

THE EFFECT OF A COMPREHENSIVE WEIGHT REDUCTION PROGRAM
ON BLOOD VARIABLES, BODY COMPOSITION, AND
NUTRIENT INTAKE IN OBESE BLACK WOMEN

by

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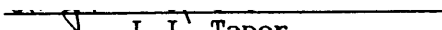
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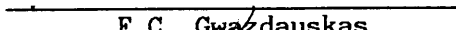
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(ABSTRACT)

The effect of comprehensive weight reduction program on selected blood parameters, body composition, and nutrient intake was investigated in 56 obese black women, 22 to 51 years old. Subjects attended nutrition and behavior modification classes once per week for 3 months. Twenty subjects participated in a low impact aerobics programs 3 times per week for 3 months. A 3.8 kg average weight loss was observed. A 2.6% significant reduction in body fat (BF) was observed in the exercise group. Total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-C) fell significantly from 198 to 175 mg/dL, and 130 to 109 mg/dL, respectively, in the exercise group. High density lipoprotein₂-cholesterol (HDL₂-C) was lowered significantly from 24 to 17 mg/dL in the non-exercise group. Triglycerides, HDL-C, TC/HDL-C ratio, glucose, thyroxin, and insulin values were not significantly altered. Significant reductions in protein, fat, and cholesterol intake were reported. A comprehensive weight loss treatment promoted gradual weight loss, changes in body composition and food intake patterns, and significantly altered some blood lipid levels in obese black females.

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INTRODUCTION

The American population spends millions of dollars a year on diet and nutritional paraphernalia in an attempt to lose weight, and gain a more healthy, physically fit, and acceptable body in our thin-minded society. Despite the present concern for better health and slimness, obesity remains a prevalent and serious problem in the United States. According to data collected during the National Health and Nutrition Examination Survey II (NHANES II, 1976-80) an estimated 28% of the adult population 25 to 74 years of age was obese, generally defined as a body weight exceeding 20% desirable body weight and a body mass index (BMI) above the 85th percentile (U.S. Department of Health and Human Services and Agriculture, 1986). In addition, the prevalence of obesity was higher among women (29.6%) than men (26.3%), occurred more frequently among black women (60%) than white women (30-36%) 45 years and older (Figure 1), and was more common among women below poverty level (Figure 2).

In general, obese individuals are assumed to have energy intakes which exceed their energy needs. The obese individual is often believed to be either hyperphagic, sedentary, or possess both characteristics. Presently, there is no clear relationship that exists between an individuals' kcal intake and degree of adiposity (Rolland-Cachera and Bellisle, 1986). An investigation of the differences of kcal intakes among black and white females indicated that the median kcal intake for the black female population was similar to or actually slightly below that of age-matched white females (Abraham et al, 1980),

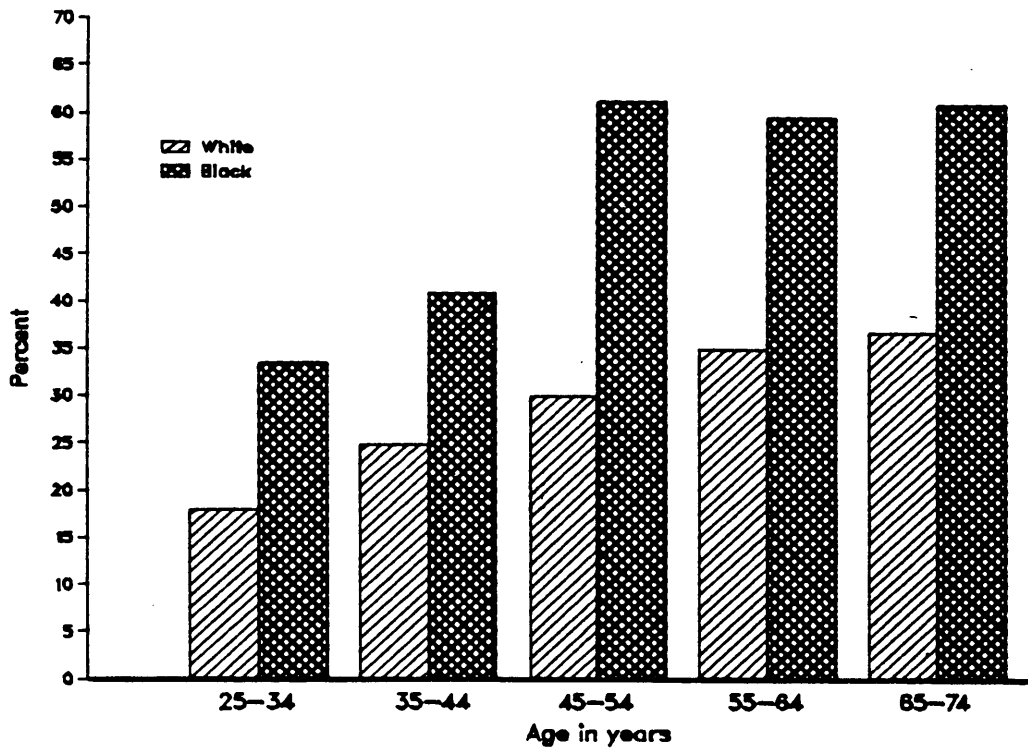


Figure 1. Percent of females overweight, by race and age: 1976-80.

From: U.S. Department of Health and Human Services and Agriculture. Nutrition Monitoring in the United States, Washington, DC: U.S. Government Printing Office, 1986. (DHHS publ no [PHS] 86-1255).

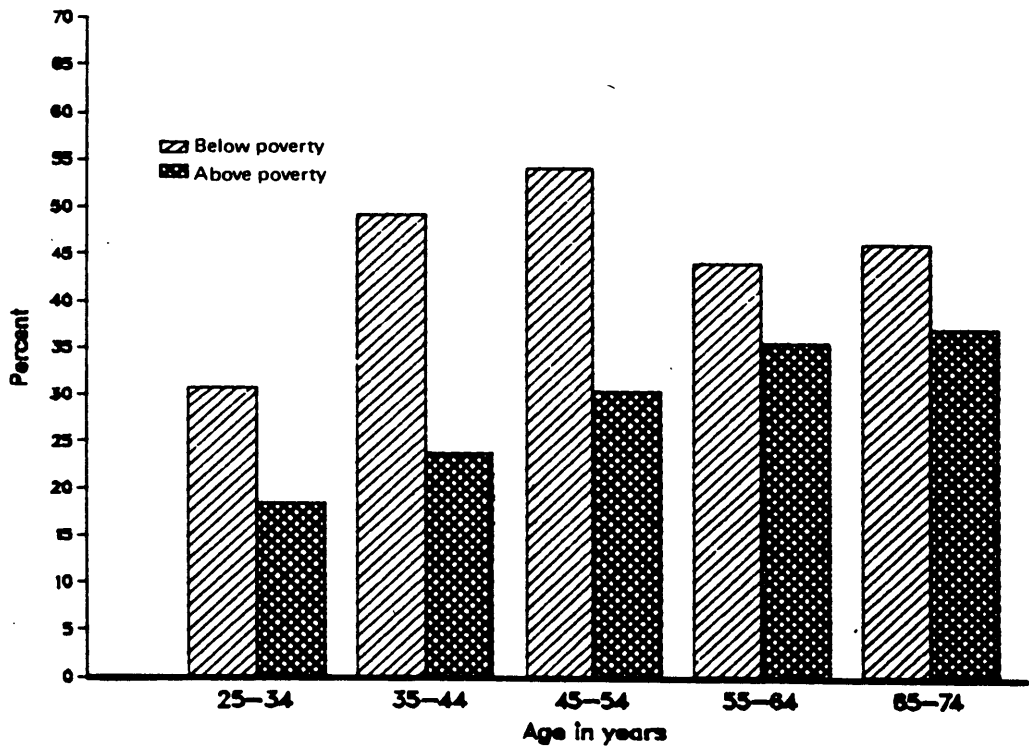


Figure 2. Percent of females overweight, by poverty status and age: 1976-80.

Note: Data based on nonpregnant females only.

From: U.S. Department of Health and Human Services and Agriculture. Nutrition Monitoring in the United States, Washington, DC: U.S. Government Printing Office, 1986. (DHHS publ no [phs] 86-12555).

despite the higher prevalence of obesity within the black population. In addition to excess kcal intake and low physical activity, metabolic and endocrine abnormalities may play a role in the development of obesity.

According to the National Institutes of Health Consensus Development Panel, 1985, overwhelming evidence exists that obesity unfavorably affects health and longevity. Epidemiological studies indicate that the incidence of hypertension, hypercholesterolemia, non-insulin dependent diabetes mellitus (NIDDM), and certain cancers are higher among obese than non-obese individuals (U. S. Department of Health and Human Services and Agriculture, 1986). The high incidence of hypertension and hypercholesterolemia play primary roles in the development of cardiovascular disease (CVD), which in addition to cancer and diabetes mellitus, constitute several of the leading causes of death in the United States (Van Itallie, 1980, Gotto, 1986).

The importance of a method to control and treat the obese condition is evident, yet thus far, a successful program for weight loss has not been identified. Most treatments for weight loss have resulted in minimal weight reduction, usually averaging no more than 5.4 kg (Wing and Jeffery, 1979). A recent report by The Panel on Energy, Obesity, and Body Weight Standards (Jegquier et al, 1987) suggested that a multifactorial approach toward the treatment of obesity, including diet, exercise, behavior modification, and nutrition education, may be important in improving diet and treatment adherence and aiding maintenance of weight loss, in addition to promoting weight loss through a chronic kcal deficit. Weinsier et al (1984) previously

discussed the importance of an integrated and multifactorial approach toward weight loss. According to these investigators, diet, exercise, and behavior modification were closely interrelated and interdependent, and when prescribed together were likely to increase the success of long term weight control.

Most studies to date have focused on one or two of the treatment components (diet, exercise, behavior modification, nutrition education) when investigating weight loss in obese individuals. In addition, all investigations have been conducted using a white population, and no research inquiring into weight loss treatment of obese black individuals has been performed. In particular, the black female is at a high risk for developing obesity, and the increased morbidity and mortality associated with the obese condition clearly indicates and stresses the tremendous need for the development of a successful weight loss treatment for this population. The present study was designed to determine the effect of a three month integrated and multifactorial approach on weight loss in adult obese black females. The effect of weight loss with or without exercise on blood lipid and lipoprotein levels, plasma glucose, and serum insulin and thyroxin levels was investigated. The bioimpedance analysis (BIA) method was used to evaluate changes in body composition during and immediately following treatment.

REVIEW OF LITERATURE

Etiology

Bone, muscle, fat and water comprise the total body weight of an individual, and weight loss or gain may be due to variation in any one or a combination of the components (Abraham and Johnson, 1980). Obesity is characterized by overfatness, or an accumulation of fat in excess of that necessary for basic adipose tissue function in the body (Dillon, 1980). Weight control and prevention of obesity focuses on overweight due to fat, which is normally thought to result from excess kcal intake relative to energy expenditure (Abraham and Johnson, 1980).

Although an energy imbalance is generally regarded as the basic underlying cause of obesity, the question of why some individuals are in such a state of imbalance and others are not remains unanswered. Theories include environmental, behavioral, genetic, metabolic, endocrinologic, and nutritional factors which may or may not influence the incidence and cause of obesity in humans (Garn, 1976). For the most part, obesity appears to be a multifaceted disorder which affects a heterogeneous population.

Despite the general belief that obese individuals consume excessive kcal, the actual relationship between kcal intake and degree of adiposity has not been clearly established (Rolland-Cachera and Bellisle, 1986). Most available data indicate that similar levels of energy intake exists between obese and nonobese persons (McCarty, 1966; Bingham et al, 1981). This has been observed when the kcal intake

between black and white adult female populations was compared. Although a higher incidence of obesity was observed in the black female population, no significant difference in energy intake was evident between the two groups (Bray, 1979). In addition, incidence of excessive kcal intake was present in some individuals in both lean and obese groups (Garrow, 1974).

Rolland-Cachera and Bellisle (1986) formed an interesting hypothesis concerning the relationship between food intake and adiposity. In their study of 2,440 children, no relationship in kcal intake was found between different corpulence classes, yet when grouped according to socioeconomic level, a higher prevalence of obesity and fatness was found among children of lower social class, where energy intake was higher. Thus, although an individual relationship was not present, a significant relationship between obesity and energy intake was present when different populations with different eating practices were observed. The authors suggested "that at a given level of culturally or socially determined caloric intake, obesity will develop in predisposed individuals who do not eat differently than normal weight people of the same social group." Because food intakes of certain black populations were different than white populations (Sempos et al, 1987), this may be a feasible explanation for the high incidence of obesity in this population. In fact, a higher percentage of adult obese black females were in the population below poverty level than above poverty level (Bray, 1979). Whether or not this was attributable to differences in energy intake or food patterns between social groups was not discussed and the relationship between race and obesity has, thus far, not been identified.

Obesity was observed more often in populations with a low activity level (Bray, 1979). Obviously, a sedentary lifestyle was conducive to the development of obesity due to the difficulty in balancing energy intake to output. Obese individuals have been observed to be extremely economical with their energy expenditure: ie. "They rarely use the stairwells if they can wait for an elevator" (Dillon, 1980). Because an individuals' kcal intake and activity patterns are highly susceptible to environmental influences (Bray, 1979), it becomes apparent that lifestyle may play a large role in the development of obesity.

In addition to external factors, several internal factors may be related to excessive weight gain. Because of the role of thyroid hormones in metabolic regulation (Ryan et al, 1975), impaired thyroid function was often believed to be responsible for an individuals' obesity. However, an actual hypothyroid condition was present in only one to two percent of the population (Rimm et al, 1975), and therefore obesity cannot be explained solely on the basis of endocrine dysfunction (Armstrong et al, 1950).

Investigations of the metabolic efficiency of obese individuals have yielded conflicting results. In a study by Jung et al (1979), decreased thermogenesis was reported in six obese and post obese female subjects when compared to lean subjects. The obese and post obese subjects were from families with a history of obesity. These investigators studied the change in resting metabolic rate (RMR) associated with infusion of noradrenaline. Although a similar increase in plasma noradrenaline levels was observed in all subjects, the RMR of the obese and post obese subjects increased only one-half that reported in the

lean subjects. Thus, this study indicated that abnormal thermogenesis may underly the imbalance present in obese and previously obese persons. In contrast to this latter finding, it has been reported that obese patients generally have higher metabolic rates, although wide variation exist at any degree of obesity (Garrow, 1980).

Presently, the existence of a genetic factor which is responsible for obesity in some individuals is unknown. Although obesity is more prevalent among individuals with a family history, this may be due to the adoption of family exercise and eating habits, rather than a genetic factor (Dillon, 1980, Bray, 1979). The observation that adopted children of obese parents often follow the same pattern of obesity indicated the role of lifestyle in the development of obesity (Garn et al, 1976). Yet Jung et al (1979), reported that a genetic thermogenic abnormality may be implicated. Thus, although the study of obesity has continued for decades, the actual physiological, environmental and/or psychological factors involved in the development of excessive accumulation of adipose tissue in certain individuals remain elusive.

Health Implications

The increased risk of accelerated mortality associated with obesity has been recognized for some time (Armstrong et al, 1950; Van Itallie, 1980; Gordon and Kannel, 1973; Keys et al, 1972; Garrow, 1980). The relative increase in mortality rate with respect to increased levels of ideal body weight is demonstrated in Figure 3 (Van

Itallie, 1980). When discussing increased body weight, it is important to associate the excess weight to adipose tissue. The body mass index ($BMI = kg/m^2$) is often used as an indirect measure of adiposity (Abraham and Johnson, 1980; Keys et al 1972), because of its high correlation with the independent measures of body fat (Sims, 1979; Jackson et al, 1986; Keys et al, 1972). A BMI over 25 indicates a percent body fat over 35%, and is associated with a decreased life expectancy (Garrow, 1980). A BMI of 35 is associated with a mortality ratio of approximately 200% (Garrow, 1980).

The metabolic profile of obesity is predictable and the high incidence of hyperinsulinemia, impaired glucose tolerance, hypertension, and hyperlipidemia have been well documented (Armstrong et al, 1950; Gibson et al, 1975; Pyorala et al, 1985; Moak et al, 1987; Ashley and Kannel, 1974; Simopoulos, 1985). (Figure 4). These features increased the susceptibility to the development of certain disease states which contributed to the premature death observed in obese individuals. Obesity is believed to be a primary contributing factor in the development of adult-onset diabetes, hypertension, and arthritis, and is recognized as being associated with cardiovascular disease (Rimm and White, 1979). The increased mortality from disease resulting from increasing body weight and assuming increased adiposity is shown in Figure 5.

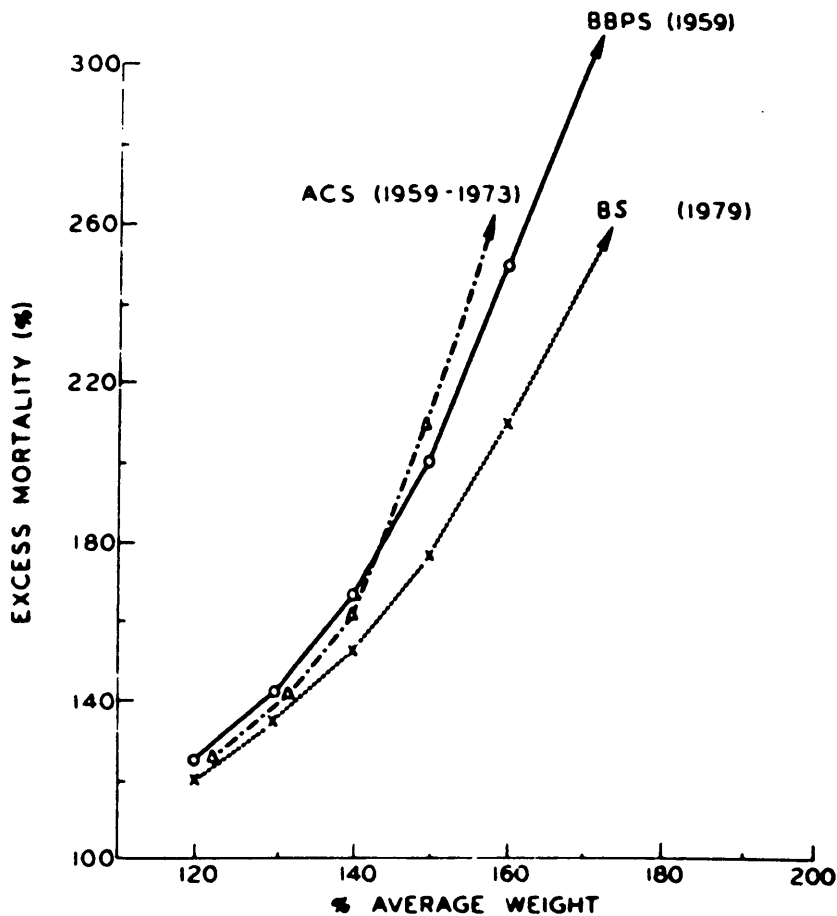


Figure 3. Acceleration of excess mortality among men and women as their overweight becomes increasingly severe. The segments of the lines beyond 140% of average weight in the case of ACS study and beyond 160% in the Build and Blood Pressure Study (BBPS) 1959 and the Build Study (BS) 1979 are extrapolations.

From: Van Itallie, 1980.

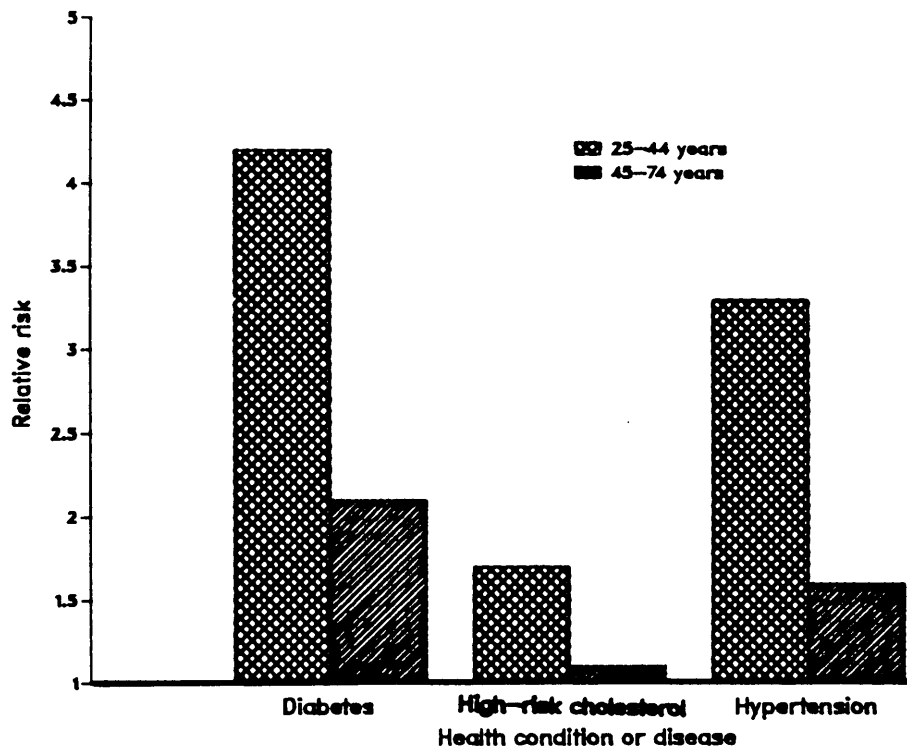


Figure 4. Relative risk of diabetes, high-risk serum cholesterol level, and hypertension for overweight persons (relative to not overweight persons), by age: 1976-80.

From: U.S. Department of Health and Human Services and Agriculture. Nutrition Monitoring in the United States, Washington, DC: U.S. Government Printing Office, 1986. (DHHS publ no [phs] 86-1255).

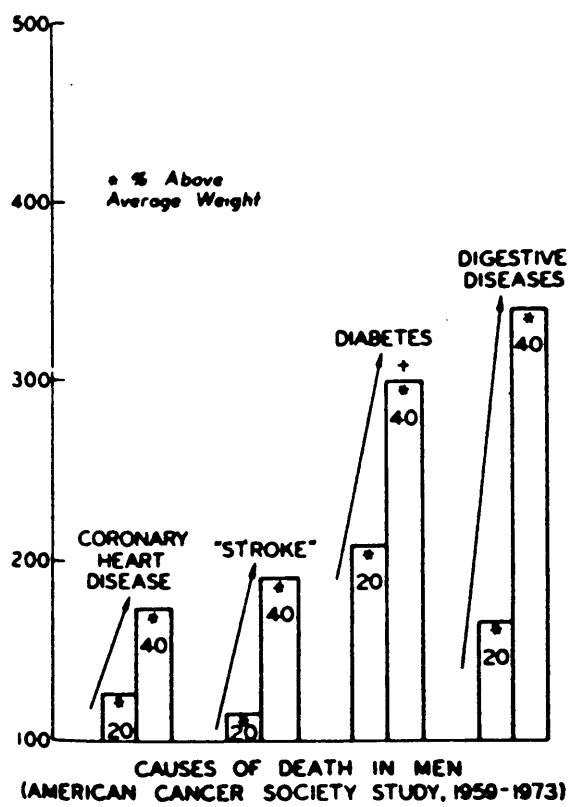


Figure 5. Relation of excess weight to cause of death in men.

