244	175	63	35	125
I25	208	122	63	24	121
J1	232	71	71	14	147
J25	189	74	68	15	106
K1	240	235	58	47	135
K25	206	98	69	20	117
L1	225	199	34	40	151
L25	218	159	42	32	144
M1	191	158	40	/	/
M25	195	129	48	26	121
N1	176	92	46	18	99

	CHOL	TG	HDL	VLDL	LDL
N25	163	44	62	9	92
O1	249	143	36	29	/
O25	198	97	47	19	132
P1	145	69	36	14	93
P25	104	39	31	8	5
Q1	181	147	32	29	/
Q25	25	100	34	20	130

Appendix F

Abbreviations

Age - years
Height- centimeters
Body Mass - kilograms
CBF/Circumference Body Fat - %
IBF/Infra-red Body Fat - %
FBM/Fat Body Mass - kilograms
LBM/Lean Body Mass - kilograms
TBW/Total Body Water - %
AREE/Actual Resting Energy Expenditure - kcal
PREE/Predicted Resting Energy Expenditure - kcal
REE/Resting Energy Expenditure - kcal
RQ/Respiratory Quotient
VLCD - very-low-calorie-diet
CBC - complete blood count
SMA-12 - serum chemistry panel

Appendix G

Normal Laboratory Values

Sodium - Na (mmol)	135-146
Potassium - K (mmol/L)	3.5-5.1
Cholride - Cl (mmol/L)	100-112
Glucose (mg/dL)	70-115
BUN (mg/dL)	6-20
Creatinine (mg/dL)	.5-1.6
Total Protein (gm/dL)	6.2-8.0
Albumin (gm/dL)	3.4-5.0
Calcium (mg/dL)	8.5-10.6
Phosphorus (mg/dL)	2.4-4.5
Cholesterol (mg/dL)	0-200
Uric Acid (mg/dL)	2.4-7.5
White Blood Cells (10^3/uL)	5.0-10.0
Red Blood Cells (10^3/uL)	4.5-6.2
Hemoglobin (g/dL)	14-18
Hemotocrit (%)	43-56
Total Lymphocyte Count (%)	20-40