From: Van Itallie, 1980.

METABOLIC AND ENDOCRINE CHARACTERISTICS

Plasma Lipids

The possibility that obesity may influence lipid metabolism was evident by the association present between obesity and hyperlipoproteinemia (Kannel et al, 1979; Hare, 1986). Epidemiological studies demonstrated that the levels of plasma lipids influenced the atherogenic process (Ashley and Kannel, 1974; Gotto 1986). A positive relationship between plasma total cholesterol (TC) levels and incidence of cardiovascular disease was shown (Pyorala et al, 1985), and evidence indicated that the distribution of plasma cholesterol among the different lipoprotein classes better assessed the risk of cardiovascular disease (Gotto, 1986). Generally, a positive relationship is found between levels of low density lipoprotein-cholesterol (LDL-C) and incidence of cardiovascular disease, whereas a strong inverse association is present with levels of plasma high density lipoprotein-cholesterol (HDL-C) (Gotto, 1986; Castelli et al, 1977; Despres et al, 1985; Miller et al, 1981; Ballantyne et al, 1982).

The low density lipoprotein acts as the main carrier of cholesterol to the cells in the body (Ballantyne et al, 1982). At plasma LDL-C concentrations below 40 to 80 mg/dl, the transfer of LDL-C into the arterial walls is thought to be slight, but at concentrations above 80 mg/dl, lipids and protein are deposited and arterial plaque forms (Gotto, 1986). The high density lipoprotein acts in an opposite manner, by transporting cholesterol away from tissues for esterification

and removal from the body (Ballantyne et al, 1982). In addition, HDL-C is thought to inhibit smooth muscle cellular uptake of LDL-C (Oster et al, 1981), thus impeding the influx and aiding the efflux of cholesterol into and out of the cell.

The HDL-C class is heterogeneous, and is made up of the subfractions, HDL₂-C and HDL₃-C. Recent evidence indicated that the HDL₂-C subfraction may possess the antiatherogenic qualities associated with the HDL-C particle (Ballantyne et al, 1982). This has been supported by research which has looked at the blood lipid levels of survivors of myocardial infarction compared to control subjects (Ballantyne et al, 1982), and patients undergoing coronary angiography (Miller et al, 1981). In both studies, the HDL₂-C subfraction was significantly lower in survivors of myocardial infarctions than controls, and in subjects with a high number of angiographically defined coronary stenoses, while the relation of HDL₃-C was not significant. The HDL₂-C particle is rich in apoprotein E, which competes with the LDL-C particle for receptor-mediated uptake by peripheral cells (Miller et al, 1981).

Serum triglyceride levels are positively correlated with body weight (Dillon, 1980), and inversely correlated with HDL-C levels (Kannel et al, 1979). A positive relationship is often observed between LDL-C and obesity, which may be due to an increased synthetic rate of the LDL particle in the obese patient (Kesaniemi et al, 1985). To a lesser extent, plasma TC levels are correlated with body weight (Dillon, 1980). Thus, high plasma triglyceride and LDL-C, and more

inconsistently, high TC levels in conjunction with low HDL-C levels, represents the blood lipid profile of the obese person. In an investigation by Kannel et al (1979), increased body weight was associated with higher blood lipid values, with the exception of HDL-C, which was lower. Significantly lower HDL-C levels were observed in obese subjects when compared to nonobese subjects, in a study conducted by Howard et al (1987). The results of these studies indicated that atherogenic traits worsen in a population in proportion to excess weight.

The high plasma LDL-C and low HDL-C seen in the obese, predisposed these individuals to the development of cardiovascular disease, a condition which accounts for more than fifty percent of the deaths in the United States (Gotto, 1986). The fact that obese individuals are at an increased risk of cardiovascular disease was demonstrated by Ashley and Kannel (1974). The relation of energy balance, as reflected by weight change, to cardiovascular risk factors was investigated using the 5,209 Framingham participants involved in the study since 1948. A positive relationship existed between increased body weight and increased cardiovascular risk characteristics, as well as incidence of cardiovascular disease. In 1983, Hubert et al, reexamined the Framingham cohort for the incidence of cardiovascular disease and similarly found obesity to be a significant independent predictor of cardiovascular disease. According to Gordon and Kannel (1973), at 135% ideal body weight, cardiovascular disease is 1.6 and 1.4 times as great in males and females, respectively. In addition, these authors speculated that if all Framingham participants above ideal body weight were at their ideal

weight, the incidence of coronary heart disease in this population would be reduced by 25%.

Fasting Insulin

The resistance to the action of insulin on glucose uptake into fat, hepatic, and muscle cells is a well documented metabolic alteration associated with condition of obesity (Van Itallie, 1980; Armstrong et al, 1950; Modan et al, 1986; Zavoroni et al, 1985; Howard et al, 1987). In addition, impaired glucose tolerance is often observed in these same individuals (Dillon, 1980; Salans, 1979; Van Itallie, 1980; Armstrong et al, 1950; Pyorala et al, 1985). Pyorala et al (1985), observed increasing plasma insulin levels in men as relative weight increased and level of physical activity decreased. Thus, both obesity and physical inactivity appeared to be related to the cells' decreased sensitivity to insulin, resulting in a higher than normal insulin level necessary for the maintenance of normal blood glucose values.

The mechanism responsible for the insulin resistance and subsequent hyperinsulinemia present with obesity has not yet been elucidated, but several theories pertaining to this phenomenon have been developed. Some investigators hypothesized that fasting serum insulin and insulin response to glucose increased relative to the enlargement of the adipose cell (Dillon, 1980; Salans, 1979; Farguhar et al, 1975; Woods et al, 1985). According to Dillon (1980), abnormally elevated levels of insulin may be necessary to induce fat cell hypertrophy for additional fat and carbohydrate storage. This was illustrated when

observing changes in fat cell size with a high kcal intake associated with or without a high energy expenditure. With excessive kcal intake without adequate energy expenditure, high fasting plasma insulin levels in conjunction with increased fat cell size was observed. In contrast, consumption of a high kcal diet along with a high energy expenditure did not result in fat cell hypertrophy or increased insulin levels (Kalkhoff and Ferron, 1971). These results were in agreement with the following theory. It had been proposed that hyperinsulinemia resulted from excessive kcal intake, especially from a carbohydrate source, which over a period of time, resulted in a chronic hyperinsulinemic state, which further resulted in lessened tissue sensitivity to insulin (Bray, 1979; Farguhar et al, 1975). The tissue insensitivity was believed to be the consequence of negative feedback action on the insulin binding receptors from the high insulin levels, which resulted in a reduced number and availability of receptors which, as an adaptive mechanism served to protect against hypoglycemia (Bray, 1979; Modan et al, 1986).

According to other investigators, insulin resistance was present in obese individuals and led initially to diminished glucose disposal and resultant hyperglycemia which further promoted the characteristic hyperinsulinemia observed with obesity (Dillon, 1980; Salans, 1979). A reduced number of insulin-binding receptor sites per surface area of enlarged adipocyte was often believed to cause the initial resistance to insulin (Dillon, 1980; Salans, 1979).

With respect to the direct correlation between basal insulin levels and degree of adiposity, Woods et al (1985) discussed an

interesting possible mechanism concerning a relationship between the brain and the hormone insulin with regard to regulation of food intake and adiposity. In the past, the brain was thought to utilize glucose independently of insulin, thus no direct link was believed to exist between insulin and the nervous system. Evidence exists that certain cells in the brain are directly responsive to insulin, in particular, insulin receptors have been found present in the hypothalamus, which is known to participate in the control of food intake and body weight. The authors speculated that the hormone insulin, which had been found present in the cerebrospinal fluid (CSF), acts as a messenger to the brain relating the relative state of adiposity to the feeding centers. In normal individuals, an increase in CSF insulin levels is thought to be interpreted by the brain as increased adiposity, which would result in decreased food intake and subsequent weight loss. This phenomenon had been observed with experimental animals upon infusion of insulin into the CSF. In obese Zucker rats, insulin levels were almost undetectable in the brain, yet high levels were present in the CSF and the plasma. Thus, insulin may not have gained access to the brain, possibly due to insufficient insulin transport, or lack of functional brain insulin receptors, resulting in a false perception of leanness, which resulted in overeating and obesity.

Although the underlying mechanism remained unclear there was clearly an alteration in glucose metabolism and insulin secretion associated with the condition of obesity, and hence this disorder played a major role in the development of adult-onset diabetes (Type II) (Modan et al, 1986; Rimm et al, 1975). The interrelationship of

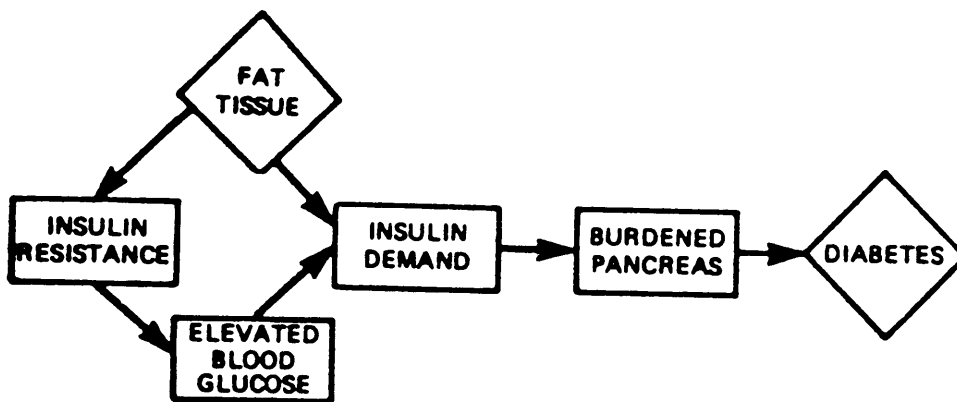


Figure 6. Model depicting interrelationship of obesity and adult-onset diabetes.

From: Rimm and White, 1979.

obesity and adult-onset diabetes is shown in Figure 6. Diabetes was believed to develop as a result of pancreatic beta cell exhaustion due to the chronic hyperinsulinemia discussed previously (Modan et al, 1986). The increased risk of developing adult-onset diabetes with increasing relative weight is demonstrated in Figures 7 and 8. It is evident that the occurrence of diabetes in obese women becomes more prevalent with increasing age and weight. According to The National Commissions on Diabetes, degree and duration of obesity were the factors most strongly associated with development of adult-onset diabetes. This was also demonstrated by Modan et al (1986), who studied past and concurrent BMI values relative to the incidence of Type II diabetes, glucose intolerance, and hyperinsulinemia in 2,140 subjects. According to their results, impaired glucose tolerance became evident shortly after the BMI was equal to or greater than 27. In contrast, the development of Type II diabetes and reduced insulin response appeared to require a prolonged period of obesity. These authors suggested that a BMI greater than or equal to 27 indicated a "critical mass of fat tissue which accelerates deterioration of glucose intolerance" (Modan et al, 1986).

Available evidence indicates that a high plasma insulin level may promote the development of cardiovascular disease (Pyorala et al, 1985; Smoak et al, 1987; Zavaroni et al, 1985; Welborn and Wearne, 1979). Pyorala et al (1985), investigated the occurrence of cardiovascular disease in 982 men, ages 35 to 64, who entered the study free of cardiovascular disease. After a period of 9.5 years, a nonlinear association between 1 and 2 hour plasma insulin and the incidence of nonfatal

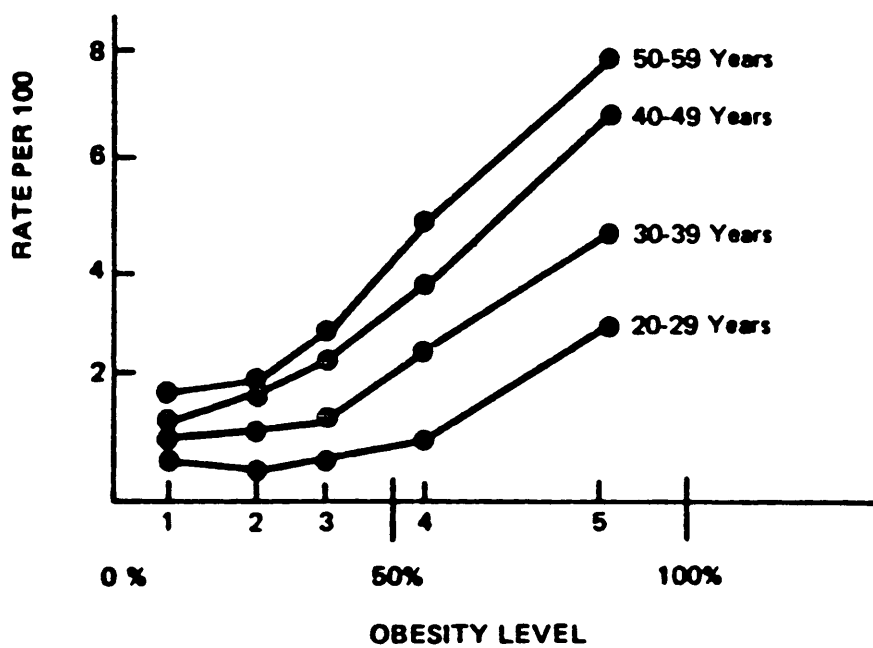


Figure 7. Obesity and age specific occurrence rates for women with a history of adult-onset diabetes.

From: Rimm et al. 1985

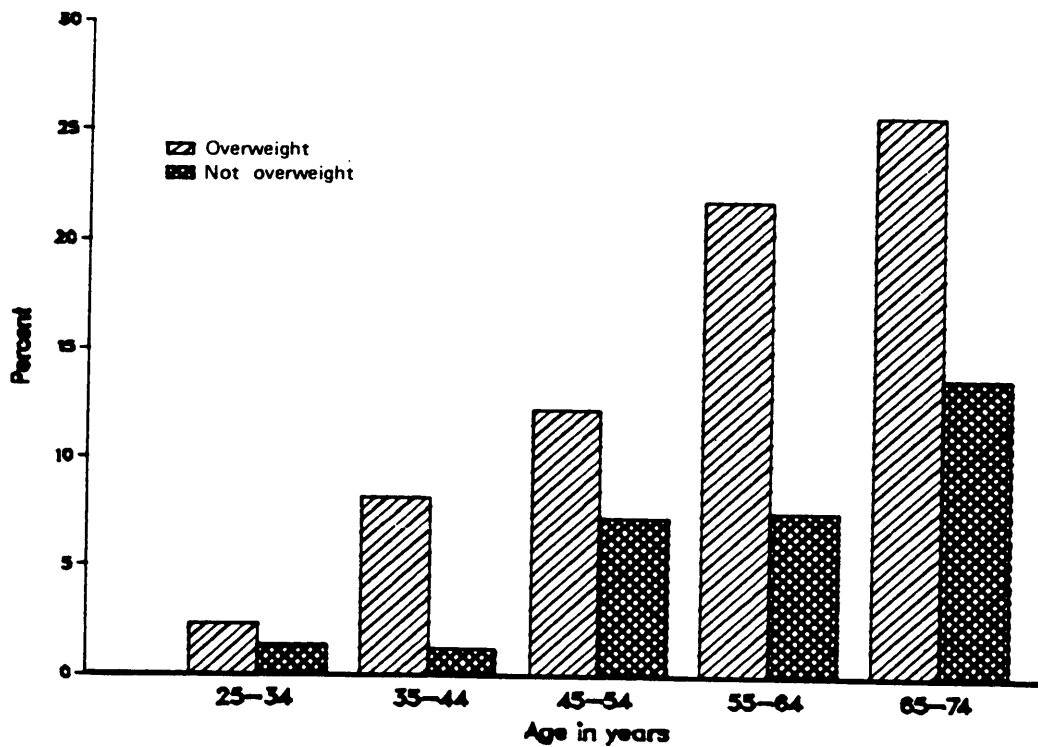


Figure 8. Percent of adults with diabetes, by overweight status and age: 1976-80.

From: U.S. Department of Health and Human Services and Agriculture. Nutrition Monitoring in the United States, Washington, DC: U.S. Government Printing Office, 1986. (DHHS publ no [PHS] 86-1255).

myocardial infarction, or death from cardiovascular disease ($P < 0.002$) was observed. The investigators concluded that plasma insulin was an independent predictor of cardiovascular disease. This association was not as strong in females.

METABOLIC AND ENDOCRINE INTERACTION

An interrelationship has been demonstrated among many of the endocrine and metabolic characteristics which comprise the biochemical profile observed in the obese state. Several investigators have found the hormone insulin levels positively correlated to levels of blood glucose (Pyorala et al, 1985; Gibson et al, 1975), plasma triglycerides (Pyorala et al, 1985; Howard et al, 1987; Zavaroni et al, 1985; Gibson et al, 1975; Farguhar et al, 1975), and inversely related to HDL-C levels (Zavaroni et al, 1985). In addition to biochemical interactions, plasma insulin was found to be positively related to BMI, blood pressure, and inversely related to exercise level (Pyorala et al, 1985).

Zavaroni et al (1985) studied 607 male and female subjects to determine whether a relationship existed between plasma insulin levels and both HDL-C and triglyceride levels. A significant negative relationship between plasma insulin and HDL-C levels was observed, and both HDL-C and insulin were significantly correlated to the degree of hypertriglyceridemia, degree of obesity, and level of glucose tolerance. The strongest relationship observed was between the degree of hyperinsulinemia and hypertriglyceridemia.

The correlation between obesity, fasting insulin, and fasting triglycerides was observed by other investigators (Gibson et al, 1975; Howard et al, 1987; Kesaniemi et al, 1985). Howard et al (1987) and Kesaniemi et al (1985) observed an increased production of very low density lipoprotein (VLDL)-triglycerides (TG) and VLDL apolipoprotein (apo) B levels in obese individuals, as well as a significant correlation between plasma insulin levels and production of VLDL-TG and VLDL-apo B. VLDL-TG and VLDL-apo B are produced in the liver and transport endogenous triglyceride. These investigators suggested that the hyperinsulinemia was responsible for the hypertriglyceridemia observed in obese individuals because it induced overproduction of both VLDL-apo B and VLDL-TG. Farquhar et al, (1975), further speculated that the association present between obesity, hyperinsulinemia, and fasting hypertriglyceridemia occurred as a consequence of insulin resistance developed in the enlarged adipocyte and development of secondary hyperinsulinemia and increased insulin-stimulated hepatic triglyceride synthesis.

BODY COMPOSITION

Body weight is the simplest index of overweight, yet it does not always reflect the body's stores of adipose tissue. Body composition analysis serves to determine the nature of overweight and to distinguish those individuals with excess musculature from those with excess fat. Relative body fat varies with age, sex, and race, with fat content increasing with age, women being relatively fatter than men,

and black females having significantly greater subscapular skinfold thicknesses than white females (Sims, 1979). Morbidly obese individuals often have a percent body fat greater than 50%, which is often attributed to hypertrophy and hyperplasia of the adipose tissue cells (Hare, 1986; Dillon, 1980). The determination of body fat content is important in that excess fat in the abdominal and subscapular body regions is associated with increased plasma lipids, and hence, increased cardiovascular disease risk (Despres et al, 1985; Krotkiewski et al, 1983).

More conventional anthropometric indices include body weight as a measure of leanness or obesity, triceps skinfold which measures subcutaneous fat stores, and mid arm muscle circumference which indicates amount of lean body mass. Determination of body fat content in obese individuals, and especially in obese persons experiencing edema, is difficult due to technical errors which often occur with these conventional measurements. The errors in reporting body composition in obese individuals result from differences in fat compressibility among different body types, which is disregarded in the triceps skinfold method, and assumptions made concerning total body water distributions, hydration state, and estimation of lean body stores (Bencich et al, 1986). In addition to the need to determine the body composition at a stable weight, the need for a reliable method to assess body composition changes during weight loss is also evident.

As mentioned earlier, the body mass index (BMI) is one measurement used to estimate degree of adiposity when a more direct measurement is not available. Recent reports by The Panel on Energy, Obesity, and

Body Weight Standards (Jequier et al, 1987) discuss the use of the Quetelet's index, or BMI as a system for classifying obesity. Three grades of obesity have been established: a BMI of 25-29.9 corresponds to Grade I obesity, a BMI of 30-40 indicates Grade II obesity, and a BMI greater than 40 indicates Grade III obesity. Grade III obesity is synonymous with morbid obesity.

The bioelectrical impedance analysis (BIA) is a technique more recently investigated as a method for determining body composition. The application of this method to clinical nutrition appears to provide a fairly accurate measurement of body composition during stable weight and weight loss conditions. The principle of the BIA is based on the different dielectric properties of lean and fat tissues. Lean tissue is comprised largely of electrolyte containing water, and readily conducts electrical current, whereas fat acts as an insulator. The electrical properties of biological systems were initially discussed by Nyboer in 1959. Application of a constant low level alternating current at radio frequency was demonstrated to result in an impedance whose resistance was inversely related to ionic conductive volume (Nyboer, 1959).

Soon after Nyboer's report on the electrical properties of biological systems, Thomasset (1962; 1963) and Hoffer et al (1969), demonstrated the application of body impedance to determining total body water (TBW). Hoffer et al (1969) reported a direct relationship between impedance and TBW in normal healthy males and in patients with various degrees of previously established levels of hydration. The findings by these investigators that a relationship between body

impedance and TBW exist did not stimulate further investigation into the applicability of this method for body composition determination until the 1980's.

One of the more recent studies was conducted by Lukaski et al (1985), who attempted to develop a method to use BIA for assessing fat free mass (FFM), studied the reliability of impedance measurements and investigated the validity of the measurements by comparison with standard estimates and indices of human body composition in 37 healthy men. FFM was determined by hydrodensitometry, TBW by D₂O dilution, and total body potassium (TBK) from whole body counting. Linear relationships were found between resistance (R) values and FFM, TBW, and TBK. Significantly greater correlation coefficients were found when the predictor Height² (HT)/R was regressed against FFM, TBW, and TBK. The significant relationship between HT²/R measurement and the parameters mentioned has been documented by other investigators (Kushner and Schoeller, 1986; Lubanski et al, 1987). In addition, test-retest correlation coefficient was 0.99 for a single R measurement and the reliability coefficient for a single R measurement was 0.99 over 5 days. These authors concluded that the BIA is a reliable and valid method for the estimation of human body composition in normal, healthy populations.

A more heterogeneous population was investigated by Segal et al (1985) who studied 75 male and female subjects ranging in age from 17 to 59 years, and ranging in body fat from 4.9 to 54.9%. Compared with existing body composition methods, LBM predicted from BIA by use of prediction equations provided with the instrument was significantly

correlated with densitometrically determined LBM (LBMd) ($r = 0.912$) in nonobese subjects, but overestimated LBM compared with LBMd in obese subjects. This latter finding was previously documented by Kushner et al (1984). The authors indicated that factors such as abnormal hydration, variability in bone density, variation in proportions of muscle and bone to total LBM, and changes in nutritional status may cause variability in the water and potassium content of LBM which would also affect the density of LBM. The derivation of a new prediction equation which included Ht^2/R , height, weight, and sex, alleviated the systematic error observed when the previous equation was applied to this heterogeneous sample. According to these investigators, the BIA method was a convenient, rapid, and safe technique for determining human body composition in stable weight individuals (Segal et al, 1985).

The estimation of TBW by BIA was compared with a deuterium-isotope dilution (D_2O -TBW) method in 58 subjects, grouped by sex and weight (Kushner and Shoellers, 1986). Sex specific and group formulas for assessing TBW from BIA were developed and subsequently validated on a heterogeneous group of patients who were obese and either had diabetes mellitus, or inflammatory bowel disease. The results indicated that Ht^2/R was again the most independent predictor of D_2O -TBW, and the prediction was further improved by adding body weight to the equation. In contrast to Segal et al (1985), the height term was not found to greatly contribute to the predicted LBMd. The data for the D_2O -TBW predicted from BIA calculated formulas and the measured D_2O -TBW are shown in Figure 9, for obese and nonobese females (Kushner and

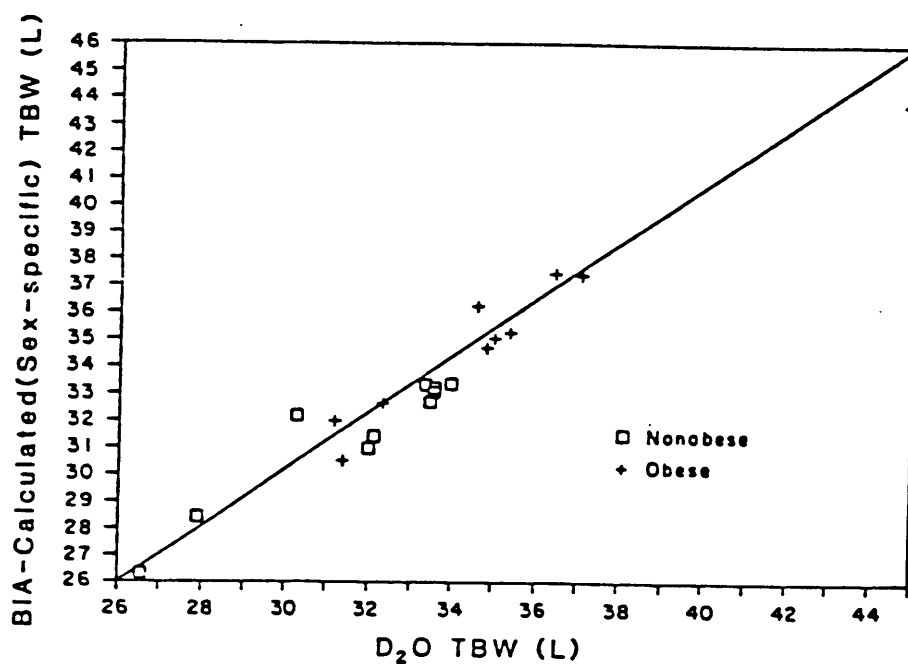


Figure 9. BIA-calculated, sex-specific equation for predicted TBW compared to the measured D₂O dilution space for nonobese and obese females. $R=0.975$.

From: Kushner and Schoeller, 1986.

Schoeller, 1986). D₂O-TBW was predicted well using the BIA for both nonobese and obese males and females with both the group and sex-specific equations. In addition, the predictive equations compared to the measured D₂O dilution space in the patient sample (Kushner and Schoeller, 1986).

In a more recent study, Kushner and Haas (1987) investigated the prediction of LBM determined by BIA (LBM-BIA) compared to LBM determined by skinfold thickness (LBM-SA) in 59 weight stable patients grouped according to whether they were lean, normal, obese, or super-obese. The results indicated a mean percent error less than 1.1% between LBM-BIA and LBM-SA in all groups except super-obese which demonstrated a 3.04% difference. In addition to LBM determination during stable weight conditions, these researchers looked at the mean difference between change in LBM-BIA and LBM-SA following a $9.7\% \pm 3.6\%$ weight loss. The difference observed between the two methods was 1.2 ± 1.65 kg LBM. These authors concluded that excellent agreement exists between BIA and SA determined LBM in individuals ranging from lean to super obese during stable weight and weight loss conditions (Kushner and Haas, 1987).

The need to monitor body composition changes during weight loss is evident, particularly in those individuals on very-low kcal dietary treatment. The ability of arm circumference (C), skinfold thickness (ST), BIA, and hydrodensitometry (HD) to detect changes in body fat during weight loss were compared (Johnson et al 1987). Fourteen obese male and female subjects were placed on a low kcal diet consisting of 50 to 1500 kcal per day for 21 days. Significant differences among the

means for each measurement time was observed, as well as significant differences between the four measurements at each time. Each method was reported to accurately determine change in body fat during weight loss, although the BIA and HD methods appeared better able to detect small changes. A study performed by Preuss et al (1986) also indicated that BIA is a reliable method for following changes in adipose and nonadipose tissue during weight loss treatment.

TREATMENT

The therapeutic approach to weight loss often involves manipulation of existing diet, activity, and/or behavior patterns. Studies have focused on one, or a combination of two, or all three of the areas, yet weight loss by any treatment has thus far resulted in limited success. Weight loss treatment for obese persons is often associated with weight loss followed by weight gain, a cycle often referred to as the "yo-yo syndrome". According to Stunkard (1958) "... of those who remain (on a low-calorie diet) most will not lose much weight; of those who lose weight, most will regain it." Over the past 30 years, the prognosis for the treatment of obesity has not improved substantially. There is general agreement that no more than 25% of obese persons undergoing weight loss treatment will lose as much as 20 lbs, and only 5% will lose as much as 40 lbs (Penick et al, 1971; Musante, 1974). Wing and Jeffery (1979) reviewed the results of 145 outpatient treatments of obesity conducted during the previous eleven years. Different treatment methods, including behavior modification,

exercise, diet and anorectic drugs, produced similar weight losses which usually averaged no more than 5.4 kg. Weight losses of 14.5 kg or more were associated with treatments of one year duration, whereas smaller weight losses were observed with short term duration. It was acknowledged that improvement of treatment results would accompany increased duration of treatment (Stunkard, 1987).

The adoption of a more integrated approach to the treatment of obesity, including behavior modification, nutrition education, diet, and physical activity, appears to be gaining increasing favor over more traditional and radical treatments (Frankle, 1985; Stunkard, 1987; Weinsier et al, 1984). The interrelationship and interdependency of these different approaches make this method desirable and offers hope for better treatment success.

Diet

The increased consensus for adoption of a more integrated approach to weight loss results in part from the minimization of lean body mass (LBM) loss observed with a moderate kcal restriction, or low kcal diet, as opposed to very low kcal diet treatments or total starvation (Weinsier et al, 1984; Hagan et al, 1986). Energy deficit and rate of weight loss are related, yet the type of tissue lost differs with different rates of weight loss. Excess weight in obese people is comprised of approximately 75% fat and 25% LBM (Garrow, 1987). The optimal treatment should strive for a similar composition of weight loss, so that loss of LBM is kept to a minimum. According to Garrow

(1980) loss of adipose tissue was observed with a slow rate of weight loss of under 1 kg per week, whereas a faster rate of weight loss of 4 kg per week was associated with a large loss of LBM, and subsequent decrease in metabolic rate. LBM is the major determinant of resting metabolic rate (RMR), and during starvation, tissue loss is approximately 50% fat and 50% LBM, and a large decrease in metabolic rate occurs (Garrow, 1987).

The type of diet also contributes to the rate of weight loss. This can be observed when comparing the weight loss differences between ketogenic and non-ketogenic diet treatments. A much larger rate of weight loss is initially observed with consumption of a ketogenic diet, which is attributed to large losses in body water (Dillon, 1980; Yang and Van Itallie, 1976). This effect often misleads the person to believe a more substantial weight loss has occurred with that particular dietary treatment. In addition to a loss of LBM associated with a moderate kcal restriction, a more nutritionally adequate diet is more easily achieved, there is increased probability of subject compliance, and the energy intake allows for participation in an exercise program (Hagan et al, 1986).

Exercise

Although the contribution of physical activity to weight loss is marginal, the incorporation of an exercise program into the treatment of obesity is observed to limit LBM loss and induce loss of adipose tissue, to limit reductions in metabolic rate, and is thought to

suppress appetite (Weinsier et al, 1984; Dillon, 1980; Stunkard, 1987). When exercise was included in conjunction with kcal restriction, loss of body fat weight more closely approximated loss of total body weight (Lewis et al, 1976). The effects of a 17 week exercise program, in addition to self determined kcal restriction was studied in 22 obese women (Lewis et al, 1976). At the end of the program, a 5% reduction in body fat which corresponded to a loss of 5.4 kg body fat was observed, and closely paralleled the total loss of body weight of 4.2 kg. Hagan et al (1986) found similar results in 96 obese male and female subjects participating in an exercise program five days per week for 12 weeks, and consuming a 1,200 kcal diet. The treatment group participating in both the exercise program and consuming the reduced kcal diet showed significantly greater body weight and fat weight losses than did either the diet alone, or the exercise alone treatment groups.

Nutrition Education

Kcal restriction accompanied by an effort to improve nutrition education promotes a gradual change in the composition of the diet in addition to weight loss, and offers hope for the continuation of new food habits following treatment and increased probability of weight maintenance (Stunkard, 1987; Weinsier et al, 1984). It has been suggested that the alteration of the macronutrient composition, in addition to kcal reduction, is important for successful weight reduction. Other researchers do not agree, and believe that a "calorie

is a calorie" regardless of the food source. The manipulation of macronutrient composition, may in fact, alter energy intake without a conscious effort to restrict kcal intake. This was demonstrated by a manipulation of fat intake by Lissner et al (1987) who studied the effects of varying fat composition of 15 to 20%, 30 to 35%, and 40 to 45% of total energy intake, on twenty-four obese and nonobese females. Regardless of whether the subject was obese, food intake ad libitum among the three groups differed significantly. The energy intake on the low-fat diet was 11.3% below, and the energy intake on the high fat diet was 15.4% above, that consumed on the medium fat diet. These researchers suggested that habitual intake of a low fat diet may effectively reduce weight.

Behavior Modification

In addition to moderate kcal restriction, physical activity and nutrition education, the incorporation of behavior modification into weight loss therapy has demonstrated positive outcomes for obese individuals (Stunkard, 1987; Weinseir et al, 1984; Musante, 1974). Behavioral therapy defines obesity as a learned behavior disorder (Musante, 1974), and promotes weight loss through changes in eating behavior (Jeffery et al, 1978). The factors involved in behavior modification include identifying eating behaviors which need to be changed, learning to control stimuli that precede eating, development of techniques to control eating, reinforcement of desired behaviors, and cognitive restructuring to change attitudes toward eating

(Stunkard, 1987; Weinsier et al, 1984). It is generally agreed by advocates of behavior modification that obese individuals do not know when they are physiologically hungry or satiated (Rosenstock, 1969; Schachter, 1968; Dillon, 1980). The difference between the normal weight and the obese persons' response to physiological hunger has been documented in studies which manipulated gastric motility and glycemic state, and observed eating behavior (Rosenstock, 1969; Schachter, 1968). Rosenstock (1969) found that self report of hunger coincided with gastric motility 71% in normal weight, and only 48% in obese people. Schachter (1969) reported that eating was triggered by different sets of stimuli in obese and normal weight subjects. Obese individuals are thought to eat in response to external rather than internal cues (Dillon, 1980).

Behavior modification usually resulted in moderate short term weight loss in addition to newly formed attitudes toward eating and the increased probability of maintaining the weight loss (Follick et al 1984). In addition, behavior therapy was accompanied by low attrition rates (Weinsier et al, 1984), which rarely exceeded 15% (Stunkard, 1987).

Behavioral therapy resulted in successful weight loss (Foreyt et al 1982). They studied the effects of 8 weekly sessions of behavior modification on weight loss in 817 men and women, treated in groups of 12-15 people. Treatment resulted in an average weight loss of 6.8 and 4.3 kg, in men and women, respectively, and an additional 0.9 and 1.0 kg weight loss was reported at the time of follow-up. At one year

following treatment, 30% of the patients continued to lose weight, 43% showed no change, and 27% regained some weight.

A study by Penick et al (1971) assessed the effectiveness of a three month behavior modification program compared to traditional psychotherapy in the treatment of 32 obese men and women. Results indicated greater weight loss in the behavioral group when compared to the psychotherapy treated group, with 3% losing more than 40lbs and 53% losing more than 20 lbs. The authors reported that much more variation in weight loss was observed for the behavior modification group than the psychotherapy group, and thus suggested that behavioral therapy may be remarkably effective for some individuals and quite ineffective for others.

To determine whether group or individual intervention differed in success of behavioral therapy, Adams et al (1986) investigated 153 obese people, of which 82% attended group therapy, and 18% were seen individually. No significant difference in weight loss between individual and group therapy was seen, and average weight loss for both treatments was 11.8 lbs, with a range of -57.5 to +11.5 lbs. Average accumulated weight loss at follow up was -6.9 lbs, with a range of -150.8 to +61.5 lbs. It appeared that men lost more weight in group therapy, and women were more successful in individual therapy. In addition, these researchers looked for factors which could predict outcome of weight control programs. Both percentage overweight and amount of weight change during the program predicted treatment success. The more overweight the subject was, the more weight lost following the treatment, whereas the more weight lost during treatment,

the more gained following the program. Subjects who gained weight during the program, lost the most weight after treatment. Also, the more weight loss programs an individual had tried previous to participation in this study was inversely related to the program success, and the number of sessions attended during the program was significantly related to weight loss.

WEIGHT LOSS INDUCED METABOLIC AND ENDOCRINE ALTERATIONS

Plasma Lipids

Improvement of the lipid and lipoprotein profile of obese individuals is generally observed with weight loss (Ashley and Kannel, 1974; Gotto, 1986; Keys et al, 1972; Dillon, 1980). This alteration in blood lipids would apparently reduce their risk for coronary heart disease. The short and long term effects of weight loss on lipid and lipoprotein levels were observed in 42 obese women, ranging from 10 to 60% above ideal body weight (Follick et al, 1984). Subjects attended a 10 session behavioral weight loss program, and a 4 session weight maintenance program. A 1,200 kcal diet was recommended. Treatment resulted in an average 4.3 kg weight loss, and reductions in both TC and LDL-C levels. At the time of the six-month follow-up, lipid values remained reduced despite a 1.6 kg weight gain. Changes in lipid values were correlated significantly with BMI. No change in HDL-C or the ratio of HDL-C/LDL-C was seen during treatment, but significantly higher values were reported at follow-up. These authors suggested that a reduction

in HDL-C levels accompanied active weight loss, but with weight stabilization HDL-C levels returned to initial values. It was concluded that weight loss produced consistent and favorable changes in lipid and lipoprotein concentrations. In obese hypercholesterolemic patients, dietary induced weight loss resulted in significantly lower TC and LDL-C levels from 273 to 249 mg/dL and 209 to 186 mg/dL, respectively, but no effect on HDL-C levels was observed (Davis et al, 1985).

In contrast to the latter two studies, significantly reduced HDL-C levels were reported in 30 obese women who underwent acute, rapid weight reduction of 5.6 ± 0.7 kg within 15 days by very low kcal diet treatment (Bosello et al, 1985). A significant reduction in VLDL-C, LDL-C, and TC levels also was observed, and the HDL₂-C subfraction was significantly lower. No correlation between variation of HDL-C and change in body weight was present. It was speculated that the decrease in HDL-C and HDL₂-C was due to kcal restriction, rather than weight loss, and may reflect a reduced formation of HDL-C particles, particularly HDL₂-C.

In addition to weight loss, altering the diet composition may result in blood lipid and lipoprotein changes. A review of the literature revealed that high plasma TC levels were often seen with high intakes of total and saturated fats, and less consistently with high intakes of dietary cholesterol (Grundy, 1987; Pyorala, 1987). Upon substitution of saturated fat in the diet with polyunsaturated fatty acids, a reduction in plasma TC, LDL-C, and HDL-C was generally seen (Schlierf et al, 1983; Grundy, 1987). Increased consumption of

monounsaturated fatty acids was accompanied by a decrease in plasma TC and LDL-C, without affecting HDL-C levels (Grundy, 1987). The improvement of diet composition, along with kcal restriction necessary for weight loss, improved the obese persons' lipid levels.

Fasting Insulin

High fasting insulin levels often characterized in the obese condition were reduced during kcal restriction and approached normal values following weight reduction (Dillon, 1980; Neufeld et al, 1986; Henry et al, 1986a). The fall in plasma insulin levels upon restriction of kcal occurred simultaneously with an increase in insulin receptors (Gould et al, 1982; Kolterman et al 1979). Reduction of fasting insulin to normal levels and increased insulin binding to monocytes was observed following weight loss in 10 non-diabetic obese individuals (Neufeld et al, 1986). A decrease in cell membrane phospholipid to protein ratio occurred, and it was concluded that the increased insulin receptor number affinity associated with weight loss resulted from changes in the chemical composition of the cell membrane.

In obese subjects with non-insulin dependent diabetes, improvement of glucose homeostasis occurred with weight loss, in addition to reduced fasting insulin levels (Henry et al, 1986a; Henry et al, 1986b). The normalization of elevated glucose and insulin levels in 10 obese non-insulin dependent diabetics and 5 obese non-diabetic subjects was seen after a 36 day very low kcal diet treatment. From the above research it appeared the obesity was a primary factor in the development of non-insulin dependent diabetes. With weight loss, blood

glucose and insulin levels showed improvement and would therefore be of primary importance for the treatment and control of this disease.

Serum Thyroxin

Resistance to weight loss is often reported in obese individuals trying to lose weight after a period of kcal restriction and some weight reduction. This phenomenon is often explained by reduced energy expenditure or resting metabolic rate believed to result from altered peripheral metabolism of thyroid hormones during hypocaloric intake or total starvation (Hramiak and Nisker, 1985; Palmbald et al, 1977; Aziz, 1978; Danforth et al, 1979). The hormone, thyroxin (T_4), is converted to either serum reverse triiodothyronine (rT_3), or serum triiodothyronine (T_3) by monodeiodination in the liver (Hramiak and Nisker, 1985). Serum T_3 is the major thermogenic hormone and is the active metabolite responsible for energy expenditure, whereas rT_3 is an inactive metabolite. The regulation of serum concentration of thyroid hormones, and the proportion of T_3 and rT_3 produced from T_4 appears to be influenced by kcal intake and diet composition.

Following hypocaloric intake, or total starvation, T_4 levels normally remain unchanged, or decrease slightly, while serum T_3 levels decreased, and rT_3 levels increased in obese individuals (Welle et al, 1984; Visser et al, 1978; O'Brian et al, 1980; Serog et al, 1982; Mathieson et al, 1986). The opposite effect is observed with experimental overfeeding. Serum T_3 levels increased and serum rT_3 levels decreased (Danforth et al, 1979). Carbohydrate restricted diets

also appeared to alter the thyroid hormone levels similar to hypocaloric feeding, and some investigators discussed the possibility that an intake of a high carbohydrate diet may have decreased further the reduction in serum T_3 seen with kcal restriction (Mathieson et al, 1986; Serog et al, 1982).

The effect of kcal restriction on levels of serum thyroid hormones was investigated in 9 obese individuals who consumed a 300 kcal diet for 6 weeks (Visser et al, 1978). Body weight was reduced by 12%, and serum T_3 fell to 70% and rT_3 rose to 180% of the pretreatment values. Serum T_4 levels remained unchanged. These results were later confirmed by Mathieson et al (1986), who studied the effects of a very low kcal diet treatment for 28 days, followed by a 1,000 kcal refeeding diet for 1 week. Subjects consumed either a high or low carbohydrate diet, and the effects of kcal restriction and diet composition on thyroid hormone levels and resting metabolic rate (RMR), were investigated. Serum T_4 levels decreased with increased duration of the treatment, and become significantly lower during and after the 1,000 kcal refeeding diet. A reduction in serum T_3 occurred in both groups, but decreased faster and to a greater extent with the low carbohydrate diet. A similar increase in serum rT_3 was observed with both diets, and both serum T_3 and rT_3 returned to pretreatment levels following the 1,000 kcal diet. Reduction in RMR was similar for both the low carbohydrate and the high carbohydrate diet, despite the different reductions in serum T_3 between the two groups. In addition, RMR remained lower during refeeding at which time serum T_3 and rT_3 returned to normal levels. It was suggested by these authors

that factors other than reduced serum T₃ levels play a role in the reduction in RMR observed with kcal restricted and low carbohydrate diets, and that the persistence of a lower RMR upon refeeding may account for the weight gain often experienced in obese people following weight loss.

In a study conducted by Welle et al (1983) RMR was reduced by 9.4% and mean serum T₃ concentrations reduced by 46% in 6 obese women following consumption of a very low kcal diet for 5 weeks. Despite the fact that a reduced energy expenditure, or RMR has often been used to explain the resistance to further weight loss and subsequent weight gain in individuals consuming a hypocaloric diet, these investigators identified a mean RMR of 1328 kcal per day following kcal restriction and reduced serum T₃ levels. This value was similar to that observed in lean women, which was 1,241 kcal per day. It was suggested that even after weight loss, the RMR of obese or previously obese individuals was not abnormally low compared to nonobese individuals. Thus, these researchers concluded that decreased metabolic rate did not explain the inability of obese individuals to lose weight on kcal restricted diets.

EXERCISE INDUCED METABOLIC AND ENDOCRINE ALTERATIONS

Plasma Lipids

Results from cross-sectional studies indicate that physically fit individuals possess a more desirable blood lipid profile than do sedentary individuals. It has been well documented that active individuals

had higher HDL-C and HDL₂-C levels, and lower plasma triglycerides, VLDL-C, and LDL-C levels (Wood and Haskell, 1978; Hostmark, 1982; Goldbert and Elliot, 1985; Berg et al, 1981; Moore et al, 1983; Haskell, 1984). A comparison of blood lipid levels of long distance runners, joggers, and inactive women by Moore et al (1983) indicated that long distance runners have lower triglyceride and higher HDL-C levels than both joggers and inactive women. TC levels did not differ between groups.

It is believed that participation in an exercise program may result in desirable changes in blood lipids of sedentary people. Review of the literature revealed that a reduced plasma triglyceride, VLDL-C, LDL-C, and increased HDL-C level are found in initially inactive individuals undergoing endurance training programs (Hostmark, 1982, Wood and Haskell, 1978). The effects on plasma TC have been more difficult to identify, and are less conclusive. An altered lipid profile is not always observed in individuals that begin an exercise program, and thus a relation between physical activity and blood lipid levels in previously sedentary individuals has been difficult to establish (Goldberg and Elliot, 1985). The effects of exercise on plasma lipid and lipoprotein levels may be augmented by weight loss, and changes in diet, and influenced by duration of exercise period (Goldberg and Elliot, 1985; Schlierf et al, 1983; Haskell, 1984).

In one study by Lewis et al (1976) participation in a 17 week exercise program did not alter plasma triglyceride or TC levels despite a weight loss of 4.2 kg. The HDL-C/LDL-C ratio increased significantly, indicating that exercise and/or weight loss positively

influenced HDL-C and/or LDL-C levels. Sopko et al (1985) found that exercise and weight loss independently influenced HDL-C levels in obese men. In this study, weight loss without exercise and exercise without weight loss both resulted in higher HDL-C levels. The greatest increase in this lipoprotein was observed when weight loss and exercise occurred together. Reductions in triglyceride and VLDL-C levels were seen with the group that lost weight and exercised, but was not indicated in the group that exercised but did not lose weight. Thus, it was observed that both exercise and weight loss positively influence HDL-C levels.

Investigations of the exercise effects on blood lipids in normal weight, sedentary people have been conducted by Frey et al (1982) and Morgans et al (1983). In the study by Freyal et al (1982) 16 healthy, sedentary females participated in an aerobic exercise program for 10 weeks, 3 times per week. Although reduction in body fat percent occurred, no change in HDL-C, or triglyceride levels were observed at the end of the program. When participation in an aerobic exercise program was increased to 6 months, Morgans et al (1983) still observed no change in plasma HDL-C, TC, or triglyceride values in 5 normal weight, inactive male and females. The pretreatment lipid and lipoprotein levels appear to be important in the extent to which exercise influences blood lipid levels. Normally, exercise influences blood lipid levels to a greater extent in individuals with high plasma TC, LDL-C and triglyceride, and low HDL-C levels.

Fasting Insulin

Physical conditioning resulted in reduced insulin levels with increased sensitivity to insulin in peripheral tissues (Bray, 1979; Pedersen et al, 1980). The effects of kcal restriction with or without exercise on insulin receptors in obese subjects was studied by Neufeld et al (1986). Increased insulin receptor concentration, and decreased fasting insulin levels were seen with both treatments. Membrane fluidity, which was reduced prior to treatment, was restored to normal levels in subjects who both exercised and consumed a hypocaloric diet, but not in individuals who were subjected to kcal restriction alone. Normally, changes in the membrane fluidity either promote or impair the interaction of receptors with their respective ligand. The insulin receptor has been shown to be susceptible to alteration by the lipid environment (Gould et al, 1982). An increase in circulating free fatty acids was observed simultaneously with reduced fasting insulin levels in subjects that exercised and consumed a kcal restricted diet. The elevated free fatty acid levels may have contributed to the restoration of membrane fluidity to normal levels.

Serum Thyroxin

Physical exercise has been acknowledged to aid in development of lean body mass (LBM), and thus has a proteoanabolic effect on the human body. Kcal restriction and weight loss have an opposite effect, and are often associated with decreased LBM, serum T_3 , and resting metabolic rate (RMR). The ability of physical exercise to restrict the

alterations of the thyroid hormones and limit the reduction in RMR associated with low kcal intake has been investigated (Bosello et al, 1981; Mathieson et al, 1986). In a study by Bosello et al (1981) a low kcal protein sparing modified diet with or without exercise for 28 days was compared for the effect on thyroid hormones in 11 obese subjects. No significant difference in weight loss was found between the two groups. Consumption of the protein sparing modified diet prevented the reduction in serum T_3 normally observed with fasting and hypocaloric diets, yet a transient rise in rT_3 was still present. The increase in rT_3 was not observed in the group that exercised, thus these investigators concluded that physical activity promoted the formation of the active thyroid hormones (T_3) and limited the activity of the pathway responsible for formation of the inactive thyroid hormone (rT_3).

Project Objectives

1. To evaluate the effectiveness of a comprehensive weight reduction program on weight loss in obese black women for 12 weeks. Intervention included:

- a. Diet (1200 kcal exchange diet).
- b. Behavioral modification
- c. Nutrition Education
- d. Exercise

2. To evaluate the effect of weight reduction with or without exercise on blood lipid and lipoprotein levels.

- a. Total cholesterol (TC)
- b. Low-density lipoprotein-cholesterol (LDL-C)
- c. High-density lipoprotein-cholesterol (HDL-C)
- d. High-density lipoprotein₂-cholesterol (HDL₂-C)
- e. Triglycerides

3. To evaluate the effect of weight reduction with or without exercise on fasting insulin and thyroxin levels

4. To evaluate the effect of weight reduction with or without exercise on body composition.

- a. Total Body fat (Kg)
- b. Percent Body fat

- c. Total lean body mass (Kg)
- d. Percent lean body mass
- e. Percent body water

5. To determine the effectiveness of the bioimpedance analysis (BIA) technique in measuring body composition changes during and following weight loss in a obese black female population.

6. To evaluate alterations in nutrient intake following behavior modification and nutrition education classes.

7. To determine the effect of change in dietary intake on measured blood lipid and lipoprotein levels.

Journal Article

The effect of a comprehensive weight reduction program on blood variables, body composition, and nutrient intake in obese black women.

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Footline: Metabolic effects of weight reduction.

Abstract

The effect of a comprehensive weight reduction program on selected blood variables, body composition, and nutrient intake was investigated in 56 obese black women, 22 to 51 years old. Subjects attended nutrition and behavior modification classes once per week for 3 months. Twenty subjects participated in a low impact aerobics program 3 times per week for 3 months. A 3.8 Kg average weight loss was observed. A 2.6% significant reduction in bodyfat (BF) was observed in the exercise group. Total cholesterol (TC) and low-density lipoprotein-cholesterol (LDL-C) fell significantly from 198 to 175 mg/dL, and 130 to 109 mg/dL, respectively, in the exercise group. High density lipoprotein₂-cholesterol (HDL₂-C) was lowered significantly from 24 to 17 mg/dL in the non-exercise group. Triglycerides, HDL-C, TC/HDL-C, glucose, thyroxin (T₄), and insulin values were not significantly altered. Significant reductions in protein fat, and cholesterol intake were reported. A comprehensive weight loss treatment promoted gradual weight loss, changes in body composition and food intake patterns, and significantly altered some blood lipid levels in obese black females.

Key words: Obesity, body composition, lipids, lipoproteins, insulin, behavior therapy, food habits

INTRODUCTION

The negative health implications associated with the obese condition have been well documented (1-3), yet little progress has been made in the development of a successful long term weight loss treatment to reduce the health related risks of these individuals. Most research has focused on the obese white population, while very little attention has been given to the black population. In particular, the black female is at a high risk for developing obesity in the adult years (4). According to data obtained from the Health and Nutrition Examination Survey II (NHANES II) (4), a greater percentage of black than white women are obese, regardless of age or income.

Treatments utilizing techniques for kilocalorie (kcal) restriction (5), exercise (6), and behavior modification (5) to promote weight loss have resulted in limited success (7). The adoption of a more integrated and comprehensive approach to the treatment of obesity, including classes on behavior modification and nutrition education, kcal restriction and increased physical activity can result in greater weight loss success, and the possibility of maintaining that weight loss due to the development of new food and activity habits (8). Evidence exist that obesity unfavorably affects health and longevity (50). Epidemiological studies indicate that the incidence of hypercholesterolemia and hyperinsulinemia are higher among obese than non-obese individuals, placing the obese person at a higher risk for the development of cardiovascular disease and non-insulin-dependent diabetes mellitus (NIDDM) (4). The present report describes the effects of an integrated program for

weight reduction on nutrient intake, blood lipids, plasma glucose, serum thyroxin and insulin, and body composition in healthy obese black females.

METHODS

Subjects

98 black obese females volunteered to participate in a 6 month weight loss program conducted at Virginia State University. This paper will report the progress of the subjects following the first 3 months of treatment. All potential subjects completed a medical history questionnaire and were examined prior to participation in the program. Subjects with medical contraindications (eg. coronary heart disease, diabetes mellitus, and uncontrolled hypertension) were excluded from the study. At the time of the 3 month measurement, 42 subjects had dropped out of the program leaving 56 subjects for evaluation. All subjects signed a consent form and the study was approved by the Virginia State University Human Subject Committee.

The physical characteristics of the subjects are presented in Table 1. The participants ranged from 22 to 51 years of age (mean age 36), were at least 20% above ideal body weight (Metropolitan Life Insurance Co. tables), and were between 25.6 and 59.1 on the body mass index (BMI) (Kg/m^2) (mean BMI 35.7). Their pretreatment body weights ranged from 65.6 to 149.7 kg. (mean body weight 92.4 kg). All subjects were residents of Chesterfield County.

Table 1.

Physical Characteristics of 56 Obese Black Female Subjects

	Mean $\pm$ SEM	Range Limits
Age, year	36.4 $\pm$ 0.14	22 - 51
Height, cm	160.9 $\pm$ 0.02	148.1 - 180.1
Body weight, kg	92.5 $\pm$ 2.2	65.6 - 149.7
BMI	35.7 $\pm$ 0.7	25.6 - 59.1

Procedures

Weight Reduction Classes

For 12 weeks, all subjects met with an instructor once per week for classes on nutrition education, behavior modification and stress management. The lessons were adapted from the Diet, Exercise and Behavior Modification (DEB) program (9). The class period lasted for two hours and included a weigh-in time, and group discussion, in addition to the scheduled lesson. The participants were given the opportunity to discuss problems they were having losing weight, and to hear problems that others were having. The lessons focused on educating the subjects to choose more nutritious foods, to count kcals, and on the behavioral skills necessary to lose weight. A daily intake of 1,200 kcal was recommended, but subjects monitored their own kcal intake and modified their diet as they wanted.

Exercise Program

Participation in the exercise program was voluntary, and depended on the ability of the subject to make it to the scheduled class time, and on the physicians' assessment of the physical examination. Subjects participated in a carefully supervised program of jogging, brisk walking, stretching and low impact aerobics, for one hour, three times per week, for ten weeks. The intensity of the exercise was monitored twice during each session by carotid artery pulse palpitation, and was maintained so that the subjects began the 10 week exercise program at approximately 55% of their age adjusted maximal heart rates, and gradually increased to 75%, over the duration of the program. An activity

questionnaire was administered to those subjects that did not participate in the exercise program on a regular basis in order to determine the level of unsupervised activity in those individuals.

Blood Samples

Once at the beginning of the study, before exercise and nutrition classes started, and again following the 3 month treatment, all subjects reported to a physician's office in the morning following a 12 hour fast. A 15 ml venous blood sample was obtained from each subject using vacutainers containing EDTA and held on ice until centrifuged at 3000 rpm for 20 min. The blood was divided for the determination of plasma total cholesterol (TC), triglycerides (TG), and glucose, serum insulin and thyroxin, and the fraction of TC contained in the high-density lipoprotein (HDL-C), and the subfraction of HDL-C, HDL₂-C. Plasma TC was determined using the Lieberman-Burchard colorimetric reaction (10), and plasma TG was determined enzymatically (11). Plasma glucose was measured directly using a glucose test kit (Stanbio Laboratory Inc., San Antonio, TX). Serum values for insulin and T₄ were determined using radioimmunoassay kits (Diagnostic Products Corporation, Los Angeles, CA). HDL-C was determined following heparin-MnCl₂ precipitation of very-lowdensity lipoproteins (VLDL-C) and low-density lipoproteins (LDL-C) (12). HDL₃-C was determined following dextran sulfate precipitation of the less dense HDL-subfraction, HDL₂-C (13). HDL₂-C was then calculated by subtracting the HDL₃-C from the total HDL-C value. The LDL-C was determined using the following

equation (14):

$$\text{LDL-C} = \text{TC} - (\text{TG}/5 + \text{HDL-C})$$

Anthropometric Measures

Body weight was determined at the beginning of the study, once per month during, and immediately following the treatment period using a Kg scale. Body composition measurements were not taken until one month following the beginning of the program due to a delay in receiving the bioelectrical impedance machine. Measurements were then taken once per month during the 3 month treatment period. Body weight and body composition measurements were taken on the same day during treatment.

Body composition of the subjects was determined using the four electrode bioelectrical impedance method using a bioelectrical impedance analyzer (Model BES 200Z, Bioelectrical Sciences, La Jolla, CA). A discussion of the principle of electrical impedance plethysmography is presented in a monograph by Nyboer (15). The subject was placed in a supine position on a dry nonconducting surface, and positioned so that no contact was made between limbs. The detector electrodes were placed on the distal side of a line bisecting the prominent bones of the right wrists, and between the lateral and medial malleoli at the ankle. The current electrodes were placed on the dorsal surfaces of the same hand and foot at the distal metacarpals and metatarsals, respectively. Electrolyte gel was applied to each electrode before application to the skin. The bioimpedance measurement was then determined by pressing the indicated button for two seconds or until a

stable number was obtained. Two readings were taken each time to assure that a reliable measurement was obtained. Prior to each bioimpedance measurement, each subject was administered a questionnaire to evaluate their hydration status and to approximate the day of their menstrual cycle. Due to possible water weight-gain immediately before and during the first day of the menstrual cycle, bioimpedance data from those particular individuals was not used.

Dietary Analysis

Two 24-hour food recalls were obtained prior to and following the treatment period. Both recall days were analyzed together using the Nutritionist II Computer Program (16), and divided by 2 to obtain a daily 24 hour intake.

Statistical Analysis

Pre- and post-treatment mean differences were analyzed statistically by paired t-test. Correlation coefficients were determined by the non-parametric Kendall Tau B analysis. Statistical significance was set prior to the study at $p < 0.05$.

RESULTS

Exercise Class Attendance

Subjects were divided into three exercise groups depending on their percent attendance at the exercise class. The exercise group consisted of 20 subjects that attended the exercise class at least 70%

of the time. An additional 14 subjects started the exercise program, but discontinued sometime during the 10 weeks, and did not attend 70% of the time. Because the subjects were exposed to some exercise (attended more than 30%, but less than 70% of the classes), they were placed in a "light" exercise group. The remaining 22 subjects were placed in the non-exercise group.

Behavior Modification and Nutrition Class Attendance

Subjects were divided into two nutrition groups depending on their attendance at the nutrition and behavior modification classes. This was done in order to evaluate the change in nutrient intake and food habits among those subjects that attended class on a regular basis, and those that did not. The first group included 40 subjects that attended the scheduled classes at least 80% of the time (Nutrition group 1). The remaining 16 subjects were placed in the second group (Nutrition group 2).

Anthropometric Means

Weight loss averaged 3.8 kg (4.1% of total body weight) for all subjects as one group, during the 3 month treatment (Table 2). The maximum weight lost was 14.5 kg (14.8%) and the maximum weight gain was 2.2 kg (2.8%). 15.6% of the subjects gained weight during treatment (average weight gain, 1.26 kg). Divided by age groups, subjects 22 to 29 years old lost the most weight, 5.2 kg, while subjects 30 to 39 years old experienced the least weight loss, 2.1 kg, and subjects 40 to 51 lost 4.0 kg.

Table 2.

Body Weight, Body Mass Index (BMI), and Body Composition Measurements During Weight Loss and Following Treatment in 56 Obese Black Females

	$\bar{X} \pm \text{SEM}$			
	Pre-treatment	1 month	2 month	Post-treatment
Total body weight (kg)	92.5 $\pm$ 2.2 ^a	90.9 $\pm$ 1.5	89.3 $\pm$ 1.6	88.7 $\pm$ 2.3 ^a
BMI (kg/m ²)	35.7 $\pm$ 0.7	35.1 $\pm$ 0.7	34.3 $\pm$ 0.7	34.3 $\pm$ 0.7
Lean body mass (%)	---	59.7 $\pm$ 0.6 ^b	61.6 $\pm$ 0.6	61.3 $\pm$ 0.6 ^b
Lean body mass (kg)	---	53.6 $\pm$ 1.8	54.1 $\pm$ 0.8	53.2 $\pm$ 0.7
Total body fat (%)	---	40.2 $\pm$ 0.6 ^c	38.3 $\pm$ 0.6	38.6 $\pm$ 0.6 ^c
Total body fat (kg)	---	36.9 $\pm$ 1.0 ^d	34.7 $\pm$ 1.1	34.6 $\pm$ 1.0 ^d
Total body water (%)	---	38.9 $\pm$ 0.5 ^e	40.3 $\pm$ 0.5	40.2 $\pm$ 0.5 ^e

a,b,c,d,e mean values with same letter are significantly different at $p \leq 0.005$

The differences in body weight within and between the three exercise groups are present in Table 3. The subjects in the exercise group lost an average of 4.3 kg (5.1%) over the three month period time ($p < 0.0005$). Subjects within the light exercise group demonstrated a 2.1 kg (2.3%) average total body weight loss over the 3 month period ($p < 0.005$). The nonexercise group lost 4.9 kg (5.3%) ($p < 0.05$). Looking at differences between groups showed that the exercise group was significantly lighter than the non-exercise group prior to and following treatment ($p < 0.02$).

The body composition parameters determined using the bioelectrical impedance analysis (BIA) technique during and following the weight loss program are presented in Table 2. An average reduction in body fat (BF) and corresponding increase in lean body mass (LBM) of 1.6% was observed. A significant 1.3% increase in percent total body water (TBW) was observed ($p < 0.05$). The BMI was used as a measure of obesity (17) and the change during weight reduction is seen in Table 2. BMI fell from 35.7 to 34.3 during treatment. A significant negative correlation was observed between BMI and BIA determined percent LBM ($r = -0.49$, $p < 0.001$) and TBW ($r = -0.50$, $p < 0.0001$), and a positive correlation with percent BF ($r = 0.50$, $p < 0.0001$). Total body weight was also negatively correlated with BIA determined percent LBM ($r = -0.50$, $p < 0.0001$) and TBW ($r = -0.50$, $P < 0.0001$), and positively correlated with percent BF ($r = 0.50$, $p < 0.001$).

The changes in body composition within the different exercise groups are presented in Table 3. A mean 2.6% and 1.5% reduction in BF was observed in the exercise and non-exercise groups, respectively.

Table 3.

Body Weight and Body Composition of Subjects Divided Into Exercise Groups During Treatment

	X $\pm$ SEM			
	Pre-Treatment	One Month	Two Months	Post-Treatment
EXERCISE GROUP (N=20)				
Total body weight (kg)	85.5 $\pm$ 3.6 ^{1a}	83.8 $\pm$ 2.5	82.8 $\pm$ 2.5 ¹	81.2 $\pm$ 3.6 ^b
Lean body mass (%)	—	61.2 $\pm$ 1.0 ^c	63.7 $\pm$ 1.0 ¹	63.8 $\pm$ 1.0 ^{1d}
Lean body mass (kg)	—	50.2 $\pm$ 1.3 ¹	51.7 $\pm$ 1.3 ³	50.0 $\pm$ 1.2 ³
Total body fat (%)	—	38.7 $\pm$ 1.0 ^e	36.2 $\pm$ 1.0 ⁷	36.1 $\pm$ 1.0 ^{6h}
Total body fat (kg)	—	32.5 $\pm$ 1.7 ^{3g}	30.0 $\pm$ 1.8 ⁹	28.8 $\pm$ 1.8 ^j
Total body water (%)	—	39.9 $\pm$ 0.8 ⁱ	41.9 $\pm$ 0.8	42.0 $\pm$ 0.8
LIGHT EXERCISE GROUP (N=14)				
Total body weight (kg)	94.9 $\pm$ 4.3 ^k	92.5 $\pm$ 3.0	90.6 $\pm$ 3.1 ^{1,2}	92.8 $\pm$ 4.4 ²
Lean body mass (%)	—	58.4 $\pm$ 1.2	61.1 $\pm$ 1.3 ^{3,4}	58.9 $\pm$ 1.2 ²
Lean body mass (kg)	—	53.6 $\pm$ 1.5	54.6 $\pm$ 1.5 ^{5,6}	53.9 $\pm$ 1.4 ^{3,4}
Total body fat (%)	—	41.5 $\pm$ 1.2 ⁴	38.8 $\pm$ 1.3 ⁸	41.0 $\pm$ 1.2 ⁶
Total body fat (kg)	—	38.8 $\pm$ 2.0	35.9 $\pm$ 2.2 ^{9,10}	38.5 $\pm$ 2.0
Total body water	—	37.7 $\pm$ 0.9	39.8 $\pm$ 1.0	38.2 $\pm$ 0.9
NON-EXERCISE GROUP (N=22)				
Total body weight (kg)	97.2 $\pm$ 3.4 ^{2m}	96.5 $\pm$ 2.4	94.3 $\pm$ 2.4 ²	92.3 $\pm$ 3.8 ⁿ
Lean body mass (%)	—	59.5 $\pm$ 0.9 ²	60.0 $\pm$ 1.0 ⁴	61.0 $\pm$ 1.0 ^{1,2}
Lean body mass (kg)	—	56.8 $\pm$ 1.2 ²	56.0 $\pm$ 1.2 ⁶	55.8 $\pm$ 1.1 ⁴
Total body fat (%)	—	40.4 $\pm$ 0.9 ^{4,0}	39.8 $\pm$ 1.0 ⁸	38.9 $\pm$ 1.0 ^{6p}
Total body fat (kg)	—	39.5 $\pm$ 1.6	38.3 $\pm$ 1.7 ¹⁰	36.4 $\pm$ 1.7
Total body water (%)	—	38.7 $\pm$ 0.7	39.2 $\pm$ 0.8	39.9 $\pm$ 0.8

a-p = mean values within rows with different letters are significantly different at $p < 0.05$.

1-10 = mean values within columns for a single variable with different numbers are significantly different at $p < 0.05$.

Using the total body weight value measured at one month, the exercise group lost 2.6 Kg by the end of treatment, which corresponded to a significant reduction in BF weight of 3.7 Kg ($p < 0.0002$) (Table 3). A 4.2 Kg total body weight reduction was seen from one month until the end of treatment for the non-exercise group. The total body weight loss was reflected by a significant 3.1 Kg loss of BF ($p < 0.05$) and a slight but not significant 1.0 Kg reduction in LBM. The light exercise group maintained about the same percent BF and LBM composition from one month to the end of treatment. From month one to two, the light exercise group showed a reduction of BF and increase in LBM of 2.7%. BF and LBM values returned to month one values at the end of treatment. This trend appeared to follow the change in total body weight, which fell 1.9 kg from month one to two, and returned to month one mean values at the time of the three month measurement.

Blood Variables

The effect of weight reduction on the blood lipids and lipoproteins is presented in Table 4. Plasma total cholesterol (TC) levels fell 19 mg/dL (10.3%) from 203 to 184 mg/dL ($p < 0.005$). A similar and significant decrease in LDL-C from 134 to 116 mg/dL (-18 mg/dL, 13.4%) was observed ($p < 0.05$), which closely paralleled the fall in TC levels. HDL₂-C levels were also significantly reduced from 25.9 to 20.2 mg/dL ($p < 0.0005$). Plasma triglycerides (TG), HDL-C and TC/HDL-C ratio were not significantly different.

BMI was used to assess the association between obesity and plasma

Table 4.

Mean Blood Values for Fasting Lipids and Lipoproteins, Glucose, Insulin, and Thyroxin Before and After Weight Reduction in Obese Black Females

	$\bar{X} \pm \text{SEM}$		P value
	Pre-treatment n = 56	Post-treatment n = 53	
Total cholesterol (mg/dL)	203 $\pm$ 4.8	184 $\pm$ 4.8	<0.005
Triglycerides (mg/dL)	98.8 $\pm$ 8.0	93.0 $\pm$ 8.0	NS
HDL-C (mg/dL)	49.6 $\pm$ 1.7	48.3 $\pm$ 1.7	NS
HDL ₂ -C (mg/dL)	25.9 $\pm$ 1.1	20.2 $\pm$ 1.1	<0.0005
LDL-C (mg/dL)	134 $\pm$ 4.4	116 $\pm$ 4.5	<0.05
TC/HDL-C ratio	4.2 $\pm$ 0.02	4.0 $\pm$ 0.02	NS
Glucose (mg/dL)	96.4 $\pm$ 3.0	91.8 $\pm$ 3.0	NS
Insulin (uIU/mL)	23.4 $\pm$ 1.8	20.3 $\pm$ 1.7	NS
Thyroxin (ug/dL)	8.8 $\pm$ 0.31	8.6 $\pm$ 0.30	NS

NS = not significantly different

lipid levels. The only significant correlation was between initial mean BMI and HDL-C levels ($r = -0.19$, $p < 0.05$). Initial total body weight was also correlated with initial HDL-C values ($r = -0.24$, $p < 0.01$) but the significant relation was not present following weight loss.

The plasma lipid changes among the three exercise groups before and after treatment are shown in Table 5. A significant change in plasma TC and LDL-C was evident only for the subjects in the exercise group ($p < 0.05$). A reduction of 23 mg/dL (13.0%) and 21 mg/dL (19.3%) for TC and LDL-C levels, respectively, was seen in the group that attended greater than 70% of the exercise classes. A nonsignificant 20 mg/dL fall in both plasma TC (9.7%) and LDL-C (14.7%) was observed in the group of subjects ($n = 14$) who inconsistently attended the exercise class. A 16 mg/dL (9.5%) and 12 mg/dL (11.5%) reduction in TC and LDL-C was observed in the non-exercise group. HDL-C levels remained relatively stable within the exercise and light exercise groups, but fell slightly (3.9 mg/dL) in the non-exercise group. HDL₂-C decreased significantly 7.4 mg/dL (43.8%) in the non-exercise group ($p < 0.05$), decreased 5.8 mg/dL in the light exercise group, which approached significance ($p < 0.06$), and was not significantly reduced in the exercise group (-3.8 mg/dL, 18%). No significant changes in TG were observed for any group. No change in the TC/HDL-C ratio was observed for any group after weight loss. Differences between exercise groups included significantly higher plasma TC levels within the light exercise groups for both pre- and post-treatment measurements ($p < 0.05$).

The plasma lipid changes among the two nutrition groups are

Table 5.

Mean Blood Values for Fasting Lipids and Lipoproteins, Glucose, Insulin, and Thyroxin Between Exercise Groups Before and After Weight Reduction In Obese Black Females

	X $\pm$ SEM					
	Exercise group (n=20)		Light exercise group (n=14)		Non-exercise group (n=22)	
	pre-treatment	post-treatment	pre-treatment	post-treatment	pre-treatment	post-treatment
Total cholesterol (mg/dL)	198 $\pm$ 7.8 ^a , ^a	175 $\pm$ 7.8 ^a , ^c	227 $\pm$ 9.4 ^a , ^b	207 $\pm$ 9.4 ^c , ^d	184 $\pm$ 7.5 ^b	168 $\pm$ 7.5 ^d
Triglycerides (mg/dL)	84.0 $\pm$ 13.2	79 $\pm$ 13.2	107 $\pm$ 15.8	96 $\pm$ 15.8	106 $\pm$ 12.6	104 $\pm$ 12.6
HDL-C (gm/dL)	51.0 $\pm$ 2.7	50.2 $\pm$ 2.7	50.2 $\pm$ 3.3	51.2 $\pm$ 3.4	47.4 $\pm$ 2.6	43.5 $\pm$ 2.6
HDL ₂ -C (mg/dL)	24.9 $\pm$ 1.8	21.1 $\pm$ 1.8	28.6 $\pm$ 2.2	22.8 $\pm$ 2.2	24.3 $\pm$ 1.8 [*]	16.9 $\pm$ 1.7 [*]
LDL-C (mg/dL)	130 $\pm$ 7.3 [*]	109 $\pm$ 7.3 [*]	156 $\pm$ 8.6	136 $\pm$ 9.0	116 $\pm$ 6.9	104 $\pm$ 6.9
TC/HDL-C ratio	3.9 $\pm$ 0.07	3.5 $\pm$ 0.06	4.5 $\pm$ 0.10	4.0 $\pm$ 0.09	3.9 $\pm$ 0.06	3.9 $\pm$ 0.06
Glucose (mg/dL)	92 $\pm$ 4.9	85 $\pm$ 4.9	99 $\pm$ 5.9	89.5 $\pm$ 5.9	98 $\pm$ 4.7	101 $\pm$ 4.7
Insulin (uIU/mL)	18.3 $\pm$ 2.7 ^e	15.7 $\pm$ 2.8 ^f	21.9 $\pm$ 3.8	21.4 $\pm$ 3.3	30.1 $\pm$ 2.6 ^e	23.8 $\pm$ 2.6 ^f
Thyroxin (ug/dL)	8.8 $\pm$ 0.51	8.4 $\pm$ 0.48	8.8 $\pm$ 0.63	8.5 $\pm$ 0.58	8.7 $\pm$ 0.48	8.8 $\pm$ 0.46

* pre- and post-treatment means significantly different at p < 0.05.

a-f = mean values with same superscript are significantly different at p < 0.05.

presented in Table 6. Significant changes in plasma TC, LDL-C, and HDL₂-C levels were observed in both nutrition groups. An 18 mg/dL (9%) and 24 mg/dL (12%) reduction in TC levels was observed for the first and second groups, respectively. The reductions in TC were closely paralleled by the drop in LDL-C of 15 mg/dL (11.6%) and 25 mg/dL (18%) for the two groups respectively. Similar reductions in HDL₂-C levels of 6 mg/dL (24.1%) and 5.5 mg/dL (20.1%) for the two groups, respectively, was seen, but no difference in plasma HDL-C and TG were observed for both groups. The TC/HDL-C value remained approximately the same during weight loss for both groups.

Change in glucose, insulin, and T₄ levels during the weight reduction program for all subjects are shown in Table 4. Values for all three variables from the beginning to the end of the study were not significantly different. Significant correlations between initial BMI and glucose ($r = 0.26$, $p < 0.05$) and insulin ($r = 0.38$, $p < 0.0001$) values were present. A negative correlation between BMI and thyroxine T₄ approached significance ($r = -0.18$, $p < 0.06$). Following weight loss, positive correlation between BMI and glucose ($r = 0.33$, $p < 0.001$), insulin ($r = 0.36$, $p < 0.0005$), and a negative correlation with T₄ ($r = -0.23$, $p < 0.05$) was seen. Body weight was also correlated with glucose ($p < 0.05$), and insulin ($p < 0.0005$) before and after treatment. Pre-treatment fasting insulin was significantly correlated to fasting glucose ($r = 0.37$, $p < 0.0001$) and triglycerides ($r = 0.22$, $p < 0.05$). Post-treatment fasting insulin was significantly correlated to fasting glucose ($r = 0.28$, $p < 0.005$) and triglycerides ($r = 0.20$, P

Table 6.

Mean Blood Values for Fasting Lipids and Lipoproteins, Glucose, Insulin, and Thyroxin Between Nutrition Groups Before and After Weight Reduction in Obese Females

$\bar{X} \pm \text{SEM}$				
	Nutrition group 1 (n=40)		Nutrition group 2 (n=16)	
	pre-treatment	post-treatment	pre-treatment	post-treatment
Total Cholesterol (mg/dL)	198 $\pm$ 6.1*	180 $\pm$ 6.1*	205 $\pm$ 9.6*	181 $\pm$ 9.6*
Triglycerides (mg/dL)	104 $\pm$ 9.3	96.1 $\pm$ 9.3	82.3 $\pm$ 14.8	85.7 $\pm$ 14.8
HDL-C (mg/dL)	48.4 $\pm$ 1.9	46.7 $\pm$ 2.0	51.8 $\pm$ 3.1	46.7 $\pm$ 2.0
HDL ₂ -C (mg/dL)	24.9 $\pm$ 1.3*	18.9 $\pm$ 1.3*	27.4 $\pm$ 2.1*	21.9 $\pm$ 2.0*
LDL-C (mg/dL)	129 $\pm$ 5.5*	114 $\pm$ 5.6*	138 $\pm$ 8.8*	113 $\pm$ 8.8*
TC/HDL-C ratio	4.1 $\pm$ 0.05	3.8 $\pm$ 0.06	4.0 $\pm$ 0.06	3.9 $\pm$ 0.06
Glucose (mg/dL)	97 $\pm$ 3.5	95 $\pm$ 3.5	92.9 $\pm$ 5.6	86.4 $\pm$ 5.6
Insulin (uIU/mL)	24.3 $\pm$ 2.1*	19.8 $\pm$ 2.1*	21.3 $\pm$ 3.4	21.5 $\pm$ 3.2
Thyroxin (ug/dL)	8.5 $\pm$ 0.35	8.4 $\pm$ 0.33	9.6 $\pm$ 0.56	9.0 $\pm$ 0.51

* pre- and post-treatment means significantly different at $p \leq 0.05$.

< 0.05), but the correlation was not as strong as the pre-treatment correlation.

The changes in insulin, glucose, and T₄ within the different exercise groups are shown in Table 5. No significant change was seen for fasting glucose or insulin levels, within any group. Serum T₄ levels remained stable within all groups over time. Within the exercise group, initial insulin and triglyceride values were significantly correlated ($r = 0.39$, $p < 0.05$). The insulin and glucose values within the non-exercise group were significantly correlated during both initial ($r = 0.44$, $p < 0.005$) and post-treatment ($r = 0.34$, $p < 0.05$) measurements.

A closer look at the data reveals that 15.6% ($n=8$) of the subjects had high insulin levels prior to treatment. The average initial fasting insulin levels was 50.1 uIU/mL (range 36.8 to 98.7 uIU/mL) for this subgroup. Initial body weight of this subgroup was 101.6 kg, which was 9.1 kg above the mean body weight for the entire sample. An average weight loss of 4.3 kg was observed for this subgroup, which was accompanied by a 16 uIU/mL fall in insulin levels to 34.0 uIU/mL (range 14.6 to 51.4 uIU/mL). Only 5.7% of the entire sample had elevated insulin values following weight loss. In addition, preliminary values for fasting triglycerides and glucose within this subgroup were 151 mg/dL, and 110 mg/dL, respectively. Following weight loss, triglyceride and glucose levels were 121 mg/dL and 134 mg/dL, respectively. The increase in the mean glucose levels can be explained by one of the subjects who had high initial insulin and glucose and experienced a large drop in insulin and a subsequent doubling of her glucose level. The

subject may have initially been prediabetic. Without this subject's glucose values included, the mean glucose values for this subgroup decreased from 105 to 97 mg/dL.

Nutrient intake means

The mean values of the nutrient intakes before and after treatment are shown in Table 7. Kcal intake was significantly reduced by 503 ± 72 kcal per day. Significant reductions in subject intake of total protein, total carbohydrate, total fat, saturated fat, monounsaturated fat, polyunsaturated fat, dietary cholesterol, sodium, and iron were reported.

Table 8 presents the nutrient intake difference between and within the two groups. Both groups reported similar reductions in kcal intake of 510 and 498 kcal per day, respectively ($p < 0.005$). This similar reduction in kcal intake reported by the subjects did not coincide with the measured weight loss, which differed between the two groups. A 5.4 ± 2.7 weight loss was noted for group 1, which was higher than group 2, which only experienced a mean 2.0 ± 4.3 kg weight loss. Compared to group 2, group 1 consumed significantly less total grams of monounsaturated fat, and polyunsaturated fat, and had significant reductions in the percent energy intake derived from fat sources between pre- and post-treatment food recalls ($p < 0.05$). Both groups reported reductions in grams of total and saturated fat consumed ($p < 0.05$). With respect to correlations between nutrient intakes and blood lipids, there was a relatively low, but significant negative correlation between dietary fiber intake and plasma TC ($r = -0.19$, $p < 0.05$) and LDL-C ($r = -0.20$, $p < 0.05$) levels for all subjects as one group, following treatment.

Table 7.

Energy and Nutrient Intakes Before and After Treatment in Obese Black Females on a Weight Reduction Program

	$\bar{X} \pm \text{SEM}$		P value
	Pre-treatment n = 56	Post-treatment n = 53	
Energy (Kcal)	1693 $\pm$ 70	1190 $\pm$ 75	<0.0001
Nutrients sources (g)			
Total protein	62.4 $\pm$ 2.7	52.6 $\pm$ 2.8	<0.05
Total carbohydrate	189.6 $\pm$ 6.7	131.5 $\pm$ 7.8	<0.05
Total fat	76.1 $\pm$ 3.6	45.7 $\pm$ 3.6	<0.0001
Saturated fatty acids	25.5 $\pm$ 1.2	14.4 $\pm$ 1.2	<0.0001
Monounsaturated fatty acids	26.6 $\pm$ 1.5	14.7 $\pm$ 1.5	<0.0001
Polyunsaturated fatty acids	13.4 $\pm$ 0.97	8.0 $\pm$ 0.98	< 0.005
P/S ratio	0.55 $\pm$ 0.006	0.57 $\pm$ 0.007	NS
M/S ratio	1.04 $\pm$ 0.005	1.07 $\pm$ 0.008	NS
Percent daily energy intake			
Protein	15.1 $\pm$ 0.7	19.5 $\pm$ 0.8	<0.002
Fat	40.3 $\pm$ 1.3	36.1 $\pm$ 1.3	NS
Carbohydrate	44.9 $\pm$ 1.4	44.2 $\pm$ 1.4	NS
Fiber (g)	3.3 $\pm$ 0.3	3.5 $\pm$ 0.3	NS
Cholesterol (mg)	258 $\pm$ 15	200 $\pm$ 15.6	<0.01
Vitamins			
Vitamin A (IU)	2484 $\pm$ 294	3026 $\pm$ 302	NS
Vitamin B6 (mg)	0.90 $\pm$ 0.64	0.85 $\pm$ 0.65	NS
Folacin (ug)	128 $\pm$ 9.2	140 $\pm$ 9.4	NS
Minerals			
Sodium (mg)	2254 $\pm$ 131	1679 $\pm$ 135	<0.05
Magnesium (mg)	137 $\pm$ 8.5	133 $\pm$ 8.8	NS
Iron (mg)	9.5 $\pm$ 0.4	7.7 $\pm$ 0.4	<0.002
Zinc (mg)	5.9 $\pm$ 0.4	5.5 $\pm$ 0.4	NS
Calcium (mg)	387 $\pm$ 27	374 $\pm$ 28	NS

NS = not significantly different

Table 8.

Energy and Nutrient Intakes Between Nutrition Groups Before and After Treatment
In Obese Black Females on a Weight Reduction Program.

$\bar{X} \pm \text{SEM}$				
		Nutrition group 1 (n=40)		Nutrition group 2 (n=16)
		pre-treatment	post-treatment	pre-treatment post-treatment
Energy (Kcal)		1602 $\pm$ 74 ^a	1092 $\pm$ 74 ^a	1786 $\pm$ 118 ^b 1288 $\pm$ 130 ^b
Nutrient source (g)				
Total protein		61.2 $\pm$ 3.1	51.1 $\pm$ 3.1	66.7 $\pm$ 4.9 57.1 $\pm$ 5.5
Total carbohydrate		171 $\pm$ 7.0	126 $\pm$ 6.7	195 $\pm$ 7.2 128 $\pm$ 7.2
Total fat		74.9 $\pm$ 0.64 ^c	42.8 $\pm$ 0.49 ^c	82.3 $\pm$ 2.0 ^d 61.0 $\pm$ 2.6 ^d
Saturated fat		25.3 $\pm$ 0.23 ^e	13.1 $\pm$ 0.18 ^e	27.3 $\pm$ 0.70 ^f 19.9 $\pm$ 0.90 ^f
Monounsaturated fat		26.2 $\pm$ 0.29 ^g	13.5 $\pm$ 0.18 ^g	28.1 $\pm$ 0.78 20.7 $\pm$ 1.03
Polyunsaturated fat		13.5 $\pm$ 0.22 ^h	6.6 $\pm$ 0.10 ^h	14.2 $\pm$ 0.55 10.6 $\pm$ 0.48
P/S ratio		0.56 $\pm$ 0.009	0.56 $\pm$ 0.009	0.54 $\pm$ 0.02 0.60 $\pm$ 0.02
M/S ratio		1.05 $\pm$ 0.007	1.08 $\pm$ 0.012	1.02 $\pm$ 0.01 1.01 $\pm$ 0.01
Percent daily energy Intake				
Protein		14.9 $\pm$ 0.10 ⁱ	19.6 $\pm$ 0.18 ⁱ	15.2 $\pm$ 0.17 18.3 $\pm$ 0.52
Carbohydrate		45.5 $\pm$ 0.22	45.4 $\pm$ 0.30	43.7 $\pm$ 0.66 42.3 $\pm$ 0.67
Fat		39.8 $\pm$ 0.19 ^j	34.8 $\pm$ 0.25 ^j	41.3 $\pm$ 0.59 39.5 $\pm$ 0.83
Fiber (g)		3.4 $\pm$ 0.005	3.5 $\pm$ 0.06	3.3 $\pm$ 0.11 3.7 $\pm$ 0.13
Cholesterol (mg)		220 $\pm$ 2.32	185 $\pm$ 2.50	329 $\pm$ 9.3 243 $\pm$ 8.4

a-j = means with same letter are significantly different at $p < 0.05$

DISCUSSION

It has been suggested that an integrated approach to attain weight loss, including diet, exercise, and behavior modification may be more effective in the treatment of obesity than more traditional methods (8). In the present study, the subjects experienced an average weight loss of 3.8 kg during three months of treatment which incorporated diet, exercise, behavior modification, and nutrition education. This average weight loss was lower than weight losses reported by many other investigators which attempted treatment using one or a combination of two methods (7). Follick et al (18) observed a similar weight loss of 4.2 kg in their group of female subjects that underwent a ten-week behavior modification program without exercise.

The subjects in this study who did not exercise lost slightly more weight (-4.9 kg) than the subjects that exercised on a regular basis (-4.3 kg), and considerably more than those that inconsistently attended exercise class (-2.1 kg). The composition of weight loss in the non-exercise group was approximately 66% body fat (BF) and 33% lean body mass (LBM). This is similar to the recommended composition of weight loss (75% BF, 25% LBM) (20). The exercise aided in the maintenance of LBM in the exercise group during weight loss and was composed of 95% BF and 5% LBM. The light exercise group experienced no total body weight or body composition changes during the period that body composition was measured. Similar results were reported by Lewis et al (21) who studied the effects of exercise two times per week for 17 weeks on weight loss. A loss of 4.2 kg total body weight, which

corresponded to a 5.4 kg loss of BF and 1.2 kg increase in LBM, was observed in 22 obese females. Following a 12 week study, Hagan et al (6) concluded that diet and exercise, when compared to diet alone, produced greater reductions in total body weight and BF.

According to Garrow (22), a BMI over 25 was indicative of BF in excess of 35%, assuming that the overweight condition was due to overfatness and not overmusculature. Our subjects had a mean BMI value above 35, which corresponded to a BIA determined BF of 40.2%. Use of the BIA technique is relatively new, and its use in measuring body composition in obese populations during stable weight or weight loss conditions has been criticized (23). In addition, we are unaware of any studies that have utilized this technique in black-normal or overweight-populations. Nonetheless, a reliable method for determining body composition changes in obese individuals undergoing weight loss was necessary in order to monitor loss of BF and/or LBM. In obese white populations, determination of LBM (24, 25), TBW (26), and BF (27) by BIA has compared well with more conventional anthropometric indices. Segal et al (25) found BIA determined % LBM to compare well with densitometrically determined % LBM in male and female subjects who ranged in % BF from 4.9 to 54.9%. More recently, Kushner and Haas (24) found that LBM determined by BIA compared well to LBM determined by skinfold thickness, with less than 1.1% difference between values, in lean, normal weight, and obese subjects during stable weight and weight loss conditions. Johnson et al (27) studied changes in BF during weight loss and found that the BIA determined BF changes were similar

to changes detected by arm circumference, skinfold thickness, and hydrodensitometry. Within our subjects, BMI was significantly inversely correlated to BIA determined percent LBM ($r = -0.49$, $p < 0.001$) and TBW ($r = -0.49$, $p < 0.0001$), and correlated positively with percent BF ($r = 0.50$, $p < 0.0001$). Other investigators have studied the correlation between the body composition variables and more conventional anthropometric measures. The significant correlations between the BMI value and the BIA determined body composition measurement in this study, suggested that this instrument accurately measured the body composition of obese black as well as obese white populations during weight loss.

The condition of obesity has been observed to negatively influence blood lipids (28) and thus increase the risk of cardiovascular disease in obese individuals (32,33). Risk of cardiovascular disease was positively related to high levels of plasma TC and LDL-C, and was negatively associated with HDL-C levels (29). A similar blood lipid profile has been observed in the obese person in other studies (3, 28, 29, 30, 31). In contrast, the mean lipid values within the subjects of the present study were within normal ranges, although much variation existed within and between the exercise groups.

The effect of weight loss on plasma lipid and lipoprotein levels has produced inconsistent results. In addition, the effect of physical activity in initially sedentary individuals on the status of blood lipids has been difficult to establish (34). Our results indicated that a reduction in body weight of 3.8 kg resulted in significantly lower TC, and LDL-C levels. When divided by exercise groups significant

reductions were observed only for the subjects that exercised consistently. No significant changes were observed for the light and non-exercise groups. The reductions in plasma TC and LDL-C levels observed in this study have not been reported by some investigators (6,21) despite similar pretreatment values, similar reductions in total body and fat weight, and subject participation in an exercise program. On the other hand, Follick et al (18) observed significant reductions in both plasma TC and LDL-C levels following weight loss unaccompanied by exercise.

Reduced plasma triglycerides have been reported in initially inactive individuals participating in an aerobic type exercise program (35) and during weight loss (36). Most often, this phenomenon occurred in subjects with initially elevated plasma triglyceride levels. The subjects on this study did not have extremely high pretreatment plasma triglyceride values, and no change was observed. Previous investigators have reported similar results (6,18,21) with exercise and weight loss. Lewis et al (21) suggested that weight loss which resulted from increased physical activity and kcal restriction did not lower plasma triglyceride levels in subjects whose values were within normal range.

Recent evidence has indicated that evaluation of cardiovascular disease risk was improved by the determination of the TC distribution among the different lipoprotein classes (29). A strong inverse association was present between levels of plasma HDL-C and risk of cardiovascular disease (29) and, more recently, evidence indicated that the HDL₂-C subfraction may possess the antiatherogenic qualities

associated with the HDL-C particle (37). No reductions were seen in plasma HDL-C levels for any exercise group. HDL₂-C levels were reduced significantly only in the non-exercise group (-7.4 mg/dL). No significant reductions in HDL₂-C were observed in the light exercise (-5.8 mg/dL) and exercise (-3.8 mg/dL) groups. No improvements were seen in the TC/HDL-C ratio in any of the exercise groups.

Research has indicated that higher HDL-C and HDL₂-C levels exist among active individuals than sedentary individuals (34,35). It has not been determined whether or not this phenomenon occurred as a result of exercise or some other factor (38), and it has not been determined whether increased exercise in normally sedentary individuals will elicit an increase in the HDL-C and/or HDL₂-C particle. Morgans et al (38) reported no significant change in HDL-C in five initially sedentary, normal-weight subjects who participated in an endurance type exercise program, five days per week for six months. In 16 healthy, normal-weight, sedentary women, Frey et al (39) similarly found no improvement in levels of HDL-C, despite changes in fitness, following ten weeks of aerobic exercise, three times per week.

In studies which have evaluated the effect of physical activity accompanied by weight loss, similar observations have been reported (6,18,21). Follick et al (18) noted that both the HDL-C levels and the HDL-C/LDL-C ratio increased significantly above pretreatment levels at six months following weight loss, although immediately following weight loss no significant change was detected. Lewis et al (21) reported an elevated HDL-C/LDL-C ratio directly following weight loss, yet no

significant increase in HDL-C levels were observed. In contrast, Sopko et al (40) found that both exercise and weight loss independently raised HDL-C levels in obese men. Significantly reduced HDL-C and HDL₂-C levels were observed in 30 individuals who lost weight rapidly (41). The authors found no correlation between variation of HDL-C and change in body weight, and speculated that the decrease in HDL-C and HDL₂-C was due to kcal restriction, rather than weight loss, and may have reflected a reduced formation of HDL-C particles, particularly HDL₂-C. The significantly reduced HDL₂-C level among the non-exercise group in the present study suggested that exercise may have protected the HDL₂-C molecule against the effects of kcal restriction during weight loss.

The mean serum insulin levels for the subjects of this study were within normal range, thus large reductions in insulin values were not expected for the subjects as one group, or for any of the exercise groups. Within the exercise groups, slight reductions in serum insulin levels were observed for the exercise and non-exercise groups (-6.3 uIU/ml), respectively. The non-exercise group had significantly higher pretreatment insulin values compared to the exercise group. No reduction in serum insulin occurred in the light exercise group (-0.5 uIU/ml). Among obese individuals with hyperinsulinemia, both kcal restriction and increased physical activity have resulted in reduced serum insulin levels, increased insulin receptor concentrations, and increased sensitivity to insulin in peripheral tissues (42,43,44). It appeared that both consistent, regular exercise and substantial weight

loss resulted in lowering the serum insulin levels, although it was not significant. Neufeld et al (43) reported that fasting insulin levels fell to normal levels following weight loss in 10 obese individuals. In the present study, when only those subjects (n=8) with high pretreatment serum insulin levels (mean 50 uIU/ml) were observed, a 16 uIU/ml reduction in fasting insulin values was seen. Thus these results supported previous investigations (43,44,45) and substantiate the importance of weight loss and exercise in obese individuals with high insulin levels to establish normal insulin levels.

Several investigators have found insulin levels positively correlated to levels of blood glucose (3, 45), plasma triglycerides (3, 45, 46), and inversely related to HDL-C levels (46) in obese individuals. Zavaroni et al (46) reported a significant negative relationship between one- and two-hour plasma insulin levels and HDL-C levels in female subjects, but no relationship was observed for fasting insulin levels. We similarly did not observe a significant correlation between HDL-C and fasting insulin levels. Previous studies (3,45,46) have observed serum insulin levels to correlate significantly to degree of hypertriglyceridemia, degree of obesity, and level of glucose tolerance. Zavaroni et al (46) noted the strongest relationship was between the degree of hyperinsulinemia and hypertriglyceridemia. Our results indicated that fasting insulin and fasting plasma glucose ($r = 0.37$) demonstrated the strongest correlation between blood variables. Body weight and BMI have been reported significantly correlated to serum insulin, and HDL-C levels (3,46). We similarly observed that body

weight and BMI both correlated significantly to both fasting serum to both fasting serum insulin ($r = 0.38$) and plasma HDL-C ($r = -0.19$) levels. Our results and those of other researchers have demonstrated the interrelationship present among many of the endocrine and metabolic characteristics which comprise the biochemical profile observed in the obese individual.

Kcal restriction accompanied by behavior modification and nutrition education is believed to promote weight loss more successfully by promoting new and more healthy food habits, and offering the individual the skills necessary to maintain the weight loss following treatment. Our results indicated that those subjects that attended the nutrition and behavior modification classes regularly experienced a greater weight loss than those subjects that attended on an inconsistent basis. In addition, although both groups consumed significantly less grams of total fat, only those individuals who consistently attended class were successful at significantly reducing their percent daily energy intake from fat.

The effect of weight loss on the blood variables in obese individuals have yielded confusing and nonconclusive results. In addition to degree of weight loss, rate of weight loss, pre-treatment blood levels, and physical activity, altering the diet composition may have resulted in blood lipid and lipoprotein changes. A review of the literature revealed that high plasma TC levels were often seen with high intakes of total and saturated fats, and less consistently with high intakes of dietary cholesterol (47, 48). However, we observed significant

negative correlations between plasma TC levels and dietary total and saturated fat intake. No correlation between TC and dietary cholesterol was observed. We also noted significant positive correlations for plasma HDL-C levels and dietary total fat, saturated fat, monounsaturated fat intake, percent energy intake as fat, and a negative correlation with fiber intake. Other investigators have reported similar relations between HDL-C and fat intake (47, 49) although Grundy et al (47) reported that consumption of monounsaturated fatty acids did not affect HDL-C levels. Most of the studies that evaluated the role of food nutrients on blood lipid and lipoprotein levels have controlled for body weight, physical activity, and other factors known to influence these blood levels. This study incorporated the effects of weight loss, increased physical activity, and altered food intake patterns on blood lipid and lipoprotein levels. Thus it is not surprising that the many factors present would act to alter the effect of any one of the factors on the blood values. The present study demonstrated that moderate kcal restriction in conjunction with consistent attendance at nutrition and behavior modification classes was successful in promoting gradual weight loss, positive alterations of body composition, and beneficial changes in nutrient intake in obese adult black women. In addition, the consistent participation in a supervised exercise program aided in minimizing the loss of LBM while promoting loss of BF, and positively influenced the blood lipid and lipoprotein profile of those individuals.

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SUMMARY AND CONCLUSIONS

The present study was conducted to evaluate the effects of an integrated approach to weight loss on nutrient intake, blood lipids, plasma glucose, serum insulin and thyroxin, and body composition in black obese females. Fifty-six women, ages 22 to 51, and at least 120% of ideal body weight participated in this 3 month weight loss program. Forty of the subjects met once a week and attended at least 80% of the lessons on nutrition and behavior modification. Sixteen of the subjects attended these classes fewer than 80% of the time and were placed in a second group for comparison. Out of the 56 subjects, 20 participated regularly in a supervised low impact aerobic exercise class program three times per week, 14 subjects attended the exercise on an irregular basis, and 22 of the subjects attend no exercise class. Body weight was measured monthly, and body composition was determined monthly beginning one month after the program began, using the bioelectrical impedance analysis (BIA) method. Plasma lipids, plasma glucose, serum insulin and thyroxin were each measured prior to and immediately following the 3 month treatment. Twenty four-hour dietary recalls were used to measure nutrient intakes before and after treatment.

An average weight loss of 3.8 kg (4.1% of total body weight) was observed following the 3 month treatment. The subjects in the exercise group lost an average of 4.3 kg (5.1%), subjects in the light exercise group had a mean weight loss of 2.1 kg (2.3%), and subjects in the non-exercise group lost 4.9 kg (5.3%). From the one month body weight

and body composition measurement, the exercise group maintained a stable lean body mass (LBM)(kg) and lost significant BF (-3.7 kg). The non-exercise group showed significant weight loss which corresponded to 66% fat and 33% LBM (-3.1 kg BF, -1.0 Kg LBM). The light exercise group lost weight and BF at the two month measurement, but returned to one month levels immediately following treatment and thus no change occurred. For all 56 subjects as one group, significant correlations were observed between body mass index (BMI) and percent LBM ($r=-0.5$), TBW ($r=-0.5$), and BF ($r=0.5$) determined by BIA.

Significant reductions in plasma total cholesterol (TC) (23 mg/dl) and low-density lipoprotein cholesterol (LDL-C) (21 mg/dl) were present in the exercise group after weight loss. A nonsignificant decrease of 20 mg/dl for both TC and LDL-C was observed in the light exercise group, and 16 mg/dl and 12 mg/dl decreases were observed for TC and LDL-C, respectively, in the non-exercise group. Levels of high-density lipoprotein₂-cholesterol (HDL₂-C) were significantly depressed in the non-exercise group (-7.4 mg/dl), and approached significance in the light exercise group (-5.8 mg/dl). No significant change in plasma triglycerides (TG), TC/HDL-C ratio, glucose, or serum insulin and thyroxin was seen for any exercise group. Within the exercise groups, HDL-C levels remained relatively stable. No differences were seen between the two nutrition group blood lipid responses to weight loss. Both groups had significant reductions in plasma TC, LDL-C, and HDL₂-C levels. In subjects with elevated pretreatment insulin levels ($\bar{x} = 0.50$ uUI/mL) a reduction of fasting insulin levels of 16 uUL/mL was observed following weight loss in this group.

Significant correlations between initial BMI and glucose ($r=0.26$) and insulin values ($r=0.38$) were seen. Following weight loss a significant negative correlation was present between BMI and T_4 ($r=-0.23$) levels. Fasting insulin levels were correlated significantly to fasting glucose ($r=0.37$) and triglycerides ($r=0.22$).

Subjects reported a reduction in kcal intake of 503 kcal/day. Significant reductions in subject intake of total protein, total fat, total carbohydrate, saturated fat, monounsaturated fat, polyunsaturated fat, dietary cholesterol, sodium, and iron were reported. Compared to nutrition group 2, group 1 consumed significantly less total grams of protein, monounsaturated fat, and polyunsaturated fat, and had significant reductions in percent energy intake derived from fat sources between pre- and post-treatment 24-hour food recalls.

A significant negative correlation between dietary fiber intake and plasma TC ($r=-0.19$) and LDL-C ($r=-0.20$) levels was evident following treatment for the subjects in nutrition group 1. In the same group, significant negative correlations between plasma TC levels and dietary total fat and saturated fat intake were observed. In group 2, significant positive correlations were noted for plasma HDL-C levels and dietary total fat, saturated fat, monounsaturated fat intake, and percent energy intake as fat, with a negative correlation with fiber intake.

Most attempts at weight loss are met by high attrition rates, moderate weight reduction, and a tendency to gain the weight back once the weight is lost. In most studies evaluating the effects of a

particular treatment on weight loss, there was little mention of the motivation and attitude of the subjects. These inherent characteristics, although difficult to measure and control, may play an important role in the success of weight loss in a particular group. Within our group of subjects, there appeared to be three groups, each with large differences in motivation. When the study originally began, 98 subjects started the program. Shortly after the beginning of the study, 42 subjects had dropped from the program. This represented a very high attrition rate, and these subjects obviously lacked the motivation to do the hard work associated with losing weight. Another group of subjects, mainly those that attended the exercise class and nutrition class on an inconsistent basis represented a group of individuals who had committed themselves to the program, but lacked the motivation required to lose weight. It appeared that these subjects started off motivated, but soon were tired of the work and gave up. The body weight change of the light exercise group revealed that this group started off similar to the exercise and non-exercise groups, but just did not keep up with it. On the other hand, most subjects in both the exercise group and non-exercise group were outwardly motivated and had positive attitudes toward their weight loss. It appeared that in addition to the type of weight loss program an obese person attempted, the success of weight loss was, in part, dependent on their desire to lose weight and their motivation to work hard for it and change eating habits.

In conclusion, the results of this study indicated that moderate kcal restriction, increased nutrition knowledge, modification of eating

habits, and regular participation in an exercise program successfully promoted loss of body weight in the form of BF, while sparing LBM, in obese black females seeking treatment to lose body weight. Positive changes in blood lipids occurred with weight loss, although these change were greater for those individuals who exercised while losing weight. Exercise also aided in the maintenance of HDL-C and HDL₂-C levels which were reduced in the non-exercise group with weight loss. For those individuals who may have health-related problems which prevent them from exercising, successful weight loss may occur with only slight reductions in LBM, if a moderate kcal restricted diet is followed, in addition to modifying food habits and nutrient intake through increased awareness of good nutrition.

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A P P E N D I C E S

APPENDIX A

Theory And Method of Bioelectrical Impedance Analysis (BIA)

The hypothesis that bioelectrical impedance can be used to measure body composition parameters in human subjects is based upon the different dielectrical properties of lean and adipose tissue, and upon the principle that the impedance is related to conductor length and configuration, the cross-sectional area, and signal frequency, in a geometrical system. Electrical conduction is related to water and ionic distribution in biological systems, and impedance is inversely related to lean body mass (LBM) which is highly conductive due to the presence of water and electrolytes. Adipose tissue contains virtually no electrolytes and acts as an insulator.

Subjects were placed on a dry, nonconducting surface in a supine position with the limbs not touching each other or trunk. This was done to assure a clear transmission pathway from the right arm to the right leg. Current electrodes were placed on the distal metacarpals and metatarsels on the distal side of the right hand and foot, respectively. Detector electrodes were placed on the right wrist at the right pisiform prominence and on the right ankle between the medial and lateral malleoli. Before application to skin, a thin layer of electrolyte gel was applied to each electrode. The impedance measurement was obtained by introducing an excitation current of 800 μ A at 50 KHz into the subject at the distal electrodes of the hand and foot and measuring the voltage drop by the proximal electrodes. Two consecutive measurements were averaged on each subject to obtain a more precise measurement.

APPENDIX B

Individual Pretreatment Blood Lipid (mg/dL), Glucose (mg/dL),
Thyroxin (ug/dL), Insulin (uIU/mL) Values
for 56 Obese Black Women

Subject	TC	TG	LDL-C	HDL-C	HDL ₂ -C	TC/HDL-C	GLC	INS	THY
201	160	77	97	48	22	3.3	101	32.5	9.6
206	166	77	103	48	29	3.4	100	40.9	-
207	205	50	151	44	19	4.6	80	20.8	8.4
209	249	77	160	74	47	3.4	87	19.6	14.6
218	128	57	104	34	13	3.8	117	47.4	-
222	187	68	121	52	21	3.6	85	12.7	10.0
230	203	184	107	59	40	3.4	115	28.4	13.5
235	161	62	106	43	15	3.7	86	17.2	8.8
242	326	122	261	41	12	8.0	88	12.8	9.1
248	232	93	164	49	28	4.7	86	23.9	8.5
252	233	202	134	59	22	3.9	82	23.9	9.3
253	175	58	121	39	21	4.5	78	11.2	11.4
256	217	102	148	49	31	4.4	91	19.0	-
302	283	145	226	28	17	10.1	81	31.8	7.0
303	202	430	81	35	5	5.8	140	36.8	9.9
307	199	103	137	41	26	4.8	86	26.3	8.3
308	210	87	137	56	39	3.8	73	11.7	10.1
312	243	84	177	49	27	5.0	86	14.6	6.9
319	176	58	114	50	26	3.5	87	10.6	10.1
322	226	64	164	49	-	4.6	78	-	-
329	169	93	95	55	27	3.1	88	10.7	6.6
333	210	52	153	47	21	4.5	90	-	7.8
336	144	57	85	48	25	3.0	98	10.1	8.2
347	175	69	108	53	33	3.3	92	17.2	8.4
352	180	53	105	64	-	2.8	107	23.2	9.6
354	166	65	112	41	23	4.1	97	21.2	7.5
355	269	62	173	84	42	3.2	115	-	-
358	192	124	128	39	21	4.9	70	-	7.4
361	122	39	66	48	33	2.6	76	11.5	10.5
363	195	68	134	47	27	4.1	96	47.5	8.4
364	205	130	140	39	15	5.2	76	18.9	8.1
366	247	153	171	45	30	5.5	100	41.5	6.5
402	176	69	95	67	25	2.6	100	25.7	7.7
404	151	117	83	45	30	3.4	103	22.8	9.1
405	287	154	207	49	22	5.8	95	31.1	8.0
407	236	83	167	52	26	4.5	91	6.6	9.9
408	166	39	110	48	30	3.4	83	7.0	-
409	161	106	106	34	22	4.7	108	46.8	8.1
410	171	74	103	53	29	3.2	87	14.4	11.3
413	170	56	108	51	30	3.3	117	28.6	6.6
414	197	55	121	65	26	3.0	117	16.4	6.1

APPENDIX B (Continued)

Individual Pretreatment Blood Lipid (mg/dL), Glucose (mg/dL),
Thyroxin (ug/dL), Insulin (uIU/mL) Values
for 56 Obese Black Women

Subject	TC	TG	LDL-C	HDL-C	HDL ₂ -C	TC/HDL-C	GLC	INS	THY
416	191	69	135	42	28	4.5	126	33.1	7.1
417	158	88	97	43	22	3.7	88	19.9	8.4
421	190	60	114	64	38	3.0	103	18.8	7.2
422	192	121	125	44	25	4.4	92	12.0	-
423	162	247	82	31	16	5.2	102	41.2	7.0
424	218	129	138	54	33	4.0	103	21.1	6.4
425	230	127	174	31	20	7.4	117	27.3	6.5
428	230	42	169	53	19	4.3	96	13.5	9.4
429	204	188	125	41	24	5.0	101	25.5	-
434	131	68	82	35	23	3.7	115	98.7	8.3
435	165	71	99	52	15	3.2	101	33.3	8.3
437	194	37	105	82	36	2.4	109	15.4	9.5
440	188	62	128	48	29	3.9	97	24.8	10.4
441	293	162	188	73	31	4.0	85	8.8	12.9
444	275	112	200	53	-	5.2	117	-	-

APPENDIX C

Individual Post Treatment Blood Lipid (mg/dL), Glucose (mg/dL),
Thyroxin (ug/dL), and Insulin (uIU/mL) Values
For 56 Obese Black Women

Subject	TC	TG	LDL-C	HDL-C	HDL ₂ -C	TC/HDL-C	GLC	INS	THY
201	158	84	88	53	19	3.0	102	17.5	9.8
206	148	40	96	44	-	3.4	92	25.4	11.8
207	187	49	136	41	19	4.6	82	14.3	8.0
209	227	112	129	76	39	3.0	83	17.0	13.0
218	138	63	80	45	12	3.1	104	47.8	7.4
222	182	72	111	57	26	3.2	76	12.0	10.3
230	187	150	94	63	30	3.0	85	18.8	11.8
235	159	79	105	38	20	4.2	96	25.3	9.0
242	210	63	158	39	17	5.4	80	4.1	6.0
248	210	108	141	47	19	4.5	87	18.9	6.8
252	184	165	101	50	24	3.7	90	31.5	7.8
253	155	114	94	38	19	4.1	90	17.1	10.8
256	202	74	128	59	28	3.4	84	10.0	12.7
302	246	97	191	36	12	6.8	84	28.3	10.0
303	169	133	80	22	10	7.7	286	14.6	11.0
307	176	101	122	34	14	5.2	84	27.0	7.2
308	186	92	115	53	25	3.5	78	13.2	9.9
312	201	50	137	54	19	3.7	82	14.2	6.8
319	185	38	130	47	18	3.9	88	6.7	11.8
322	188	68	131	43	11	4.4	83	22.0	8.4
329	180	40	135	37	12	4.9	98	10.4	6.0
333	209	51	-	-	-	-	86	19.2	7.6
336	125	58	77	36	12	3.5	83	11.9	8.4
347	181	81	114	51	14	3.5	82	-	-
352	156	60	79	65	26	2.4	86	25.1	8.6
354	139	90	88	33	13	4.2	88	21.5	7.5
355	232	67	144	75	28	3.1	89	25.2	6.4
358	175	78	125	34	9	5.1	81	25.3	9.2
361	135	50	72	53	17	2.5	75	10.7	8.9
363	183	52	125	47	15	3.9	100	26.9	9.1
364	220	220	145	31	9	7.0	84	16.1	9.5
366	207	117	137	47	-	4.4	61	30.1	6.1
402	162	78	90	56	25	2.9	91	22.2	8.2
404	115	100	48	47	20	2.4	96	29.3	8.0
405	278	118	197	57	17	4.9	94	22.2	5.5
407	193	73	134	44	23	4.4	94	30.2	6.2
408	155	50	103	42	16	3.7	83	12.0	10.1
409	141	111	85	34	11	4.1	109	51.4	7.5
410	151	61	72	67	31	2.2	86	18.5	8.9

APPENDIX C (Continued)

Individual Post Treatment Blood Lipid (mg/dL), Glucose (mg/dL),
Thyroxin (ug/dL), and Insulin (uIU/mL) Values
For 56 Obese Black Women

Subject	TC	TG	LDL-C	HDL-C	HDL ₂ -C	TC/HDL-C	GLC	INS	THY
413	170	68	97	59	25	2.9	92	18.9	7.0
414	145	50	75	60	32	2.4	94	10.4	7.0
416	190	87	133	40	17	4.8	100	-	-
417	153	109	87	44	15	3.5	87	29.1	8.0
421	173	71	100	59	27	2.9	84	15.4	10.7
422	200	135	141	32	15	6.2	83	7.9	5.1
423	174	176	106	33	6	5.3	102	-	-
424	203	76	129	59	29	3.4	85	30.1	6.0
425	206	163	145	28	19	7.4	89	15.8	7.0
428	186	50	125	51	26	3.6	74	17.4	8.7
429	220	181	138	46	12	4.8	97	21.7	7.7
434	136	74	97	24	12	5.7	114	41.7	10.5
435	134	42	69	57	17	2.4	103	12.8	6.5
437	160	29	175	79	41	2.0	78	8.6	5.5
440	193	73	132	46	16	4.2	85	13.7	9.2
441	207	208	112	53	23	3.9	92	13.0	16.2
444	223	117	136	64	41	3.5	110	25.2	7.4

APPENDIX D

Individual Body Weights (kg) of 56 Obese Black Women

Subject	Pre-treatment	1 month	2 months	Post-treatment
201	118.4	113.9	109.8	105.6
206	106.7	104.9	104.3	103.5
207	90.6	88.1	87.3	85.8
209	78.6	78.0	76.3	76.8
218	113.2	111.6	-	114.3
222	81.2	75.6	72.1	69.5
230	97.1	91.0	89.2	88.6
235	129.2	126.4	124.5	-
242	80.0	75.4	74.1	72.5
248	119.3	116.0	115.9	111.0
252	86.0	84.2	84.4	83.8
253	95.0	93.4	91.8	90.7
256	66.9	68.4	67.5	68.6
302	83.8	82.5	81.1	79.8
303	73.5	76.0	75.4	72.0
307	94.6	95.2	93.6	95.7
308	72.3	72.2	72.2	71.5
312	98.8	98.1	99.2	101.1
319	79.4	77.1	75.8	76.2
322	94.2	92.8	-	91.4
329	84.9	84.5	83.6	-
333	93.8	89.5	-	87.0
336	103.2	102.6	100.7	102.0
347	69.1	69.4	68.1	67.2
352	76.1	77.7	75.9	76.4
354	103.3	99.4	100.4	99.4
355	96.6	96.4	97.0	98.2
358	65.8	60.8	60.6	60.5
361	78.3	76.3	76.3	75.0
363	119.1	117.4	115.4	115.2
364	85.1	85.9	85.0	84.7
366	82.3	81.6	78.5	79.5
402	90.3	88.3	86.7	87.3
404	103.3	103.4	101.0	101.4
405	70.0	69.2	-	66.6
407	78.4	76.8	79.1	-
408	65.6	66.6	63.9	63.2
409	106.3	106.6	106.3	-
410	76.4	76.5	75.1	75.4
413	94.1	94.7	93.1	92.7
414	76.6	77.5	76.9	78.8
416	149.7	144.6	142.1	142.0
417	79.4	80.2	77.5	78.4

APPENDIX D (Continued)

Individual Body Weights (kg) of 56 Obese Black Women

Subject	Pre-treatment	1 month	2 months	Post-treatment
421	90.0	87.7	85.8	83.8
422	112.6	106.8	102.1	98.1
423	102.1	103.2	101.2	-
424	111.5	108.4	-	109.6
425	109.9	108.8	106.9	106.4
428	90.4	89.2	89.1	88.0
429	105.8	102.5	101.4	100.3
434	109.4	101.7	101.7	99.4
435	86.4	85.0	83.4	82.9
437	75.1	75.1	76.5	75.0
440	102.7	103.0	99.9	98.5
441	93.4	89.7	83.4	80.4
444	80.0	80.5	79.1	80.8

APPENDIX E

Individual Lean Body Mass Measurements (kg)
for 56 Obese Black Women

Subject	1 month	2 month	Post-treatment
201	59.8	60.0	58.5
206	62.6	65.1	61.4
207	53.8	54.3	54.0
209	53.3	52.8	54.1
218	63.5	-	66.1
222	50.6	49.3	49.2
230	47.2	54.7	48.7
235	65.3	67.1	-
242	50.1	51.5	51.0
248	63.5	62.8	59.6
252	49.5	53.1	52.1
253	49.6	51.1	55.4
256	44.2	42.3	42.7
302	51.7	51.9	51.2
303	49.6	49.1	47.3
307	44.4	44.4	49.9
308	45.2	46.7	47.6
312	52.7	56.0	55.2
319	43.8	48.3	44.3
322	55.5	-	59.2
329	46.8	47.2	-
333	49.0	-	49.9
336*	57.4	61.9	59.7
347	44.6	46.0	44.6
352	55.0	51.9	52.9
354	64.1	66.3	64.8
355	48.3	48.6	46.1
358	41.3	43.5	42.9
361	45.0	47.9	46.1
363	51.4	47.9	50.0
364	54.0	53.6	50.1
366	47.2	45.7	48.6
402	54.9	57.8	54.5
404	71.9	67.9	66.7
405	43.1	-	41.6
407	44.0	49.2	-
408	43.1	43.4	42.1
409	60.1	57.0	-
410	51.6	52.7	54.1
413*	58.6	58.3	56.0

APPENDIX E (Continued)

Individual Lean Body Mass Measurements (kg)
for 56 Obese Black Women

Subject	1 month	2 month	Post-treatment
414	55.1	55.6	57.4
416	83.4	83.4	79.9
417	53.0	53.4	53.8
421*	53.4	55.9	51.0
422*	61.4	65.3	58.9
423	65.3	64.7	-
424	62.5	-	64.2
425	59.7	59.2	55.0
428	44.2	44.8	46.1
429	64.6	59.4	58.0
434	57.2	55.1	60.6
435	53.2	53.8	52.1
437	52.1	52.3	50.3
440	63.5	59.8	63.3
441	48.3	50.0	44.1
444	48.9	48.8	51.4

* Data not included due to menstrual cycle.

APPENDIX F

Individual Percent Lean Body Mass Measurements
for 56 Obese Black Women

Subject	1 month	2 month	Post-treatment
201	52.5	54.6	55.4
206	59.3	62.4	59.3
207	61.0	62.6	62.9
209	68.4	69.2	70.4
218	56.9	-	57.9
222	67.0	68.4	70.0
230	51.9	61.3	55.0
235	71.7	53.9	-
242	66.4	69.5	70.4
248	54.8	54.2	53.7
252	58.8	62.9	62.2
253	53.2	55.7	61.0
256	64.7	62.7	62.1
302	62.6	64.1	64.2
303	65.3	65.1	65.7
307	46.7	47.5	52.1
308	62.6	64.7	66.5
312	53.7	56.4	55.1
319	56.9	63.8	58.1
322	59.8	-	64.8
329	55.4	56.4	-
333	54.7	-	57.3
336*	56.0	61.5	58.5
347	64.3	67.6	66.4
352	70.9	68.4	69.2
354	64.4	65.3	65.1
355	50.1	50.1	46.9
358	67.9	71.8	70.9
361	59.0	62.8	61.5
363	43.8	41.5	43.4
364	62.8	63.0	59.2
366	57.8	58.3	61.2
402	62.2	66.6	62.4
404	69.5	67.2	65.8
405	62.6	-	62.4
407	51.3	62.3	-
408	64.7	67.9	66.6
409	56.4	53.7	-
410	67.5	70.2	71.7
413*	61.8	62.7	60.4
414	71.1	72.3	72.8
416	57.7	58.7	56.3
417	66.1	68.9	68.6
421*	61.0	65.2	60.9

APPENDIX F (Continued)

Individual Percent Lean Body Mass Measurements
for 56 Obese Black Women

Subject	1 month	2 month	Post-treatment
422*	57.5	64.0	60.1
423	63.3	63.9	-
424	57.7	-	58.6
425	54.9	55.4	51.7
428	49.5	50.3	52.4
429	63.0	58.5	57.8
434	53.6	54.1	61.0
435	62.7	64.5	62.9
437	64.9	68.4	67.1
440	61.6	59.9	64.3
441	53.8	59.9	54.8
444	60.1	61.7	63.6

* Data not included due to menstrual cycle

APPENDIX G

Individual Body Fat Measurements (kg)
for 56 Obese Black Women

Subjects	1 month	2 months	Post-treatment
201	54.0	49.7	47.0
206	42.6	39.1	42.0
207	34.2	32.9	31.7
209	24.6	23.4	22.6
218	48.0	—	48.1
222	24.9	22.7	20.2
230	43.7	34.4	39.8
235	61.0	57.3	—
242	25.2	22.5	21.4
248	52.4	53.0	51.3
252	34.6	31.2	31.6
253	43.7	40.6	35.2
256	24.1	25.1	26.0
302	30.7	29.1	28.5
303	23.6	26.2	24.6
307	50.7	49.1	45.7
308	26.9	25.4	23.8
312	45.3	43.1	44.8
319	33.2	27.4	31.8
322	37.2	—	32.1
329	37.6	36.3	—
333	40.4	—	37.0
336	45.1	38.7	42.2
347	24.7	22.0	22.5
352	22.6	23.9	23.4
354	35.2	35.0	34.5
355	48.0	48.3	52.0
358	19.4	17.0	17.5
361	31.2	28.3	28.8
363	65.9	67.4	65.1
364	31.8	31.6	34.5
366	34.3	32.9	30.8
402	33.3	28.8	32.7
404	31.4	33.0	34.6
405	26.0	—	24.9
407	32.9	29.8	—
408	23.8	20.4	21.0
409	46.6	49.2	—
410	24.9	22.3	21.2
413	36.0	34.7	36.6
414	22.3	21.2	21.3

APPENDIX G (Continued)

IndividualBody Fat Measurements (kg)
for 56 Obese Black Women

Subjects	1 month	2 months	Post-treatment
417	27.1	24.0	24.5
421	34.2	29.8	32.7
422	45.3	34.7	39.1
424	45.8	-	45.3
425	49.0	47.6	51.3
428	37.8	41.9	42.2
434	49.4	46.5	38.7
435	31.7	29.5	30.7
437	22.9	24.1	24.6
440	39.4	40.0	35.1
441	41.3	33.3	36.2
444	31.5	30.2	29.3

APPENDIX H

Individual Percent Body Fat Measurements
for 56 Obese Black Women

Subjects	1 month	2 months	Post-treatment
201	47.4	45.3	44.5
206	40.6	37.5	40.6
207	38.9	37.7	37.0
209	31.5	30.7	29.5
218	43.0	-	42.0
222	32.9	31.5	29.2
230	48.0	38.6	44.9
235	48.2	46.0	-
242	33.5	30.4	29.5
248	45.1	45.7	46.2
252	41.1	37.0	37.7
253	46.7	44.2	38.9
256	35.2	37.2	37.7
302	37.3	35.8	35.7
303	34.6	34.8	34.2
307	53.2	52.4	47.8
308	37.3	35.2	33.4
312	46.2	43.5	44.5
319	43.0	36.1	41.8
322	40.1	-	35.1
329	44.5	43.5	-
333	45.2	-	42.6
336*	43.9	38.4	41.4
347	35.6	32.3	33.5
352	29.0	31.5	30.7
354	35.5	34.6	34.8
355	49.8	49.8	53.0
358	32.0	28.1	29.0
361	40.9	37.1	38.4
363	56.1	58.4	56.5
364	37.1	36.9	40.7
366	42.1	41.6	38.7
402	37.7	33.3	37.5
404	30.4	32.7	34.1
405	37.7	-	37.5
407	42.6	37.6	-
408	35.2	32.0	33.3
409	32.0	46.2	-
410	32.4	29.7	28.2
413*	38.1	37.2	39.5
414	28.8	27.6	27.1
416	42.4	41.2	43.6

APPENDIX H (Continued)

Individual Percent Body Fat Measurements
for 56 Obese Black Women

Subjects	1 month	2 months	Post-treatment
417	33.8	31.0	31.3
421*	38.9	34.7	39.0
422*	42.4	35.9	39.8
423	36.6	36.0	-
424	42.2	-	41.3
425	45.0	44.5	48.2
428	50.4	49.6	47.5
429	36.9	41.4	42.1
434	46.3	45.8	38.9
435	37.2	35.4	37.0
437	38.5	31.5	32.8
440	38.3	40.0	35.6
441	46.1	40.0	45.1
444	39.1	38.2	36.3

APPENDIX I

Individual Percent Total Body Water Measurements
for 56 Obese Black Women.

Subjects	1 month	2 months	Post-treatment
201	33.2	34.7	35.2
206	38.1	40.7	38.2
207	39.5	40.5	41.1
209	45.6	46.6	47.8
218	36.3	-	37.1
222	44.6	45.9	48.1
230	32.8	39.8	34.9
235	32.6	34.1	-
242	44.1	46.9	47.8
248	34.8	34.4	34.0
252	37.8	41.1	
253	33.7	35.4	39.5
256	42.6	40.9	40.4
302	40.9	42.0	42.2
303	43.1	42.9	43.5
307	29.6	30.0	33.0
308	40.9	42.6	44.2
312	34.1	36.0	35.0
319	36.3	41.8	37.3
322	38.5	-	42.6
329	35.3	36.0	-
333	34.8	-	36.6
336*	35.6	39.9	37.5
347	42.2	45.1	44.1
352	48.2	45.9	46.6
354	42.4	43.1	43.0
355	31.6	31.6	29.7
358	45.5	49.0	48.2
361	37.9	41.0	39.9
363	27.9	26.7	27.7
364	41.0	41.2	38.1
366	37.0	37.4	39.7
402	40.5	44.3	40.7
404	46.9	44.8	43.5
405	40.5	-	40.7
407	36.6	40.6	-
408	42.6	45.4	44.3
409	36.0	34.0	-
410	45.1	47.5	49.0
413*	40.2	70.9	39.1
414	48.4	49.6	50.0
416	36.9	37.7	35.9

APPENDIX I (Continued)

Individual Percent Total Body Water Measurements
for 56 Obese Black Women

Subjects	1 month	2 months	Post-treatment
417	43.8	46.3	46.1
421*	39.5	43.0	39.4
422*	36.8	42.0	38.8
423	41.4	41.9	-
424	36.9	-	37.6
425	34.9	35.2	32.6
428	31.6	31.7	33.1
429	41.2	37.6	37.0
434	34.0	34.3	39.5
435	40.9	42.4	41.1
437	46.8	45.9	44.7
440	40.0	38.6	42.2
441	34.1	38.7	34.8
444	39.4	40.1	41.7

APPENDIX J

Individual Pretreatment Nutrient Intakes for 56 Obese Black Women

Subjects	kcal	Protein (g)	Carbohydrate (g)	Fat (g)	Saturated Fat (g)	Monounsaturated Fat(g)
201	2027	90.5	266	119	31	38
206	2720	85.2	356	108	39.2	41.9
207	2297	84.4	288	92.9	24.5	16.0
209	2646	87.3	321	118	37.5	46.7
218	1428	43.3	147	73.0	28.4	25.4
222	1290	47.8	142	60.4	21.6	18.7
230	1194	48.5	137	52.5	19.2	23.2
235	1941	79.2	195	74.5	30.5	29.9
242	983	63.0	65	51.2	18.2	17.3
252	754	30.9	194	32.5	-	-
253	1551	64.6	199	60.3	21.6	22.7
256	1604	60.6	83	113	28.6	39.2
302	909	58.3	182	80.0	30.9	36.4
303	2244	55.04	310	91.9	21.9	39.8
307	1378	64.5	131	69.0	16.3	17.1
308	1145	61.4	115	48.2	16.1	12.7
312	1116	31.3	89.0	72.5	24.1	30.2
319	1116	36.1	161	33.8	12.9	17.0
329	1289	62.1	88.0	77.3	35.1	25.8
333	853	40.5	99.0	35.1	10.4	12.9
336	1841	53.8	199	92.0	31.1	19.7
347	1995	77.9	219	93.0	26.9	24.2
352	2802	94.4	365	111	52.6	39.2
354	1940	55.3	234	88.4	31.4	39.1
355	1388	63.7	162	53.7	12.4	15.0
358	926	43.2	103	39.7	10.3	13.8
361	2133	77.3	197	166	38.4	38.9
363	2648	94.8	252	125	44.2	46.8
364	2013	69.8	236	93.0	30.0	31.0
366	1747	62.4	168	95.0	38.1	34.3
402	1095	52.4	102	52.8	-	-
404	1646	36.0	226	72.8	27.5	24.9
405	1408	36.6	208	52.3	23.9	16.2
407	1114	52.8	122	45.3	18.1	13.1
408	2274	80.1	177	132	36.1	50.6
409	1875	100	231	59.7	14.9	12.4
410	1572	32.7	224	64.2	27.4	22.3
413	1634	71.2	197	61.4	20.6	20.3
414	1844	76.7	180	89.4	20.6	39.3
416	1973	104	241	71.0	22.2	25.9
417	1954	48.8	256	82.2	25.0	27.5

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 obese black women

Subjects	kcal	Protein (g)	Carbohydrate (g)	Fat (g)	Saturated Fat (g)	Monounsaturated Fat(g)
421	913	27.3	113	40.5	12.8	12.8
422	1602	58.9	252	41.4	13.2	12.3
423	1342	49.6	138	66.5	21.6	24.0
424	1616	60.7	169	77.7	25.6	29.0
425	2322	90.8	200	131	41.7	48.9
428	1389	48.7	213	40.8	14.4	12.4
429	1747	77.0	159	90.1	30.8	34.2
434	1849	62.5	213	84.5	34.1	31.8
435	2256	77.6	205	134	49.2	46.4
437	2329	64.4	378	67.1	22.7	24.5
440	1537	78.5	152	68.0	24.9	24.6
441	1622	84.6	148	79.0	28.5	25.2
444	1494	51.2	196	59.2	17.1	21.0

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Polyunsaturated fat	P/S	M/S	%Protein	%Carbohydrate	%Fat
201	38.0	1.22	1.0 (1.22)	14	43	43
206	15.8	0.40	0.37 (1.06)	12	52	36
209	18.7	0.76	1.16 (0.05)	15	50	36
218	8.8	0.30	0.89 (1.24)	13	48	40
222	11.9	0.55	0.86	15	44	42
230	4.4	0.23	1.2	16	46	40
235	23.6	0.77	0.82	16	40	44
242	9.6	0.52	0.95	26	27	47
248	16.9	0.48	1.28	14	41	46
252	-	-	-	10	66	25
253	7.2	0.34	1.06	16	50	34
256	41.5	1.45	1.02	15	21	64
302	4.2	0.14	1.12	14	43	43
303	30.8	1.44	1.87	10	55	37
307	90.	0.55	1.05	19	38	45
312	11.3	0.47	1.26	11	31	58
319	3.7	0.30	1.0	13	57	27
322	8.9	0.47	0.90	13	44	43
329	6.1	0.19	0.74	19	27	54
333	5.9	0.57	1.24	19	45	36
366	6.5	0.20	0.90	16	44	42
352	16.3	0.38	0.92	13	54	33
354	12.2	0.39	1.24	11	48	41
355	17.3	1.40	1.21	18	47	35
358	9.4	0.91	1.34	18	44	38
361	19.0	0.49	1.01	14	37	49
363	15.1	0.34	1.06	15	39	43
364	23.7	0.79	1.03	14	53	34
366	6.6	0.17	0.90	14	38	48
402	-	-	-	19	37	43
404	11.0	0.40	0.90	8	53	38
405	40.8	0.20	0.68	10	59	33
407	9.1	0.50	0.72	19	44	37
408	30.0	0.83	1.40	14	35	51
409	15.8	1.06	0.83	21	50	29
410	6.2	0.23	0.81	8	56	36
413	8.9	0.43	1.0	18	48	34
414	18.8	0.91	1.91	16	39	43
416	11.3	0.51	1.17	21	48	32
417	22.1	0.88	1.10	9	54	37

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Polyunsaturated fat	P/S	M/S	%Protein	%Carbohydrate	%Fat
421	9.9	0.77	1.0	12	49	39
422	6.5	0.49	0.93	15	62	23
423	16.1	0.74	1.11	15	41	44
424	9.2	0.36	1.13	15	42	43
425	22.0	0.53	1.17	16	34	51
428	3.8	0.26	0.86	14	61	26
429	11.6	0.38	1.11	18	36	46
434	9.8	0.29	0.93	14	46	41
435	26.1	0.53	0.94	14	36	53
437	13.3	0.58	1.08	11	65	26
440	8.9	0.36	1.0	20	40	40
441	16.5	0.58	0.88	21	36	43
444	10.7	0.62	1.23	13	52	35

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Fiber(g)	Cholesterol(mg)	Sodium(mg)	Magnesium	Iron	Zinc
201	2.8	200	3172	159	12.9	4.3
206	3.4	324	4239	162	15.7	4.0
207	2.4	231	2868	149	9.5	3.6
209	3.6	250	3533	170	15.6	5.7
219	0.98	148	1941	115	6.5	7.0
222	1.25	150	7722	105	6.4	5.0
230	0	174	2131	32	5.8	0.78
235	3.4	220	2695	156	11.9	6.7
242	0.95	310	586	98	6.5	6.4
252	1.39	66	1947	38.6	5.7	1.60
253	7.3	187	2170	158	13.3	8.1
256	3.2	466	2640	96.1	6.4	5.0
302	0.34	294	2037	39.5	10.8	5.4
303	6.8	222	3213	115	8.9	4.5
307	5.5	316	2073	124	10.9	7.1
308	3.3	246	1530	147	5.5	4.7
312	3.5	133	957	155	6.7	5.6
319	1.0	279	2428	53.4	5.7	2.8
322	1.6	139	1490	103	6.2	5.1
329	3.1	223	2179	119	6.8	9.7
333	4.3	86	710	98.5	7.6	3.6
336	2.2	384	1711	92.3	8.9	8.2
347	5.3	224	2550	113	8.6	4.8
352	1.4	432	3066	115	12.8	9.2
354	2.0	174	2212	118	8.0	3.6
355	1.11	296	1490	126	8.8	5.4
358	4.4	495	1058	124	10.1	6.1
361	3.9	492	1767	176	11.1	12.3
363	4.7	356	3808	161	14.7	9.3
364	4.1	95	3644	122	7.0	2.9
366	7.4	232	1651	220	11.5	9.0
402	2.0	159	1875	78.4	6.5	2.2
404	6.7	205	902	163	8.6	5.7
405	3.1	140	957	214	8.2	6.7
407	1.8	175	3122	114	7.8	5.4
408	4.9	193	2734	258	9.9	10.9
409	3.1	255	1715	186	11.8	7.5
410	2.1	170	1521	94.2	7.9	3.1
413	2.6	325	2065	108	10.9	7.0
414	3.1	217	1396	113	10.2	4.21
416	8.2	379	900	438	14.8	10.9

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Fiber(g)	Cholesterol(mg)	Sodium(mg)	Magnesium	Iron	Zinc
417	6.7	158	1384	127	7.2	4.7
421	1.7	100	1220	113	4.6	2.6
422	6.2	187	2287	160	9.9	6.8
423	2.0	104	2049	73	8.1	2.3
424	0.66	589	2193	89.9	7.8	5.6
425	3.1	505	2584	268	10.0	10.9
428	2.3	290	4103	145	8.5	3.4
429	2.1	531	2915	132	11.3	10.0
434	2.5	312	2341	81.5	8.7	5.7
435	4.2	233	3682	178	9.3	7.8
437	4.1	174	2290	221	11.6	5.1
440	45.3	200	1161	188	8.1	8.7
441	4.8	255	2723	130	12.5	7.4
444	7.7	248	1853	148	7.6	5.5

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Vitamin A	Vitamin B6	Calcium(mg)	Folacin
201	2355	1.44	269	125
206	5789	0.62	995	116
207	1093	1.12	436	88
209	1587	0.84	500	110
218	1595	0.28	360	103
222	1265	0.64	364	156
230	470	-	222	13.2
235	-	0.53	929	191
242	949	0.77	155	113
248	4883	0.94	388	88.1
252	2418	0.72	202	125
253	310	1.22	440	194
256	1719	0.91	350	188
302	382	0.18	43	22
303	747	1.17	300	109
307	2095	1.18	351	150
308	2016	1.25	398	157
312	4887	0.68	369	121
319	782	0.54	465	85.0
322	1450	0.42	401	126
329	-	1.04	810	160
333	1575	0.57	214	106
336	1841	5.38	199	92
347	2015	1.18	315	87.7
352	1628	0.71	539	97.5
354	813	1.41	304	94.8
355	1272	0.90	177	154
358	-	1.38	178	240
361	998	0.91	426	121
363	1816	0.87	527	746
364	440	0.48	546	141
366	-	0.85	645	218
402	1936	0.47	417	63.2
404	2951	0.57	519	195
405	5022	1.64	496	116
407	1478	0.35	587	62.3
408	1800	1.63	344	170
409	1969	1.83	356	76.5
410	2624	0.67	388	109
413	2791	0.75	241	64.3

APPENDIX J (Continued)

Individual Pretreatment Nutrient Intakes
for 56 Obese Black Women

Subject	Vitamin A	Vitamin B6	Calcium(mg)	Folacin
414	2315	0.77	365	100
416	8615	5.03	221	291
417	3139	0.84	279	83.2
421	5005	0.76	182	89.6
422	1876	1.43	157	155
423	767	0.40	266	56.1
424	1183	0.63	232	126
425	3473	1.12	716	210
428	5793	0.65	316	179
429	-	1.90	213	257
434	1517	0.57	400	70.9
435	1979	1.23	830	112
437	1868	1.22	481	185
440	1466	1.44	342	123
441	2871	0.95	358	113
444	6338	1.45	611	155

APPENDIX K

Individual Post-treatment Nutrient intakes
for 56 Obese Black Women

Subjects	kcal	Protein (g)	Carbohydrate (g)	Fat (g)	Saturated Fat (g)	Monounsaturated Fat(g)
201	775	41.6	113	14.7	5.25	4.80
206	1679	94.8	135	86.8	31.1	24.5
207	934	39.6	141	28.4	7.47	7.30
209	2241	94.3	241	102	30.8	33.2
218	-	-	-	-	-	-
222	933	50.7	106	36.4	10.6	11.5
230	623	33.6	81	20.6	4.3	4.72
235	1126	108	66	43.9	14.7	16.3
242	685	56	48	28.0	10.3	8.9
248	1237	30.1	156	55.6	5.8	20.4
252	844	39.6	111	29.2	6.12	5.6
253	-	-	-	-	-	-
256	1018	49.0	67.0	61.2	19.0	20.8
302	857	33.3	87.0	44.9	14.3	15.8
303	1383	27.8	203	51.5	17.0	19.7
307	770	44.7	113	19.3	6.51	5.91
308	958	52.6	111	35.8	11.5	11.5
312	522	33.6	22.0	33.2	12.2	13.0
319	892	74.4	39.0	47.2	12.9	-
322	979	50.4	92.0	45.6	12.3	12.3
329	473	23.9	56.0	18.8	3.22	2.15
333	774	61.6	93.0	18.6	7.79	5.13
336	1458	70.9	153	61.2	22.0	20.04
347	1256	77.0	123	50.7	16.4	15.3
352	1900	66.8	232	82.3	34.0	28.3
354	1280	40.4	203	36.6	11.2	12.9
355	832	51.5	93.0	31.2	6.3	7.8
358	-	-	-	-	-	-
361	1916	56.8	166	118	34.7	47.2
363	1141	45.7	144	76.2	24.3	29.1
364	1929	87.3	216	82.0	25.9	30.6
366	966	28	130	37.3	13.7	12.1
402	920	49.8	113	34.0	10.2	7.2
404	1228	56.2	204	27.8	9.9	7.98
405	1328	40.9	163	60.4	24.3	20.1
407	771	15.6	111	32.2	8.51	9.07
408	1245	56.6	212	19.8	6.4	6.05
409	1314	42.7	141	64.0	13.1	17.0
410	1493	44.1	174	72.7	27.4	24.9
413	1175	42.0	147	46.4	18.4	17.6

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subjects	kcal	Protein (g)	Carbohydrate (g)	Fat (g)	Saturated Fat (g)	Monounsaturated Fat(g)
414	2124	74.8	292	78.3	22.5	29.8
416	730	42.4	45.0	43.0	13.6	18.3
417	1122	59.0	130	43.4	13.9	13.9
421	671	48.4	111	88.9	2.78	2.07
422	630	20.0	54.0	34.0	12.2	14.6
423	945	59.0	91.0	35.6	9.66	12.5
424	1228	56.2	116	58.0	20.4	22.2
425	1314	103	156	32.6	11.1	7.17
428	4879	45.7	114	31.4	8.74	8.13
429	960	36.8	138	27.9	9.13	10.6
434	1906	49.4	242	85.9	27.4	-
435	807	40.6	86.0	34.8	13.6	12.7
437	1906	62.2	187	108	39.5	36.9
440	1079	60.2	113	44.9	11.5	14.3
441	682	51.5	65	25.7	9.1	9.1
444	1572	63.2	202	59.6	9.2	12.3

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subject	Polyunsaturated fat	P/S	M/S	%Protein	%Carbohydrate	%Fat
201	2.23	0.42	0.91	21	58	17
206	3.59	0.11	0.79	22	32	46
207	3.11	0.42	0.98	16	58	26
209	24.0	0.78	1.08	17	43	41
218	-	-	-	-	-	-
222	4.15	0.39	1.08	21	44	34
230	4.07	0.95	1.10	21	50	29
235	5.47	0.37	1.11	40	24	36
242	3.54	0.34	0.86	33	29	38
248	14.3	2.5	3.5	10	50	40
252	1.90	0.31	0.92	18	51	30
253	-	-	-	-	-	-
256	16.8	0.88	1.09	19	26	54
302	5.81	0.41	1.10	15	39	46
303	8.67	0.51	1.16	8	59	33
307	3.39	0.52	0.91	22	56	22
308	6.21	0.54	1.00	22	45	33
312	1.95	0.16	1.06	26	17	57
309	7.47	0.58	-	33	17	48
322	9.62	0.78	1.0	21	37	42
329	2.35	0.73	0.67	19	47	34
333	4.37	0.56	0.66	31	47	21
336	11.6	0.53	0.93	19	41	37
347	10.8	0.66	0.93	25	39	36
352	6.60	0.19	0.83	13	48	38
354	7.45	0.66	1.15	12	62	25
355	3.88	0.62	1.24	24	43	33
358	-	-	-	-	-	-
361	11.1	0.32	1.36	12	34	56
363	16.9	0.69	1.20	13	40	47
364	11.2	0.43	1.18	18	45	38
366	5.74	0.42	0.88	12	54	35
402	10.1	1.0	0.70	21	47	32
404	3.67	0.37	0.81	17	63	19
405	6.5	0.32	0.83	12	48	40
407	10.0	1.18	1.07	8	56	36
408	3.25	0.51	0.94	18	68	14
409	13.6	1.04	1.30	13	43	44
410	10.1	0.37	0.91	12	46	43

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subject	Polyunsaturated fat	P/S	M/S	%Protein	%Carbohydrate	%Fat
413	5.8	0.32	0.96	14	50	36
414	17.1	0.76	1.32	14	54	32
416	6.0	0.44	1.34	23	24	53
417	8.6	0.62	1.00	21	45	34
421	1.0	0.36	0.74	27	62	11
422	3.6	0.29	1.20	13	36	51
423	5.74	0.59	1.29	26	40	35
424	7.72	0.38	1.09	19	38	43
425	4.46	0.40	0.64	31	47	32
428	6.34	0.72	0.93	20	50	31
429	4.07	0.44	1.16	15	58	26
434	16.4	0.60	-	10	51	40
435	3.16	0.23	0.93	20	42	38
437	16.3	0.41	0.93	13	38	49
440	8.37	0.73	1.24	24	39	37
441	4.48	0.49	1.00	30	37	33
444	7.71	0.84	1.30	16	51	34

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subject	Fiber(g)	Cholesterol(mg)	Sodium(mg)	Magnesium	Iron	Zinc
201	2.75	130	703	117	5.15	2.93
206	1.55	424	2420	222	13.4	14.3
207	7.45	74	1879	156	13.1	5.03
209	2.99	299	3273	196	11.2	9.07
218	-	-	-	-	-	-
222	2.94	170	1532	118	8.49	4.16
230	4.04	204	865	89.1	3.78	1.83
235	2.32	283	910	109	10.5	9.81
242	3.86	158	582	67.2	5.34	4.77
248	2.60	367	1112	83.6	7.16	4.04
252	4.91	96	1295	130	7.70	4.87
253	-	-	-	-	-	-
256	2.62	241	1776	71.6	6.0	4.21
302	1.28	81	844	108	3.38	4.33
303	3.98	83	2379	70.8	6.58	3.48
307	3.80	109	1442	147	6.73	4.76
308	6.10	188	1318	196	9.54	5.03
312	1.42	137	1241	63.3	3.31	5.51
319	0.66	218	604	77.3	5.66	8.45
322	2.26	497	1087	123	7.25	5.75
329	2.97	73	2280	68.2	2.91	0.83
333	2.99	158	1013	129	4.91	4.36
336	5.20	296	1710	205	14.2	10.3
347	5.40	226	1166	152	8.99	10.0
352	3.09	222	1460	142	6.80	7.81
354	2.52	93	911	85.2	6.07	2.73
355	1.96	400	2432	97.1	5.33	4.22
358	-	-	-	-	-	-
361	2.04	321	2924	121	8.94	6.52
363	3.22	186	1173	161	8.41	6.67
364	2.49	334	2628	147	11.7	13.8
366	2.15	88	1123	80.2	4.87	3.14
402	7.31	298	2805	236	9.21	6.54
404	7.51	117	1738	238	10.9	5.1
405	2.29	178	2293	85.9	7.46	4.01
407	4.34	174	1816	78.3	4.35	1.61
408	8.51	235	1310	154	7.62	5.10
409	6.52	117	2394	79.1	7.81	1.90
410	1.97	247	2058	117	6.79	5.53
413	0.89	91	3283	159	4.78	0.64
414	4.39	126	1858	257	9.4	8.67

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subject	Fiber(g)	Cholesterol(mg)	Sodium(mg)	Magnesium	Iron	Zinc
416	1.69	134	1421	62.9	5.0	4.03
417	5.65	140	1273	204	8.7	12.2
421	9.74	118	575	222	10.7	5.62
422	0.50	54	1986	30.4	4.40	2.90
423	0.85	399	1441	85.7	6.44	3.37
424	3.21	275	2637	147	7.03	6.67
425	7.95	324	1607	232	12.0	11.3
428	6.4	99	1068	206	9.70	4.60
429	1.24	119	1669	102	6.98	4.05
434	3.01	172	2145	129	8.97	5.03
435	3.21	200	1316	132	6.15	4.85
437	1.99	277	2568	162	7.98	7.99
440	2.55	251	1563	92.2	6.80	3.03
441	4.99	133	2594	245	6.74	5.28
444	2.13	122	1985	156	9.20	3.25

APPENDIX K (Continued)

Individual Post-treatment Values Nutrient Intakes
for 56 Obese Black Women

Subject	Vitamin A	Vitamin B6	Calcium(mg)	Folacin
201	9507	0.86	245	249
206	6681	0.62	855	138
207	4180	1.14	348	277
209	2264	1.94	314	182
218	-	-	-	-
222	5683	1.23	359	203
230	-	0.56	488	150
235	6249	1.09	150	45.0
242	871	0.64	90	82.5
248	1778	0.76	260	96.0
252	3401	1.24	295	259
253	-	-	-	-
256	1069	0.66	230	75.1
302	910	0.28	445	49.0
303	730	0.58	193	118
307	1770	0.66	530	118
308	5969	1.59	423	187
312	700	0.41	100	61.0
319	730	0.83	67	67
322	2286	0.67	133	84.0
329	232	0.88	122	36.3
333	3770	1.26	344	181
336	-	0.66	547	120
347	960	1.35	266	86.2
352	2229	0.68	439	117
354	270	0.62	73	32.3
355	587	0.43	244	64.0
358	-	-	-	-
361	2422	0.59	388	125
363	1239	0.45	391	85.0
364	1746	0.33	690	109
366	3795	1.22	325	115
402	3669	1.75	564	233
404	-	1.86	843	345
405	5746	0.29	398	47.0
407	3944	0.29	340	102
408	3325	1.16	388	187
409	1521	0.33	210	72.4
410	1657	0.66	470	119
413	3511	0.25	791	220

APPENDIX K (Continued)

Individual Post-treatment Nutrient Intakes
for 56 Obese Black Women

Subject	Vitamin A	Vitamin B6	Calcium(mg)	Folacin
414	3010	1.12	707	205
416	1193	0.63	197	52.4
417	1052	0.83	415	117
421	-	1.78	580	352
422	-	0.15	80	340
423	5324	1.02	663	116
424	4630	0.85	546	214
428	-	0.70	647	167
429	5427	0.80	114	107
434	3360	0.55	180	123
435	6399	0.77	559	96.2
437	4381	0.56	532	77.0
440	1630	0.42	499	196
441	9758	1.27	315	196
444	689	0.50	374	124

APPENDIX L

MEDICAL HISTORY OF SUBJECT AND FAMILY, FORM NO. 2

OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE _____

It is important that we ask the following questions because your medical background, certain drugs, smoking, alcoholic consumption and your menstrual cycle can affect the outcome of some of the analyses we are doing. Your replies will be kept in strict confidence.

1. DOES THE SUBJECT HAVE THE SICKLE CELL TRAIT? YES ___ NO ___
2. ARE YOUR MENSTRUAL PERIODS REGULAR? YES ___ NO ___
DATE OF LAST PERIOD _____
3. ARE YOU TAKING BIRTH CONTROL PILLS NOW? YES ___ NO ___
NAME _____
4. DO YOU HAVE ANY KNOWN ILLNESS OR CONDITIONS? YES ___ NO ___
NAME _____
5. ARE YOU TAKING MEDICATION FOR ANY ILLNESS OR CONDITIONS? NAME _____ DOSAGE _____ YES ___ NO ___
6. HAVE YOU TAKEN ANY MEDICINE DURING THE PAST WEEK? NAME _____ DOSAGE _____ YES ___ NO ___
7. IS THERE AN ILLNESS OR CONDITION THAT YOU SUFFERED FROM IN THE PAST, BUT NO LONGER HAVE? YES ___ NO ___
NAME _____ HOW LONG? _____
8. ARE YOU TAKING ANY PRESCRIPTION MEDICINE, GENERIC OR BRAND NAME MEDICATION FOR WEIGHT LOSS? YES ___ NO ___
NAME _____ DOSAGE _____ HOW LONG? _____
9. DO YOU SMOKE? HOW MUCH/WEEK? _____ YES ___ NO ___
10. DO YOU CONSUME ALCOHOLIC DRINKS? HOW MUCH/WEEK? _____ YES ___ NO ___
11. DO YOU HAVE HIGH BLOOD PRESSURE? YES ___ NO ___
12. DO YOU HAVE DIABETES? YES ___ NO ___
13. AMONG THE SUBJECT'S BROTHERS, SISTERS, PARENTS, AUNTS, UNCLAS, OR GRANDPARENTS, IS THERE A KNOWN CASE OF:

HEART ATTACK	NO. CASE _____	YES ___ NO ___
HIGH BLOOD PRESSURE	NO. CASE _____	YES ___ NO ___
DIABETES	NO. CASE _____	YES ___ NO ___
CANCER	NO. CASE _____	YES ___ NO ___
GOUT	NO. CASE _____	YES ___ NO ___
GALL BLADDER DISEASE	NO. CASE _____	YES ___ NO ___
EMOTIONAL/MENTAL PROBLEMS	NO. CASE _____	YES ___ NO ___

APPENDIX M

PHYSICAL EXAMINATION/MEDICAL APPROVAL FORM NO. 11
OBESITY IN BLACK WOMENNAME OF SUBJECT _____ SUBJECT NO. _____
DATE _____

1. BODY TEMPERATURE _____
2. BLOOD PRESSURE SYSTOLIC _____ DIASTOLIC _____
3. FINDINGS OF EXAMINATION:

 DEB PROGRAM

_____ is interested in participating in the series of weight reduction lessons in the DEB Program conducted by the VSU Nutrition Research Project. This educational program is designed to help women evaluate and restructure eating habits and activity patterns to bring about weight loss. It uses principles of behavior modification to encourage changing actions or habits related to overeating and/or underactivity. The program has been given the acronym DEB--diet, exercise, and behavior modification.

The diet plan used is based on the seven food group exchange system used for diabetic diets. The minimum calorie level used is 1200. Participants are encouraged to determine a calorie level that will bring about a weight loss of one to two pounds per week.

Participants in the DEB program are encouraged to increase their physical activity to help burn calories for weight reduction. Sports, low impact aerobic, walking, jogging, and jumping rope are suggested. However, any increase in physical activity is completely up to the participant.

On the basis of a physical examination and/or the medical history of this patient, do you feel this patient is physically able to participate in the weight reduction program? No _____ Yes _____

Activity limitations _____

Dietary limitations _____

Signature M.D.

APPENDIX N

FORM #4

VIRGINIA STATE UNIVERSITY

OBESITY STUDY IN BLACK WOMEN

SUBJECT _____

DATE _____ YEAR _____

BIOCHEMICAL DATA

SICKLE TRAIT (Hg-S) _____

HEMATOCRIT _____ %

HEMOGLOBIN _____ g/dl

SERUM IRON _____ ug/dl

TIBC _____ ug/dl

TOTAL CHOLESTEROL _____ mg/dl

HDL CHOLESTEROL _____ mg/dl

T. TRIGLYCERIDE _____ mg/dl

FASTING GLUCOSE _____ mg/dl

INSULIN _____

PLASMA CALCIUM _____

PLASMA MAGNESIUM _____

PLASMA ZINC _____

PLASMA COPPER _____

RBC ZINC _____

GIRDLE:	PANTY-TYPE	KNEELENGTH	LONG-LINE

APPENDIX O (Continued)

ANTHROPOMETRIC MEASUREMENTS, FORM NO. 3

OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE _____ YEAR _____

CLOTHING LIST CONTINUED:

SLACKS: POLYESTER (250 G) COTTON (360 G) JEANS (440 G)

SKIRT: LIGHT (250 G) MEDIUM (360 G) HEAVY (420 G)

JACKET: LIGHT MEDIUM HEAVY

BLOUSE: LIGHT (100 G) MEDIUM (190 G) HEAVY (280 G)

SWEATER: LIGHT (320 G) MEDIUM (390 G) HEAVY (440 G)

BELT: LIGHT (60 G) MEDIUM (100 G) HEAVY (140 G)

DRESS: LIGHT MEDIUM HEAVY

OTHER: LIST AND WEIGH SIMILAR ITEMS

APPENDIX F

BIOIMPEDANCE QUESTIONNAIRE

SUBJECT'S NAME: _____ NO: _____

1. Is your menstrual period regular? Yes _____ No _____
Date of last period _____
2. How much coffee, tea and/or soda have you consumed today and yesterday?

DRINKS	TODAY'S AMOUNT	YESTERDAY'S AMOUNT
coffee	_____	_____
tea	_____	_____
soda (type)	_____	_____
(type)	_____	_____
(type)	_____	_____

3. Have you consumed any alcoholic drink today and yesterday?
No _____ Yes _____ Today's amount _____
Yesterday's amount _____
4. Have you taken a diuretic or blood pressure medication within the last 48 hours? No _____ Yes _____ When _____
5. How much liquid have you consumed within the last 24 hours? Include water and all other beverages. Amount _____ Is this your normal intake?
Yes _____ No _____

DATE: _____

APPENDIX Q

VSU Obesity Study

NAME _____
 DATE _____
 ACTIVITY SCORE _____

EXERCISE ACTIVITY LEVELS

1. Dancing(aerobic)	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>6.4</u>
2. Bicycling	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>5.0</u>
3. Swimming	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>4.0</u>
4. Stretches	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>4.5</u>
5. Golfing	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>5.0</u>
6. Walking (circle) Outdoor: a. slowly b. moderate c. rapidly d. upstairs	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>5.0</u>
7. Tennis	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>7.0</u>
8. Running	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>14.0</u>
9. Jogging	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>9.0</u>

Intensity: light, mild, moderate, vigorous
 1 2 3 4

APPENDIX Q (Continued)

10.	Skating, Ice	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>7.0</u>
11.	Skating, Roller	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>7.0</u>
12.	Bowling	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>3.0</u>
13.	Calisthenics	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>4.5</u>
14.	Farming or Gardening	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>3.7</u>
15.	Snowskiing	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>8.0</u>
16.	Housework (Standup work)	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>3.7</u>
17.	Volleyball	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor <u>5.5</u>
18.	Other (Please specify)	Frequency _____ # of months _____ # of days/week _____	minutes _____ intensity _____ activity factor _____

Intensity: light, mild, moderate, vigorous
 1 2 3 4

APPENDIX R (Continued)

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE OF RECALL _____ YEAR _____

5. HOW MANY DAYS OF EACH WEEK DO YOU USUALLY EAT SOMETHING BETWEEN BREAKFAST AND LUNCH? (CIRCLE ONE) 0 1 2 3 4 5 6 7

6. DID YOU EAT A MORNING SNACK YESTERDAY? YES NO

7. WHERE DID YOU EAT THE FOOD IF YOU ATE SOMETHING BETWEEN BREAKFAST AND LUNCH? (CIRCLE ONE)

1. HOME

2. FAST FOOD TYPE, GROCERY, VENDING MACHINE, SNACK BAR

- ### 3. RESTAURANT, CAFETERIA

4. OTHER _____

8. WHAT DID YOU EAT AND/OR DRINK BETWEEN BREAKFAST AND LUNCH YESTERDAY?
TIME

[illegible]This image shows a single sheet of white paper with horizontal blue or grey ruling lines. The lines are evenly spaced and run across the width of the page. There are approximately 20 lines visible. The paper has a slightly textured appearance and some minor discoloration or shadows, suggesting it's a physical scan. There is no handwriting or other markings on the paper.

OBESITY IN BLACK WOMEN

DATE OF RECALL _____ YEAR _____

11. WHERE DID YOU EAT THE FOOD IF YOU ATE SOMETHING FOR LUNCH?

3. RESTAURANT, CAFETERIA

12. WHAT DID YOU EAT AND/OR DRINK FOR LUNCH YESTERDAY? TIME

A. FOOD/DRINKS	B. AMOUNT IN HOUSEHOLD UNITS	C. FREQ OF UNITS	D. NAS ID	E. WT IN GRAMS
----------------	---------------------------------	---------------------	-----------	-------------------

This image shows a single sheet of white paper with horizontal blue or grey ruling lines. The lines are evenly spaced and run across the width of the page. There is no handwriting or other markings on the paper.

APPENDIX R (Continued)

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE OF RECALL _____ YEAR _____

13. HOW MANY DAYS OF EACH WEEK DO YOU USUALLY EAT SOMETHING BETWEEN NOON AND THE EVENING MEAL? (CIRCLE ONE) 0 1 2 3 4 5 6 7

14. DID YOU EAT AN AFTERNOON SNACK YESTERDAY? YES NO

15. WHERE DID YOU EAT THE FOOD IF YOU ATE SOMETHING FOR BETWEEN NOON AND THE EVENING MEAL? (CIRCLE ONE)

1. HOME

2. FAST FOOD TYPE, GROCERY, VENDING MACHINE, SNACK BAR

- ### 3. RESTAURANT, CAFETERIA

4. OTHER _____

16. WHAT DID YOU EAT AND/OR DRINK FOR AN AFTERNOON SNACK YESTERDAY?
TIME

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OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE OF RECALL _____ YEAR _____

4. OTHER

24. WHAT DID YOU EAT AND/OR DRINK FOR AN EVENING SNACK YESTERDAY?
TIME

A. FOOD/DRINKS	B. AMOUNT IN HOUSEHOLD UNITS	C. FREQ OF UNITS	D. NAS ID	E. WT IN GRAMS
----------------	---------------------------------	---------------------	-----------	-------------------

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DATE OF RECALL _____ YEAR _____

29. HOW MANY TIMES PER WEEK DO YOU USUALLY TAKE NUTRITIONAL SUPPLEMENTS SUCH AS VITAMINS, MINERALS, OR PROTEIN IN ADDITION TO THE FOODS YOU EAT? (CIRCLE ONE) 0 1 2 3 4 5 6 7
30. IF YOU TAKE SUPPLEMENTS, WHO RECOMMENDED THAT YOU TAKE THE SUPPLEMENTS?
- | | |
|--------------|------------------|
| 1. PHYSICIAN | 5. GIRLFRIEND(S) |
| 2. MOTHER | 6. BOYFRIEND(S) |
| 3. FATHER | 7. MEDIA |
| 4. SELF | |
31. WHAT SUPPLEMENTS DID YOU TAKE YESTERDAY, HOW MANY CAPSULES OR TABLETS AND WHAT TIME WERE THEY TAKEN?

(BE SURE IF THE SUPPLEMENT IS A SINGLE NUTRIENT THAT THE CONCENTRATION IN EACH TABLET IS OBTAINED. UNDER COLUMN E BELOW, RECORD THE MEAL CODE OR SNACK CODE CORRESPONDING TO THE TIME PERIOD WHEN THE SUPPLEMENT WAS CONSUMED.)

A. VITAMIN/MINERAL SUPPLEMENT	B. CONC OF TABLET	C. FREQ	D. NAS ID	E. TIME OF DAY	F. HOW LONG
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[illegible]

APPENDIX R (Continued)

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO. _____

DATE OF RECALL _____ YEAR _____

THE FOLLOWING INFORMATION NEEDS TO BE ANSWERED BY THE INTERVIEWER.

A) SUBJECT'S AGE

B) YESTERDAY WAS (CIRCLE ONE) SU M TU W TH F SA

C) IS THIS RECALL BEING TAKEN ON THE DAY BLOOD IS DRAWN? (PLEASE CIRCLE)

YES = 1

NO = 2

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